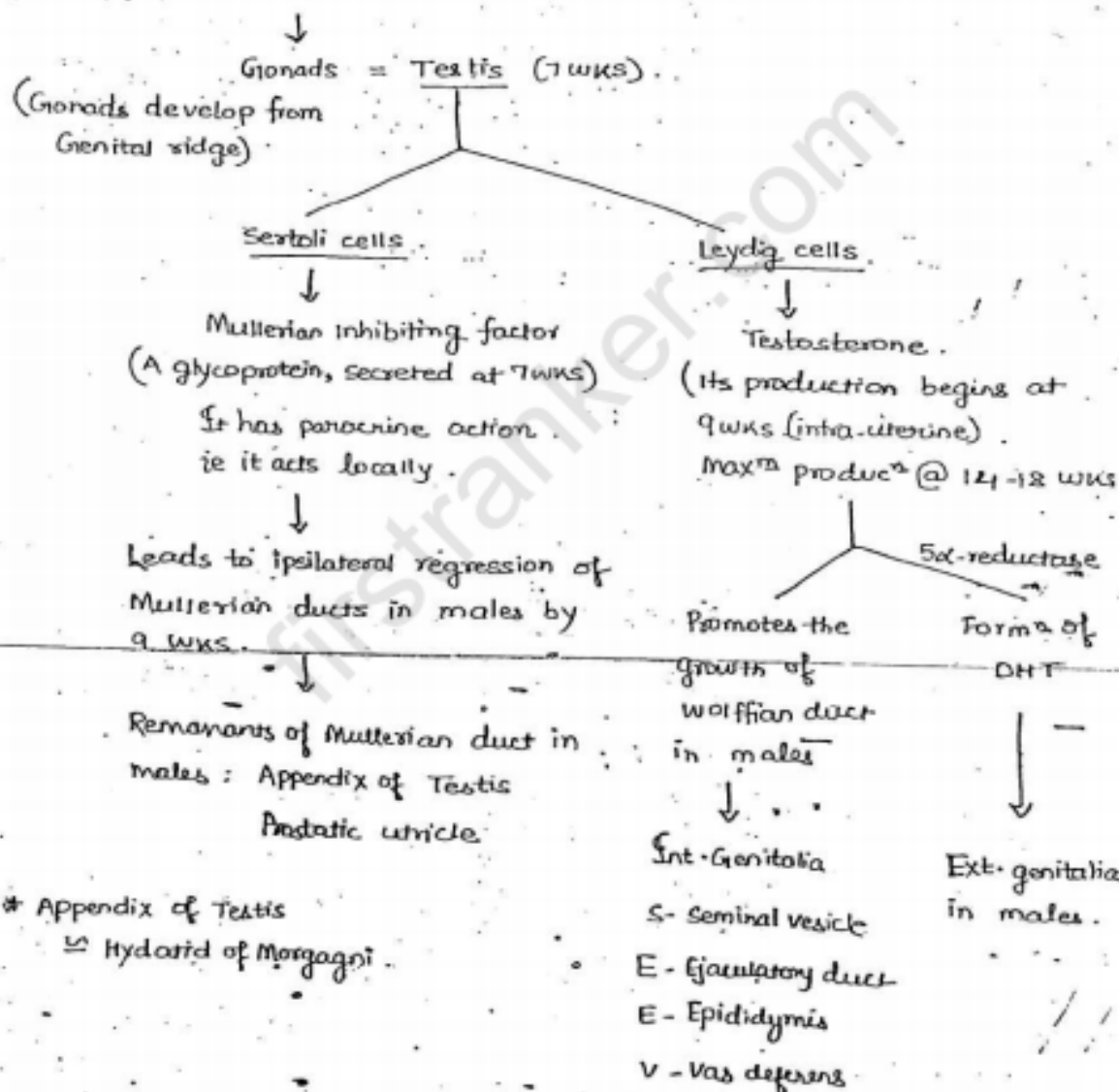


Stem cell differentiation:

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

Others :

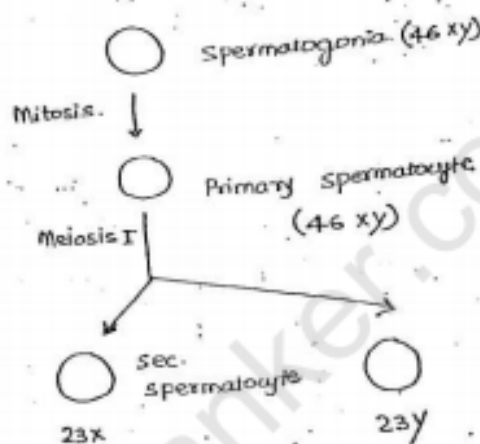
If SRY gene is present.



* Appendix of Testis
= Hydroid of Morgagni

21. Cells which lie outside blood testis barrier.

Spermatogenesis.



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).

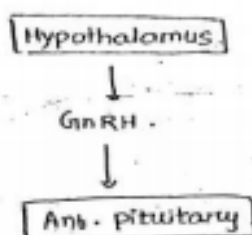
Sperms

○ 55 μ m.

○ Life span - 72 hrs.

○ Sperms attain motility & motility in - Cranial end of epididymis.

Hormonal support of spermatogenesis :



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

3.

Sperm pathway -

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



Motility & maturity @ Epididymis (cranial end).



Vas deferens.



They are released along with seminal vesicle fluid. (60% vol^m + 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 liquifacⁿ occurs in 60 min : 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 with congenital absence of vas def., suffer from seminal vesicle agenesis (CAVD).

They have low semen volume, low pH & low fructose.

Spermatogenesis is, however, normal.



- * Absence of spermatozoa } in semen.
But presence of fructose }

Likely diagnosis may be: Mumps orchitis.

Capacitation:

- Ability of sperm to fertilize ova.
- Begins in female reproductive tract (cervix).
- Major part occurs in Fallopian tube.
- Time taken: 7 hours
- After capacitation, sperm 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 hyaluronidase, acrosin, etc. are released.



In females

SRY gene ~~into~~ Absent



Gonads = ovary.

Sertoli cells absent



Mullerian I.F. absent



Mullerian duct grows into

- Fallopian tube
- uterus
- Cervix
- upper $\frac{3}{5}$ th Vagina.

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

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-o-phoron / organ of Rosenmüller
 - Caudal end. — — — — — Para-oophoron.
3. Wolffian duct proper — — — — — Gartner's duct.

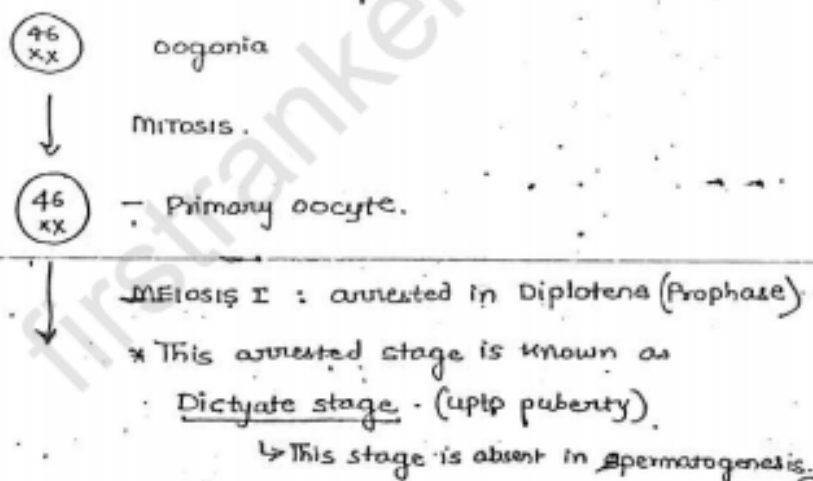
All remnants of Wolffian duct is present in lateral part of broad ligament, except paramesonephros, which is present in medial part of broad ligament.

- * Wolffian duct $\approx$ Mesonephric duct
- Mullerian duct $\approx$ Para-mesonephric duct.

* Organ of Rosenmüller :

- consists Vertical tubules. (Not Horizontal tubules)
- Lined by cuboid epithelia (Not by columnar epithelia).

Oogenesis



At puberty, Meiosis I is resumed.

Meiosis is hormone dependent. $\rightarrow$ LH

$\therefore$ Meiosis I is resumed 24-36 hours before ovulation.

5.

At puberty :

The arrest is over.

(23X)

Sec. oocyte.

(23X)

1st polar body.

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

○

Sec. oocyte.

MEIOSIS II: (arrested in METAPHASE Completed @ Fertilization)

○

Female pronucleus.

○

Second polar body.

Size of ova : 120 microns (largest cell of body).



Follicles

PRIMORDIAL follicle . (size : 0.02 mm)

* Size of follicle just before ovulⁿ = 18-20 mm

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

At birth : 1-2 millions

— 6-7 millions.

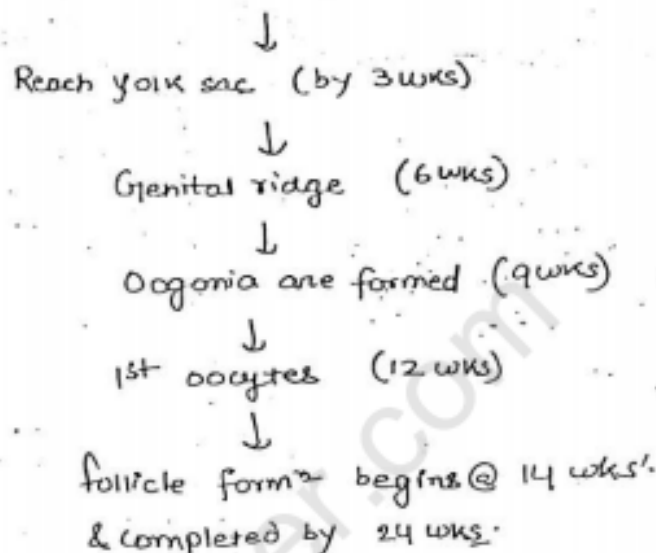
At Puberty : 4-5 lakhs.

400-500 follicles mature in entire lifetime.

1000 follicles

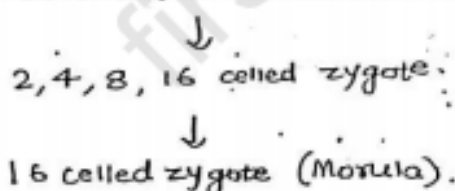
every month.

- Germ cells are derived from Epiblast/ectoderm.



- Fertilization:

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



* zygote stays in Fallopian tube for 3 days.

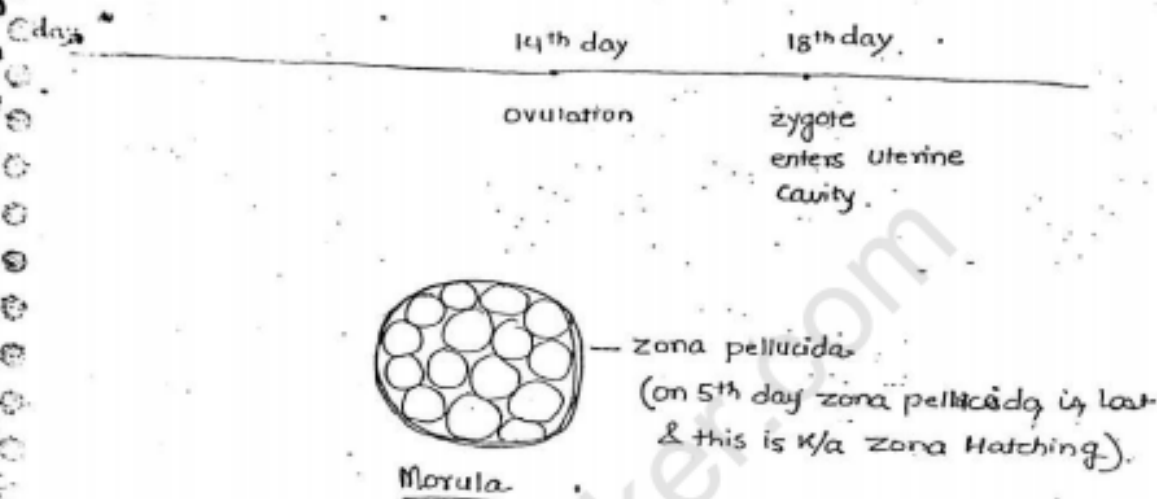
Then it moves towards uterine cavity d/t:-

- (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.

6.

1st zygote enters the uterine cavity : Day 4 after fertilizⁿ.



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

In Blastocyst, cells are arranged in 2 manners.

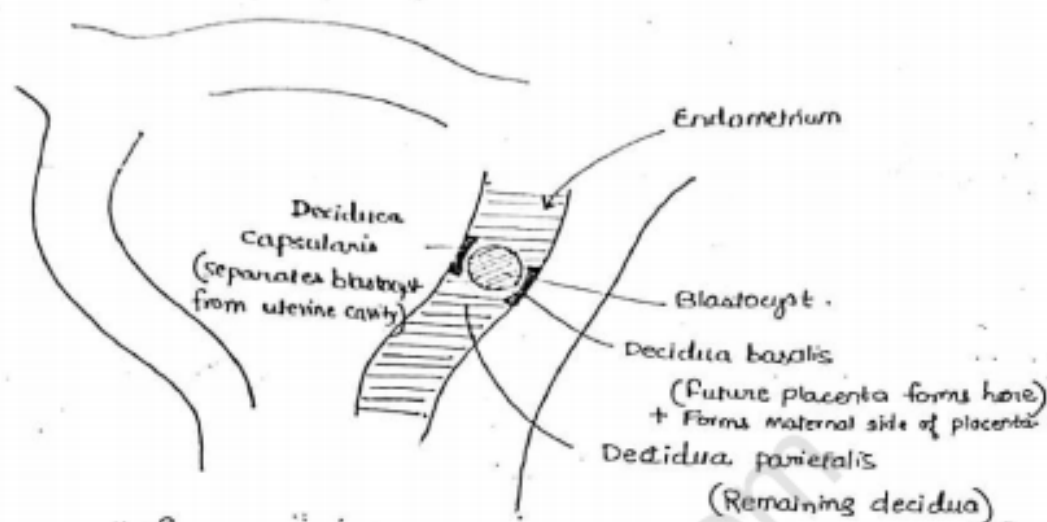
- ~~same~~ TROPHOBLAST : which lines the Blastocyst
- Inner cell mass

Implantation :

occurs in the form of Blastocyst.

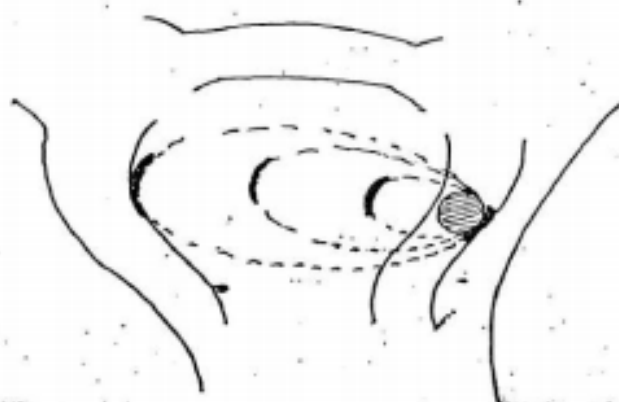
6-7 ~~days~~ days after fertilization : Beginning

Completed by 10 days after fertilization



• Interstitial implantation.

- Thickness of endometrium at time of implantation = 8mm.
- In some females, bleeding occurs at the time of implantation.
 - HARTMAN'S sign.
- After Implantation, Endometrium is k/a Decidua.
- As the fetus grows, Decidua basalis & decidua parietalis fuse by 16 wks. (uterine cavity obliteration).
- ∴ superfetation could be possible only before uterine cavity obliteration. i.e. by 16 wks.



7.



Double ring sign is absent in Ectopic Pregnancy.

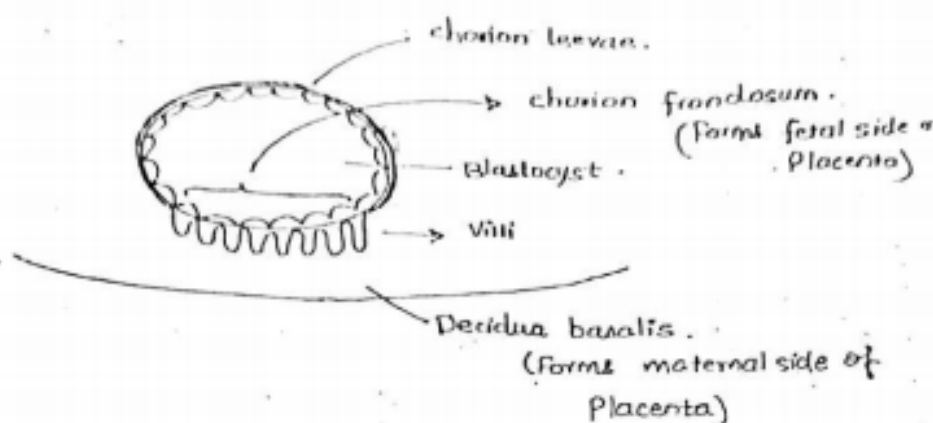
Embryonic period: 3-8 wks after fertilization.
(5-10 wks after pregnancy)

→ Most teratogenic period.

Fetal period: From 9 wks after fertilization upto delivery.

First germ layer formed in fetus: Endoderm.

Trophoblast	{ Cytotrophoblast Syncytio-trophoblast		This differentiation occurs 8th day after fertilization.



• Fetal membranes.

Amnion.

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

Chorion.

Formed 8 days after fertilisation.

- Chorion laevae: fuses to amnion.
- Chorion frondosum

Yolk sac.

First site for hematopoiesis

Allantois.

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

- * Prostaglandin predominantly present in fetal memb. - PGI_2
- ∴ When fetal memb. are ruptured during labour → Prostaglandins are released. & labour proceeds @ faster rate



8.

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 2,3DPG. | - More 2,3DPG. |
| $\therefore$ Higher affinity for O_2 . | $\therefore$ Less affinity for O_2 . |
| - Less of carbonic anhydrase. | - More: |
| - Resistant to acid & alkali. | - Sensitive to acid & alkali |
| | $\therefore$ gets hemolysed. |

* Apt test / singer alkali denaturation test.

It is a qualitative test done in vasa previa, to differentiate
b/w maternal & fetal blood.

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 to & 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



9.



Villous = Fetal blood = 350 ml.

Inter-villous space = Maternal blood (150 ml).

$\therefore$ 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 do not mix & each other & are separated by

- | | |
|-----------------------------------|---|
| (i) cyto & syncytio-trophoblast | } Placental membrane
or
Placental barrier |
| (ii) Extra-embryonic mesoderm | |
| (iii) Fetal capillary endothelium | |

Wt of placenta at term = 500 grams.

Ratio of wt. of placenta : Fetus = 1:6.

Fetal side of placenta

- (i) - Shiny & grey.
- (ii) - Membranes attached to it. Cord attached to it.
- (iii) - Formed by trophoblast or chorion frondosum.
- (iv) - Forms $\frac{4}{5}$ th part of placenta.

Maternal side of placenta

- (i) - Dull red
- (ii) - Divided into polygonal areas - lobes.
• Each lobe is divided into lobules / cotyledon.
- (iii) - Formed by decidua basalis
- (iv) - Forms $\frac{1}{5}$ th 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 cytotrophoblast
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

10.

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 circulaⁿ 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 circulaⁿ 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.

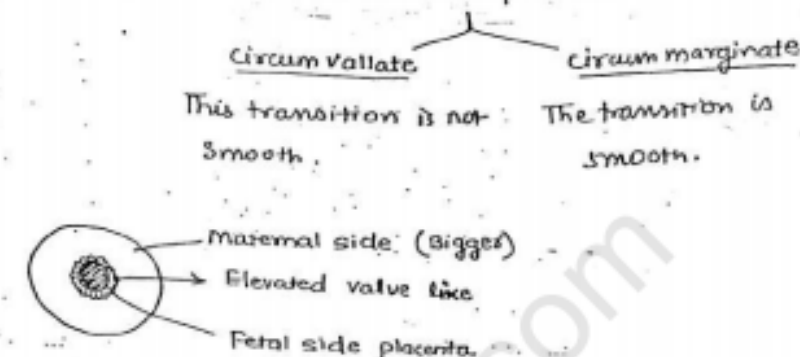
	umbilical artery	umbilical vein.
O_2 Satura ⁿ	50-60%	70-80%
Pressure	60-80 mm Hg	8-10 mm Hg
Remnant	Medial umbilical lig.	Ligamentum teres.

- Medial umbilical ligament is a remnant of urachus.

Anomalies of placenta

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

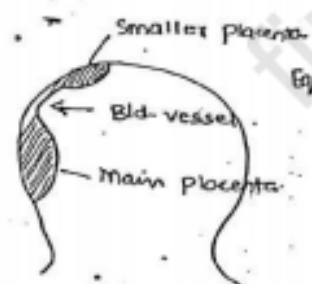
But if fetal side is small $\Rightarrow$ Extrachorial placenta.



CIRCUMVALLATE

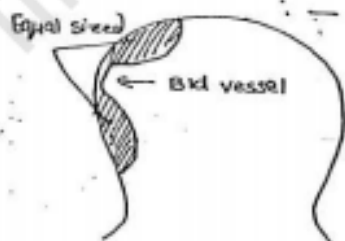


CIRCUMMARGINATE



Placenta Succenturiata

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



Placenta Bilobata



Placenta spuria.



- When umbilical cord is not attached to centre,
but on any one side $\Rightarrow$ w/a Lateral insertion of cord.
& Such a placenta is w/a **BATTLEDORE PLACENTA**.

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 attached~~

invades into myometrium

Pl. attached to serosa

- Decidua basalis is absent

Nitabuch's layer "

" - Histologically } in above conditions

- Highest Risk factor for placenta accreta: ~~prev~~ placenta previa in present pregnancy

placenta previa in previous preg.

is not a RISK FACTOR

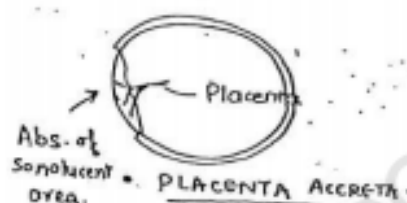
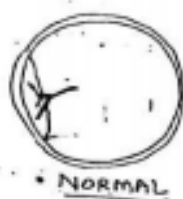
- Previous caesarean (2nd High)
- Previous D&C
- Previous MTP

Presentation:

If undiagnosed :- Presents as refractory PPH.

If Diagnosis :- In antenatal period.

Tvs :- 1) Absence of subplacental sonolucent area (which represents decidual basalis)



2) Myometrial thickness $< 1\text{mm}$

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

4) on Doppler's - Vascularity is $\uparrow$

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

chandraharan, from chennai, has proposed PPP (12)
Procedure for Placenta accreta (when pt. refuses hysterectomy)

- P - Peri-operative placental localisⁿ
- P - Pelvic devascularisation
- P - Placental non-separation treated $\pm$ myometrial excision

Functions of placenta :-

Mainly Endocrinal.

HORMONES :

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

* Atax stelleria rxn :- Adenomatous changes that occur in endometrium (d/t Progesterone).

II. Estrogen

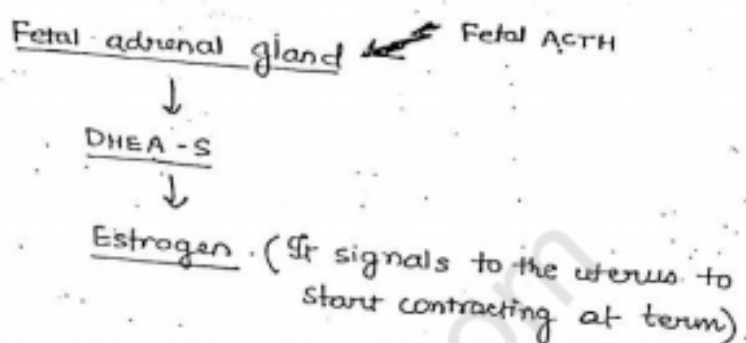
- Placenta lacks 17 hydroxylase enzyme, so it can't

synthesise estrogen using maternal precursor.

It depends on fetal DHEA Sulphate for estrogen synthesis

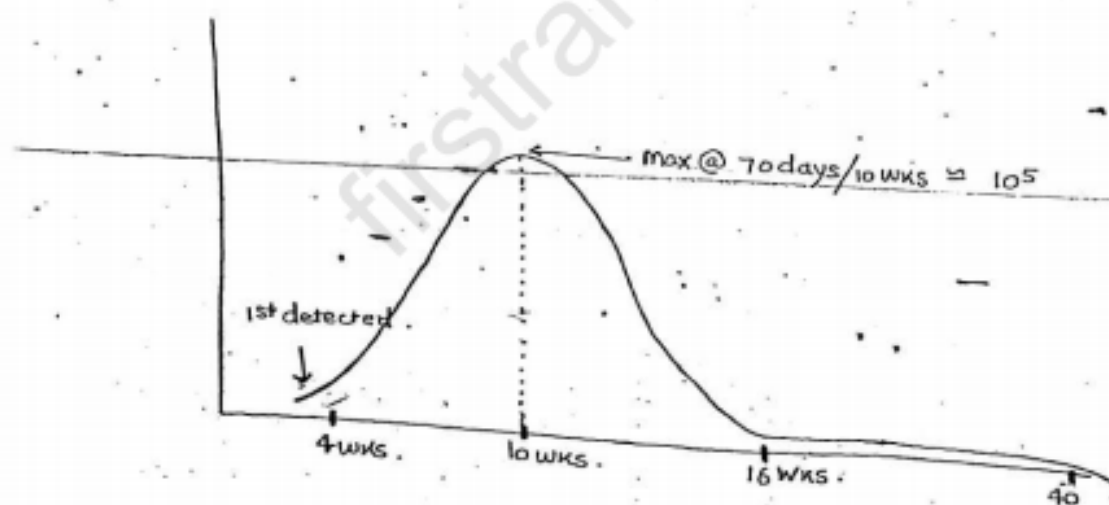
* Main product of fetal adrenal gland - DHEA Sulfate

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



III. HPL

IV. HCG



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

After abortion → 4-6 weeks,

Down syndrome

Trisomy 21

Extra chromosome can be d/t.

Non-disjunction.

in 95%

Recurrence = 2%.

Translocation of chromosomes.

in 4% cases

B/w chr. 3/21.

11/21.

21/21 :- Balanced

translocn.

(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 & 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.

- (i) PAPPA (Pregnancy associated Plasma protein A) or. (Schwanger shaft- protein) $\rightarrow$ Low
- (ii) HCG $\rightarrow$ High.
- } Dual test

2nd trimester :

- ① USG : Nuchal fold/skin thickness.

$\rightarrow$ Measured in trans cerebellar view.
 $\rightarrow$ If $\geq 6\text{mm}$ $\rightarrow$ Then +ve for D.S.
 $\rightarrow$ Measured b/w 15-20 wks.

- ② Biochemical test :

- (i) Triple test : HCG $\uparrow$ + AFP $\downarrow$ + unconjugated estrogen $\downarrow$.

- (ii) Quadruple test : HCG $\uparrow$ + AFP $\downarrow$ + unconj. estrogen $\downarrow$ + Inhibin $\uparrow$.

Definitive diagnosis : KARYOTYPING.

- Viable cells are needed for diagnosis.
- They are arrested at metaphase, using colchicine $\rightarrow$ G₁ Banding.
- $\therefore$ 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.

Chorionic villi sampling

Amniocentesis

14.

Fibrocytes / Fibroblasts

≥ 15 WKS

16-18 WKS.

Earlier, amniocentesis was done b/w 11-14 wks but now not done due to Post Procedure abortion.

Fetal loss : - 1 in 300-500

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%.

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 ($\therefore$ early results in 6-8 hours)



First 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. complication of chorionic villi sampling : Fetal loss.
- Mc. " " " done before 9 weeks : 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 to 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.

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 :- Acetyl cholinesterase (NTD)

Recurrence rates :- After 1 NTD = 5%.

After 2 " = 10-15%.

Low fetal adrenal gland hypoplastic/absent. → DHEAS absent
→ Estriol ↓ → post dated Preg.

umbilical cord

- Develops from connecting stalk
- In early fetal life, cord has 2 arteries & 2 veins, but later 1. umb. vein disappears, leaving only the original 1. 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 Hobboken' 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 memb. & then to placenta.

It gives rise to Vasa previa



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 not~~

APT test

Antenatally - can be detected by Doppler usn



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 wks)



Recent ACOG Guidelines

- If cs. is being done 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 steroid)

Doc: Betamethasone

- 12mg: 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.

Max volume = 36-38 wks.

Composition: 98-99% water.

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

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

17.

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

Fetal urine producⁿ at term = 650 ml/d.

(at 20 wks = 5 ml/hr, at 40 wks = 52 ml/hr)

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

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

After 42 wks : Post term

What % of females deliver on EOD = 4%.

What % of females deliver on EDD = 4%
50% deliver either 1wk before or 1wk after EDD.

1st step : Review of her menstrual his.

Congenital anomaly that leads to Post dated Preg
= Amniocentesis.

Aminiotic 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

> 25 cm: Polyhydramnios.

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

2-8 am.
 < 2 = oligo.
 > 8 = poly

Oligohydramnios

- AF < 200 ml
- 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 in oligohydramnios:

If oligohydramnios occurs early in preg.

↓
 Uterus will expand less

↓
 Less space for fetal parts to grow properly.

Pulmonary Hypoplasia (mc)

Limb reduction 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.

18.

Fetus swallows meconium stained A.P.



Meconium Aspiration syndrome.

Management: Amnio-infusion & Normal saline.

Polyhydramnios:

AFI ≥ 2 L.

AFI > 25 cms.

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.

Rest all signs (+ve at 8 wks).

3. Chadwick's / Jacquemier's sign - Bluish discolorⁿ of vagina.

4. Osiander's sign - Pulsation in lat. fornix.

5. Piskacek sign - d/t lateral implantation

Other probable signs are :-

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

Ext. & int. ballotment

Definitive signs of pregnancy :-

1. Fetal movements can be felt

2. USG evidence of preg.

3. FH's 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.

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

Presumptive signs of pregnancy - All the symptoms.

- * BMR $\uparrow$ by 10-20%.
- * Total water retained - 6.5L.
- * Na & K retention occurs during preg.
But $\downarrow$ water retention, Serum Na & K $\downarrow$.
Plasma osmolality $\downarrow$.
- * Extra calorie req: $\therefore$ 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.

