

25/4/18

OBS & GYNAE

①

RELEVANT ANATOMY

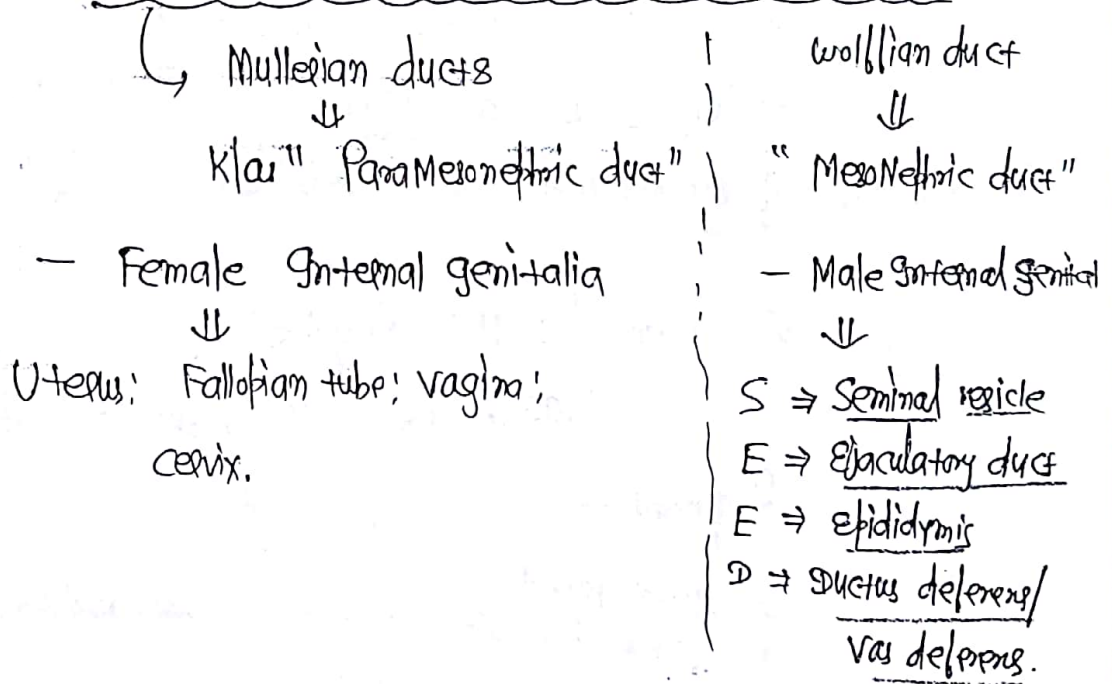
GONADS

Internal genitalia

External genitalia

Internal Genitalia ⇒ Uterus; cervix; Fallopian tube; vagina

embryological structure from which it develops ⇒



* Both the ducts appear in every foetus @ 6 weeks.

* Mullerian duct is lateral to Wolffian duct,
↳ either ♂ or ♀

* Mullerian disappears @ 9 weeks / Wolffian disappears in ♀.

Mullerian ducts

Disappear ⇒ In Male

Why?? ↓
Testis

MIS (Mullerian Inhibiting Substance)

↳ * ipsilateral
↳ Released by "Sertoli cells"
↳ Start producing @ 7 weeks

• Mullerian duct will persist in female b/c of Absence of MIS

* Estrogen is Not Required for development of uterus, vagina, Cx etc; for it absence of testosterone Required.