	<u>BMI's</u>	<u>Recommended wt. gain</u>
Under wt.	< 18.5	12.5 to 18 kg
Normal.	18.5 - 24.9	11.5 to 14 kg
Over wt.	≥ 25	7 - 11.5 kg
Obese.	≥ 30	< 7 kg.

changes in Hematological System

↑ (Increase)

↓ (Decrease)

- Blood volume (30-40%)
- Plasma " (40-50%)
- RBC " (20-30%)

- Hemodilution →

Viscosity of blood ↓

Plasma osmolality ↓

Hemodilution during pregnancy.

↓ Packed cell volume
($\frac{\text{RBC vol}^m}{\text{Plasma vol}^m}$)
Hematocrit

↳ Physiological anemia in preg.
(But Hb will never be less than 11 gm/dl)

- Hb mass ↑
(gm)

But Hb concentration ↓
(gm/dl)!

↓
O₂ carrying capacity ∴

- WBC count ↑

Platelet count ↓
(Benign Gestational
Thrombocytopenia)

- Plasma protein amount ↑

Plasma protein concⁿ ↓

Albumin ↑
Globulin

Globulin ↓
Albumin

($\frac{\text{ALBUMIN}}{\text{GLOBULIN}} = 1.7 : 1$)
(Normal)

In Preg. ⇒ 1 : 1

- Clotting factors ↑
(including fibrinogen).

Factor XI & XIII

Preg is hypercoagulable state.

* BT & CT are
unaffected during Preg.

↓
↑ ESR & C-reactive protein

- Anemia $< 11 \text{ gm/dL}$
Severe anemia $< 7 \text{ gm/dL}$
Very severe anemia $< 4 \text{ gm/dL}$
- MC: non-developing
- * MC: Iron-def. (In developing countries = Dimorphic)
↳ Iron def + folic acid def.
- * 1st sign in Iron def. anemia: $\downarrow$ S. Ferritin (most sensitive)
- * Iron-requirement during Preg: - 1000 mg
(300 mg - 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 Govt = Fe + Folic acid
(100mg) (500µg)

↓
Ferrous sulphate. (Ferrous fumarate: In ACPs)

- After giving iron,

1st parameter to $\uparrow$ = Reticulocyte count

$\uparrow$ in reticulocyte count occurs after 7 days of giving iron
Max^{re} $\uparrow$ " " 14 days " "

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

Best measure for Hb: cyanomethemoglobin method.

21.

Indications for Blood transfusion in Anemia

- If anemia is d/t hemorrhage (eg. APH)
- If a female presents $\bar{c}$ severe anemia beyond 36 wks of pregnancy.

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

Changes in GI/T

- No change in Gastric emptying time during preg
- $\uparrow$ during labor.
- $\uparrow$ chances of Reflux oesophagitis as progesterone relaxes LES
- splenic area $\uparrow$ by 50%.

Changes in Liver

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

Changes in Gall bladder

- cholestasis. (d/t estrogen)
- Progesterone $\downarrow$ tone
- Both these findings along $\bar{c}$ $\uparrow$ cholesterol favour GB stone formation.

cholestasis of pregnancy.

- 2nd m.c.c. of Jaundice in Preg. (m.c.c. - Hepatitis)
- $\uparrow$ S. Bile acid $\rightarrow$ Itching/pruritis (1st symp.) - Palms & soles
- $\rightarrow$ more common in 3rd Trimester
- Bilirubin never more than 5
- Liver enzymes are mildly $\uparrow$ (Never more than 250)

• 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 ↑ (max^m levels)

- But Incidence of Prolactinoma doesn't ↑ in preg.

Changes in CVS

Increased

- Cardiac output
= (Stroke Vol X HR)
- Preload
- O₂ requirement

Decreased

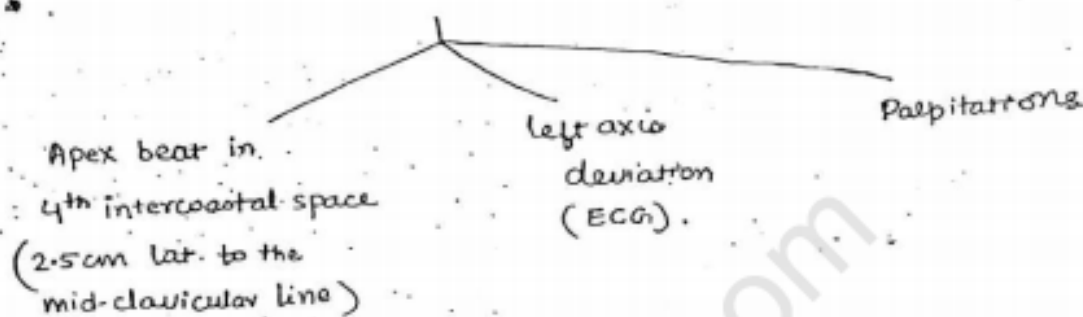
- Progesterone ⇒ smooth muscle relaxant
- Peripheral vas. resistance (∴ BP ↓)
- After load ↓
- ↓ Arterio-venous O₂ gradient

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

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

Changes in clinical indicators of Heart during Preg.

- 1) HR, Pulse $\uparrow$
- 2) BP $\downarrow$
- 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 murmur (continuous murmur).

- 6) on X-ray - mild cardiomegaly
- Straightening of left heart border

Supine Hypotension / Menzies's syndrome

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

↓
Gravid uterus presses against the IVC.

↓
Venous Return to Heart $\downarrow$ (CO $\downarrow$)

↓
Mother
HR $\uparrow$, BP $\downarrow$

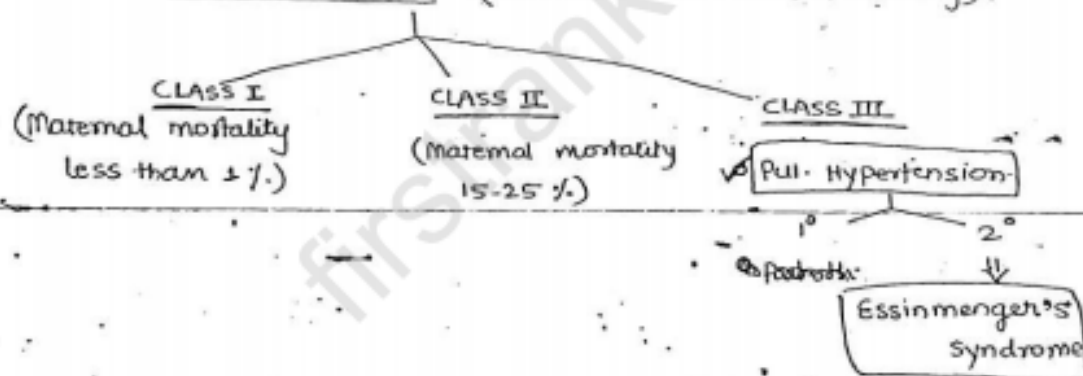
↓
Fetal distress

Indicators of Heart disease in Pregnancy.

- Clubbing, cyanosis, Raised JVP
- Persistent splitting of S₂ / 4th H.S.
- Diastolic murmur
- Systolic murmur (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 classification (Based on maternal mortality).



* 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

23. HD & max risk of maternal mortality :- Eisenmenger's syndrome.

• HD leading to mat. mortality :- MS.

• If which Preg. is C/I -

- (1) Eisenmenger's class III disease.
- (2) Ejection fraction $< 40\%$ (WHO $< 30\%$)
- (3) Severe MS (Valve area $< 1.5 - 2 \text{ cm}^2$)
- (4) Severe AS (Valve area $< 1 \text{ cm}^2$)
- (5) HD belonging to NYHA III/IV.

• Mc fetal complicⁿ of HD - Prematurity, IUGR.

Management of HD during labour -

- Left lateral position / O₂ bedside.
- Restrict PV examination.
- Restrict fluid to 75ml/hr.
- Allow pts. to go into spontaneous labour $\rightarrow$ 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 $\frac{1}{2}$ hr, then ventouse/Forceps should be used. (Vacuum or ventouse: Better b/w two)

HD where C.S. is indicated. (wherever aorta is involved)

- Aortic aneurysm
- Coarctation of aorta
- Marfan Syndrome & aortic involvement
- Severe symp. aortic stenosis
- Acute severe CHF/Recent MI



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

- Anaesthesia given during LSCS in HD : Epidural anaesthesia

* C/I of epidural anaesthesia : (GIA is to be given)

- 1) Intra-cardiac shunts
- 2) HCM
- 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 Pregn

Choice of Anticoagulant during Preg.

Period of Gestation

< 12 wks

12-36 wks.

≥ 36 wks.

Anticoagulants of choice

Heparin (warfarin causes CHONDRODYSPLASIA of fetus)

warfarin

Heparin

→ If a female is on warfarin during labor → C.S.

→ & Restart - 6 hrs after vas. del. & 24 hrs after C.S.



Why switchover to Heparin at 30 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

Fetal surgery of choice was valvotomy

Today surgery " " - Transcatheter balloon dilatation

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
(Laparoscopy - c/i) (Local anaesthesia)

Temp. methods:

→ OCPS : c/i

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

Progestrone 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 pressure ↑

Renal blood flow ↑ ∴ GFR ↑

ie. Filtering capacity ↑ → ↑ increased clearance of urea, uric acid & creatinine
↓
∴ s. urea/uric acid/creatinine ↓



Diabetes In pregnancy.

- Pregnancy is a diabetogenic state, d/w -

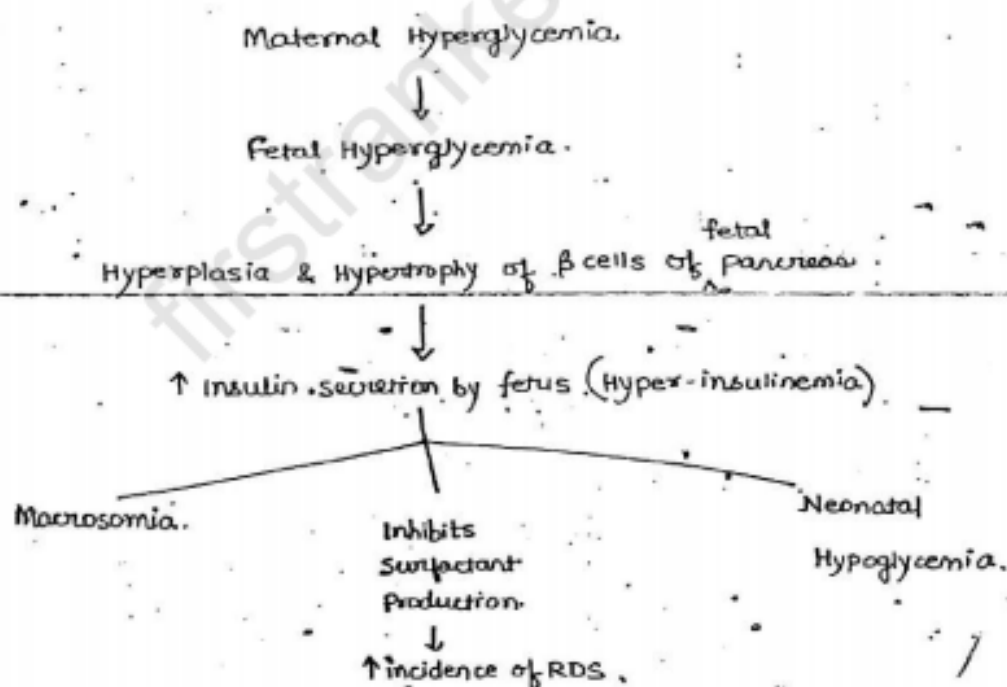
- Hyper-insulinemia
- Insulin resistance. (max: 24-28 wks).

↳ d/t: H - HPL
P - Progesterone
E - Estrogen
C - Cortisol.

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

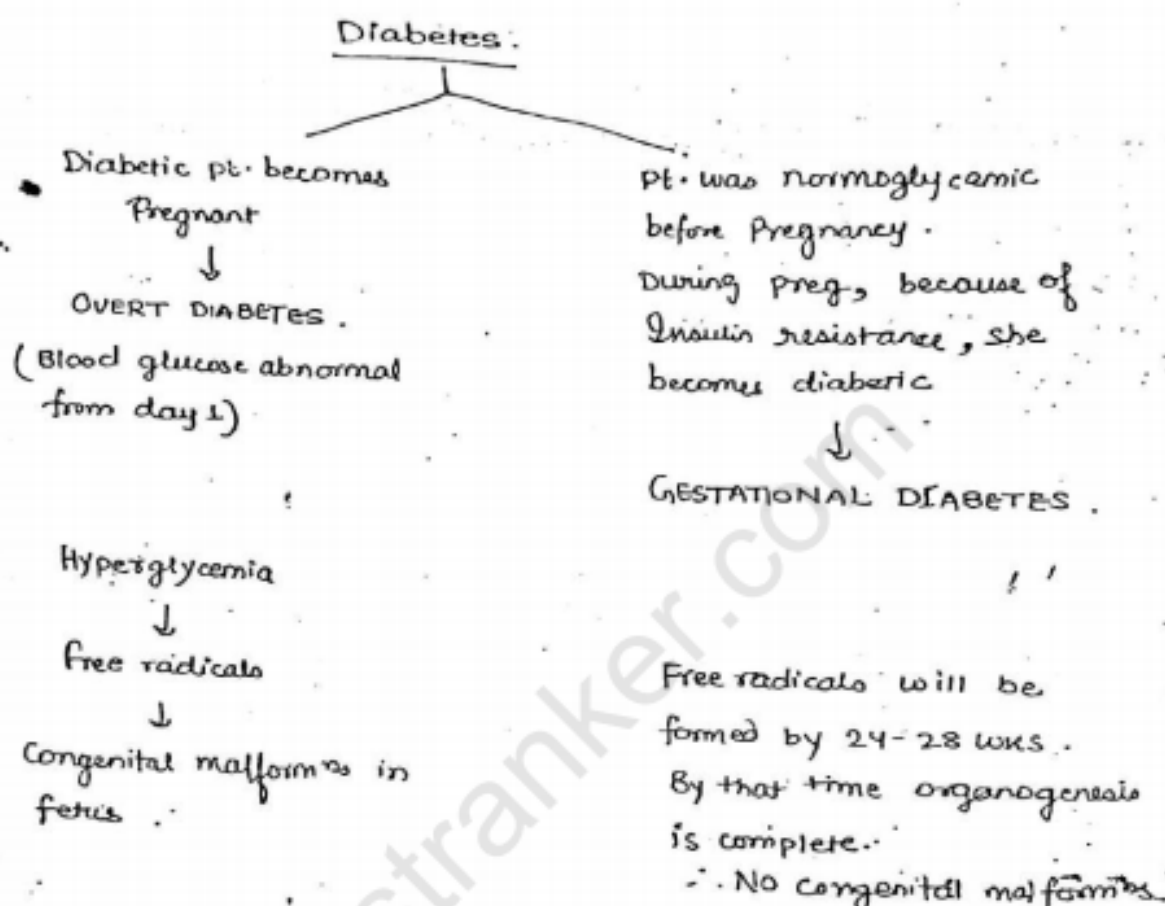
* Main source of fetal energy is glucose.

This glucose is transferred by facilitated diffusion across the Placenta using GLUT1 & GLUT3.



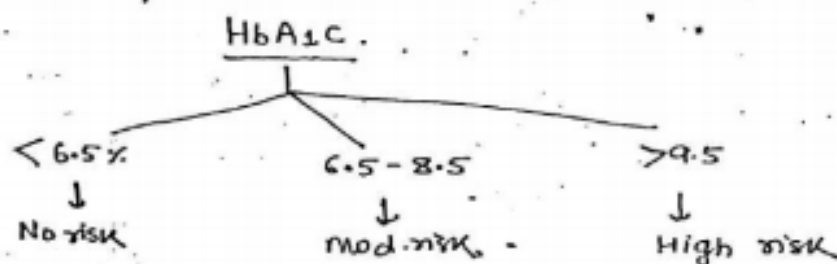
- * d/t maternal hyperglycemia → Placenta undergoes hydropic changes (as glucose imbibes water), so it becomes non-functional at places.
- This creates a disparity in demand & supply → Hypoxia → sudden IUD.

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



In Diabetic females:-

which test predicts the risk of congenital malformations:-



To detect congenital malformations - best test:- Level 2 USG @ 18-20 wks. (TIFFA)



In overt diabetics : fetal cardiography (@ 22-24 WK)
to detect heart anomalies
(Not done in Gestational DM).

mc. system involved in congenital malformations :-

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 w/ previous H/O NTDs.

(ii) Diabetic females

(iii) Females on anti-epileptic drugs

(iv) Females w/ 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

Diagnosis of GDM

2 step approach (ACOG)

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

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

1 hr < 180

2 hrs < 155

3 hrs < 140

Carpenter &
Caution
Criteria

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

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.

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

National diabetic criteria:

FBS < 105 mg/dL = (5.8 mmol/L)

1 hr < 190

2 hrs < 165

3 hrs < 145

Divide mg/dL by 18
to get into mmol/L.



Indications of screening for GDM

↓
All High risk females.

- ↳ Previous H/O GDM, Big baby, Baby $\geq$ congenital formⁿ 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 $\uparrow$, as the gestational age $\uparrow$.

But $\downarrow$ during labour. (because pt. is on liquid diet).

Overt diabetics & end pts. on insulin are admitted at 34 wks.

Time & Mode of delivery:-

- GDM controlled on diet : 40 wks.
- GDM controlled on insulin : 38 wks.

- overt diabetes : 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 Low vascular complicaⁿs :-
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 :

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



Macrosomia:

wt. of fetus ≥ 4.5 kg. (in India ≥ 4 kg).

MCC — Mother $\left\{ \begin{array}{l} \text{Diabetic} \\ \text{obese} \end{array} \right.$

• Post dated preg.

• Male fetus

• Best USG parameter to detect macrosomia — Abd. circumference

USG parameters to date:

• 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 : Johnson's formula

Management of macrosomia:

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

• Complication of macrosomia : SHOULDER DYSTOCIA.



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 maneuver/Best maneuver/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. Zavanelli's maneuver. - Push the ^{delivered} head of the baby back & Perform C.S.

* Mc complication following Shoulder dystocia: pPH
Mc fetal complication: Brachial plexus injury > #clavicle > #Humerus



IUD : Death of fetus beyond the period of viability.

1st X-ray sign to become Qive : ROBERT'S SIGN (it 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 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

All are seen in Gestational DM except.



30.

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 labourSuppose, a G₁₂ P₁₊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 stop it (ie. not a Tocolytic).

2. stop smoking3. cervical cerclageDiagnosis :-① USG :-

Normal length of cervix during preg :- 4 cm

* If length becomes < 2.5 cm $\Rightarrow$ Pre-term labour.

* U shaped cervix.

(Normal: T shaped)

* Dilatation of cervix > 2 cm $\Rightarrow$ " "② Fibronectin level :- By ELISA (B/w 24-34 wks)If Fibronectin > 50 ng/mL $\Rightarrow$ Pre-term labour.③ uterine contracⁿ : 4 contracⁿ in 20 min } $\Rightarrow$ Pre-term labour
or 8 " " 60 min }



Preterm lab. $\rightarrow$ Fetal lungs don't mature $\rightarrow$ RDS.

Management:

- Preterm lab. ≥ 34 wks. : wait & watch.
Send Rectal & vaginal swab for detecting Streptococcal infection, 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 membr. are ruptured.

Tocolytics:

- Best tocolytics : Nifedipine
- Best " in HD pts : Atosiban
- Tocolytic $\bar{c}$ max maternal side effects : β agonists / β mimetics.
(Salbutamol, Ritodrine, Isoxuprine, Terbutaline)

β mimetics can lead to Tremor (mc side effect),
Hyperglycemia, Hypokalemia, & Pul. edema.

- $MgSO_4$ acts as a Tocolytic @ concn of 9-10 meq/L.
- Indomethacin is also used as tocolytic. But it leads to closure of ductus arteriosus. ($\therefore$ it has max fetal side effect)



31.

In PROM, there is risk of chorioamnionitis,

∴ If PROM occurs > 34 wks.

Management :

- Antibiotics.
- Induce labour.
- Min^m pv-examination.
(Per speculum examⁿ is done).

Nitrazine test : For diagnosis of PROM.

Pregnancy induced hypertension.

32

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
condemns.



Chronic HT in preg.

- Increase in BP is seen before 20 wks of preg.
- BP will not come back to normal after 12 wks of delivery.

A Normotensive female becomes
hypertensive during pregnancy
(d/t Placental Pathology)



PIH.

- Increase in BP is seen after 20 wks of pregnancy.
- 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.

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
(i.e. 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.

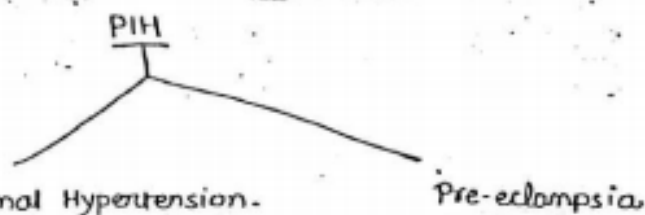
- Placenta is small in case of PIH.

* causes of big placenta:

- DM
- Rh-incompatibility
- TORCH infections

- 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.



→ All 3 characters of PIH are seen.

→ (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 proteins in urine $> 300 \text{ mg/day}$
or $> 30 \text{ mg/hour}$

Signs of End organ damage -

• Platelet count $< 1 \text{ lakh}$

• S-creatinine $> 1.1 \text{ mg/dL}$

• Raised liver enzymes to more than Normal Value.

• Pulmonary edema

• Visual symptoms / cerebral symptoms.

Mild pre-eclampsia: Pre-eclampsia without severe features.
 $\text{BP} > 140/90$ but $< 160/110$

Severe pre-eclampsia:

$\text{BP} > 160/110$ OR Signs of End organ damage

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

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

• Chronic HT & 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 $\downarrow$ IL-2 & TNF)

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

- GFR $\downarrow$ in pih, because Renal blood flow $\downarrow$

$\therefore$ 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)



But when Blindness is due to retinal detachment $\rightarrow$ Blindness is irreversible

$\therefore$ Fundal exam is done in pts.

Convulsions : Reason

$\therefore$ 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: Intracranial bleeding.

* HELLP syndrome - 1st described by Weinstein.

$\hookrightarrow$ A complication of severe pre-eclampsia.

$\hookrightarrow$ In DIC, coagulation profile is deranged. But in HELLP Syndrome, coag. profile is normal.



- In 15% of HELLP syndrome, BP can remain normal 35.
- 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 ($< 1\text{lac}$).

- criteria for diagnosing HELLP syndrome: Tennessee criteria

Platelet $< 1\text{ lac}$.

AST > 70

LDH > 600

- Mississippi classification:-

Class I: most severe.

Platelet $< 50,000$.

Class II: Platelet: $50,000 - 1\text{ lakh}$

Class III: Platelet: Normal ($1 - 1.5\text{ lakh}$), But AST & LDH $\uparrow$.

Management : Immediate termination of preg.

Pt. < 34 wks - Steroids. → Then terminate preg.

Pt. > 34 wks - Terminate.

Differential diagnosis :-

Acute fatty Liver of pregnancy (AFLP).

- ↳ Hypoglycemia
- ↳ Deranged coagulation profile
- ↳ Hepatorenal Syndrome
- ↳ Acute Pancreatitis.

→ Absent in HELLP syndrome

Management of AFLP : 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 :

- Hematocrit/concentration
- Uric acid levels
- Hypocalcemia
- ↑ 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.



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

↳ Antioxidants

↳ 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/
Hydralazine : 2nd best. acute hypertensive crisis
d. 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 10 mins

80 mg

Repeat 80 mg every 10 min.



Max dose : 220 mg

Hydralazine

5mg initial dose followed by 5-10mg

Antihypertensives c/I :-

- ACE inhibitors
- Losartan
- Diltiazem
- 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 : mgso₄
- Antihypertensive
- Def. management : Termination of preg (@ ≥ 34 wks)
Mode: vaginal del.

Indications for immediate termination irrespective of gestational age.

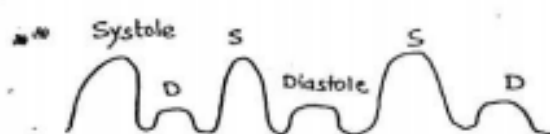
1. Impending eclampsia
2. Eclampsia
3. Fetal distress/ Placental abruption
4. Uncontrolled BP/ Rising s-creatinine

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5. HELLP syndrome

6. Reversal of end diastolic flow in umbilical artery doppler.

Umbilical artery Doppler.



Normal : • forward flow

- As $\nearrow$ period of gestation $\nearrow$, resistance of vessels $\downarrow$,
 $\therefore$ Diastolic blood flow $\uparrow$ i.e. S/D ratio $\downarrow$

Progesterone is a smooth muscle relaxant.

In PIH : S/D ratio $\uparrow$

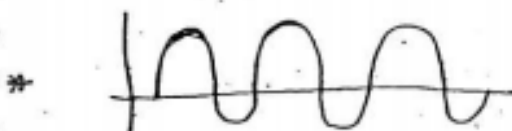
(Because : Resistance of vessels $\uparrow$)



$\rightarrow$ ABSENT END DIASTOLIC FLOW

$\hookrightarrow$ SEVERE PIH.

(Term of preg ≥ 34 wks)



$\rightarrow$ Reversal of Diastolic flow

$\hookrightarrow$ Immediate termination of pregnancy.

Management of eclampsia

- 1st line management :

- Airway management.

→ Then drug to $\frac{1}{2}$ convulsion: $MgSO_4$.

→ Antihypertensive. (Labetalol)

- Def. management : - Term² 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. ($\therefore$ should be used cautiously $\propto$ CCBs (Nifedipine)).

- MC regime for giving $MgSO_4$

loading : 4gm slow IV dose

→ 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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MgSO₄ toxicity

- 1st sign: Absence of knee jerk

↳ @ MgSO₄ ≥ 10 meq/L.

- Respiratory arrest - @ Mg = 12 meq/L

• " " arrest - @ Mg = 15 "

- Cardiac " - @ Mg = 30. "

* Continue giving MgSO₄ till 24 hrs after delivery ~
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 MgSO₄

↳ severe pre-eclampsia

↳ Impending eclampsia

↳ Eclampsia

↳ HELLP

↳ Neuroprotective - to prevent cerebral palsy

↳ Pre-term labour





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PPHNormal blood loss after vag. del $< 500\text{ ml}$ C.S. $< 1000\text{ ml}$ C.S. hysterectomy $< 1500\text{ ml}$

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

Primary

in 24 hrs



causes: 4-TS

- $\downarrow$ 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 $\rightarrow$ PPHDiagnosis:

1) Shock index -

 $\frac{\text{HR}}{\text{systolic BP}}$

Normally: 0.5-0.7

If it becomes 0.8-1.1 $\rightarrow$ Blood transfusion/Immediate resuscitation

2) Obstetrical shock index

N = 0.7-0.8

If > 1 : BT

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 separ.:

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

(Just after delivery, Fundal Ht. → just below umbilicus)

Methods of Placental separ.

Schultz method

- Mc method
- Shiny fetal side comes out first
- Retroplacental clot formed - Hemostatic
- ∴ Less Bleeding
- Placental sep. begins from centre
- 80%.

Duncan method

- Less common
- Dirty maternal side comes out first
- No retroplacental clot formed
- ∴ More Bleeding
- Periphery
- 20%.

* Line of sep. of placenta lies along zona spongiosa

Active management of 3rd stage of labour:

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① Uterotonics at the time of delivery of shoulder.

Oxytocin

- IM/IV infusion
- Never IV Bolus because it can cause Hypotension & cardiac arrest.

Dose = 10 IU.

Methergin

0.2 mg IM

- Side effect: Transient hyp

Syntometrine

= oxytocin (5U) + methyl ergometrine (0.5)

* Carbetocin = ~~100 mg~~

- 100 µg IV.
- Synthetic oxytocin
- longer $t_{1/2}$

* Misoprost = PGE₁

600 µg orally

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

OXYTOCIN

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

Route

Time of onset

Duration of action

- IM Bolus 3 min
- IV infusion Immediate

3 hrs

1 hr

$t_{1/2}$ = 3 min (3-5 min)

Stored at 2-8°C. | At room temp., its shelf life = 3 months
(Kept in fridge)

Methergin

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

Misoprost (PGI₂)

- Used in: Asthmatic Patients (PGF_{2α} causes Bronchospasm. Not used)
- Can't ~~not~~ 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 the cord clamping is delayed = 80ml (some 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.

Management of PPH :

1. Gen. Resuscitation

2.

Atonic PPH

Uterus (Not palpable per abdominally)

Traumatic PPH

(uterus palpable abdominally)

Genital tract injury

Traumatic PPH

due to :- (i) perineal injuries

(ii) Hematoma (vaginal or vulval)

(iii) Uterine rupture

(i) Perineal tear

1° = only skin of vagina is torn

2° = muscle + skin torn

3° = Anus involved

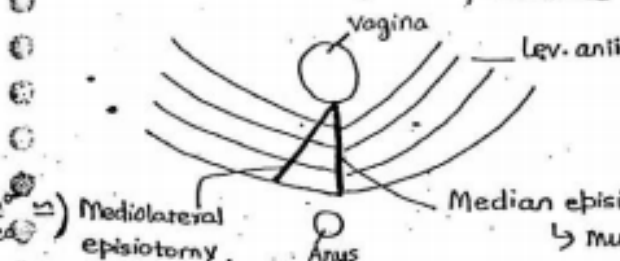
A : < 50% ext. anal sphincter torn

B : > 50% " " "

C : Ext. + Int. anal " " "

4° = All + Rectal mucosa involved

Perineal tear can be prevented by giving episiotomy.



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



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 (Pudendal Artery) vaginal (vaginal artery: source)

Pain, hypotension.
Inability to pass urine

o/e Bluish tender swelling



Conservative management ~~or~~ is done.

Surgical indicⁿ :-

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 — 0.2 mg IM or IV.

(Methyl ergometrine)

or

Ergometrine

0.25 mg IM or IV

Repeat every 15 min

• Carboprost

(PGF_{2α})

250 µg IM.

Max^m - 8 doses at

30 min interval.

side effect

↳ Diarrhoea

• Misoprost

(PGE₁)

800 µg sublingual

or

Per rectal.

• Inj. Tranexa

(Tranexamic acid)

500 mg IM/IV.



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 $\frac{1}{2}$ 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} wear antishock garment

3. ~~Ex~~ 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

• Gurnshella "

• 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 , then 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 arter



- 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.

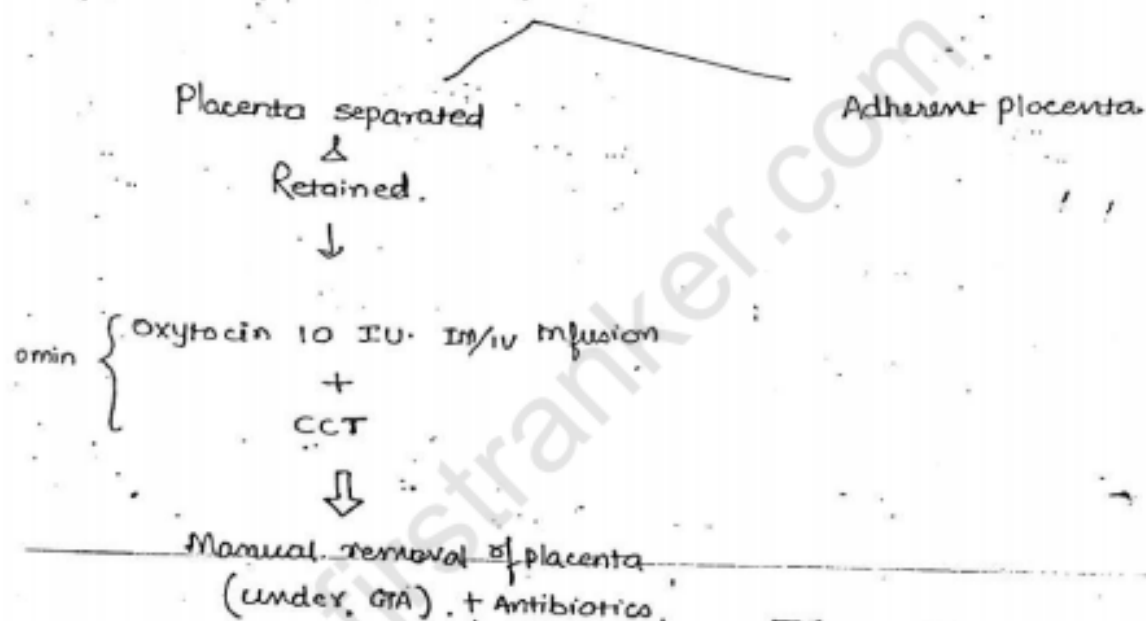
using Polyvinyl Alcohol or polyurethane foam



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:

* WHO doesn't recommend use of methyl ergometrine / prostaglandins for management of Retained placenta

* A female goes into shock after delivery $\Rightarrow$ PPH (mcc)

A female goes " " immediately after delivery $\Rightarrow$ Uterine inversion

A female goes into unexplained shock after delivery $\Rightarrow$ Amniotic fluid embolism



Uterine Inversion.

- Mismanaged 3rd stage of labour is the Mc cause.

- Initially the 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, Hattane's method)

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

Antepartum hemorrhage

Any blood loss that occurs beyond the period of viability upto delivery.

(28 WKS.
22 WKS - WHO
24 WKS - India)

Causes:Placental

- Placenta previa
- Abruptio
- Circumvallate
- Circummarginate

Fetal

- Vasa previa

Pl. previa

- Low lying placenta.

Time - USG = 3rd trimester

- Recurrence: 5%

- Incidence: 1 in 300

Abruptio

Placenta is located

12%

1 in 200

Risk fac:

- Smoking
- ↑ Mat. age
- ↑ Mat. Parity

Highest risk :- Prev. C.S.

Mc risk fac :- Prev. 1/2 Placenta
- Previa

Other risk fac:

- Myomectomy
- D&C
- Endometritis
- Twin preg., ↑ Mat. APP.
- Succenturiate lobe

mc risk :- Previous Abruptio

Other risk fac:

- Smoking, Cocaine abuse
- Folic acid-def.
- Thrombophilia, ↑ B.P.
- 1/2 abdominal trauma
- Twin preg., Polyhydramnios, Rapid Rom

Classification of Placenta Previa

Type I: Placenta is in lower ut segment.

< 2 cm from int.os (Lateral placenta previa)

Anterior

Post.

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 (Hve. !!)

↓
(Push the head of the fetus inside pelvis
→ Cord compression → Fetal distress)

Varieties of Abruptio Placentae

Concealed variety

Blood collects behind placenta

Revealed

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.



Cause of DIC in Abruptio

Premature separation of placenta



Tissue thromboplastin is released $\Rightarrow$ 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 $< 100 \text{ mg/dL}$
1 unit of FFP raises fibrinogen by 10 mg/dL .
3. Cryoprecipitate
4. Platelet transfusion : when Platelet $< 50,000$
5. Recombinant factor VIIa
6. Heparin $\pm$

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

First correct DIC $\rightarrow$ Then vaginal delivery.

Pl. previa

- S/p :- Painless, causeless
- Recurrent bleeding.
 - Bright red blood
 - Initially small bleeding w/ a warning hemorrhage followed by massive bleeding.

Gen. examⁿ

Condition of pt. & amount of blood loss

P/A

- uterus is soft, relaxed, & non tender

FHS is easily heard

Fetal parts are palpable

Ht of uterus = Period of gestation

Abruption

- H/o abd. trauma or PIH
- Pain in abdomen
- No Recurrent bleeding.
- Dark red color blood
- No warning hemorrhage.

Abruption (concealed)

condⁿ of pt. is not proportionate to amount of blood loss.

- uterus is tense, tender & rigid

FHS is not easily heard

Fetal parts not easily palpable.

Ht of uterus > Period of gestation

(Blood enters uterine myometrium → Gives it a blue color)

'COUVE LAIR'S' UTERUS

(Not an indicⁿ for hysterectomy)

P/v :- c/i

Ioc :- TVS

Malpresentation

seen

Transverse lie (mc)
Breech

clinical diag./TVS

Not seen.



Pl. previa

Abruptio (concealed)

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IUGR : Not seen

Seen (as PIH is a risk fac)

Congenital malformⁿ : Seen

Seen

Management of Abruptio

Principles $\left\{ \begin{array}{l} \text{Never wait \& watch.} \\ \text{Never give tocolytics.} \end{array} \right.$

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

Let the ♀ continue Preg.

↓
Expectant management

(Done = lungs of fetus mature)
But, don't put mother's life at risk.

↳ In hemodynamically unstable pt.

↳ Active bleeding

↳ In hemodynamically stable.

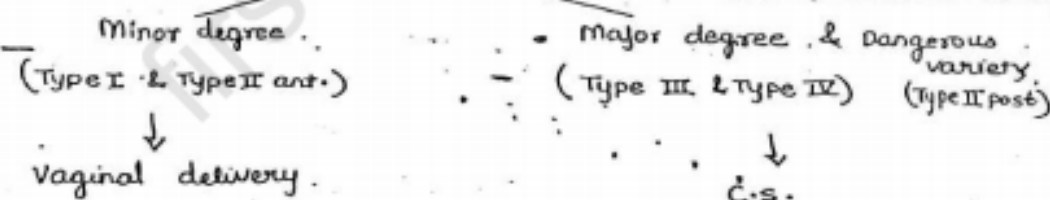
↳ No active bleeding.

- | | |
|---|---|
| <ul style="list-style-type: none"> ↳ Gest. age ≥ 37 wks Pt is in labour ↳ Fetal distress ↳ Fetal malformation on USG, which is incompatible w/ life | <ul style="list-style-type: none"> ↳ Gest. age < 37 wks. ↳ NO fetal distress. ↳ On USG, either the fetus is Normal or, any anomaly that is compatible w/ life. |
|---|---|

Steps in Exp. management :: Macaffee & Johnson regime - upto 37 wks

- ↳ Hospitalize
- ↳ Arrange blood
- ↳ Rh -ve $\rightarrow$ Anti D
- ↳ Inj corticosteroid (Lungs maturity)
- ↳ Any contraindications present :- short term tocolytic (Nifedipine)

Mode of delivery : $\left\{ \begin{array}{l} \text{USG} \\ \text{Double setup exam}^n : (\text{PV exam in or}) \end{array} \right.$



- † If pt is having persistent excessive bleeding $\rightarrow$ C.S.
- † In placenta previa, Normally Lscs is done, but if placenta is located anteriorly, classical cs. is done.
- † Placenta Previa in Present Preg. w/ previous 4/0 C.S. is a risk fac. for placenta accreta.

Molar Pregnancy

Gestational T. diseases

1. Partial mole
 2. Complete mole
- } H-mole

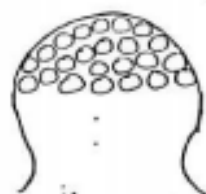
Gi. T. Neoplasia

1. Invasive mole
2. choriocarcinoma
3. placental site Trophoblastic tumor
4. Epitheloid Trophoblastic tumor

H-mole : Bening disease $\bar{c}$ malignant potential

Incidence : 1 in 400 (India)

Max^m incidence : Phillipens (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

↑ β HCG

Fundal Ht.

→ Hyper-emesis gravidarum
(Morning Sickness is $\neq$ HCG)

→ Hyperthyroidism
(Level of HCG resembles TSH)

→ Theca lutein cyst of ovary
(BHCG $\approx$ LH)



USG: Snow storm appearance

PIH (< 20 wks): 0% incomplete trophoblastic invasion

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True pelvis

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

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

Pelvis makes an angle 55° to Horizontal $\rightarrow$ Angle of inclination.

Inlet : AP diameters :-

True conjugate $\rightarrow$ upper border of Pubic symphysis $\longleftrightarrow$ sacral promontory (11 cm).

Obs. conjugate $\rightarrow$ Mid of pubic symphysis $\longleftrightarrow$ sacral promontory (10 to 10.5)

Diag. conjugate $\rightarrow$ Lower border of Pubic sym. $\longleftrightarrow$ " (12 cm)

Transverse dia of inlet : 13 cm.

Mid pelvis : Ap diameter $\rightarrow$ 12 cm

Transverse dia $\rightarrow$ Dist. b/w 2 ischial spines - 10 cm

(Bispinal/Interspinal) $\rightarrow$ Smallest diameter

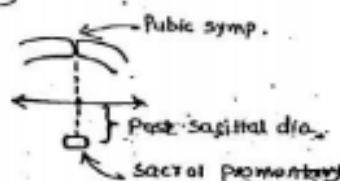
$\therefore$ Plane of Mid pelvis $\rightarrow$ Plane of least Pelvic dimension

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	7 cm



which Ap diameter of inlet can be measured clinically?

→ Diagnet. Diagonal conjugate.

Diagonal conjugate - 1 cm = True conjugate.

Diagonal " - (1.5 to 2 cm) = Obs. "

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

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

* Clinically, $\bar{c}$ one hand, both the ischial spines should not be palpated.

If both ischial spines are palpated $\bar{c}$ one hand, it means the mid-pelvis is contracted. That means interspinous dia < 8 cm

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

- Intertuberos dia < 8 cm : → outlet contracted

* Subpubic angle

♀ = 85-90° (obtuse)

♂ = 80-85° (Acute).



50.

Contracted pelvis.

Types :

- 1) Naegle's pelvis : If anyone ala of sacral bone is absent.
- 2) *Robert's pelvis : Both ala of sacral bone 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 : 1st. after vaginal del. after C.S. (VBAC)

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



A G₂P₁₊₀ female - Previous Normal del.



This time mild cpd at the level of inlet



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

A G₂P₁₊₀ female - Previous LSCS because of fetal distress.



This time previous pelvis Normal
No fetal distress.



A G₂P₁₊₀ : Previous LSCS because of fetal distress.



This time pelvis mild cpd at the level of inlet
No fetal distress.



C.S.

Best method for cpd assessment : Trial of labour

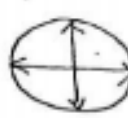
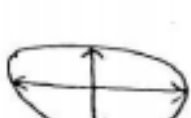
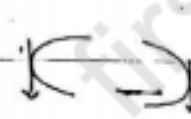
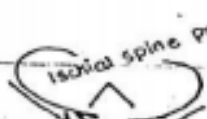
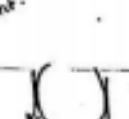
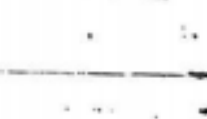
Best investigation for cpd " : MRI > CT > X-ray pelvimetry

51.

Types of Normal pelvis.

Caldwell Mahoy Classification

- Gynaecoid pelvis - 50%.
- Anthropoid pelvis - 25%.
- Android " - 20%.
- Platy pelloid " - 5%.

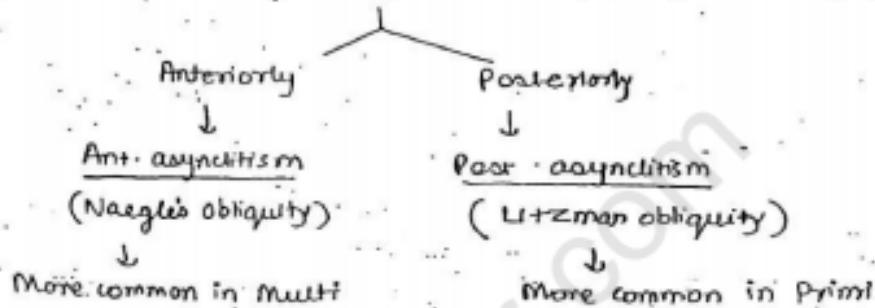
	<u>Gynaecoid</u>	<u>Android</u>	<u>Anthropoid</u>	<u>Platy pelloid</u>
Type :	Female	male		Flat bowl like
Inlet				
Diameter	T > AP.	T > AP	AP > T	T > AP
Shape	Transverse oval.	Heart shaped	A.p. oval	Bowl like
				
Pelvic side wall				
	straight	convergent	Narrow	Divergent.
Subpubic angle	obtuse	Acute		

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.



* 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.

Steps of Normal labour:

1. Engagement
2. Descent & Flexion.
3. At the level of ischial spine fetal head rotates - Int. rotⁿ ($\frac{3}{4}$ th)
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 $\frac{1}{8}$ th of the circle & lie directly behind pubic symphysis. This is visible externally in the form of ext. rotation of fetal head, such that face of the baby lies towards mother's right thigh & occiput faces in the same directⁿ, 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.



2nd stage of labour : 80-100 mmHg

Adequate uterine contractions mean - 3 contractions in 10 minutes, each lasting for 45 sec., & intrauterine pr. of 200-250 65-75 mm Hg.

* Montevideo unit

No. of contractions in 10 min $\times$ 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 w/c

Hyper-stimula

↳ seen in augmented labour (Doc-oxytocin)

oxytocin

ARM.

Abnormal uterine action:

1. Hypotonic contractions

< 3 contractions in 10 min. lasting < 45 sec.

$\&$ MV < 180 / Intrauterine pr. < 60 .

2. Hypertonic contraction

Precipitate labour

obstructed labour/Bandl's ring.



54.

Pre-labour : characterised by :

- 1) Lightening
- 2) Ripening of cervix = softening of Cx
- 3) False labour pains

False labour painTrue labour pain

opposite



- They ↑ in intensity, frequency & duration
- Regular & rhythmic
- Radiates to back & thigh
- Leads to dilatⁿ of Cx
- Show is present
- Bag of memb. present
- Never relieved by sedation & enema

Stages of labour :

1st stage	2nd stage	3rd stage
From onset of True lab. pains until Cx is fully dilated (10cm)	From full dilat ⁿ Cx to expulsion of fetus	Delivery of placenta
Latent phase (≤ 5cm dilat ⁿ of Cx) Active ph. (6-10 cm) ↓ 12 hours (Nulli) / 8 hrs (Multi) ↓ Prolonged : > 20 hrs (Nulli) > 14 hrs (Multi)	1 hour / 30 min (Nulli) / (Multi) ↓ Prolonged : 2 hrs (Nulli) / 1 hr (Multi)	15-25 min (Passive management) 5-15 min (Active M) ↓ > 30 min (Retained placenta)

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

• Latent phase.

- True lab. pain → Cx dil (5cm).
- 2 actions $\left\{ \begin{array}{l} \text{Effacement } C_x \text{ (shortening of } C_x) \\ \text{Dilatation } C_x \end{array} \right.$

D/d of prolonged latent phase : False labour.
Lab. Pain relieved on Sedation/Rest. Lab. Pain not relieved on sedation/Rest.

• Active phase.

Cx $\geq$ 6cm dilated.

- 2 actions $\left\{ \begin{array}{l} \text{Dil. of cervix} \\ \text{Descent of fetal head.} \end{array} \right.$

Dil Cx

↓
1.2 cm/hr → Nulli
1.5 cm/hr → Multi
(1cm/hr - WHO)

Descent

↓
1 cm/hr → Nulli
2 cm/hr → Multi

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

CPD

↓
C.S.

O.p. position

↓
wait & watch.

Rest condⁿ

↓
Augment labour
(oxytocin + ARM)



Active phase arrest.

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

Management : CS.

Second stage.

↳ said to be prolonged.

If Nulliparous Epidural

> 2 hrs (in Nulli)

> 1 hour (in multi)

If Multi Epidural

> 3 hrs (Nulli)

> 2 hrs (multi)

Management :

If Head station $\geq +2$: Forceps

" " $< +2$: CS.

Arrest of 2nd stage

Nulliparous epidural

> 3 hrs (Nulli)

> 2 hrs (multi)

Multi epidural

> 4 hrs (Nulli)

> 3 hrs (Multi)

Second stage arrest $\rightarrow$ obstructed lab. $\rightarrow$ C.S.



Obstructed Labour

- Mother : Exhausted, Tachypnoic, dehydrated, acidotic breathing.
- P/A : Lower uterine seg : stretched
upper uterine seg : contracted tonically.
Ring @ the junction of upper & lower seg : Bandl's ring.
FHR distress / IUD
- P/V : Dry hot vagina. (Because entire liquor has drained out.
Fresh bleeding. (Already dt. Rupture of memb.)
Caput/moulding Present.
Hematuria

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

Management :

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

Bandl's Ring

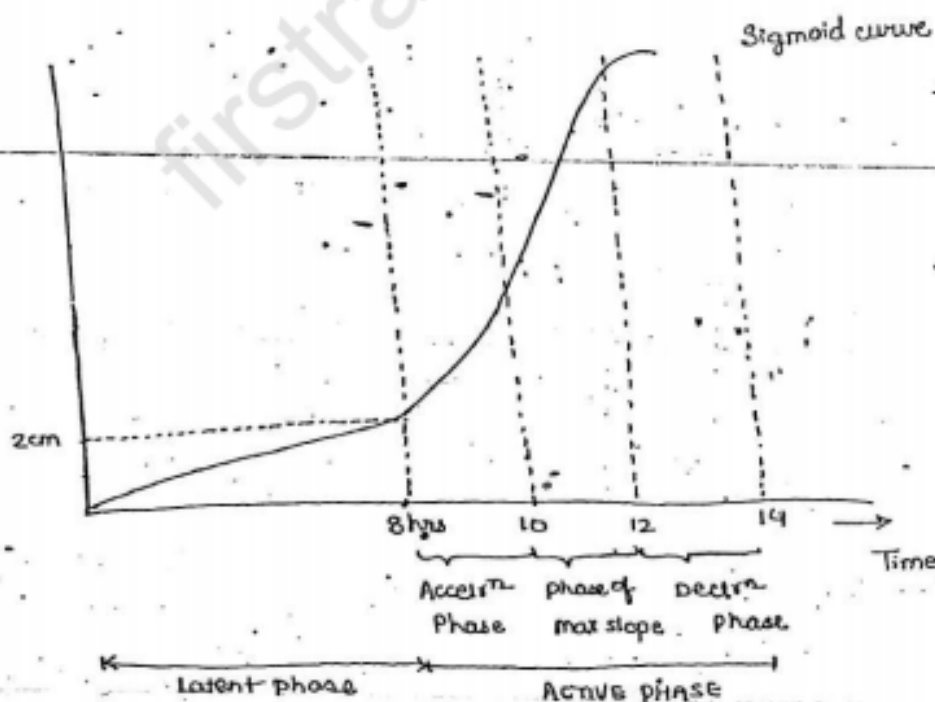
- ↳ In obstructed labour
- ↳ Pathological ring
- ↳ can be felt P/A
- ↳ Retraction ring
- ↳ Leads to Rupture uterus

Schroader's ring

- ↳ In Indiscriminate use of oxytocin / hyperstimulation of uterus.
- ↳ Physiological ring
- ↳ can be felt P/V.
- ↳ Constriction Ring
- ↳ Doesn't

Friedman curve

• 1st partograph



2nd paragraph : - philipor & cattle (1972)

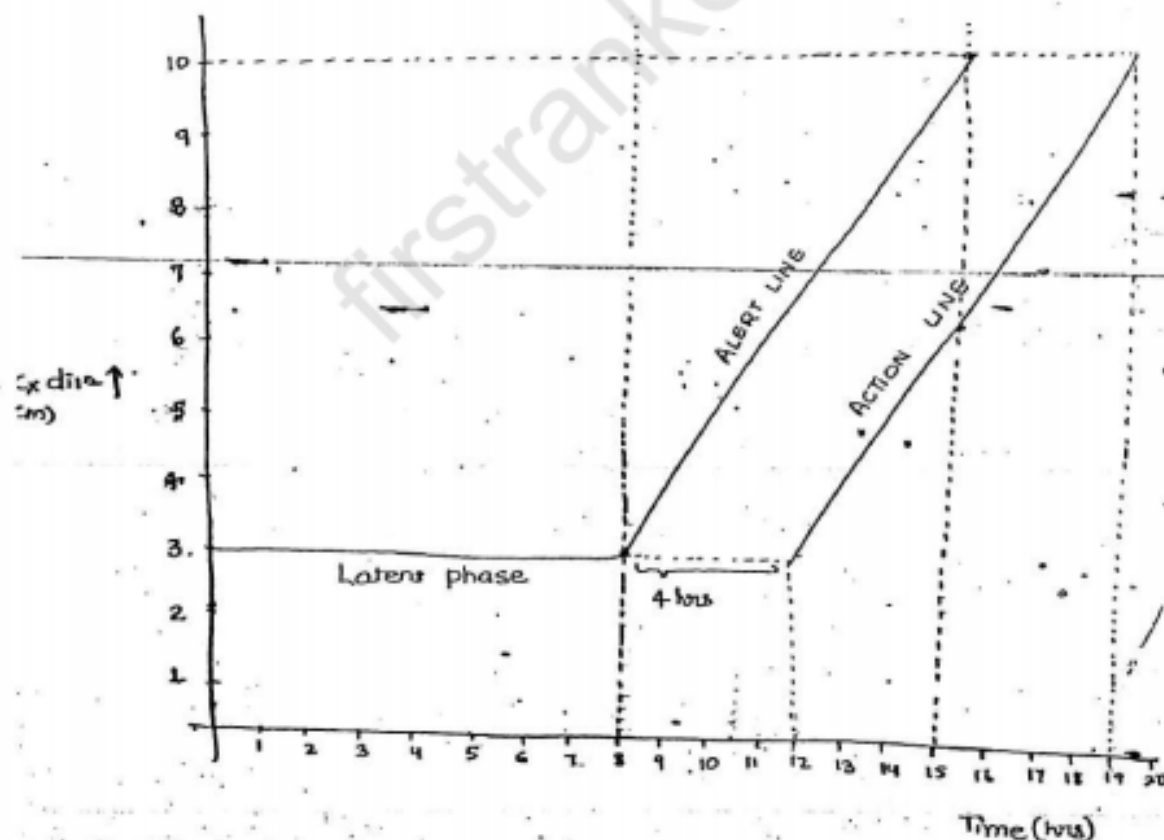
* concept of alert line & action line

* labour is said to be Normal till the time it lies to left of alert line

3rd paragraph : Composite paragraph (by WHO)

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

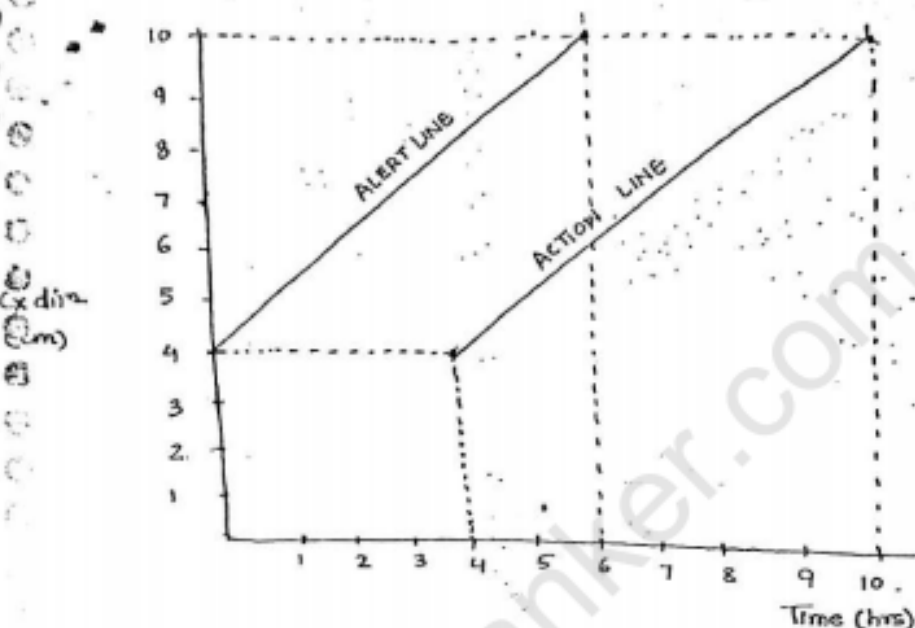
- (i) Latent phase is said to be prolonged, if it is more than 8 hrs.
- (ii) Active phase (≥ 3 cm) - Minm rate of $\dot{C}_x$ dir = 1cm/hr.
- (iii) Time gap b/w alert line & action line = 4 hours.





modified wigg postograph:

1. Latent phase removed
2. Active phase begins from 4-cm, & min^{ra} d_{ll} = 1 cm/hr.





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 - min.

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 grazer breech.
(Indicⁿ for C.S.)

Cord prolapse.

TL > Footling > knee

Management.

Fetus dead

↓
vag. del.

Fetus alive

Cx full dil

Not dilated

↓
Station of fetal head

↓
C.S.

≥ +2

< +2

↓
Forceps

↓
C.S.

Primipr., 37wks, Antenatal chk up → present.

Management of Breech.

Antenatal (≥ 36 wks) or

In labour.

Latent phase

↓
ECV

Falls in primi

Falls in multi

↓
C.S.

↓
Assisted breech.

C.S.

vag. del.

Assisted Breech

Breech extract



External cephalic Version

An opp procedure, - doesn't require any anaesthesia.

- Pre-requisites:
- Preg ≥ 36 wks.
 - liquor - Adequate.
 - memb. - Not ruptured
 - No G/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. $\therefore$ single shot of

Tocolytic is given :- Terbutaline.

In primi (with breech), if any G/I to ECV - cs.

In Multi (" "), " " - Assisted Breech

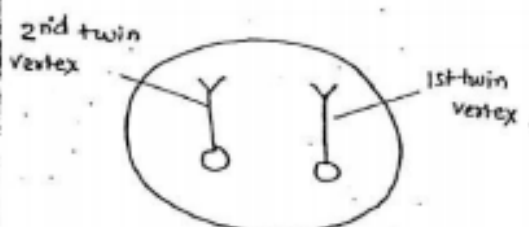
Delivery in twins.

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

If 1st twin is vertex : vaginal del.

" " Breech or : C.S.

Transverse lie



m/c presentation

↓
vag. del. of 1st

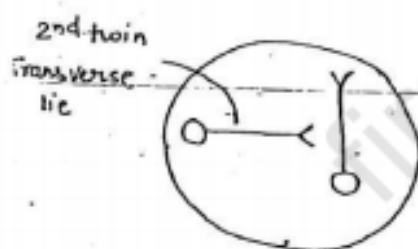
↓
Don't give methergin

↓
vag. del. of 2nd.



vag. del. of 1st

↓
Assisted Breech of 2nd



vag. del. of 1st twin

↓
Take pt. to OT & Give G.A.

Internal podalic version:

Only indicated : If 2nd twin is Transverse lie.

• Done in OT under G.A.

There is a risk of uterine rupture.

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

Breech : INT. PODALIC VERSION

↓
Complete the delivery ~~cont~~ any mat. effort → Breech extraction.



60.

Assisted Breech Delivery

Denominator — Sacrum

Mc position — LSA

Mostly in Breech position : Sacrum is anterior

If Sacrum is posterior : Then Prague manuvre is done.

Delivery :

1) Delivery of Legs & Buttocks.

Engaging diameter : Bitrochanteric dia. (10cm)

Manuvre used : 1. GROIN TRACTION

2. If legs are extended

↳ PINARD'S MANOEUVRE !
(Popliteal fossa)

2) Delivery of Shoulders.

Engaging diameter : Bisacromial dia. (12cm).

Maniuvre used : LOVSET'S MANOEUVRE.

3) Delivery of Head.

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

- Engaging diameter

Mcc of death in Breech : Intracranial hemorrhage.

Stuck head.

Preterm

Term

• Dührssen incision on Cx.

Zavanelli's maneuver

• 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
- Mentopost-face

Forcep del.
(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.

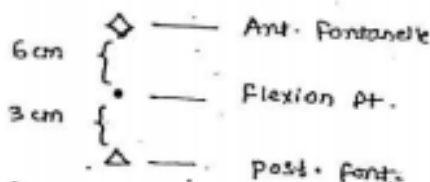
P → Pelvis s/b adequate
No CPD

Vacuum.

can be applied when $Cx \geq 6$ cms
dilated

Vacuum can rotate the head
of baby

Applied along occipito mental
diameter



Vacuum is applied at Flexion pt.

Pressure/Traction

Prim = 20 kg

Mult = 13 kg

Initial pr = 0.2 kg/cm²

Max = 0.8 kg/cm²



Artificially created
Caput: chignon

Indications:

- 1) Mat. distress.
- 2) Fetal "
- 3) Prolonged 2nd stage of lab.
- 4) Prophylactically in H.O. pts.

vacuum.

same indications except:-

- Vacuum isn't used in fetal distress

In H.O. pts. - Vacuum is preferred. (Because we don't have to bring the pt. in lithotomy position in Vacuum)

Advantages:

Presentations in which forceps can be used:

- 1) All presentations except in those where c.s. 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-term (Disadvantage)

Complications:

- 1) Lead to more of mat. injury

Fetal injuries more common 2 forcep:

- Facial N. palsy
- Brachial plexus injury.

- leads to more of fetal injury

Fetal injuries more common:-

- Cephalhematoma
- Retinal injury
- Sixth N. palsy.



max no. of pulls

↓
3

Not delivered
⇒ failed forcep.

↓
C.S.

max no. of pulls

↓
3

Not delivered
⇒ failed vacuum

↓
C.S.

Absolute c/x for Instrumental del.

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

Cephalocephaloma

→ Scalp edema.

→ Swelling above the present

• Periosteum / bone

↓

It can cross the suture lines.