↳ Medial part of broad Ligament
• Paroophoron
• Epoophoron
• Gartner's duct
• Hydatid of Morgagni
↳ Lateral part of Broad Ligament
↳ Klay's organ of Rosenmuller's
↳ all contents of broad Ligament

wolffian duct

In Female

Why? b/c absence of testosterone

• wolffian duct is persist in Male child

↓
Testes
↓
Testosterone → Leydig cells @ 8 weeks
↓
cause development of wolffian duct

* Remnants of wolffian duct

In Female

i) Epoophoron - cranial Remnants of Mesonephric tubules
ii) Paroophoron - caudal Remnants of Mesonephric tubules
iii) Gartner's duct - caudal Remnants of Mesonephric ducts

* Gartner's duct Sometimes form a cyst in the vagina

* Gartner's cyst ⇒ In Antero Lateral wall of vagina (2)

g/d ↓ B Bartholin's cyst ⇒ In Posterior Lateral wall of vagina

M/c cyst of vagina/vulva ⇒ Inclusion cyst

↓
Located @ Lateral wall.

* Remnant of Mullerian duct in Males ⇒

White { Appendix of testis = klas. "Hydatid of Morgagni".

Appendix of epididymis is Remnant of Wolffian duct.

* Mullerian duct ⇒ 2 in No.

↳ both ducts fuse & form uterus in 10 weeks

we can differentiate Internal genitalia @ 10 weeks

direction of Fusion — Fusion begins from centre then it moves cranially/caudally.

direction is from caudal to cranial.



* Initially Uterus is solid organ; Later cavity formation occurs @ 18-20 weeks.

by dissolution of a midline fibrous septum

Potential cavity

↳ to accommodate something

* if Fundus is convex upwards ⇒ No fusion abnormality

if Fundus is dipping ⇒ Fusion abnormality seen

* complete septate defect ⇒



* Incomplete septate defect ⇒



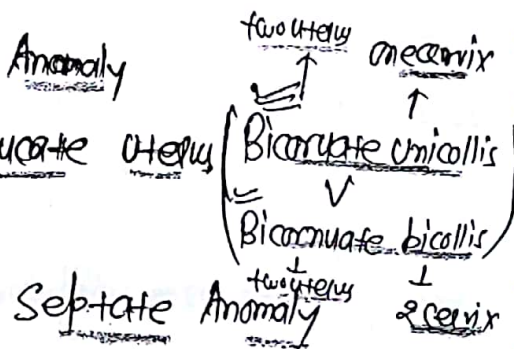
* M/c Mullerian Anomaly ⇒ Septate Anomaly

* 2nd M/c Mullerian Anomaly ⇒ Bicornuate uterus

* M/c Mullerian Anomaly also abortion

* M/c Mullerian Anomaly also infertility

* worst reproductive outcome



* In Diadelphous uterus both Mullerian duct form Anomaly (complete Lack of Fusion)



↳ complete failure of Fusion, vaginal septum

* Müllerian Anomaly = vaginal septum ⇒ Didelphus (3)

* Bicornuate Uterus has good Reproductive outcome

↳ What pregnancy complication: ↳ Pre-term Labour
Most Likely to do Abortion

* Didelphus Uterus also has good Reproductive outcome

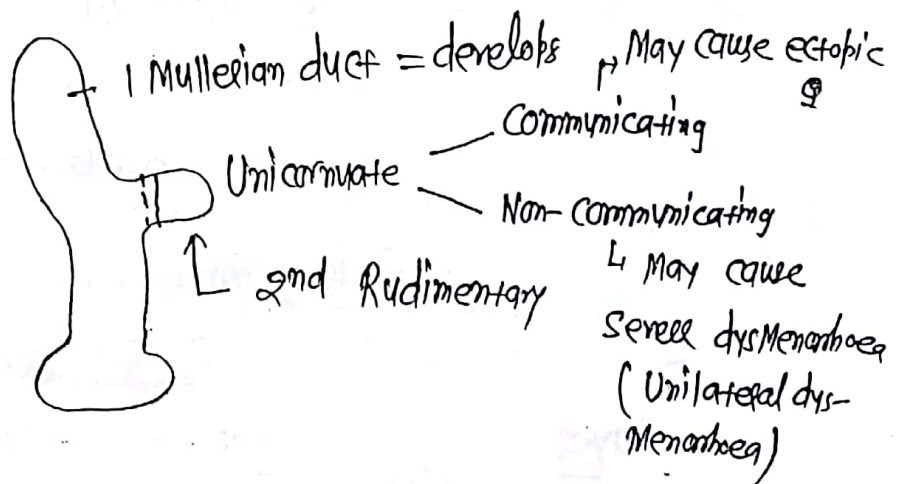
↳ What pregnancy complication ↳ Pre-term Labour
Most Likely to do