→ Present as soon as baby is born & starts ↓ after birth

Cephalhematoma

→ Subperiosteal bleeding

→ Swelling below periosteum

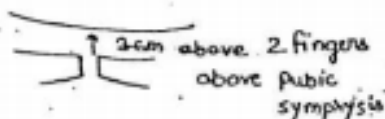
↓

It can't cross.

→ It presents few hours after birth. It ↑ for 3 days & then ↓.

Caesarean section

Skin incisions



3cm above Pubic symphysis

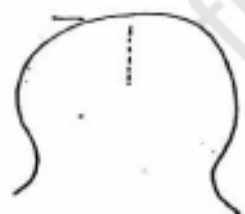
Suprapubic curved incision in which rectus sheath is opened using sharp incision w/a sharp technique.

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

PFANNENSTIEL INCISION

JOHN COHEN INCISION

Uterine incisions



Classical C.S.

Sanger's Incision.
Ut. rupture chances = 4-9%.



T-shaped incision

Rupture = 4-9%.



Rupture = 0.2-1.5%.



Rupture = 1-7%.

IC. ← Kerr incision (LSCS)

Kronig/de Lee incision (LSCS)



63.

Current indic^s 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.

Min^m " " " = 6 months.Level of anaesthesia in C.S. = T₄ 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)



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. perineal 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 if a score of 9 or more.

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



Agents used for Cx ripening & Induction of labour.

PGE_2 (dinoprost)

PGE_1 (misoprost) $\rightarrow$ ~~no~~ C/I in previous C.S. for induction of labour (oxytocin is used instead).

Augmentation of labour should be avoided in prev. C.S.

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

Rupture uterus

$\hookrightarrow$ Rupture of entire uterus along τ serosa/peritoneum.

If peritoneum is intact $\rightarrow$ K/a Scar dehiscence.

Sign of Impending Rupture.

1. Fetal Tachycardia
2. Mat. Tachy "
3. Scar tenderness.
4. Microscopic hematuria



Immediate C.S.

Sign of Rupture.

1. FETAL BRADYCARDIA.
(Mc sign/Earliest sign/
Most consistent sign).
F.H.S. absent as the baby dies.
2. Mother - Tachy, Hypotension
(shock).
3. Hematuria, Bleeding pv
4. Loss of station
5. Absent ut. contracⁿ



Immediate laparotomy



Ectopic pregnancy

↳ Pregnancy outside uterine cavity.

Mc site : Fallopian tube.



Mc - Ampulla (because fertilisation occurs in ampulla).

Lc - Interstitial (IT).

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

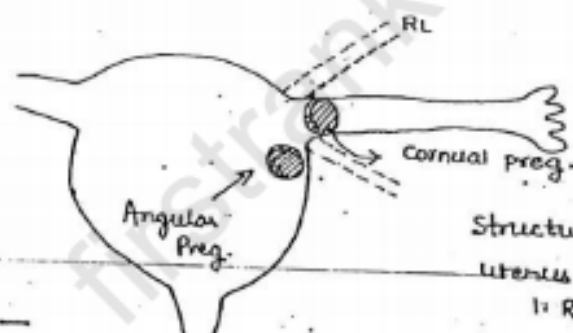
Heterotopic ectopic

↳ Twin - 1 ectopic + 1 intrauterine

Ectopic occurring Non functional horn of bicornuate uterine.

Cornual preg : Ectopic preg in interstitium of IT.

↳ 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 lg. (RL) :- cornual preg
 " medial " :- Angular "

Highest risk for ectopic.

1) Prev. H/o Ectopic Preg. $\left\{ \begin{array}{l} 1. \text{ Prev. Ectopic} = 15\% \\ 2. \text{ " " " " } = 30\% \end{array} \right.$

2) H/o Tubal surgery.

* Mc risk factor : PID

Other - Multiple sex partners.

Early age of intercourse.

In utero exp to DES (Diethylstilbestrol).

Infertility.

ART

Smoking.

Tubal adhesions (Endometriosis).

Contraception

All contraceptive agents reduce the ^{absolute} risk of Ectopic.

But if contraception fails $\Rightarrow$ Ectopic.

M/c : sterilization > IUCDs > POP.

(Mc: Progestasent).

Least common : Ocps.

Fate of ectopic pregnancy.

Abort
(= Tubal abortion)

Mc : @ Ampulla

Continues to grow

Tubal Rupture

Mc : @ Isthmus

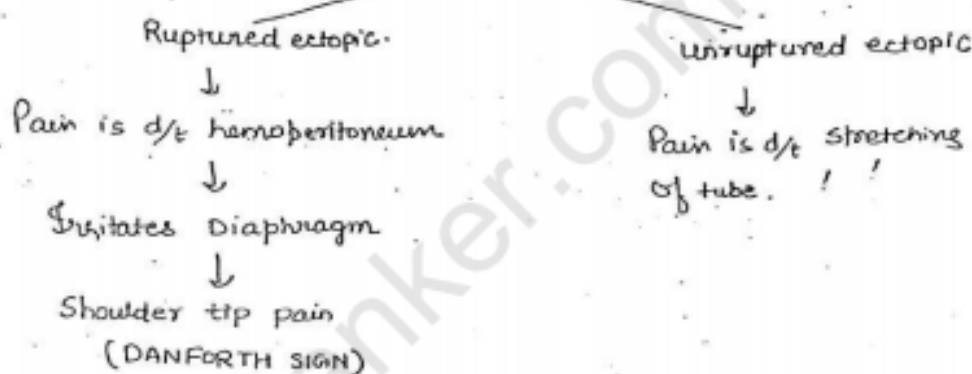
* Ectopic Preg. has shortest life span : @ Isthmus.

66.

- Ectopic preg. Lasts longest in :-
- 1) Abdominal preg.
 - 2) Heterotopic "
 - 3) Tube :- Interstitium.
(Because myometrium supports ectopic. $\rightarrow$ upto 12 wks.
 $\therefore$ most dangerous site)

Symptoms of Ectopic preg :

- Most consistent symp. : Pain abdomen.



- Triad of :-
- Abd. pain
 - Amenorrhoea (6-8 wks)
 - Bleeding pv.

Signs of Ectopic. preg :

- on P/V :-
- uterus is enlarged
 - cervical movement is tender; characteristic

Ectopic $\swarrow$ $\searrow$ PID.

- Adnexal mass can be palpated (in 50% cases).



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/mult)

* 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.

Toc = 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

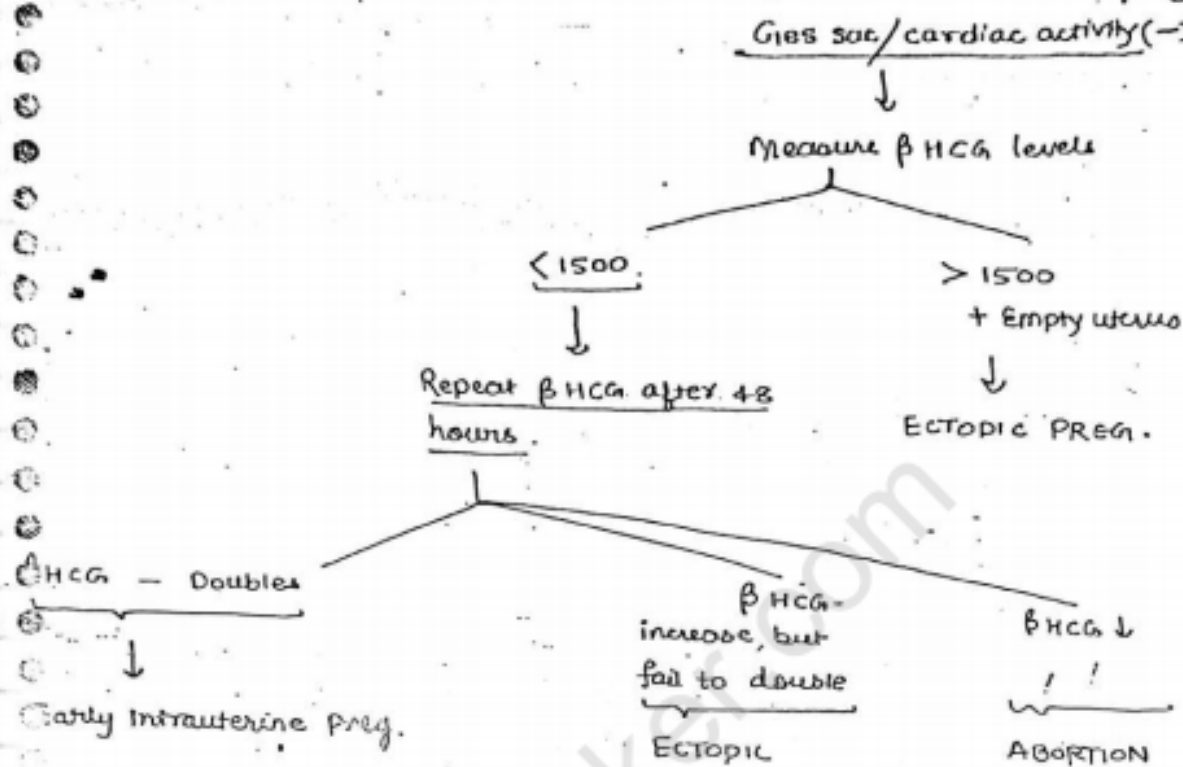
Ectopic

↑ Vascularity in Tube (RING OR FIRE APPEARANCE)

Ges sac / cardiac activity -



67



Best Investigation : TVS + β HCG.

Gold std : Laparoscopy

Other investigations : 1) S. progesterone

2) culdocentesis $\rightarrow$ Blood in case of ruptured ectopic

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



Management of unruptured ectopic.

• Expectant management : wait & watch.

Criteria → 1) Pt. hemodynamically stable
(ie. Ectopic unruptured)

2) Cardiac activity absent in Ectopic preg.

3) Size of ectopic < 3cm.

4) β HCG < 200 & should be decreasing.

* Risk of rupture in expectant management.

• Medical management :

Doc. : METHOTREXATE (single dose)

Criteria - 1) Pt. hemodynamically stable

2) Cardiac activity Preferably absent.

3) Size of ectopic < 3.5cm (if cardiac activity +)

< 4cm (if cardiac activity absent)

4) β HCG < 5000.

Doc : Methotrexate : 50 mg/m^2 I.M.

Day 1 = Injection

Day 4 = check 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.

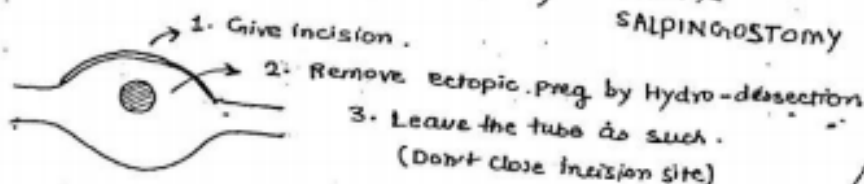
Failed Med. management : - 3 times Injection given → still the required fall in β HCG is not seen.

Surgical management : Laparoscopy (Preferred)
Laparotomy

Surgery depends on parity of female.

♀ is Multiparous + completed family = salpingectomy

♀ is Nulliparous + Not completed family = LINEAR SALPINGOSTOMY



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



Ectopic pregnancy occurs in young females.

So Salpingo-oophorectomy 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 $> 4\text{ cm}$.
- 4) If Hemostasis can't be obtained.

Site of Ectopic

1. Cervical ectopic
2. Abdominal ectopic
3. Ovarian ectopic

Diagnostic criteria!

- Rubin / Palman criteria.
- Studdiford criteria.
- Spigelberg criteria.



Abortion

Loss of fetus.

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

Abortion

Spontaneous

(single episode)

Recurrent

(≥ 3 abortion)

1st Trimester

Causes: chromosomal anomaly
(50% cases)

2nd trimester

→ same

(20% cases)

1st trim

2nd trim

Spontaneous

Time : < 8 wks.

Chromosomal anomaly →

Germplasm defect

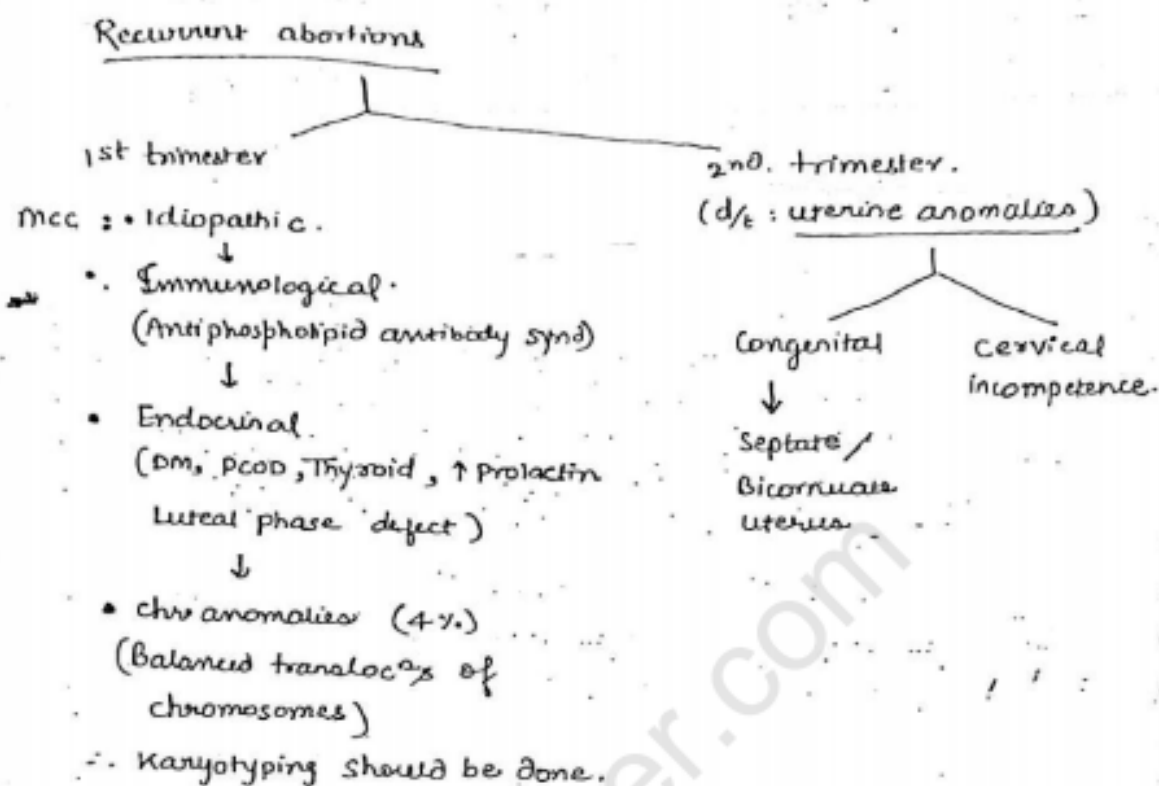
Infections

Aneuploidy ✓ (Best)

Trisomy

Monosomy X = 20% of 1st trim. abortion

Trisomy 16 = 16% of abortions



- TORCH infects never leads to Recurrent Pregnancy loss (RPL).
- only infect that leads to RPL - syphilis.

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

Cervical Incompetence:

- ↳ Sudden painless dilatation of Cx & expulsion of fetus.
- ↳ Mcc of RPL in 2nd trimester.
- ↳ Gestational age decreases & every preg. loss (Opp to syphilis)

Diagnosis:

Non-Pregnant:

- Pass Hegar dilator no. 8
- Feel any resistance (through int. os).

On Hystercervicography:

Funnel shaped appearance.

Pregnant:

• Tvs (Ioc)

- Length of Cx < 2.5 cm
- Diameter of os > 8 mm
- Shape of Cx = U shape.
- Funneling of membranes.

$$\text{Cervical Index} = \frac{\text{Funnel length}}{\text{Endocervical length}} + 1$$

Normal = 0.32

In cervical incompetence > 0.52

Management: Cervical cerclage (Put the stitch)

Prophylactic (History indicated)

- ↳ 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 dilatation < 4 cm & membrane should not be ruptured.

Contra for circlage -

- acute infection
- Membr. ruptured
- Gross congenital anomaly
- $Cx > 4cm$ diam.

Methods of doing circlage -

i) 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 β_2 microglobulin antibody

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

PIH $\rightarrow$ size of placenta decreases

Blood flow absent

Abortion IUP Still birth.

Blood flow to fetus $\downarrow$

It can lead to IUGR.



Diagnostic criteria : Revised sappro.

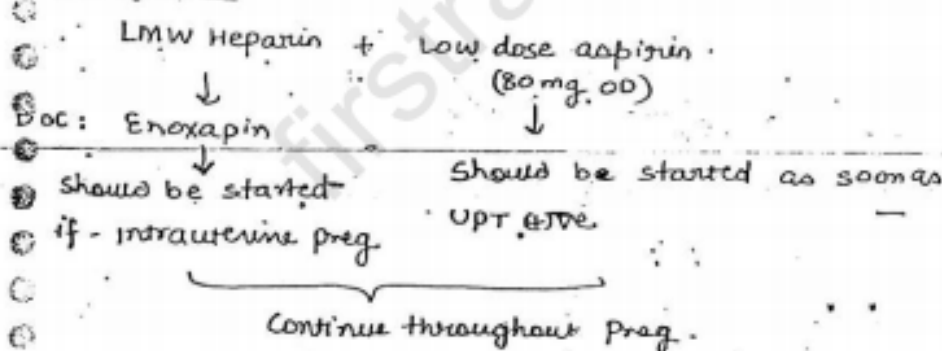
1 clinical & 1 lab criteria should be fulfilled.

- 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 anticardiolipin antibody
 - β_2 microglobulin Ab.
- ON 2 occasions 12 weeks apart.

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

Management.



Heparin stopped @ onset of lab

Aspirin " @ 5-10 days after labour



Types of abortion

<u>Type of abortion</u>	<u>complain</u>	<u>P/A</u>	<u>Int. os</u>	<u>Diagnosis</u>	<u>Imp. points</u>
• 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.	<u>Mngmnt</u> : 1) Reassurance 2) Avoid intercourse 3) Value of bed rest & Progesterone is uncertain. 4) Anti-D.
• 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 vagine misoprost to complete the process.
• Incomplete abortion Process of abortion has begun & products (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	Conservative mngmnt if
• 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	—



Missed abortion	No bleeding	MT. of ut. closed	Mean sac diameter	↓ β HCG levels.
Fetus is dead	Dirty brown discharge.	< 10cm	> 25 mm	
Outside the uterus			OR	
Sur pt. is unaware			Crown-rump length ≥ 7 mm	
			No fetal heart beat.	

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 $\bar{c}$ pain.

Anti-D is not given in :-

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

Dose = 5 μ g (< 12 wks)
300 μ g ($\geq$ 12 wks)

Methods of MTP:

Best method up to 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 vacuum aspirⁿ

2nd trim

- ↳ PGs
- ↳ Suction Evac. using Drum forcep.
- ↳ Extra-amniotic Ethacridine
- ↳ Intraminiotic
- ↳ Hypt

Med. abortion

< 7 week Pregnancy

7-9 wks. Pregnancy

- ↳ Day 1 = 200 mg Mifepristone
- ↳ Day 3 = 400 µg Misoprostol oral or vaginal
- ↳ Day 15 = clinical examⁿ to

- ↳ Day 1 = same
- ↳ Day 3 = 800 Misoprostol (vaginal)
- ↳ Day 15 = same

— ensure that abortion is complete.

* If < 1% = curettage is required

* Misoprostol 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



If perforation occurs during Procedure.

while dilating



wait & watch

while doing suction & Evac



Don't remove cannula from ut.
& Immediate laparotomy.

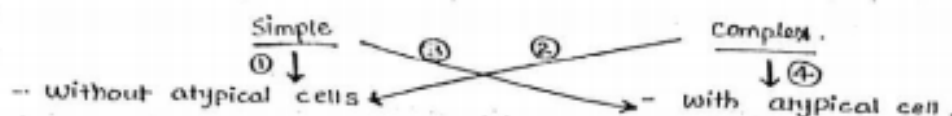
Menstrual regulation

Done $\sim$ 3 weeks after a missed period. i.e. 7 weeks of amenorrhea.

Manual vacuum aspiration

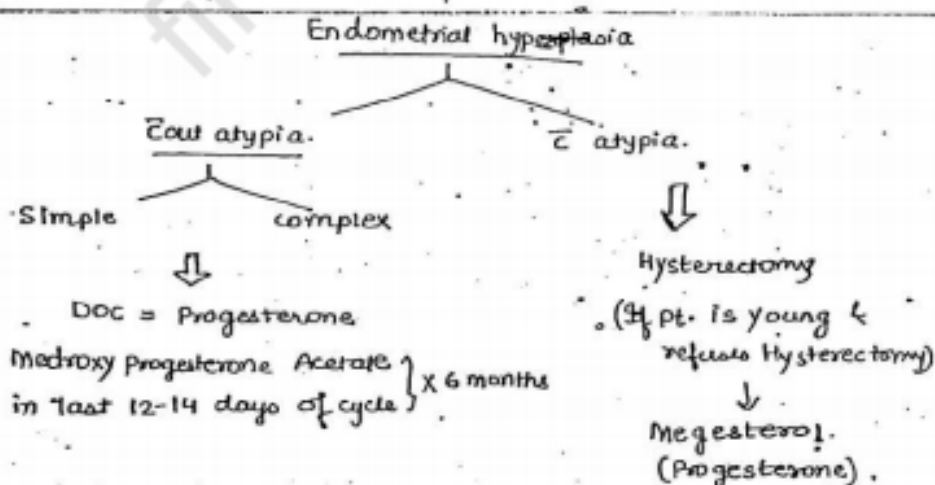
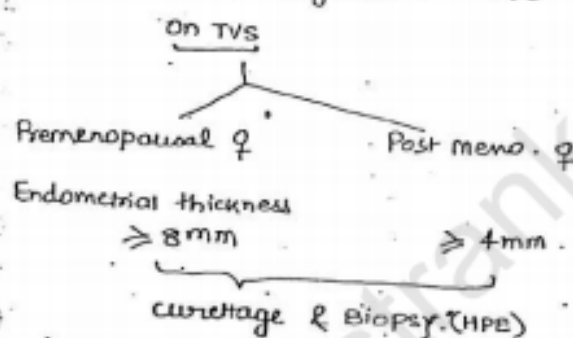
- Take a 60 ml syringe & a cannula.
- Pressure : 660 mm Hg.
- used upto 12 wks of preg.
- No electricity required.

Endometrial Hyperplasia



- ① - ma least chance of malignancy = 1% | $\approx$ cystic glandular Hyperplasia
- ② - 3%
- ③ - 8%
- ④ - 30%

- When a pt. comes w excess bleeding,
1st investigation: TVS



Endometrial ca

Type 1

- Adeno ca: Grade 1, 2

- Mainly due to excessive estrogen
- Endometrial hyperplasia Present
- More common in obese ♀
- Better prognosis
- In pre & post menopausal ♀
- White ♀
- w/ PTEN & KRAS
- Stable

Type 2

Papillary serous tumor
clean cell tumor
Adeno ca: Grade 3.

- Absent
- In Thin ♀
- only in post menopausal ♀
- Black ♀
- P53
- 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!

Post menopausal Bleeding

1st investigation :

TVS

Endometrial thickness

$< 4\text{mm}$

$\geq 4\text{mm}$

↓
Repeat TVS after 3 months

Loc:

- Endometrial aspirate biopsy using Pipelle, Vibra-aspirator & Karmann curette.

Gold std. investigation

- Fractional curettage.

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

Lymphadenectomy : Indications (in endometrial ca).

- Tumor size $> 2\text{cm}$

- Tumor Histology - Endometrioid grade 3 / papillary serous / clear cell

- More than $\frac{1}{2}$ of myometrium involved.

- Tumor extends to cervix / extra-uterine

If all cond^s are present : Pelvic & Para-aortic Lymphadenectomy

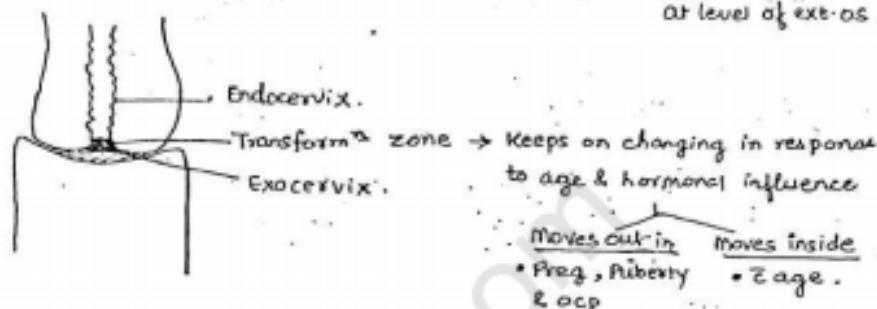
If size of tumor $> 2\text{cm}$, & no other cond^s : Pelvic Lymph.

If none of these cond^s present (eg- Adeno ca grade 1 & 2 belonging to Stage IA) $\rightarrow$ No lymphadenectomy.

Anatomy of cervix

2 parts :

- 1) Endocervix : Closer to uterus — columnar epi
 - 2) Ecto " : Closer to vagina — Squamous epi
- ← Squamo-columnar junⁿ (Transformⁿ zone) →
 ↓
 at level of ext. os.



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 endocervix changes into squamous epithelium of exocervix → k/a metaplasia

→ physiological ; Not precancerous.

Squamous epithelium of vagina & exocervix has 3 types of cells :

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

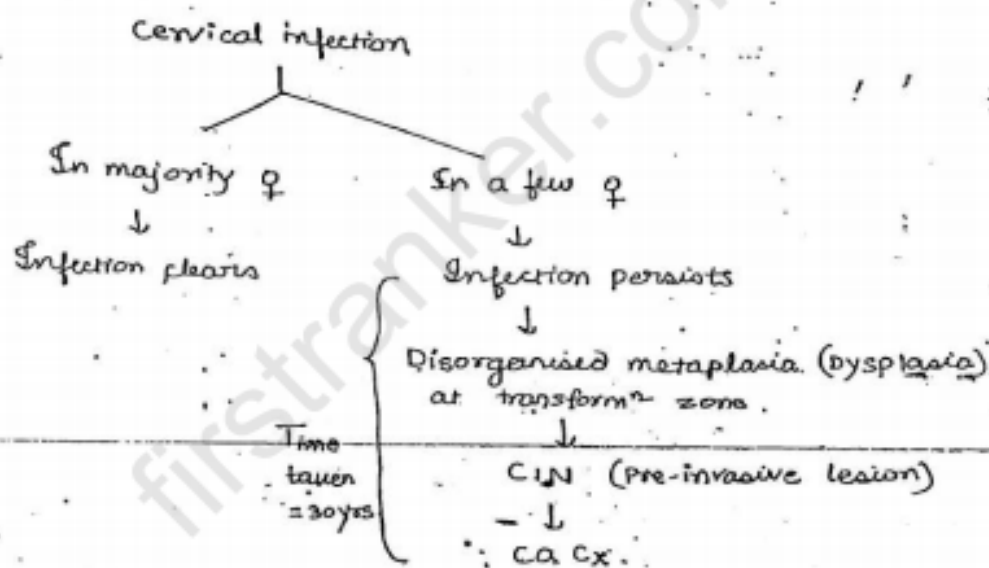
- * For Hormonal study, sample should be taken from lat. side of Vagina.

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 :

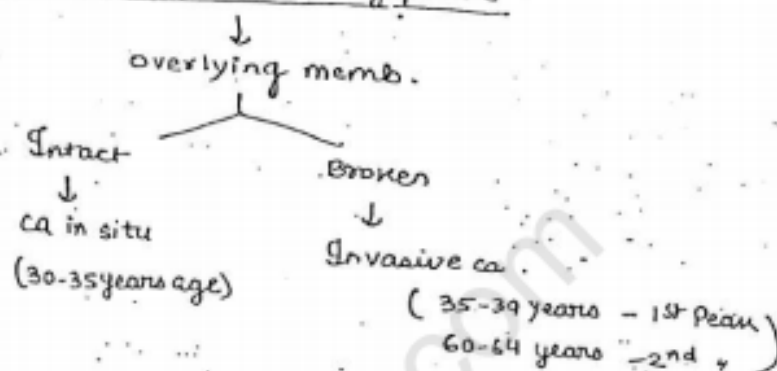
- ↳ Always pathological ; pre cancerous ; only in few females.
- ↳ Dysplastic cells - Big cells & Big nucleus & less cytoplasm.
 ∴ ↑ Nucleo-cytoplasmic ratio.
 - Mitotic figures.



Q. CIN = cervical intra-epithelial Neoplasia.

$< \frac{1}{3}$ cervical epithelial dysplastic = CIN 1
 $\frac{1}{3} - \frac{2}{3}$ " " = CIN 2
 $> \frac{2}{3}$ " " = CIN 3 } (21-30 years age group)

Complete ex epithelia dysplastic



Bethesda classification

- 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 ex.

- 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.

HPV (Booklet)

- When HPV affects cervical epithelium, the changes which are seen are k/a - Koilocytic 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 w/ Genital warts have laryngeal Papillomatosis. Hence active genital wart Pt. in labour → C.S.
- Viral protein responsible for malignant transformation.
 - E₆ - affects p 53 gene
 - E₇ - " Retinoblastoma gene.
- Viral protein required for replication : E₁ & E₂.

Screening test Cx : Pap smear

- ↳ 1st performed by - Georgios Papanikolaou
- ↳ Starts @ 21 years of age.
- ↳ B/w 21-29 years : screening should be done yearly using only Pap smear
- ↳ 30-65 years : 5 yearly screening using pap smear (increases specificity)
OR
+ HPV DNA (increases sensitivity)
3 yearly pap.
- ↳ If HIV (+), or exposed to DES : Annual screening - till we get 3 consecutive Normal. & then 3 yearly screening.

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

Pap - Normal

OR

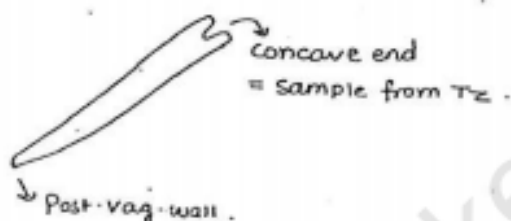
75 years - if last 10 years Pap - Suspicious.

- After vaccine - screening still continues.

- After Hysterectomy for benign cause - screening stops.

Instrument used for pap smear

① Ayer wooden spatula



② Wooden Broc Cytobrush.



Tz

1st slide

Endo Cx

2nd slide

Post-Vag-wall

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

* $\frac{1}{2}$ for pap : None

Relative c/z : Bleeding.

During preg : Pap is done in 1st ante-natal visit.

- Alternative to pap in rural areas :

1. VIA - visual inspection $\bar{c}$ 5% acetic acid - white/stained areas : Abnorm
2. VILI - " " Lugol's iodine - unstained yellow areas : Abnorm
(stained areas - Black-Brown).

* Lugol's iodine = 5% Iodine

* Schiller's " = 0.3% "

• Infection detected by pap :

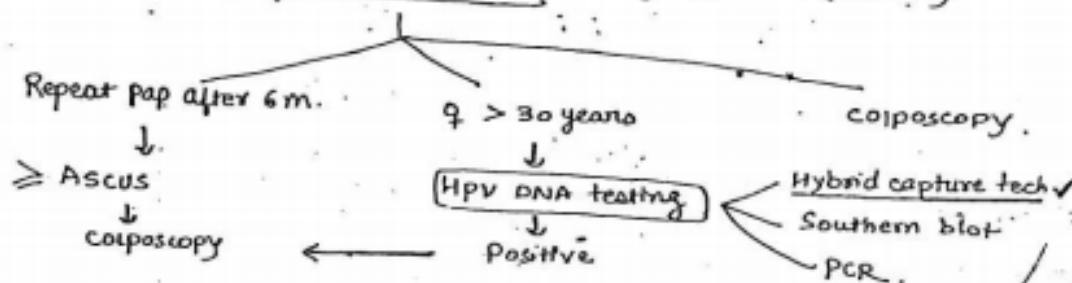
- Trichomonas
- Candida
- HSV
- Actinomyces
- HPV

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)



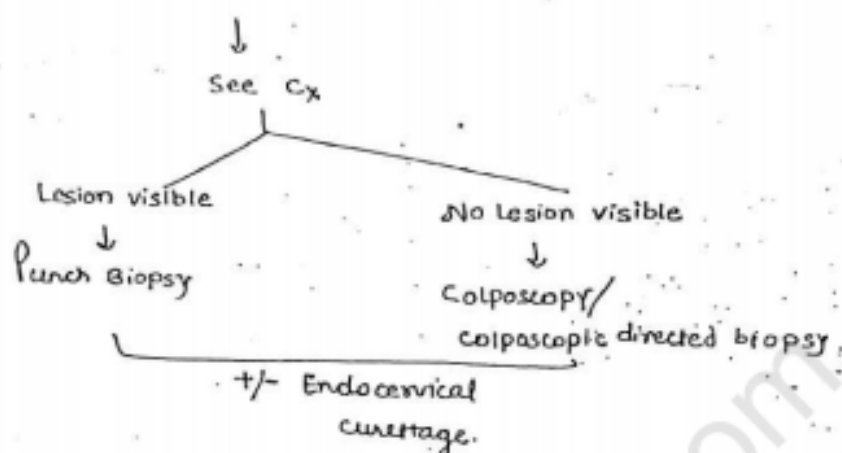
5. LSIL

6. HSIL



Management of LSIL

LSIL on pap smear.



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

Colposcope

Magnification : 5X to 30X.

Focal length : 30 cm

Can't visualize 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)

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 indic^s of cone biopsy are:-

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

Therapeutic indic^s:-

- 1) To $\frac{1}{2}$ ca in situ or stage I A I of ca cx in young female



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

Carcinoma Cervix

A pt. of Cx ca has 5cm growth which extends to lower $\frac{1}{3}$ rd of vagina, hydrouter is present, on cytology 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 + 1cm of vagina + pelvic LN are removed.
- Option for women $\geq$ stage 1A1, 1A2 & 1B1 if tumor < 2 cm

Stage wise management:

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

Dysmenorrhea: Pain during menses

Primary

No pelvic pathology
for pain

Seen in young girls
at the onset of
menarche

Rx:

① NSAIDs.

② Make the cycle
anovulatory - ocp's.

Secondary

- Pelvic pathology responsible for pain
Endometriosis, Fibroid

- Seen in Reproductive age ♀ (>30 yrs).

Rx

① Treat the underlying cause.

Amenorrhea = Absence of menstruation

Primary amenorrhea

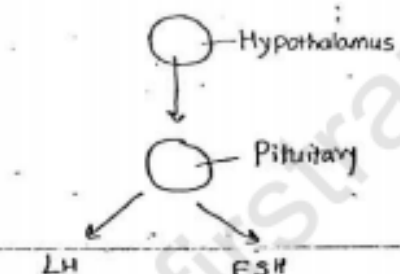
- Never experienced menstruation or Menarche.



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

sec. Amenorrhea

- Earlier menstruating normally,
but now absent menstruation



Kallman syndrome (46 XX)

Craniopharyngioma

Turner syndrome (45 XO)

Pure Gonadal dysgenesis (46 XO)

Swyer's syndrome (46 XY)

MKXH syndrome (46 XX)

Testicular feminizing syndrome

Androgen insensitivity " (46 XY)



MC cause of 1^o Amenorrhea = Turner
 2nd " " = MRKH
 3rd " " = Testicular feminising

Best Tx for 1^o Amenorrhea = Karyotyping
 " 2^o " = 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, PT, Cx & upper part of vagina are absent. (In some cases small part of PT 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).

Clinical case 1

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

• Imperforate Hymen. (Cryptomenorrhea)

↳ % : (i) Cyclic abd. pain
+
No bleeding

↳ O/E : Hymen bluish &
bulging.

P/A - mass
(uterus & blood collected
in it → Hematometra).

P/v examⁿ should not be done
in virgin females.

on Per Rectal examination:-

uterus is present & Bulky.

• MRKH Synd. & Androgen Insensitivity syndrome

↓
uterus absent.

↳ Mgmt:- Incision of Hymen:-

All the Blood will come out.

Androgen Insensitivity syndrome (AIS)

(i) Complete androgen
Insensitivity
Syndrome

(ii) Incomplete androgen
Insensitivity
syndrome

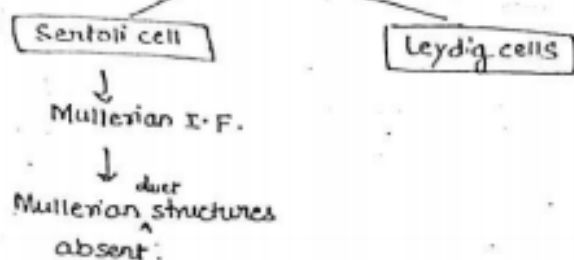
(iii) Reifenstein
Syndrome

↓
TESTICULAR FEMINISING
SYNDROME

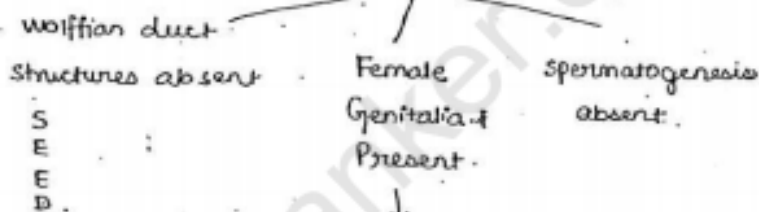
1. Complete AIS (46 XX). : Males have complete Insensitivity towards Testosterone.

X linked

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



Complete Insensitivity towards Testosterone



↓
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.

AIS

46 xy

Gonads = Testes



Barr body = Absent

Not q/w Renal anomalies

MRKH

46 XX

ovary :

Test = N

Present

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. Vaginopectasy - Tech. & Time is same like MRKH.

2. Incomplete AIS

- X-linked recessive
- less severe (little responsive to Testosterone)

Genotype = 46 xy

Gonads = Testes → Intraabdominal → Inguinal Hernia.

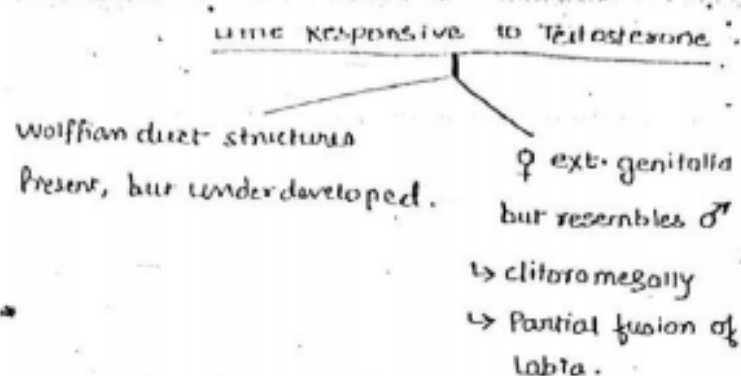
Sertoli cells

↓
MIF

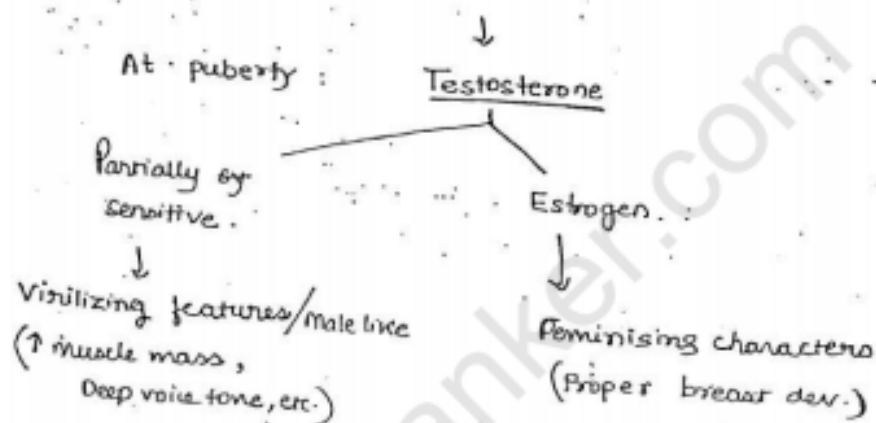
↓
Ut, Cx, Fr, Vag. absent

Leydig cells

↓
Testosterone



These babies are considered as ♀ at the time of birth



♀ ± Proper breast development (2° sex character: $\odot$)
+ Inguinal Hernia + clitoromegaly

3. Reifenstein syndrome.

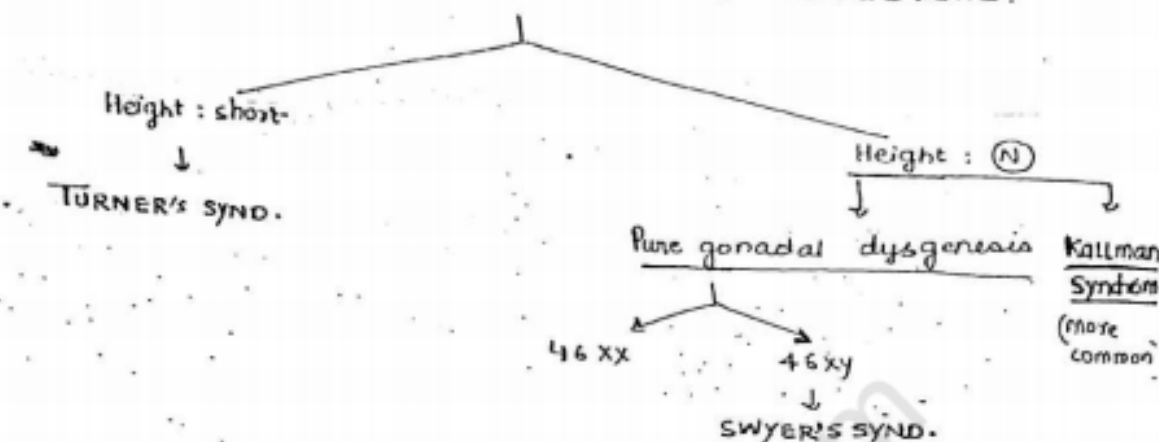
- ↳ They are males who are under virilized
- ↳ They have male genitalia, but bifid scrotum + Hypospadias.
- ↳ Int. genitalia

$\left. \begin{matrix} S \\ E \\ E \\ D \end{matrix} \right\}$ Int but less developed.

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

Clinical case II:

Pt. % 1st amenorrhea & absent sec. sex. characteristic.



- In pure gonadal dysgenesis, the pair of x chromosome responsible for form^a 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

↓
Testes

↓
Dysgenetic

Anti Mullerian
Hormone - absent

- PR
- ut
- cx +
+ upper vag.

Leydig cells
will not form
Testosterone

Wolffian duct
regress.

female genitalia

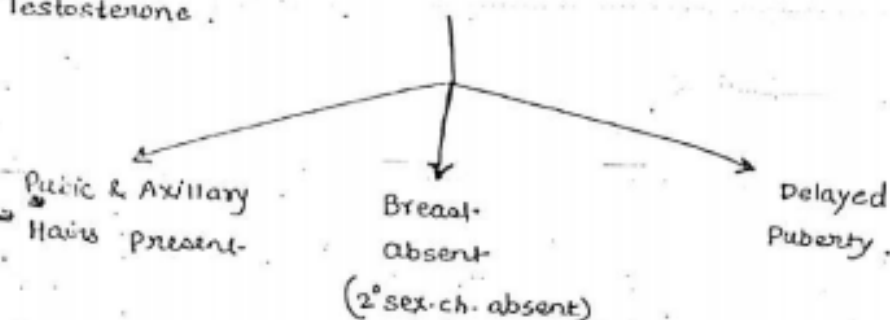
S.
E.
D.
D. } - Absent

85:1 cases = Idiopathic

15:1 cases = d/t inactivation of SRY gene



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 gene (on short arm of X chr.)

↓
codes for Anosmin 1

↓
Responsible for migration of GnRH neurons from Olfactory epithelium to Hypothalamus.

Problem :

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

Mngmnt:

↳ Estrogen + Progesterone / Gonadotropin / Pulsatile GnRH.

Secondary amenorrhea : youtube

Endometriosis

→ Occurrence 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

g/w : 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
w/ a Scar endometriosis (like cs. scar, Myomectomy scar, Episiotomy scar, Pelvic floor repair scar).



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 :

q/f :
mc. complain :- Pain (Sec. dysmenorrhea > chr. Pelvic pain > Dyspareunia)
Severity of Pain & Depth of lesion Low backache)

* 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.

Mgmt.

- If pain is minimal/mild pain : NSAIDs / ocp's.
- If pain is moderate/severe :- Reverse the disease process.
 - 1st drug - Progesterone (Pop/dmpa/minirela/implants).
 - Doc - GnRH (continuous).
 - Other drugs - Aromatase inhibitors (Letrozole)
 - Danazol (Androgenic side effects)
 - Gestrinone.

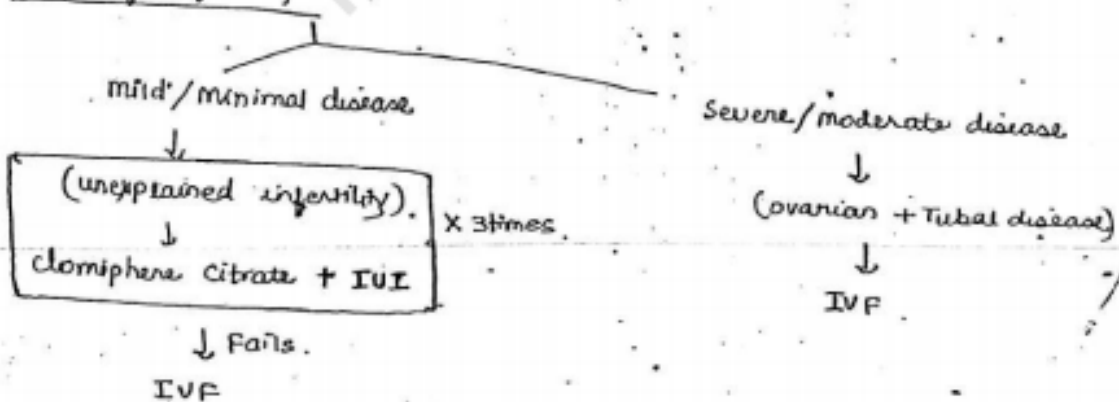
At which sites medical management not used :-

1. Ovary (for $\frac{1}{2}$ of chocolate cyst).
2. Bowel.
3. Urinary system.

Mgmt of Endometrioma.



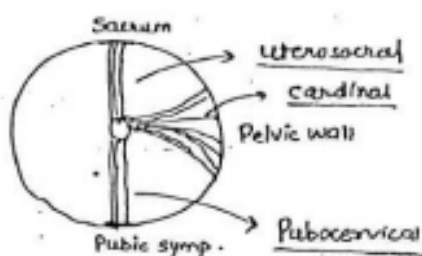
Mgmt of Infertility



Support of uterus :

I. Ligaments :-

1. Pubocervical ligament
2. Cardinal ligament or $\rightarrow$ Main
Transverse cervical lig. or
Mackenrodt's lig.
3. uterosacral lig.

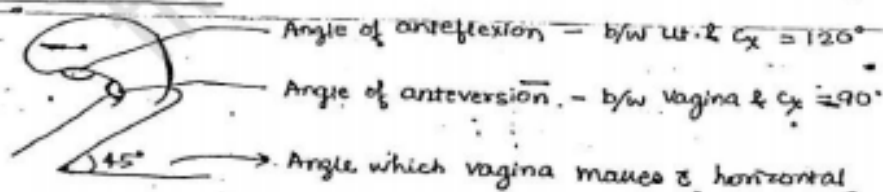


* Broad ligament doesn't support

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

1. Levator ani = Pelvic diaphragm $\rightarrow$ Main.
2. Superficial & deep transverse perineii (urogenital diaphragm)
3. Bulbospongiosus
4. Ext. anal sphincter muscle
5. " urinary "

III. Mechanical support



In anteversion : fundus points towards bladder

In Retro " " sacrum

Ligaments which prevent Retroversion : uterosacral ligaments

" " " Promote anteversion & anteversion : Round Ligament.

$\therefore$ Secondary support.

For managing Retroverted uterus, Pessary used : Hodge Smith Pessary.

De lancyes level of support of uterus.

Level 1 : uterosacral lig & Mackenrodt " Defect leads to uterine prolapse.

Level 2 : Paravaginal muscle levator ani " Defect $\frac{1}{2}$ cystocele.

Level 3 : Urogenital diaphragm & Perineal body. Defect $\frac{1}{2}$ Rectocele & urethrocele.

Vaginal prolapse



Upper $\frac{2}{3}$ rd of Ant. vag. wall prolapse : cystocele.

Lower $\frac{1}{3}$ rd " " : urethrocele

Upper $\frac{1}{3}$ rd of post. vag. wall prolapse : Enterocele

Middle $\frac{1}{3}$ rd " " : Rectocele

Lower $\frac{1}{3}$ rd " " : Laxed perineum

* Repair of cystocele & urethrocele : Ant. colporrhaphy

Repair of enterocele

- 1. Moscovitz repair } Not done
- 2. (Halban repair) } nowadays

* McCall colpoplasty is used now to repair enterocele.



Repair of Rectocele & lax perineum : Post-coeloperineo-rhaphy

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

If prolapse is seen in young nulliparous $\rightarrow$ Congenital prolapse

In congenital prolapse : cystocele is never seen.

$\rightarrow$ Risk factor :
Marfan Syndrome
Ehler-Danlos syndrome
Spina bifida

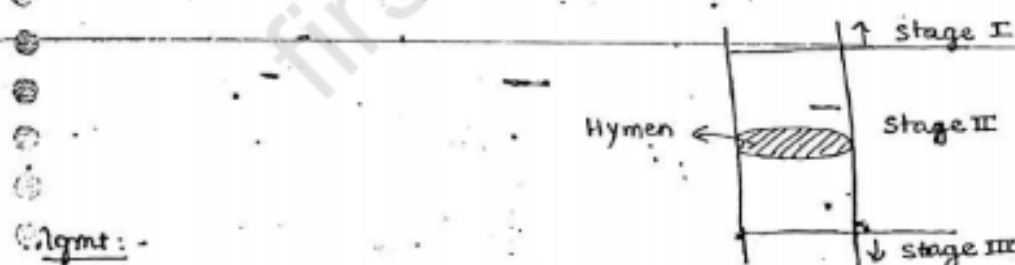
classification of uterine prolapse : Pop - Q classification

Reference point - Hymen.

If prolapsed part lies > 1 cm above hymen : Stage I prolapse.

" " lies ≤ 1 cm of hymen (either side) : Stage II "

" " lies > 1 cm below hymen : Stage III "



mgmt :-

1) Congenital prolapse : Cervicopexy / sling surgery

2) If prolapse occurs in ϕ who desires $\frac{1}{2}$ child. : Cervicopexy / sling surgery

Sling surgery

Material for making sling - Merselene 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 ♀ & 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 mesentery of sigmoid colon, it can lead to:- (i) Intestinal obstruction (ii) Injury to mesentery vessels/ureter/Genitofemoral Nerve in Psoas muscle.

Nowadays :-

Composite sling is done ; w/a virchud sling.

Rt side

↓
Shirodkar's sling

Lt. side

↓
Purandhe sling.

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

Manchester surgery / Fothergill's repair.

Amputation of Cx

The raw area of Cx should be covered by vag. epithelium
→ w/a : Steuindoff suture?

cut the cardinal lig. from side & tie them in front of uterus.

Complic^s :- Incompetent os → 1st : 2nd trimester abortion
Cx fibrosis → stenosis → ^{over} dystocia

Shirodkar's modified 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/peripartum is managed by Ring Pessary.

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 lead to VVF.

Pessary is kept @ level of bchial spine

* Relative c/x of Pessary: Decubitus ulcer.

↳ caused by venous congestion

↳ Rx = uterine packing

acriflavine & glycerine

↓
antiseptic

↓
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. Uterosacral suspension (abdominally or Transvaginally).

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

Urinary Incontinence:

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

Mgmt :- Earlier concept

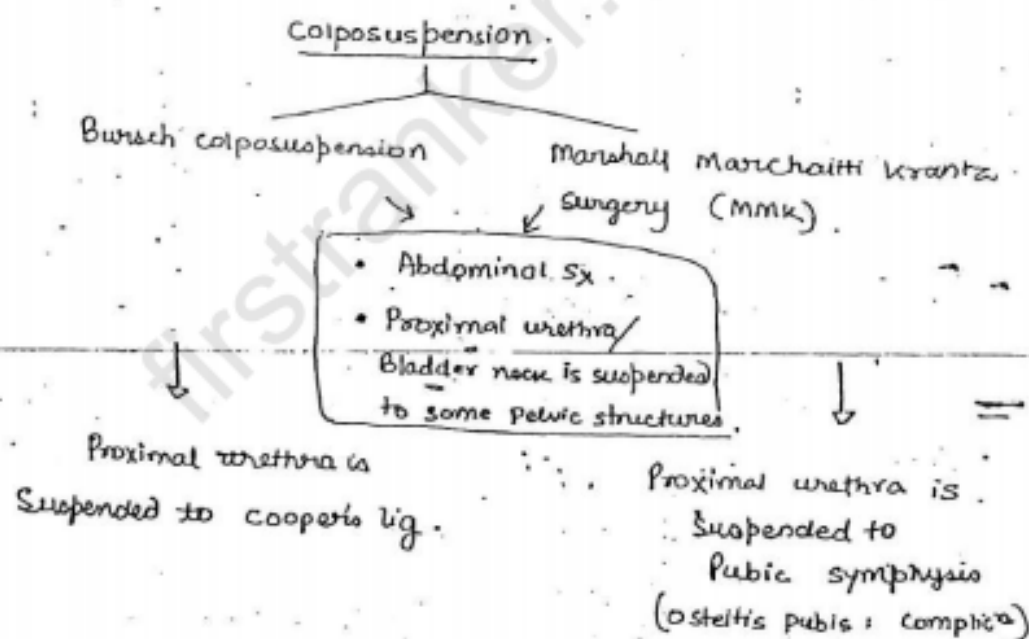
Bladder neck descent : Colposuspension

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:



Gold std. surgery till date was : Burch colposuspension.

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

Both are vaginal Sx. Middle urethra is suspended

TOT is better than TVT because

In TOT, retropubic space is not entered.

∴ less bleeding & less complications



Ovarian causes of infertility :

Tests for anovulation :

1. Indirect :

(i) Basal body temp. = $\uparrow$ by 0.5° in ovulation

(ii) cervical mucus study

Prog: - Ferning absent (Day 21) in ovulation

* Inslens score

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

In Modified Inslens score: - cellularity of mucus is also seen.

• If Ender's score > 12 : Good for sperms

(iii) Vaginal cytology

Estrogen $\rightarrow$ superficial cells

Progesterone $\rightarrow$ Intermediate "

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

Maturation Index represents - Hormonal index

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

(iv) Hormone estimation

If on Day 21, Prog. $> 2\text{gm } 3\text{ng/ml}$ $\Rightarrow$ ovulation has occurred

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 ovulation, size = 19-25 mm

- After ovulation :- Size suddenly $\downarrow$

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

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.

⑤ Clomiphene citrate challenge test - D₃

on D₃ check the level of FSH.

- Give 50 clomiphene : D₅ to D₉

D₁₀ : check FSH.

• A further drop of FSH, on D₁₀ : poor ovarian reserve.

Management :

IVF using Donor oocytes.

⑥ Tubal Infertility

Male infertility1) semen analysis

- Vol $> 1.5\text{ml}$
- PH = 7.2
- Total sperm count > 39 million per ejaculate
- Viscosity < 3
- Sperm concⁿ > 15 million/ml.
 - $< 15 \rightarrow$ oligospermia
 - $< 5 \rightarrow$ Severe "
 - No sperm in semen - Azoospermia
 - No semen - Aspermia.
- Total motility $> 40\%$
 - $> 32\%$ should be progressively motile
 - ($< 32\% \rightarrow$ 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 : IVP

Severe oligospermia (< 5 million/ml) : ICSI

$> 58\%$ of sperm should be viable

Leucocytes s/b < 1 million/ml

Round cells < 5 " "

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 these

Female :-

- 1) cervical factor infertility
- 2) cervical stenosis.

IUI can be done $\bar{c}$ Husband's semen = IUI H
 or " " " Donor's Sem = IUI D

Ix for IUI D :-

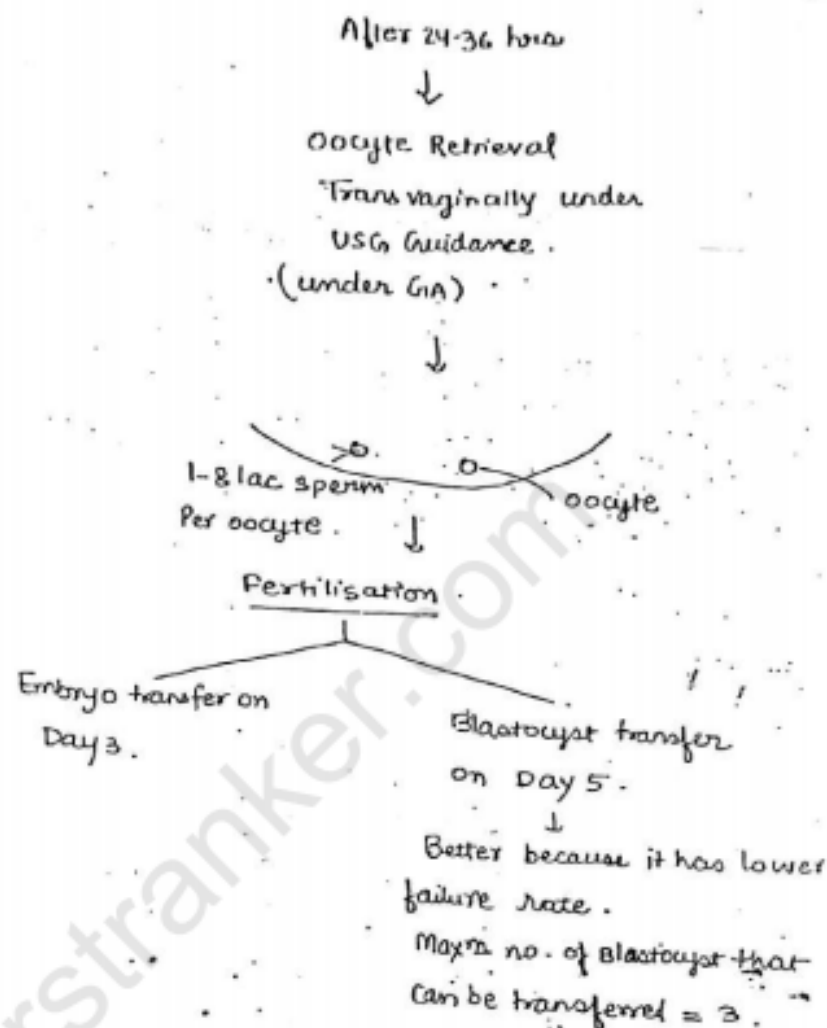
- 1) If male partner has genetic disease, which could be transmitted to off spring
 0.5ml of washed, processed, concentrated sperm are technique

- Swim-up tech.
 - Swim down tech.
 - Density gradient centrifugation (best)
- } Methods of sperm concn

IUF :

♂
 ↓
 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 $\geq 17-18$ mm.
 & endometrium = 8mm. → Give ovula
 trigger (HCG)
 ↓



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 → $\bar{c}$ Donor eggs
- 3) Mullerian agenesis (MRKH) - IVF + Surrogacy
- 4) If ♀ has severe genetic disease & has a risk of transferring → $\bar{c}$ Donor eggs
- 5) oligospermia → males

→ Females

ICSI : Intracytoplasmic sperm injection

Single spe

Single viable sperm $\xrightarrow{\text{Injected into}}$ 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 ~~TESEA~~ = Percutaneous : Testicular sperm aspiration
- TESE = Testicular sperm extraction
- MESA = Microsurgical Epididymal sperm aspiration
- PESA = Percutaneous epididymal sperm aspirⁿ

For testicular causes : Epididymal aspirⁿ is not an option.

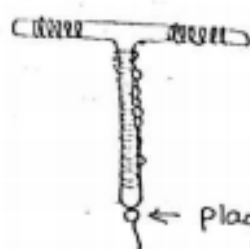
+ In semen analysis, the most imp. factor to

Sperm morphology $>$ motility $>$ concⁿ

Contraception

95

CuT 380 A



plastic ball
↓
Prevents Perforation.

380 = Total surface area of Cu wire.

314

↓
Vertical stem

33 mm²

↓
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 mm²
- 2 flexible arms & fins (to keep it in its position)
- Arms are radiolucent
Stem is Radioopaque.
- Available in a preloaded inserter conf. a plunger.
- Life span = 5 years.

Mirena



No copper wire.

↳ 52 mg of LNG; elutes LNG @ 20 mcg/day.