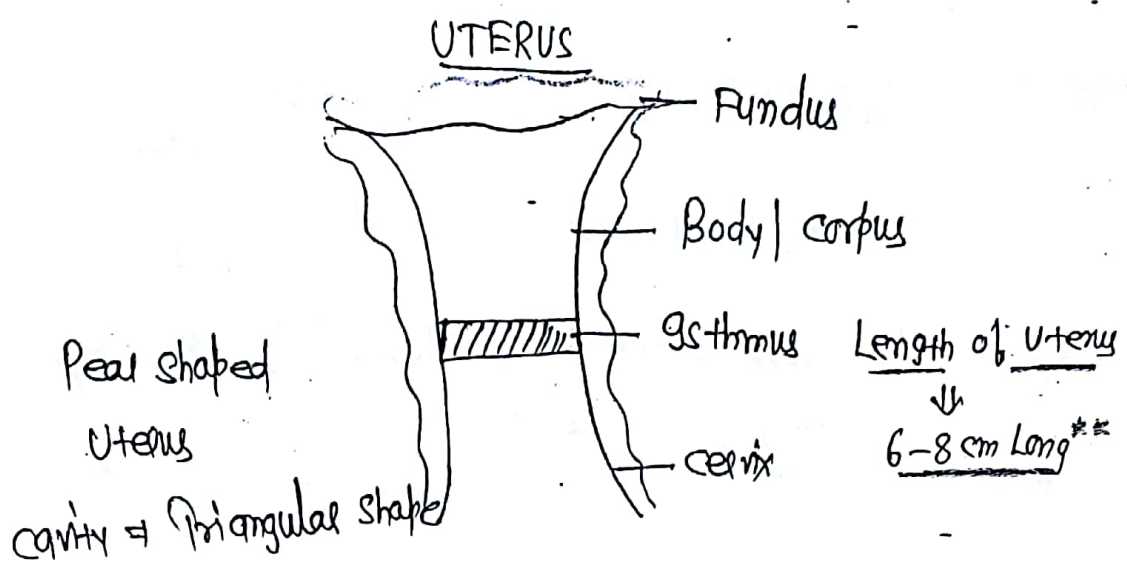
* Usually Corrective Surgery is Not Required

↳ if we want then do Unification Surgery

⇓
"STARSMAN METROPLASIA"

* Unicornuate Uterus ↳





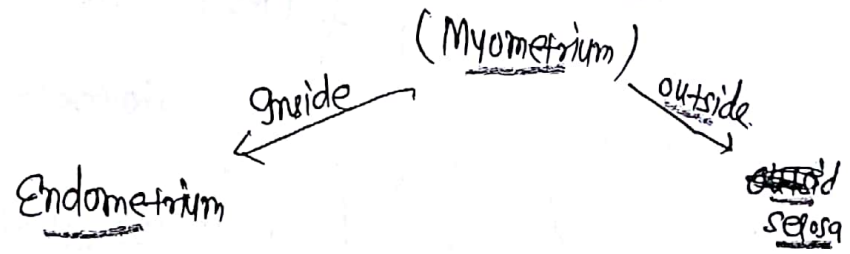
Wt. → Multiparous (80 gm)
 Nulliparous (50-70 gm)
 In pregnancy (term) = 1000 gm (1.1 kg)
 1 kg

} dt + hypertrophy
 ↓
 hyperplasia

volume of Non-pregnant = 10ml } dt + hypertrophy >> hyperplasia
 volume of pregnant = 5L