↳ Life span = 5 years

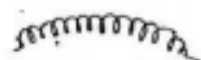
↳ Decreases Blood loss : ∴ used in menorrhagia

↳ 1/6 of dysmenorrhea, Endometriosis, Fibroid adenomyosis

↳ HRT.

↳ Drawback :- Irregular bleeding
Can't be used as emergency
Contractive, unlike COCP.

Essure



↳ spring like device

↳ Introduced in FT via vagina using hysteroscopy.

Inside FT → Initiates Tissue
↓
Inflammation

↳ Success rate : 99%.

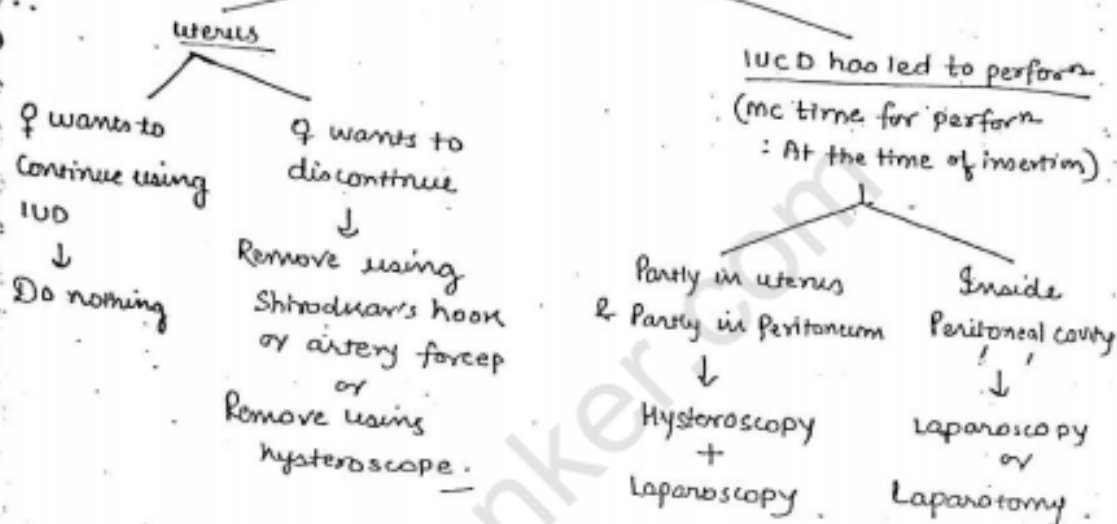
↳ Available in India.

↳ For first 3 months, use backup method



Missing thread of IUDs

↓
Do a TVS to detect whether IUD is inside the uterus or
Perforⁿ has occurred & it has gone inside peritoneum



If a ♀ becomes pregnant w IUCD.

Options

- 1) Abortion
- 2) USG Guided Removal of IUD
- 3) Continue her preg. w IUCD in.

- ↓
- (1) IUCD is not teratogenic.
 - (2) Complic^{ns} :- Abortion, Infectⁿ, Pre-term lab, PROM.

Emergency Contraception:-

Best time = ≤ 72 hours of unprotected intercourse.
Can be taken upto 120 hours.

Drug used

= Cu = Copper containing IUCDs.

= OCs = OCs.

= M = Mifepristone

= E = Estrogen

Lets = LNG

Catch = Centchroman (Saheli)

U = Ulipristal (Ella)
 $\downarrow$
Unavailable in India.

Dosage.

upto 5 days; Failure rate: 0-1%

yuzpee method $\left\{ \begin{array}{l} \text{High dose} = 2 \text{ tab } 12 \text{ hrs } 2 \text{ tab} \\ \text{Low dose} = 4 \text{ tab } 12 \text{ hrs } 4 \text{ tab} \end{array} \right.$

15 mg

15 mg

0.75 mg $\xrightarrow{12 \text{ hrs}}$ 0.75 mg.

50 mg.

30 mg single dose
(can be given upto 120 hrs)

Most effective method: Cu containing IUCDs.

Most effective Hormonal method: Ulipristal > LNG.

Not used as emergency contraceptives: Mirena, Pop, Misoprost.

Female sterilization:

Eligibility criteria - 1 child 1 year old
Age b/w 22-49 years.

Minilaparotomy
(Small incision < 3 cm).

• mc technique: Pomeroy's

Laparoscopically

Gas = CO_2

Intra-ab. pr = 8-12 mm Hg
(max: 15)

Fallopian rings

Clips



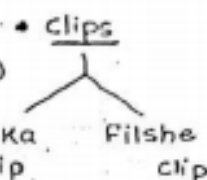
Failure rate :- 1 in 300 to 400.

Tubal damage : 3-4 cm.

Modified Pomeroy's technique



Fallope ring (more common in India)



Placed @ juncⁿ of proxima & middle 3rd of tube

- Failure rate = 0.2-1.3%

- Tubal damage = 3 cms.

(* In clips tubal damage is 4-5 mm.
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 ♀ sterilizⁿ in India: Minilaparotomy.

* Interval sterilization: Delivery → 6 wks → sterilizⁿ.

* Contraceptive C highest risk in Ectopic preg: Sterilization.

(Cawery > Madlener tech > Modified Pomeroy)

98.

Rh (-ve)

Rh antigens discovered by - Landsteiner & Wiener

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. circulaⁿ

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

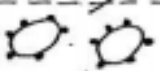
IgG

can cross placenta.

(But by that time baby is born.

∴ 1st preg is safe).

Fetus: (Rh+)



0.1ml

In next preg: This m^o occurs fast

↓
IgG antibodies formed in mother

↓
Cross the placenta

↓
In fetal circulaⁿ Ag-Ab rxn 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 due to

Rh⁺ preg

Other causes (Parvovirus infection,
Thalassemia, etc)

↓
Immune hydrops fetalis

↓
Non-Immune Hydrops fetalis



99.

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)

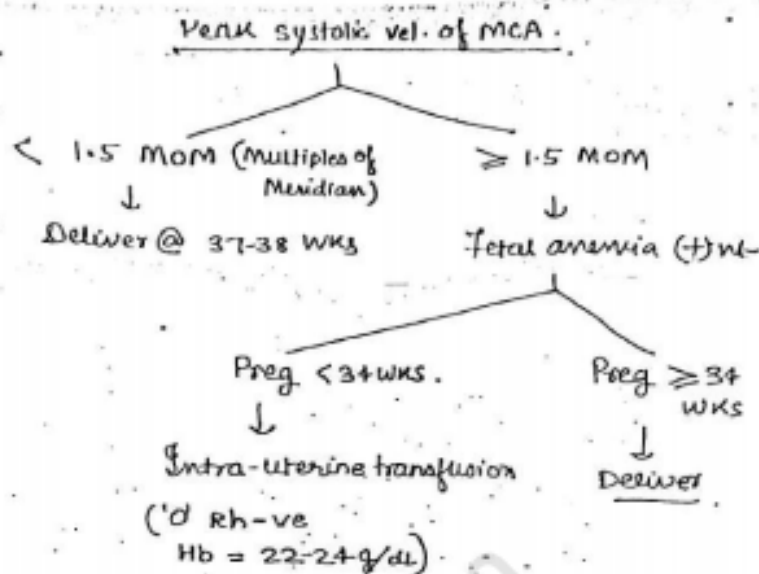
↓
Moderate Risk

CRITICAL
TITRE

{ Titre > 1:32
(Ab > 15 IU/L)
Albumin titre > 1:16

↓
Hydrops fetalis

↓
check how much hemolysis has occurred
By Peak & systolic vel. of MCA



* 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 wks, 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 coombs test = -ve

Best time to give anti-D, after delivery ⇒ in 72 hours
 Max in 28 days after delivery.

100

Ovarian cyst & Pregnancy

MC ovarian cyst diagnosed in preg. - Dermoid cyst.

MC ovarian tumor to undergo torsion in preg. - Dermoid

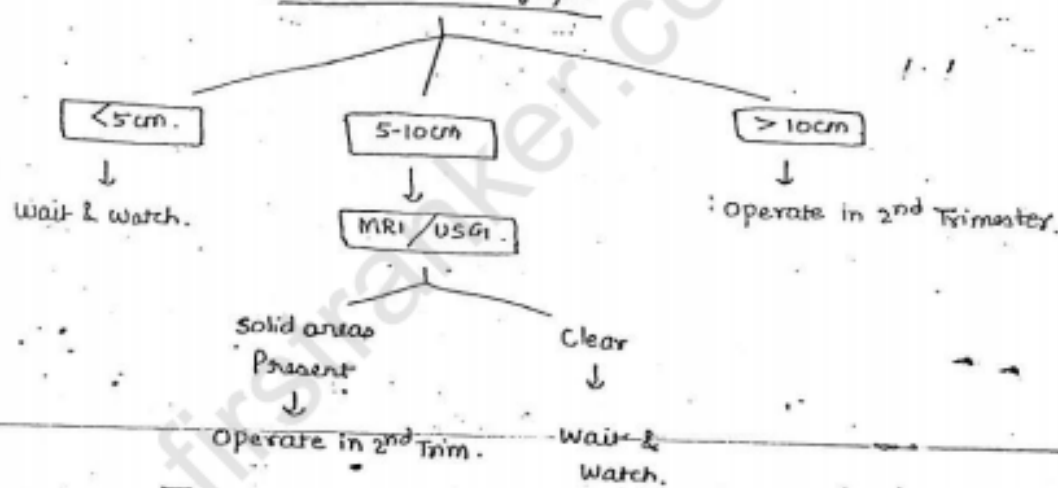
MC time for ovarian cyst to undergo " - at the end of 1st trim / Puerperium.

An ovarian cyst diagnosed during Puerperium s/b removed immediately.

MC ovarian malignancy in Preg. dysgerminoma.

Management

Depends on size of cyst.



* In case of emergency (torsion, rupture) - do surgery irrespective of size of cyst & Period of gestation.

Non-preg ♀ Ovarian mass : Indicⁿs for surgery :-

1. Any ovarian mass which has high risk features on USG.

Size $> 10\text{cm}$.

Solid

B/L.

Ascites present

LN enlargement

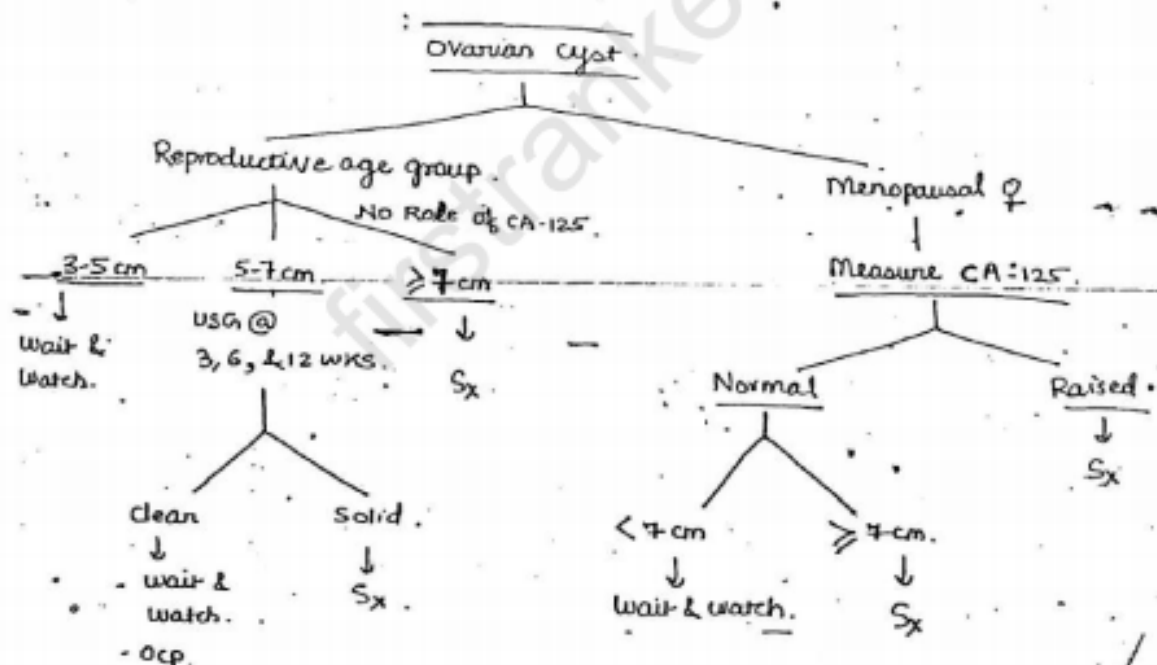
Thick septa

2. Any ovarian mass $> 7\text{cm}$] irrespective of age of pt
" adnexal mass $> 10\text{cm}$

3. Raised CA125 levels in post menopausal ♀

4. Acute complicaⁿ of cyst - Torsion

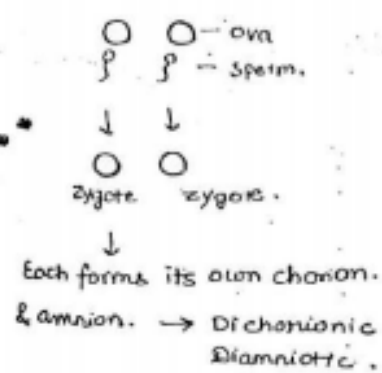
5. Diagnostic purpose.



Twin pregnancy

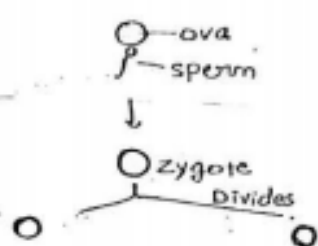
101.

Dizygotic (more common)



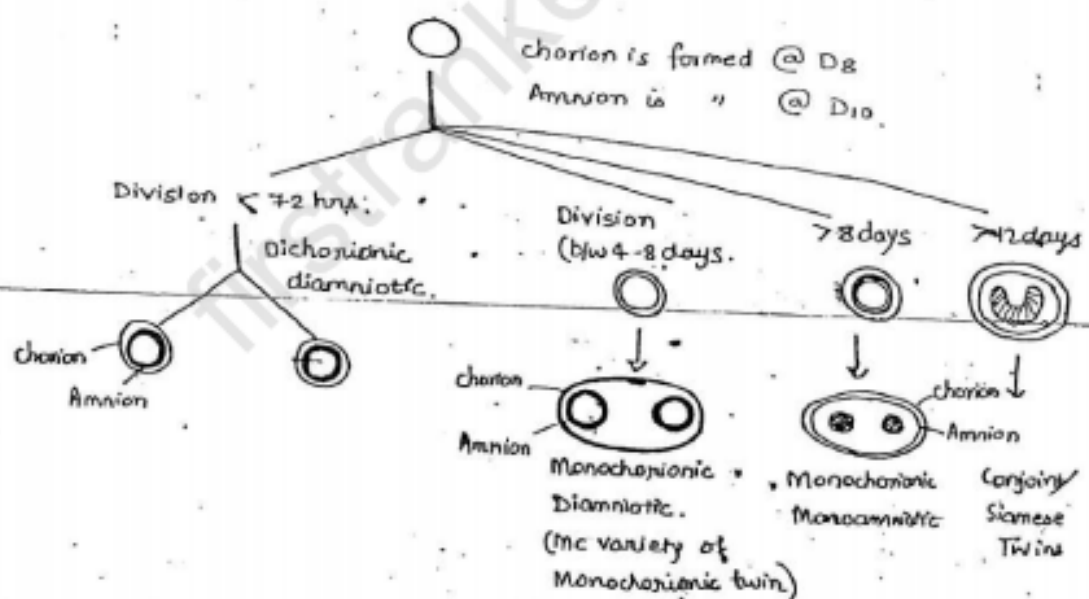
- Same/Different sex.

Monozygotic



- Always have same sex.

Monozygotic twins



Dizygotic twins

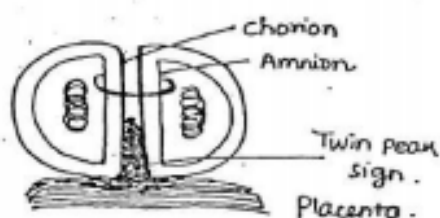
- Highest incidence - Philippines.
- Lowest " - Japan

Monozygotic: Incidence is constant throughout the world (1 in 250)

Incidence of twin is ↑ these days $\frac{d}{t} < IVF$

- * mc variety of twin preg - Dichorionic diamniotic
- * mc " of monozygotic twins - Monochorionic diamniotic.

Dichorionic Diamniotic

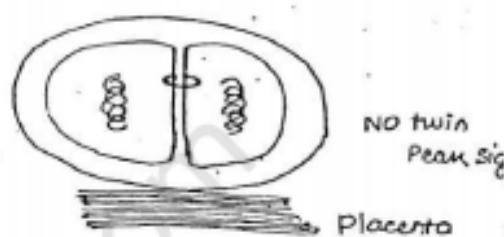


4 layers

Thickness of memb. $> 2m$

- * Twin peak sign is d/t CHORION
- * Twin peak sign: 11-14 weeks (Best time to see)

Monochorionic Diamniotic



2 layers

Thickness of memb. < 2

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).

Conjoined 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.

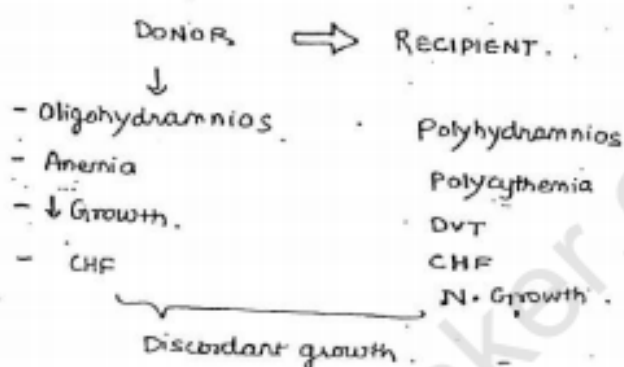
102:

Dichorionic diamniotic : 37 wks.

* Twins to previous C.S. — C.S.

Twin to Twin transfusion syndrome:-

→ Vascular connection is found b/w twins such that deep artery is of one is connected to deep vein of another.



→ More common in Monochorionic diamniotic twins.

(* In MCMA 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 in one or both.

Stage 5. — Death of one or both.



Antepartum fetal Monitoring

① Fetal Movements:-

At least 10 movts in 2 hours pd of Rest.

If pt < 40 ↓ fetal movts:-

↓
NST (Non Stress Test) :- screening test

↓
Non Reactive.

↓
Biophysical score - confirmatory test

Fetal Heart
① Baseline

N FHR = 110-160 bpm
↓ < 110 ↓ > 160
↓ ↓
Bradycardia 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 (Reactive) if FHR becomes fixed = sinusoidal heart rate pattern. Seen in fetal anemia like Vasa Previa

In 20 minutes

- ① Vasa Previa
- ② TTTS
- ③ Rh incompatibility

④ Decelerations = $\downarrow$ in FHR by 15 bpm ^{recovery} for 15 sec.

Type I deceleration = Early deceleration

Type II " = Late "

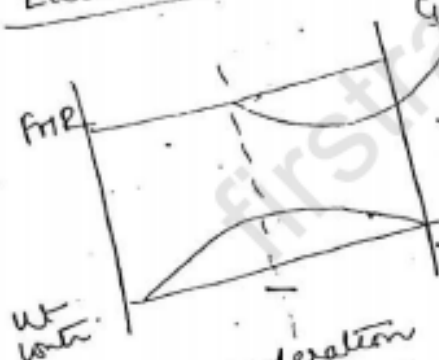
Type III " = Variable "

Early Deceleration



• occurs gradually > 30 sec.
→ seen in head compression
↓
physiological

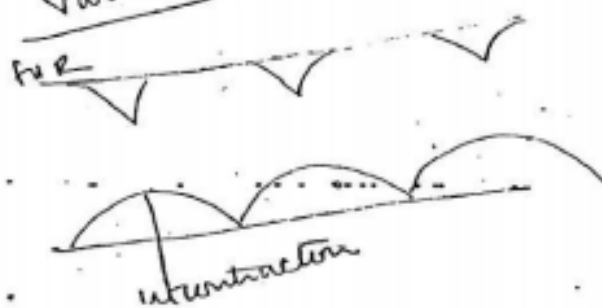
Late Deceleration



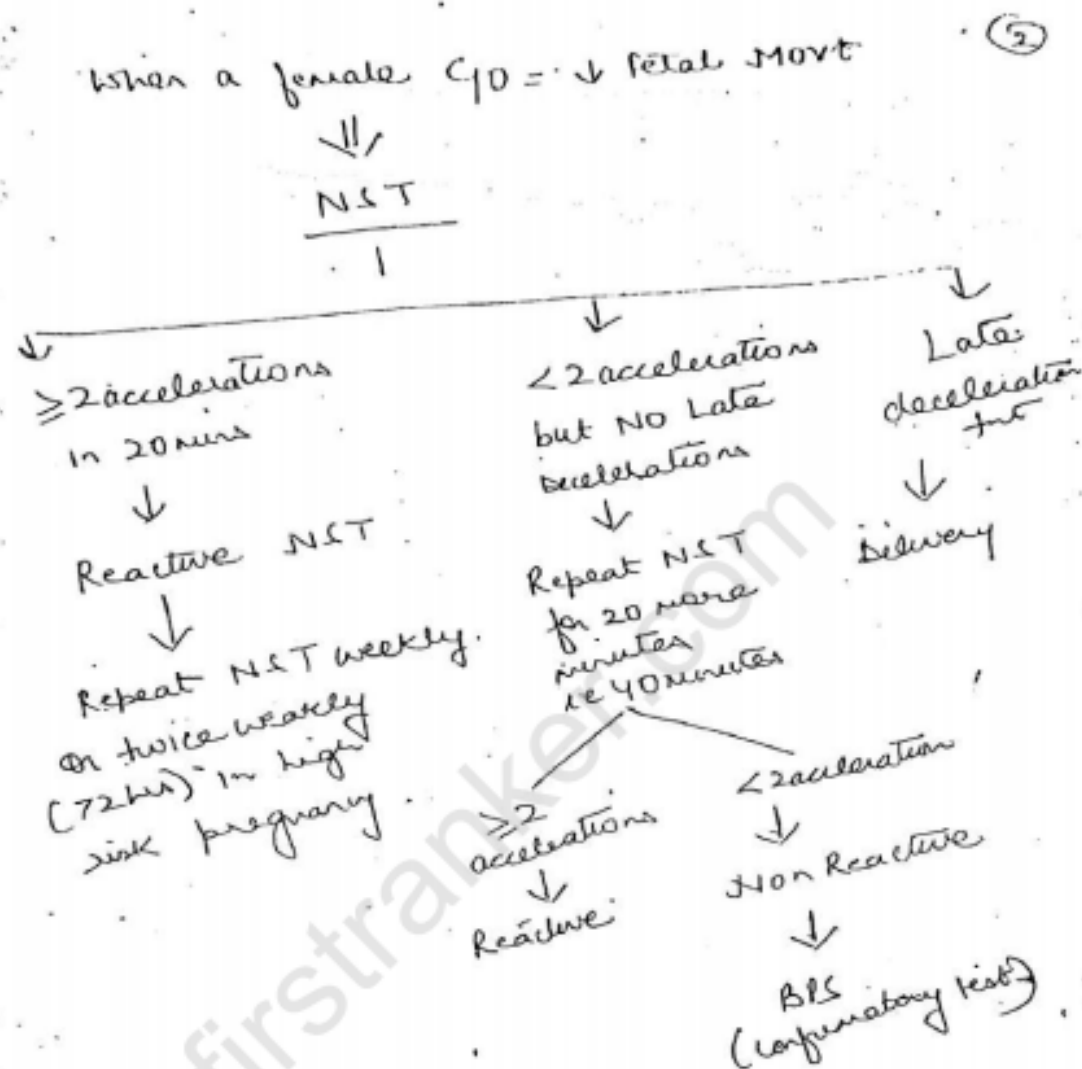
Gradual > 30 sec

→ The FHR starts dipping at uterine contraction & continues after contraction
→ seen in utero-placental insufficiency.
↓
Pathological
↓
Most ominous

Variable deceleration



No fixed relation
• sudden fall in FHR / abrupt (< 30 sec).
• Due to cord compression
↓
physiological



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.	-
Meningitis - Gross body movt	≥ 3 body movt	-
Always - Amniotic fluid volume	Single vertical pocket > 2cms	-
Noxious - 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



Biophysical score

Normal

ie 10/10 or
8/10 with fluid normal
(ie AFval = 2/2)

Management

Fetus is normal
Repeat BPP weekly

Borderline

8/10 with ↓ fluid
or
6/10

If pregnancy ≥ 36 wks
→ deliver

If preg < 36 wks
↓
Repeat BPP on same
day. If it remains
same → deliver

Abnormal

$\leq 4/10$

Deliver

→ usually LSCs needed

Modified BPS →

- > Developed by Clarke
- > NST + AF volume measured
- > ~~various parameters~~ ~~measured~~
- > If NST Reactive + AF volume index ≥ 5 → Test is Normal.
- > If test is abnormal → do a BPP

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 CI to vaginal delivery, ~~the~~ vaginal delivery is the preferred mode of delivery with continuous monitoring.
If persistent late decelerations are present → LSCS done.

Intrapartum Monitoring

- ① Continuous CTG or Intermittent auscultation of FHS.

FHS auscultation	Low Risk	High Risk
1 st stage	30 mins	15 mins
2 nd 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 - Caesarean / delivery.

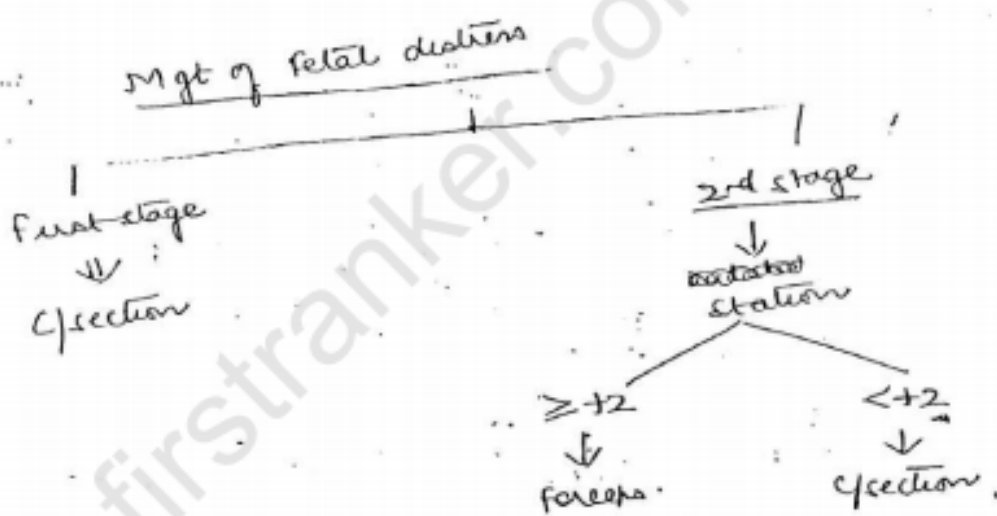
- Not to be done in nulliparous females

- Avoid vacuum till 1 hr after it.

~~Answer~~

Fetal distress term has now been replaced (4)
by Non Reassuring fetal status

- ① CTG -
 - Persistent Bradycardia
 - Persistent Late deceleration
 - Persistent variable "
- ② fetal scalp pH $\text{pH} < 7.2$
- ③ liquor - meconium stained
- ④ Low apgar score at 1 minute of birth



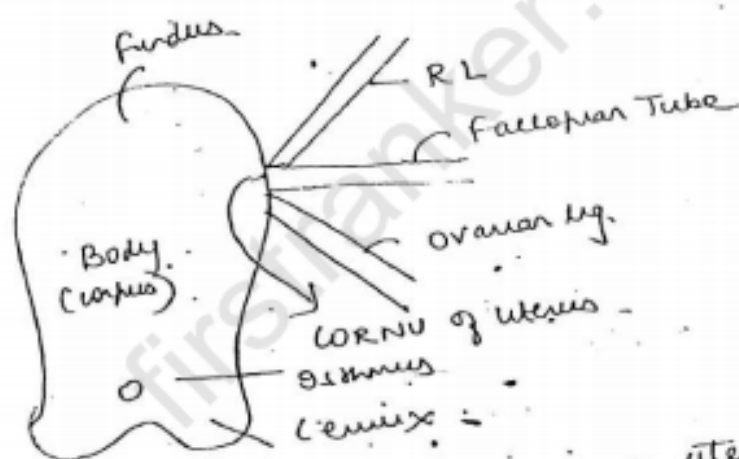
Remember:

- At station $\geq +2$, Forceps is quicker than caesarean
- Ventouse vacuum is not preferred in fetal distress

Important Points on Anatomy

UTERUS

- > Wt in Non pregnant female $\left\{ \begin{array}{l} \text{Nulliparous: } 50-70g \\ \text{Multiparous: } 80g \end{array} \right.$
- > Wt of pregnant uterus = 1000 gm.
 & when does wt of uterus become normal (non pregnant like) after delivery = 6 weeks after delivery
- > Length of non pregnant uterus $\left\{ \begin{array}{l} \text{Nulliparous: } 6-8 \text{ cm} \\ \text{Multiparous: } 9-10 \text{ cm} \end{array} \right.$
- > Length of pregnant uterus = 35 cm.
- > Capacity of uterus $\left\{ \begin{array}{l} \text{Non preg} = 10 \text{ ml} \\ \text{Preg} = 5000 \text{ ml} \end{array} \right.$



Structures attached to cornua of uterus.

Ant to Post = $\underline{R}$ = Round lig
 $\underline{T}$ = Fallopian Tube

Superior to Inferior = $\underline{O}$ - ovarian lig
 superior most = FT
 Then RT & O at same level.



- (5)
- > Round Ligament -
 - Derived from Gubernaculum
 - Keeps uterus in anteverted position
 - Attached to Labia majora
 - Suspends A, branch of uterine A runs within the ligament
 - > Canal of Nuck - Fold of peritoneum in fetus that contains the Round Ligament & ends into Gonadal canal
 - > Isthmus :-
 - Lies between Anatomical interval as 2
 - Histological int as
 - Forms lower uterine segment during pregnancy
- 70% by isthmus
 30% by CS
 Non pregnant length of isthmus = 0.5 cm
- BLOOD SUPPLY OF UTERUS.
- ① Uterine A (mainly) - br of ant dev of int Mac A.
 - ② Ovarian A.

> Note: - 2cm lateral to cx, uterine A lies just above meter k/a water under bridge & it is the M/C site of metric injury.
2nd M/C site = Pelvic brim

① Branches of uterine A to uterus

U = (Ut A Branches)

A → Arcuate A

R - Radial A

B - Basal A

S - Spinal A

② Uterine A - gives Sampson A to supply RL

③ Descending vaginal cervical A which supplies cx & upper part of vagina.

Nerve supply = T10-L1

Lymphatic drainage =

Body - Int iliac LN (M/C)

Ext "

Corpus - Superficial ing LN

Fundus - Para Aortic LN



CERVIX

(6)

- Length in preg = 4 cm
- Blood supply = Descending Cervical A
- Lymphatic Drainage

I = Int iliac
H = hypogastric LN
O = Obturator

P = Periaortic Paracervical / uterine
E = Ext iliac

- Nerve supply = S₂-S₄

Cervix : Corpus Ratio

At birth 1:1

Before puberty 2:1

After Puberty 1:2

Peak age 1:3

Menopause 1:1

pH = 7.8

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 does not have any glands
- Vagina has Döderlein bacilli
- pH of vagina

Reproductive age

During menstruation

Before puberty

After menopause

New born = 5.5-6

3.5-4.5 (4-4.5)

6.5-7.5

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 ing LN

Pain during Labor

Early stages :- due to ut contraction - T10-L1
 Later " - " " (x dilatation) = S2-S4.

Painless Labor: Epidural Analgesia

↓
 DOL - Bupivacaine

↓
 Level = T10..

During cesarean = Spinal Anesthesia

↓
 Level T4

For Instrumental delivery = Pudendal N block

↓
 Site = Sacral spine

↓
 Ligament pierced = Sacrospinous ligament.

Kab

Lab 03u Q's

Q1 G2P1 = Admitted in labor at 8am on Monday
with Cx = 2cms, 40% effaced
Krentzdays At Till 10pm → Cx = 3cms,
80% effaced.
FHR = Regular.
Membr = Intact.

Next step in Mgt. Ans = Acetaminophen Rest

Q2:- G2P1 = Admitted in labor at 8am on Monday
Cx = 2cms dilated, 40% effaced,
At 12pm (midnoon) = Cx = 6cms dilated
Station = 0, 80% effaced
At 3pm = Cx = 7cms effaced 80% effaced
Station = +1.
Membr = Intact.
FHR = Regular.
Ut contraction = int

Next step - ARM

Q3:- G2P1 = admit in labor at 8am on
Monday
Membr Ruptured →
Cx = 6cms dilated, 80% effaced.
NO meconium.
FHR = int, Regular. 14hrs period
At 12pm (midnoon) same findings.
Ut contractions = int, Adequate.

Mgt :- C-section

Q4 :- G2 P1 = admitted in labor room = ②

Time = PIV = 6x = fully dilated

8 am

Memb ruptured

liquor = clear

station of fetal head = +2

1 hr passed

At 9 am:

Prolonged labour
same findings

Mgt. forceps

At 10 am -

same findings

Mgt.

Caput tnt

Moulding tnt

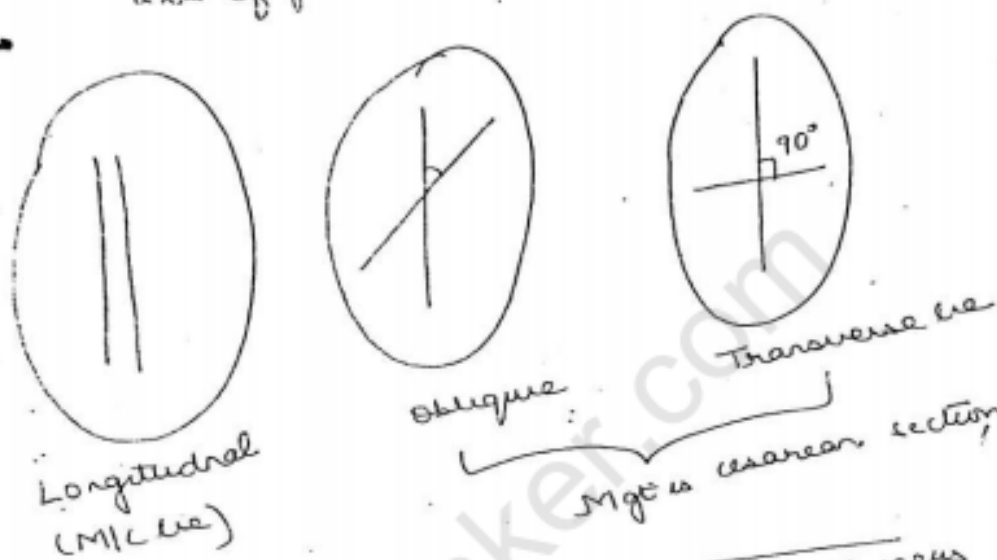
Henaternal tnt

C Section.

Second stage arrested
obstructed labour

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

MLC causes:

- Oligopathic
- Placenta previa
- Polyhydramnios

→ note - oligohydramnios can lead to malpresentation but it never leads to unstable lie.

- Ab N shape of pelvis.

Mgt = cesarean section

② Presentation Part of the fetus which lies in the lower part of uterus in longitudinal lie



Cephalic presentation (M/C)



Breech presentation

Apart from cephalic presentation, rest all presentations are malpresentations.
M/C malpresentation - Breech

in Transverse lie



Presentation - Shoulder presentation

↓
Mgt

↓
Caesarean section

(Whether the baby is alive / dead)

Presenting part:-

Part of the presentation which has directly over the interval as -

Or well flexed head - vertex (M/C)

Or fully extended " - face

Or partial extended - Brow → always do cesarean section

Denominator

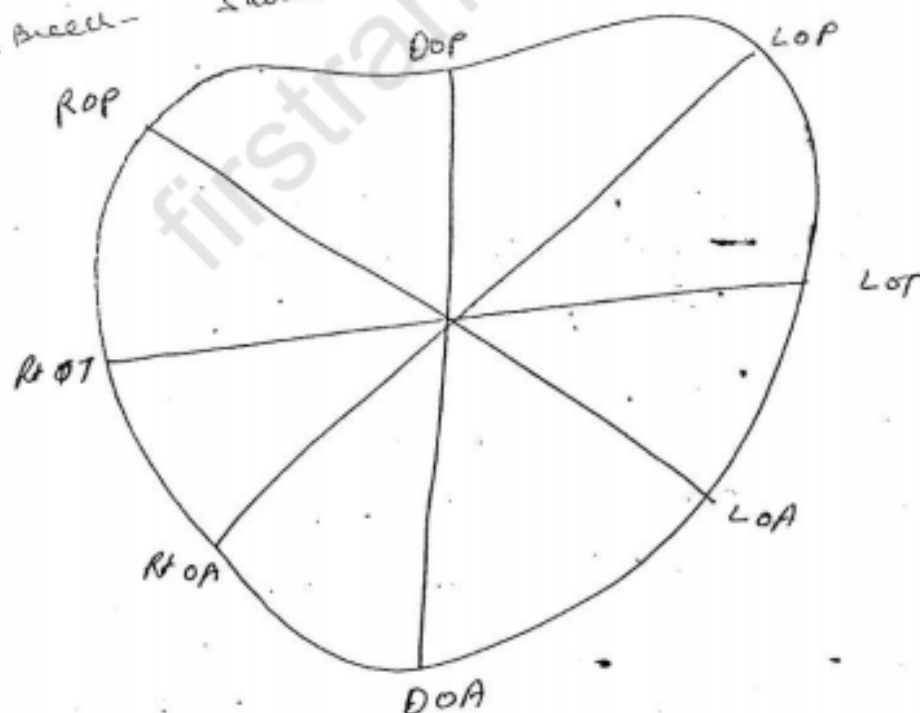
Bony fixed point of reference in presentation used to describe position -

Vertex - occiput

Face - mentum

Brow - frontal bone

Snell - sacrum



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 fontanelle and posterior fontanelle

① Vertex = B/w ant fontale and Post fontonale

② Brow = ————— on one side and Root of nose
Supraorbital ridge on other side

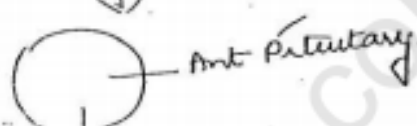
Master copy ①

OVARIAN CYCLE

At puberty

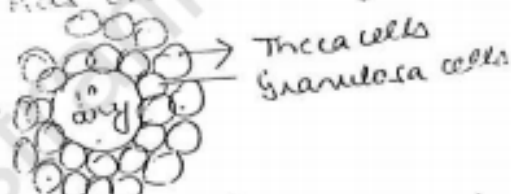
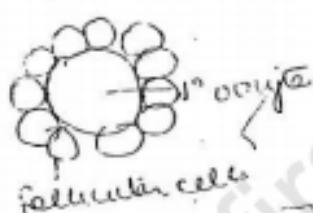
Hypothalamus becomes functional
(" is formed @ 12wks of IUL)

↓
GnRH released in a pulsatile manner



↓
FSH released

↓
Acts on follicles of ovary



↓
Granulosa cells is released

↓
Estrogen

↓
Inhibits
Negative feedback
on FSH

↓
Proliferative
effect on
Endometrium

(Regrows the
functional layer of
Endometrium which was shed
during menstruation, from
the basal layer)

↓
Negative
effect on
FSH

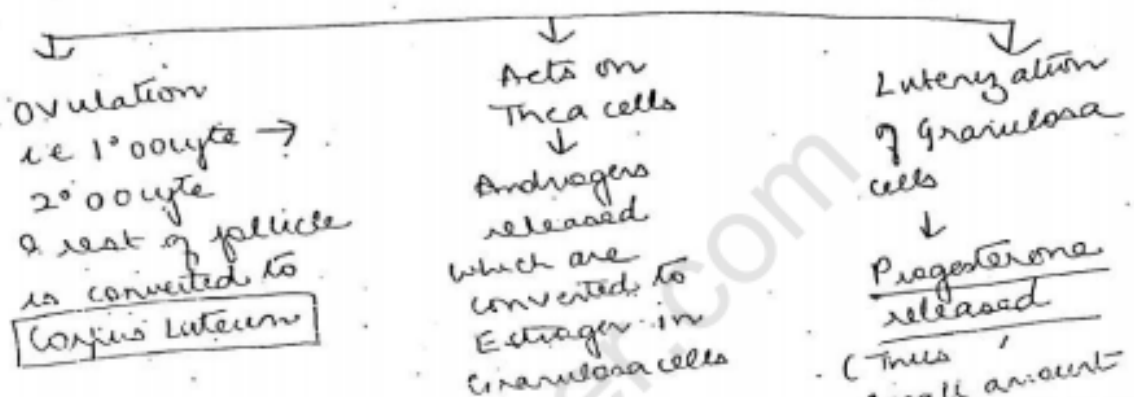
↓
+ve feedback
on LH

↓
LH suddenly ↑



sudden ↑ in LH is called (2)
as LH surge

① For LH surge Estrogen levels should be
200 pg X 48 hrs



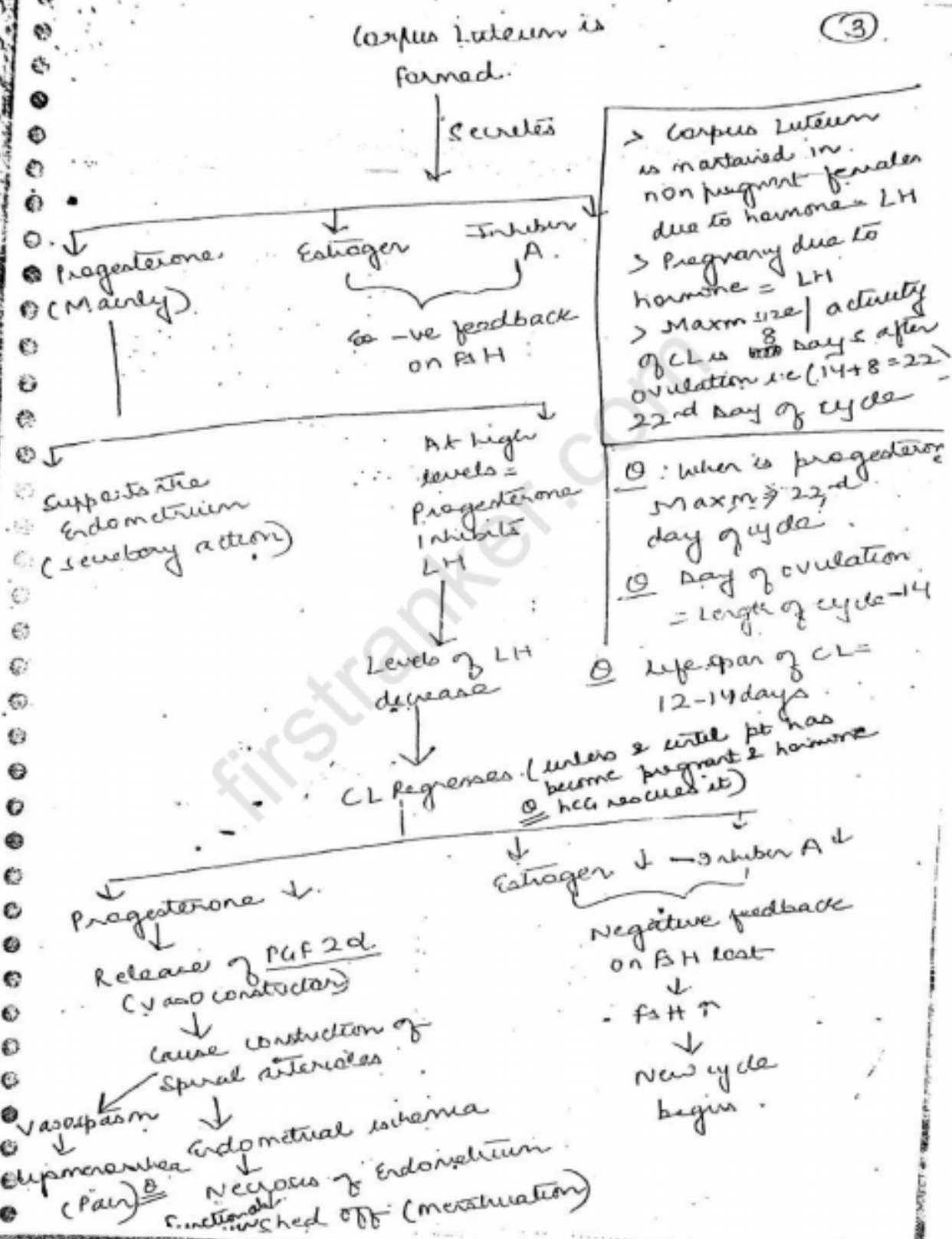
② LH surge (32-36 hrs) → ovulation
③ LH peak (24-36 hrs) → ovulation
④ LH peak (12 hrs) → ovulation
⑤ LH peak (12 hrs) → ovulation
⑥ LH peak (12 hrs) → ovulation
⑦ LH peak (12 hrs) → ovulation
⑧ LH peak (12 hrs) → ovulation
⑨ LH peak (12 hrs) → ovulation
⑩ LH peak (12 hrs) → ovulation
⑪ LH peak (12 hrs) → ovulation
⑫ LH peak (12 hrs) → ovulation
⑬ LH peak (12 hrs) → ovulation
⑭ LH peak (12 hrs) → ovulation
⑮ LH peak (12 hrs) → ovulation
⑯ LH peak (12 hrs) → ovulation
⑰ LH peak (12 hrs) → ovulation
⑱ LH peak (12 hrs) → ovulation
⑲ LH peak (12 hrs) → ovulation
⑳ LH peak (12 hrs) → ovulation
㉑ LH peak (12 hrs) → ovulation
㉒ LH peak (12 hrs) → ovulation
㉓ LH peak (12 hrs) → ovulation
㉔ LH peak (12 hrs) → ovulation
㉕ LH peak (12 hrs) → ovulation
㉖ LH peak (12 hrs) → ovulation
㉗ LH peak (12 hrs) → ovulation
㉘ LH peak (12 hrs) → ovulation
㉙ LH peak (12 hrs) → ovulation
㉚ LH peak (12 hrs) → ovulation
㉛ LH peak (12 hrs) → ovulation
㉜ LH peak (12 hrs) → ovulation
㉝ LH peak (12 hrs) → ovulation
㉞ LH peak (12 hrs) → ovulation
㉟ LH peak (12 hrs) → ovulation
㊱ LH peak (12 hrs) → ovulation
㊲ LH peak (12 hrs) → ovulation
㊳ LH peak (12 hrs) → ovulation
㊴ LH peak (12 hrs) → ovulation
㊵ LH peak (12 hrs) → ovulation
㊶ LH peak (12 hrs) → ovulation
㊷ LH peak (12 hrs) → ovulation
㊸ LH peak (12 hrs) → ovulation
㊹ LH peak (12 hrs) → ovulation
㊺ LH peak (12 hrs) → ovulation
㊻ LH peak (12 hrs) → ovulation
㊼ LH peak (12 hrs) → ovulation
㊽ LH peak (12 hrs) → ovulation
㊾ LH peak (12 hrs) → ovulation
㊿ LH peak (12 hrs) → ovulation

Note: At low levels
① Progesterone has a
+ve feedback on LH &
FSH ∴ just before
ovulation there is
both LH + FSH surge.

② Initiation of LH surge is
due to hormone → Estrogen
for maintenance of LH surge
both = E + P needed

③ When does synthesis of
Progesterone begin in ovary =
it begins at LH surge i.e.
32-36 hrs before ovulation







Remember

Estragen

- > Always has a negative feedback on FSH

> For LH

Low conc
↓
It 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 = 24-35 days

(According to Navack gynecology)
24-38 days (study based)
24-35 days (population based)

Avg = 28 days (only 15% of actually have cycle of 28 days)

Normal blood loss 5-80ml

Avg blood loss = 35ml > 50ml

Number of days women can bleed = 2-7 days
Avg = 4-5 days

If this is any deviation from above it is called as Atypical uterine bleeding = AUB

(1) Menorrhagia = cycles at Regular interval.
Blood loss > 80ml or > 8 days of bleeding.

(2) Hypomenorrhea = cycles regular.
Blood loss < 30ml 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



(6) Metrorrhagia = Normal cycles + intermenstrual bleeding.

Causes of AUB

- P = Polyps
- A = Adenomyoma
- L = Leiomyoma
- M = Malignancy

PALM COEIN classification

- C = Coagulopathy
- O = ovulatory dysfunction
- E = Endometrial cause
- I = Iatrogenic
- N = Non classified

Male & Female Differentiation

(6)

What determines sex in a fetus?
 others: Gene for Testis = SOX-9
 ovary = WNT4 & Rspo1

SRG SRY gene
 on short arm of
 Y chromosome
 Man

SRY gene int



Testis

(at 7 weeks)

Gonads develop from = Genital Ridge

1st feature which distinguishes T & O
 → formation of Testis or Ovary

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

Remnant of MD in males
 → appendix of testis
 → Prostatic utricle

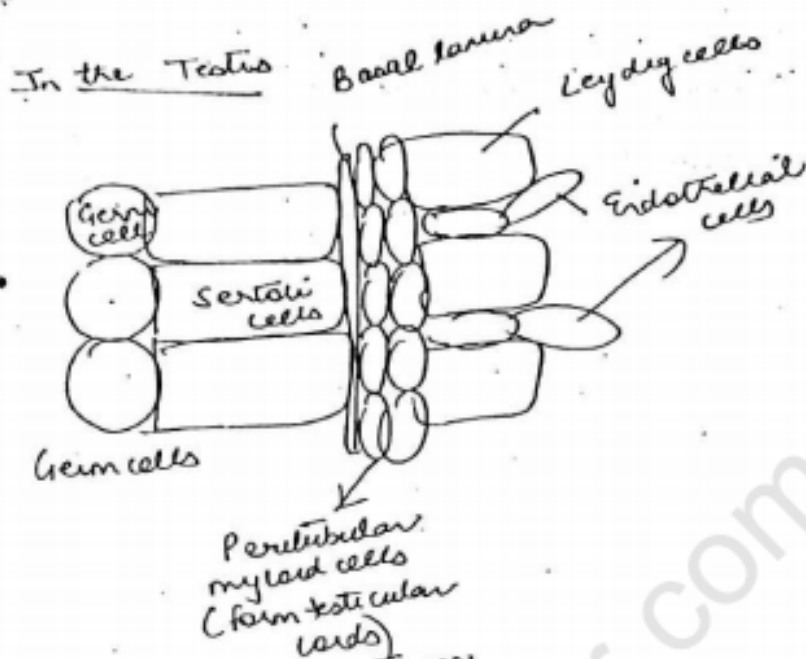
Leydig cells

Testosterone
 (Production begins 8-9 weeks)
 Max = 14-18 weeks

Promote growth of Wolffian duct in males

5 α Reductase
 Dihydrotestosterone
 Male External genitalia

S = Seminal vesicles
 E = Ejaculatory duct
 E = Epididymis
 B = Vas deferens



The tight junctions between the Sertoli cells create the blood-testis barrier which keeps blood borne substances from reaching the germ cells & keeps

allowing separation - sperm are released
Spermatogenesis occurs in seminiferous tubules of testes
↓
Epididymis (attain mat. & motility 10-14 days)
↓
Vas deferens
↓
ejaculation

of vas deferens
volume - not function
Spermatozoa
secrete in vas deferens
(B.P. & P.D.)
released along to seminal vesicle gland (60% vol. + 40% vol. of ejaculatory duct) → prostate gland
20-30 ml of semen
but if no sperm
semen = egg carrier
till 60 min → do on
but if no egg
semen



In females :-

Ovaries are formed
due to absence of Y chromosome.
(i.e. why in Turner's $45 X O =$
Gonads ovary)

↓
But for proper development of ovaries
= XX needed
(i.e. why in Turner's: Gonads = streak)

> NO Sertoli cells $\Rightarrow$ NO Mullerian factor $\rightarrow$
MS grow into

Fallopian Tube
uterus
CX
upper 3/5 of vagina

> NO Testosterone: $\left\{ \begin{array}{l} \text{Wolffian duct Regresses} \\ \text{Ext genitalia of female} \\ \text{develop} \end{array} \right.$

Remnants of WS in females

1. Hydatid of Morgagni
2. Kallit Tubercle
3. Epioophoron
4. Para-oophoron
5. Gartner duct

Q: All Remnants are present in Lateral part of Broad
lig except Para-oophoron (medial part)

Hermaphrodite

True -
Both ovary & testis
in same individual

Gonads = one sex
Genitalia = other
sex

Male Pseudo hermaphrodite

Genotype = 46XY

Gonads = Testis

External Genitalia = female

MC cause - Testicular feminizing
syndrome
(Androgen insensitivity
syndrome)

Female Pseudo-
hermaphrodite

Genotype = 46XX

Gonads = ovary

Ext Genitalia = Male

MC cause =
Congenital
Adrenal Hyperplasia

MC enzyme defect = 21 hydroxylase

Also Note

On AMH - (clinical uses)

17ONP ≥ 3500 ng/dl

(Screening test)

After ACTH stimulation test =
levels $\geq 10,000$ ng/dl

as it can't be
suppressed

Pregnant female has CAH
to prevent CAH in fetus $\rightarrow$
Hydrocortisone
DOC $\rightarrow$ ~~Hydrocortisone~~
as it crosses the placenta and $\therefore$ fetal HPA axis is not
suppressed
Pregnant female if female
fetus has CAH $\rightarrow$ DOC $\rightarrow$
Hydrocortisone Dexamethasone
as it crosses the placenta

TURNER syndrome :-

Karyotype = $45X0$

Gonads = streak gonads.

Most characteristic feature of Turner is short stature

due to deletion of SHOX gene (short stature homeobox containing gene)
Gonads are streak

(9) Note: Pure gonadal dysgenesis = $46XX$
Mosaic = $46X0/46XY$
↓
streak ↓
 Abn function
 of
 Testes
stature is normal (Ht = 11)

↓ Estrogen

Lack secondary sexual characteristics
- not Breast
- not Pubic hair & axillary hair

uterus hypoplastic

Other features of Turner

- webbing of neck
- Low post hair line
- shield shaped chest
- widely separated nipples
- CV anomalies = MLC Bicuspid Aortic valve
- Renal anomalies = MLC Horseshoe shaped kidney
- Cubitus Valgus
- short 4th metacarpal
- Auto immune dis = Diabetes / Hashimoto's Thyroiditis
- LH/FSH = ↑ (NO negative feedback)
(Hypogon. Hypergonadotropic Hypogonadism)
- IQ = Normal
- Life span ↓ due to CV problem = decreased
- As they have ↓ estrogen they will have osteoporosis
- On all pts of Turner synd = Karyotype should be done
- as if they are mosaic = $45X0/46XY$ → then gonads have high chances of developing GONADOBLASTOMA

Puberty = ♀ = 10.5 years
♂ = 11.5 years.

sequence in females

Growth spurt
↓
Thelarche
↓
Pubarche
↓
Peak height velocity
↓
Menarche

1st sign of Puberty
in males = Testicular
growth

① 1st sign of puberty in
females = Growth spurt
② 1st visible sign =
Breast budding = Thelarche

Tanner classified Breast
appearance & Pubic hair
appearance in stages -
Total 5 stages

Puberty

↓
Precocious Puberty
[Females: Puberty < 8 years, Males < 9 years]
(MIC cause = Idiopathic
2nd MIC = Brain tumor
Hamantoma = 3rd all cases
do MRI)

Delayed
Puberty

♀ = No sec sexual
characteristic by
13 years / No menarche
by 15 years
♂ = No testicular
growth by 14 years

2 types
① Central
↑ GnRH
↑ LH/FSH
Always Isosexual
MIC cause = Idiopathic
SDC = Continuous GnRH

Peripheral
Not due to activation
of HPO axis
↓
Some Edinger
adrenogen producing
Tm of ovary
Can be isosexual/
Heterosexual
or MC like Adrenogen
cushing

Note
Delayed
Puberty is
MIC in
boys = m/c
cause is
constitutional



Menopause

(11)

Absence of menses for 12 months or 1 year

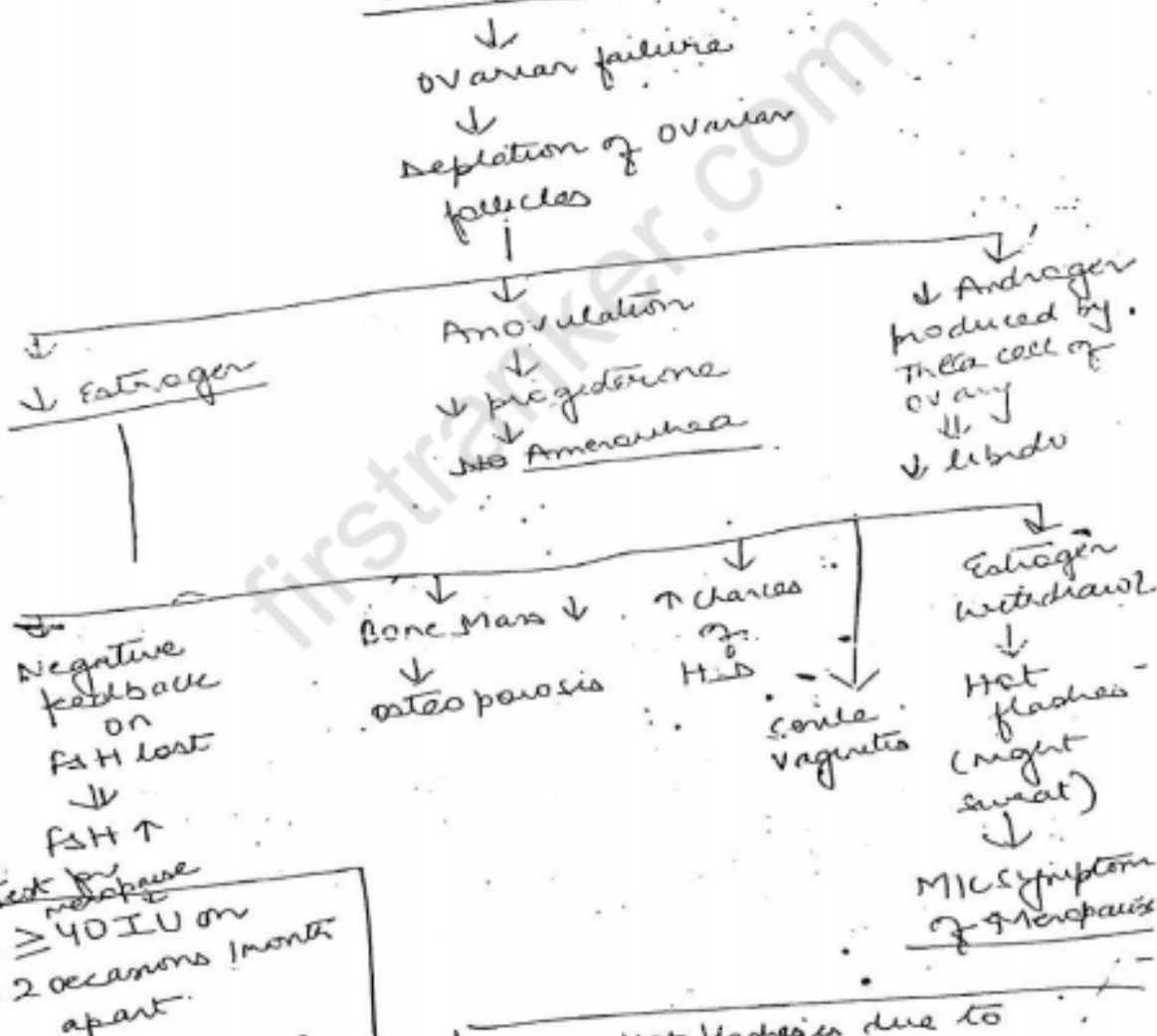
Age = 51 years.

In India = 47 ± 4 years

Delayed menopause = No menopause by 55 years.

Premature menopause = < 40 years.

Menopause



Note: Hot flashes are due to Estrogen withdrawal.
 & Well Turner's syndr pts have hot flashes → NO cause they never had Estrogen

In Menopause :-

Adrenals Normal →

Androgen

↓ Peripherally converted to
estrogen

• ~~Androgen~~
• ~~Androgen~~
• ~~Androgen~~

TEI

↓ Main hormone during
Menopause

→ ~~Androgen~~
diagnosis of Menopause

- > FSH ≥ 40 IU
- > Serum estradiol < 20 pg/ml
- > vaginal cytology = 100/0/0

Mgt of Hot flashes = HRT

- Post hysterectomy pt = E
- ♀ with uterus intact = E + P
- ♀ with venous thromboembolism with uterus intact has hot flashes = SERI - Fluoxetine

Note: Raloxifene cannot be used in mgt of hot flashes

Mgt of osteoporosis

- DOC - Bisphosphonates
- over drug which can be used - Raloxifene

Mgt of senile vaginitis

Local estrogen cream



(13)

INFERTILITY

(108)

In ability to conceive even after 1 year of unprotected intercourse

Female factors are responsible for infertility by: 40-55%

Male factors: 40%

Unexplained factors: 10%

Female factors can be:

Ovulatory: 30%

Tubal: 20-30%

Uterine: 15%

Cervical: 5%

(Unexplained: 10%)

Amovulation
↓
ovarian reserve



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 mucus: E - thin

P - Sticky (Thick)

✓
D20: Cork Screw Glands.

D22: Maximum Stromal Edema

D24: perivascular cuffing.

↓
Necrosis D28.
on Histopath

Lymphocytic Infiltration: L phase? → "Pre-menstrual phase"
Seen by D24.

Endometrial Ec: L.P.D

> 2 day lag b/w menstrual dating & HPE dating.

EC
D24: Secondary change ≈ 20 day appearance. This is called as L.P.D
Luteal phase Defect

by hist
USG for follicular monitoring:

≤ USG? TVS.

Call pt alternate day

↓
then
↓
Everyday



- Rate of Growth: 2mm/day
- When will it rupture/mature follicle: 18-20mm
- Sudden decrease in size of growing follicle & fluid in pouch of Douglas ovulated.



Mgt of Anovulation = Done in Pcos ::
ovulation inducing drugs

M.C. 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 D₃ of M.Cycle

Value: < 10 IU : (N)

Follicle less
↓ estrogen less
↓ Inhibition less
↓
FSH level ↑

10-15 IU: Borderline reserve
> 15 IU: poor reserve
> 20 IU: Suggestive of premature failure
> 40 IU: Dx of premature ovarian failure

② S. Inhibin B Levels:

More follicle = more value

Done on D₅: level < 45 pg = poor reserve

③ Clomifene Citrate Challenge Test (CCCT):

On D₃: S. FSH measured

From: D₅ - D₇: 100 mg of CC

On D₁₀: another sample: S. FSH

High value on D₃ → which further ↑ on D₁₀: Indicates poor ovarian reserve

Theoretical Not used these days

④ Best Test: Anti-Müllerian hormone -
produced by Granulosa cells of follicles.
Best test for ovarian reserve as it can be
done on any day. In ↓ ovarian reserve = ↓ AMH

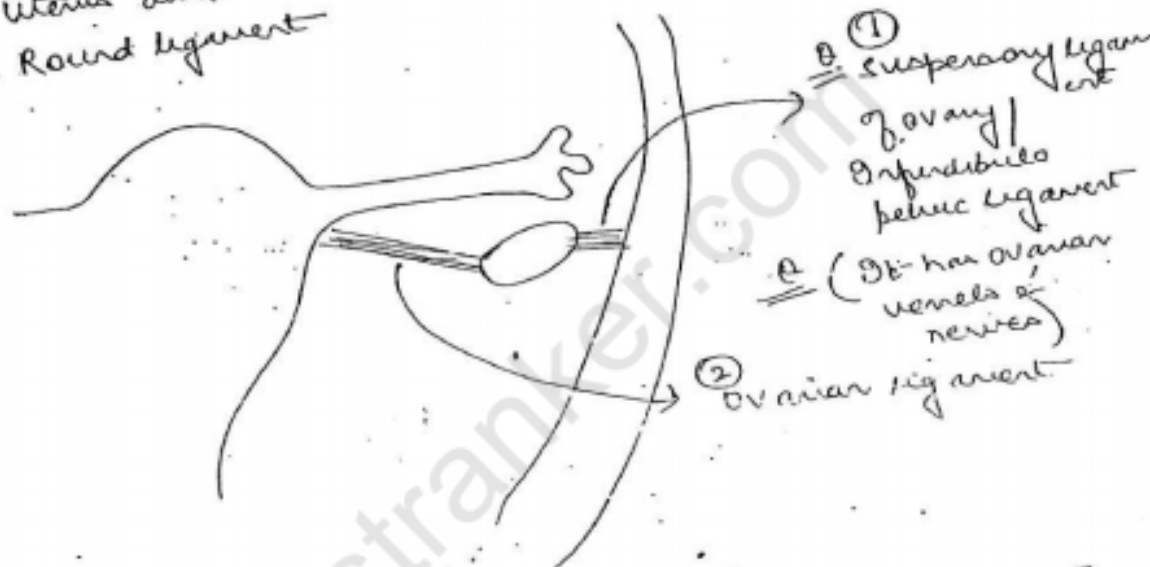
Mat: TUF using inner box



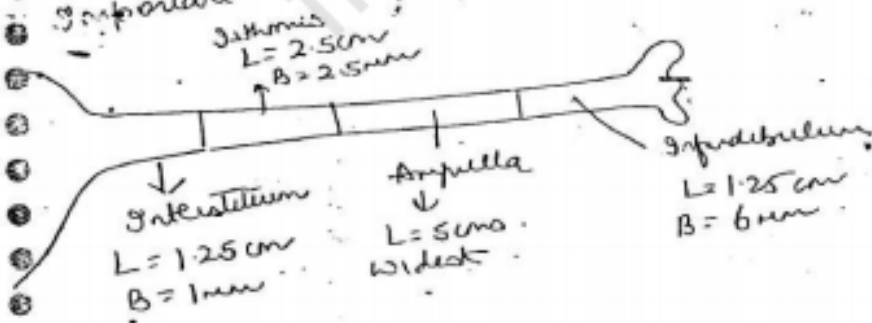
Important Points on Anatomy of ovary

(16)

1. Ovary lies in ~~PSS~~ ovarian fossa of Waldeyer in lateral pelvic wall.
 2. On 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.



(17)

Fallopian tube could

Phenomenon

Site

Fertilisation occurs

Ampulla

Ectopic preg MK

Interstitial part

Anatomic sphincter

Isthmus

Physiological "

Canal

TB causes block in

Fimbria

Gonococcus blocks

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 = (DS - DII
Bist - DIO)
OPD procedure

Canula

Leech

hook on canula

Nearest to

PI may
have
pain

Absolute 4I

- ① Genital TB
- ② Current pelvic infection
- ③ Pregnancy
- ④ Allergy to dye

Gold standard :- Laparoscopic Chromopertubation

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 MIC cause B/L cornual block is Physiological

Mgt of cornual block

Tubal cannulation under hysteroscopic or USA guidance (p is a guide wire)

+ Laparoscopic chromopertubation

↓
In most cases block is not released

↓
if not released

↓
it is pathological

MIC cause of pathological B/L cornual block -
Genital TB (Mgt = IVF)

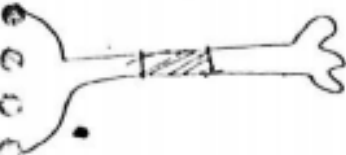
(ii) Distal block

↓
Mild
↓
fimbrioplasty

severe
IVF

Midsegmental block

(19)



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-

① apts > fallope ring > Modified

② Pomeroy > cautery

③ Type of Remnant mox -

Best with istmo-isthmus

④ Total length of tube which can be reconstructed - $\geq 4cm$ - good

⑤ Repe gang for Reversal which Ix should be done

↓
Husband semen analysis

Genital TB: Always a secondary infection.

M.C. site = Lungs > Lymph node.

M.C. Route of spread = hematogenous.

M.C. PID in virgin ♀ = Genital TB

M.C. Site: 90% Fallopian Tubes > Endometrium (50-60%) > Ovary

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 Inflamed.

Chronic phase: Tubes become Thick walled/ Adhesions

• HSG findings: BIL. tubal block

• But Remember: LEAD PIPE Appearance

String on Bead appearance

Tobacco pouch appearance

Cobblestone appearance

Hydrosalpinx

Grav. stipes appearance

Endometrium: M.C. route of Spread: DIRECT SPREAD

• acute phase: absolutely Ⓢ 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. (∴ hist. culture always)





M.C. presentation of Genital TB: Infertility > pain > Menstrual Irregularities

What % of pt's w/ TB are Infertile: Up to 75%

What % of pt's w/ Infertility have TB: { World: 10%
India: 17%

1st Menstrual Complaint: Menorrhagia

M.C. Menstrual Complaint: Amenorrhoea (or) Oligomenorrhoea

B/c 1st phase is Endometrial → Menorrhagia (just like any infection)

Findings: P/V: M/C finding in reproductive age: Normal findings.

Reproductive age women & adolescent girls

B/L adnexal masses

Least common finding

Genital TB

M/C presents w/ B/L Adnexal Masses

Best Dx: Endometrial Bx (for Acid Fast Bacilli Culture)

Rx: ATT x 6 months

Would she conceive w/ ATT? → Yes (only in Mild Injury)

But Severe Injury: ATT can't help her: For Infertility: IVF.

ATT for: T.B.

Uterine Anomals

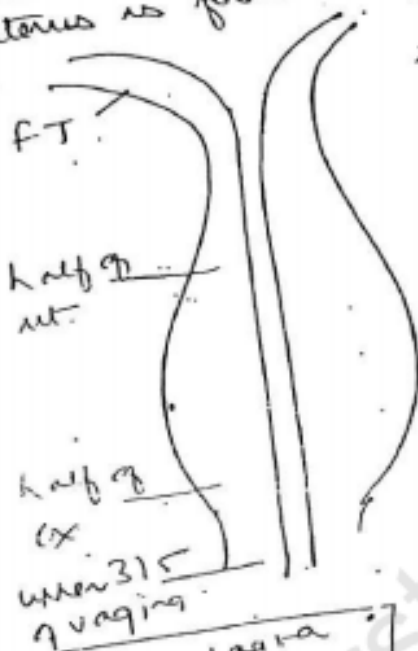
Can be due to congenital cause: Septate / Bicornuate uterus
Due to Fibroid

Important Points

uterus is formed by MD.

- > Both sides MD begin to fuse = 7-8 weeks
- > Fusion completes = 12 weeks
- > Septa fused
- > Septa Resolves by 18-20 weeks
- > Resolution of fusion begins from below upward direction.

Congenital Malformation



Lower 2/5 vagina develops from sinovaginal bulb on urogenital sinus

↓
only 1 side MD is int
↓
unicaminate uterus

↓
Both side MD int but fail to fuse
↓
uterus didelphys

FT = 2
uterus = 2
cx = 2
vagina = 2

↓
Both MD int, fuse but fusion is incomplete
↓
septa fails to rise
↓
Bicornuate uterus

Bicornuate uterus (Always have single vagina)

2 cx
Bivellus

1 cx
Univellus

MIC in uterine Malformation

MIC Malformation = Septate > Bicornuate (23)

MIC of w/ abortion/fertility = Septate

Best Reproductive outcome = Arcuate uterus > Septate
(Flat topped uterus)

Worst Reproductive outcome = Unicornuate

MC Tx for SX on uterine malformation - if it leads to ≥ 3 abortions

IOC = MRI
Gold standard. Laparoscopy

on NSG Distinguishing features of Septate & Bicornuate



Bicornuate



Septate



Angle = $> 60^\circ$

Distance b/w horns = > 4 cms.

Mgt - Hysteroscopic resection
SX



Angle $< 60^\circ$

Distance between horns < 4 cms.

Mgt - hysteroscopic
Resection of septum
Earlier - Tompkins
Jones
Metroplasty

(24)

Male Factor:

"HSA" - husband semen analysis

(178)

- Best way to obtain Semen for HSA: Masturbation.
- Done after Liquifaction of Semen.
- Max. time we 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^① (Don't do one sample)

WHO parameters: $PII > 7.2$

and ② values:

Volume > 1.5

Total count > 39 million/epididymis
(lower limit of ①)

Sperm concentration > 15 million/ml

Total motility $> 40\%$

Progressive motility (Forward) $> 32\%$

Morphology $> 4\%$

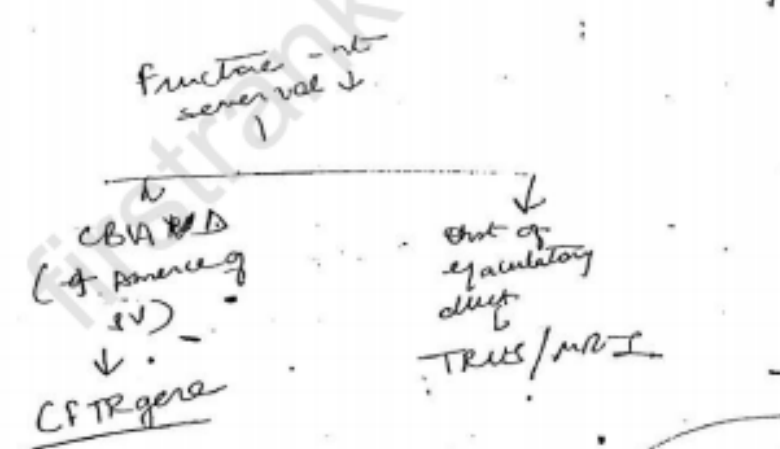
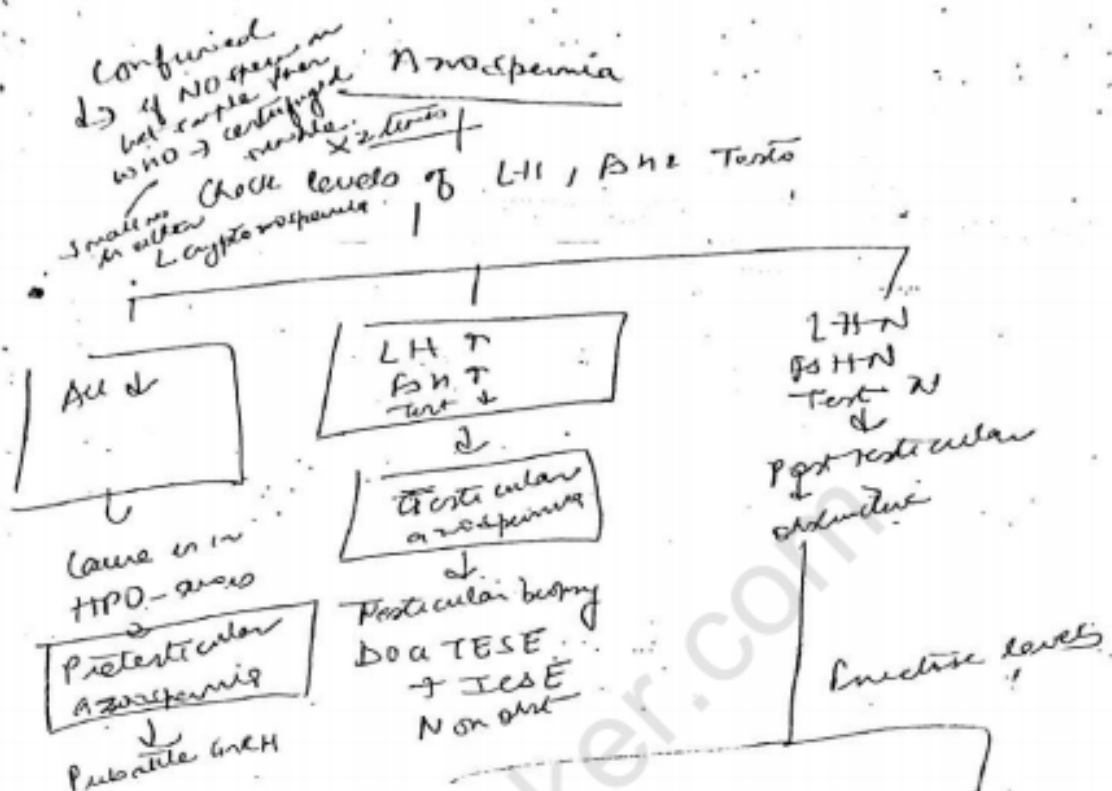
Vitality $> 58\%$

WBC count < 1 million/ml

(15)

avg count:

60-120 million/ejac



mg of Azoospermia

Varicocele - Prescribed & anastomosis
an other can - seen picture by TRUS post of TESE / PESA

↓

ICSI

On Is NOA - Retrive sperm from Testis only
NOT Sperms



Mat:-

Normal sperm count = 15 million/ml $\rightarrow$ IUI (26)

oligo spermia = IVF
(5-15 million)

< 5 million = IUI

In azoospermia = sperm are not in semen
Not in testes if cause is obstruction

In that case:-

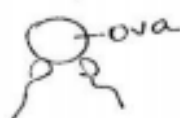
Sperm can be Retrieved from Testes using

1. MESA - Microsurgical epididymal sperm aspiration
2. PESA - Percutaneous " "
3. TESA - Percutaneous testicular " "
4. TESE - Testicular sperm extraction

and then IUI can be done

Partial Mole
Incidence in 695
Karyotype -

- Triploid ($69XX$ or $69XXY$)
- Dispermic



Hence extra Genetic material is paternal in origin

On 10% cases they are tetraploid or mosaic

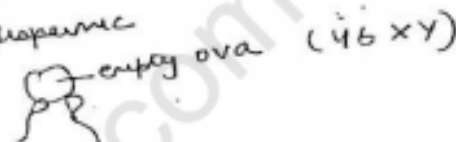
Complete Mole

In 1945

- Diploid ($46XX$)
- Empty ova Monospermic (90%)
- Sperm (duplicate)

hence all genetic material is paternal in origin

On 10% cases they are dispermic



All symptoms are less in partial mole.

Presenting P/V in features = bleeding
Ht low eaten
-nt

All symptoms more marked

- (Same)
- Preeclampsia (27%)
- Hyperemesis Gravidarum (25%)
- Thrombocytopenia (27%)
- Resp. distress due to embolization (2%)

P/A:
Ht of uterus will be = or <
POG.

Ht of uterus > POG
Singing consistency
Fns - nt
Fetal parts not palpable

<u>Partial Mole</u>	<u>Complete Mole</u>
IOC: - USG (TVS) USG picture similar to missed abortion	IOC - USG (TVS) Typical <u>SNOW STORM</u> appearance
βhCG levels - More than expected but not markedly raised	βhCG >105 mIU/ml
<u>Note</u> βhCG levels are used for followup in female after treatment	
<u>Gold standard for diagnosis</u> Histopathological examination <u>Findings</u> Hydropic degeneration - less marked Some fetal part -nt Trophoblastic swelling -nt Inclusion bodies -nt	more marked -nt -nt -nt
Theca lutein cyst -nt	BL present
Progression to GT Neoplasia 3-5%	15-20%
ChemoCA = <1%	4%



Management

- ① suction evacuation irrespective of gestational age
↓
followed by curettage
↓
Tissue sent for HPE (gold standard) ③

- ② If ♀ is more than 40 years & has completed family - hysterectomy is not done as it doesn't eliminate the need for follow up
excessive bleeding following evacuation → do hysterectomy.

- Other important points
- > Oxytocin drip should be started before evacuation as there is a risk of tumor embolization
 - > Canula size used = 10-12
 - > After procedure: Anti-D given to Rh-ve females
 - > Thela later, upto don't need any separate T/E, they regress spontaneously after 2 months of evacuation

Follow up :- on both partial & complete mole.
Measure BHC weekly
↓
Till get 3 Normal values
↓
Monthly till 6 months.

In Partial Mole, values of hcg come back to normal in 7 weeks.
In complete Mole values of hcg come back to normal in 9 weeks.

* Note
Pregnancy is 4I X 6 months in female.
contraception of choice = OCP's.

Prophylactic chemotherapy

To prevent chorio CA in female. patient is not given to all. It is given only in high risk patients.

1. Pt age ≥ 40 years.
2. hcg $\geq 10^5$ IU
3. Large uterine size.
4. B/L Thru litter cysts (≥ 6 cm)

DOC = Methotrexate

↓
If patient has Tandica then DOC = Actinomycin D

Recurrence rate after 1 molar pregnancy = 1% - 2%.

Gestational Trophoblastic Neoplasia

(5)

Diagnose

1. If any 1 of the following is present:-

1) 4 consecutive values of hcg which show plateau ($\leq 10\%$)

$\Delta 1, \Delta 7, \Delta 14, \Delta 21$

2) 3 consecutive rising values ($>10\%$)

$\Delta 1, \Delta 7, \Delta 14$

3.) On HPE:- chorion

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 thea luteal cyst.

Metastasis (lungs 80%
vagina 30%
pelvis 20%
brain 10% each)

CHORIOCARCINOMA

Chorio CA develops M/C after which type of pregnancy -
H-mole.

high risk chorio CA develops M/C after $\leq$ type
of pregnancy - Full Term pregnancy.

Q. M/C GTN which is seen after full term

pregnancy = chorio CA.

histological characteristics - Absence of villi pattern

• Extensive proliferation of cytotrophoblast and syncytiotrophoblast with haemorrhage and necrosis

Metastasis

M/C site lungs : on x-ray

M/C finding - cannon ball appearance

• Metastasis of lungs means - stage 3 of chorio CA.

2-d M/C finding -

on vagina : M/C site of metastasis is suburethral

stain appears

• Metastasis of vagina means - stage 2 of chorio CA.

Modified WHO scoring for chorio CA

Low risk (subur)

• Age

≤ 39 years

• hcg

< 103

• Type of antecedent pregnancy

Molar

• Duration of antecedent pregnancy

< 4 months

• No. of metastasis

< 4

• Site of metastasis

Lungs

• Size of Tm

< 3 cm

• Previous h/o chorio CA

single agent

high risk (subur)

≥ 40 years

> 105

Full term

> 8 months

> 8

liver, brain

> 5 cm

Multiagent

Management

of who score is ≤ 6 :- Low risk chorio CA ⑦

↓
single agent chemotherapy

↓
Methotrexate (single agent)

Follow up x 1 year

of who score is ≥ 7 :- high risk chorio CA

↓

Multi drug chemotherapy

E = Etoposide

M = Methotrexate

A = Actinomycin

C = Cyclophosphamide

O = Oncovin

called as

Bagevan's

Regime

Follow up x 2 years

Other important points

Figo staging

St. 1

choriocarcinoma limited to ~~uterus~~ uterus & vagina.

St. 2

St. 3

lung mets

St. 4

other mets like liver and Brain

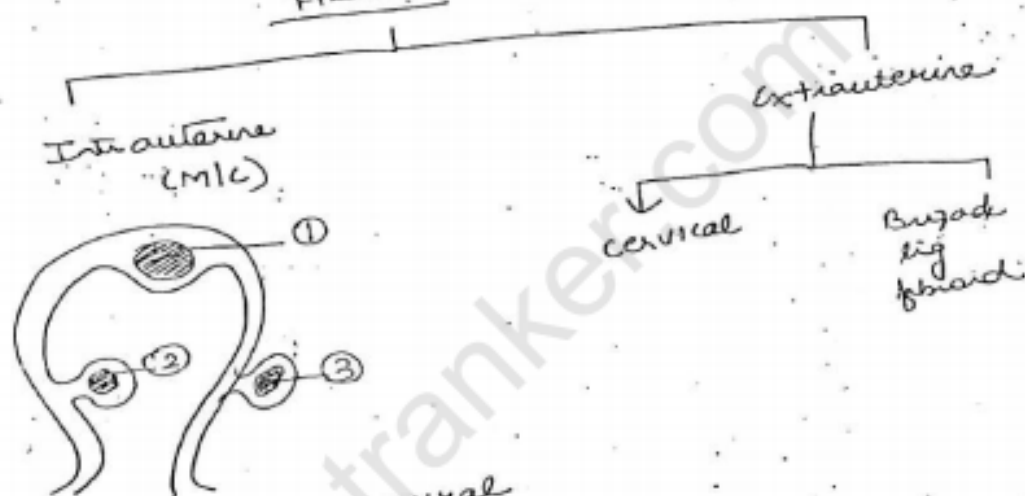
⇒ PSTT = It occurs commonly of Normal pregnancy
tumour marker in PSTT is hPL and Human placental
alkaline phosphatase = It is chemoresistant tumour
Hence the Tx is hysterectomy
• management of Invasive mole is hysterectomy

⇒ PS2 kit 2 is a gene which is maternally expressed

-	diploid	Complete mole
+	triploid	Partial mole
+	displaid	abortion

Fibroid

- > Most pelvic Tm in female
- > Most solid benign Tm in female
- ↳ Estrogen + Progesterone dependant condition
 - ↳ causes proliferation
 - ↳ prevents apoptosis
- Hence Fibroid
- MIC in Nulliparous ♀, Smoking is protective
- Fibroid



1 = Interstitial / Intramural

2 = Submucous :-

3 types

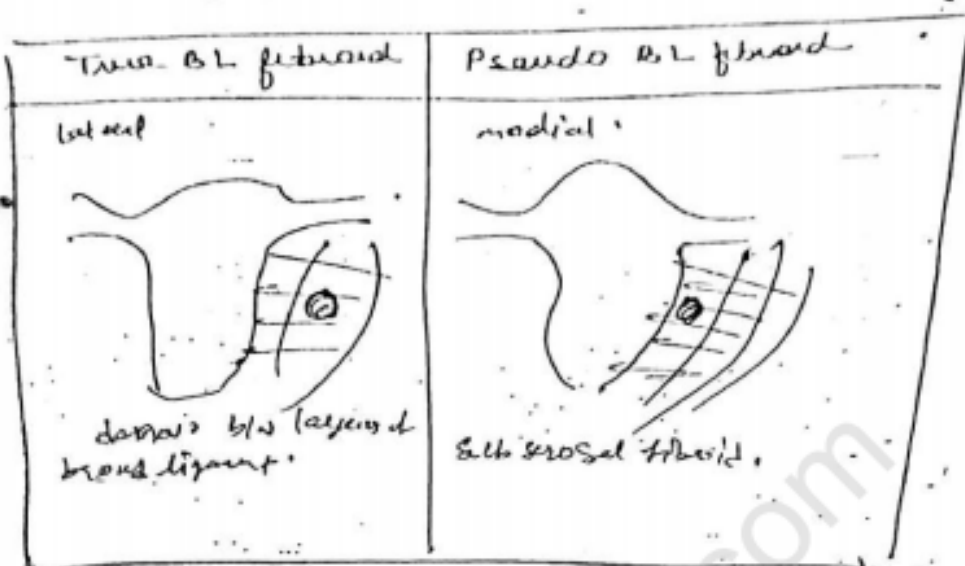
Type 0 :- Fibroid entirely intra-cavitary } can be removed hysteroscopically

Type 1 = > 50% intra-cavitary

Type 2 = < 50% " (> 50% in the myometrium)

3 = Subserosal

Broad ligament fibroid (bke)



Important points

Age of fibroid = 35-45 years

MIC in Nulliparous ♀

MIC presentation in fibroid = Asymptomatic

MIC symptom - Menorrhagia

(Menorrhagia seen in fibroid polyp)

To begin with all fibroids are - Intramural

MIC variety of uterine fibroid -

Fibroid ⊆ leads to maxm symptoms - submucosal

" " has maxm chances of abortion -

" " ⊆ has maxm chances of undergoing malignancy - submucosal fibroid

MIC extrauterine fibroid - cervical fibroid.

Fibroid with maxm urinary symptoms - cervical fibroid

Ant-Cx
↑ urinary
freq

Post-Cx
↓ urinary
Retention



Pain in fibroid

(3)

- It is never chief complain
- Dysmenorrhea can be seen
- Dyspareunia never seen
- Pain can be due to Tension

↓
MIC in

subserosal
pedunculated
fibroid

Degenerations :-

- ① Fibroid is surrounded by a pseudo capsule.
Blood vessels supplying the fibroid are seen in pseudo capsule, hence centre of fibroid is least vascular. degeneration begins from centre.

- ② MIC degeneration - hyaline degeneration.
Least common - sarcomatous change. (i.e. malignancy).

- ③ seen in <0.5% cases.
usually seen in postmenopausal female.
• fibroid ↑ in size + pain
• ≥ 10 metres / HPF

- ④ Calcification - begins from periphery as it is the most vascular part.
seen in subserosal fibroid
↓ X Ray
popcorn kernel calcification

Red Degeneration

- Red Degeneration
- > seen during pregnancy (MLC in 2nd trimester)
 - > pt. of acute abdomen in pain + Nausea + vomiting.
 - = embolism in blood vessels

Thrombosis in blood vessels
supplying fibroid
↓
Aseptic necrosis

Rever + secondary
reaction
caused by iron and
calcium

→ Mgt: conservative

510 Rate of

- Antineoplastics
- Termination of pregnancy
- Myomectomy

20/02/2020

Pregnancy complications of fibroid.

- Pregnancy Complications
1. Recurrent pregnancy loss - MLC by submucosal fibroid
 2. Prolonged labor
 3. Malpresentation
 4. Abruptio
 5. Abn Labor
 6. PPH
- submucosal

(16) PPH
fibroid can cause infertility @ CMIC = submucosal fibroid

IOC: - USG

prolonged in the uterus,

Fibroid	Adenomyosis
- cholesterol - - microcalcifications - uterus non tender - size of uterus = 16-20cm - irregular contour of uter	- menorrhagia & dysmenorrhea - tender uterus - Halban sign - not > 10-12 cm - diffuse symmetrical enlargement of uterus - capsule continuous & symmetrical thickening
IOC = USG	IOC → MRI gold standard → Histopath exam

USG appearance of Adenomyosis

1. salt & pepper appearance
 2. myometrial cyst
 3. subendometrial cyst
 4. junction blind appearance
- Thinning of junctional zone ≥ 3mm

Management of Fibroid

Medical Mgt

- Drugs with progestin synthesis don't ↓ size & blood
- Drugs → OCPs (low dose)
- Levonorgestrel IUD - uterine ballast
- Progestin only pill

Drugs ↓ size of fibroid

- GnRH agonist (continuous)
- ↓ estrogen = menopausal
- symptoms = all back up to therapy
- Small (2 + P)
- GnRH antagonist
- Uterine artery embolization
- Uterine artery
- Progestin inhibition

Drugs which ↓ blood
loss but not size of
fibroid

Drugs which ↓ size of
fibroid

surgery

conservative sx
↓
myomectomy

Radical sx
Hysterectomy

Hysteroscopy
(Morcellation)

Type 0,
Type I
submucous fibroid
≤ 5 cms

Laparoscopic

Peritoneally

- Resect all fibroids
- Laparoscopic better
in all respect except
it can lead to
higher recurrence
rate

Complications
Hwd. Retention
Bleeding
Infection
uterine perforation



Non Invasive method to treat Fibroid

1. Uterine artery embolisation
2. Magnetic Resonance guided high intensity focused ultrasound (MRgHIFU).

These both are done in females who do not desire future fertility

Ulipristal :-

- select progesterone receptor modulator.
- main use emergency contraception.
- Brand - ELLA (Ethinodiol diacetate).
- more effective than Levonorgestrel.
- also ↓ size & bleeding of fibroid size.

VE → Abt pain

- 2% of women want to conceive after myomectomy.
- fertility complete.
- infertility + Fibroid.
- recurrent bleeding after Fibroid.

Myomectomy is done
Myomectomy is done