Q8 Weight of Uterus six week after Postpartum = 80 gm

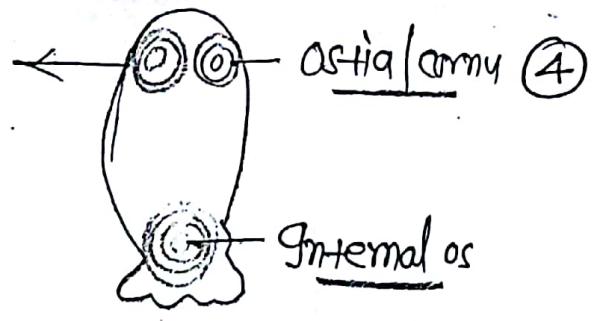
* Body ⇒ Is Made up of Smooth Muscle



- Myometrium = 2-2.5 cm thick

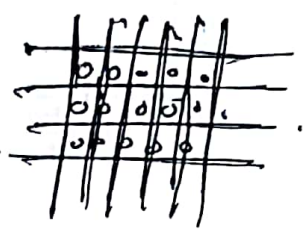
3 Layer ⇒ Inner - circular muscle
 Middle - criss-cross
 Outer - Longitudinal

Here Inner Muscle
works as sphincter



⇒ Middle Layer - cross-cross

↓
work as Living Ligature **



⇒ Endometrium

- ↳ Gland + stroma
 - ↳ Simple tubular
 - ↳ Single Layer columnal epithelium
cilia pres. only Need gland opening
- ↳ Superficial Layer = Functional ⇒ Shed off every Month
In Menses

Basal Layer = Not Shed off

- ↳ function = Regeneration
- ↳ so thickness of endometrium changes

Just after <u>Menses</u> (D5) =	Thickness
	<u>0.5mm</u>
<u>Periovulatory</u> =	<u>2-3mm</u>
<u>secretory</u> =	<u>5-6mm</u>
<u>Implantation</u> =	<u>10-12mm</u>

vigorous curettage ⇒

damage the basal layer



Result in Intrauterine Adhesion



Result in Asherman's Syndrome

Asherman's Syndrome

Highest Risk In to Manage

Post partum hemorrhage

the Post-partum hemorrhage

Pt. is Amenorrhoea

Infertility

Pt. is with Amenorrhoea infertility

⇒ outside part of Myometrium = Serosa

Anteriorly Loose fold of Peritoneum ⇒ Uterovesical fold

Posteriorly Loose fold of Peritoneum ⇒ Rectouterine Pouch

* At what level uterus opens -
into cervix! → Internal os

Anatomical (Above)

Histological (Below)

0.5cm

⇓
constriction

⇓
Kla "Gsthimus"

* Gsthimus is part b/w Anatomical & Histological Internal os.

* Gsthimus — stretch — LUS

↳ Lower uterine segment

In term LUS ... Gsthimus + cervix ; cervix comes above via

(70%) (30%) cervical abatement (taking up of cervix)

cervical effacement

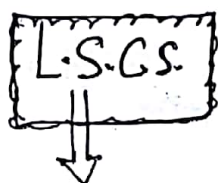
(5)

↳ Shortening + Softening

* At term LUS \Rightarrow 5cm

In complete (Manual Labour) LUS = 10cm

* LUS shortly forming after 1st Primipara



At Lower segment

How to identify?

by
Pari-toneal

Loose fold



Most common incision \Rightarrow Low Transverse Incision



Kerr's incision

Shape \Rightarrow Transverse or Pfannenstiel

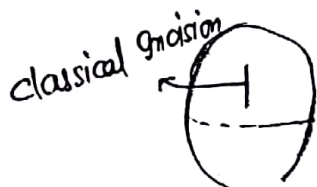
Klas "Kerr's Incision"



Kronig's incision

Low vertical incision

Klas "Kronig's Incision"



classical incision

classical incision

↳ Klas "classical incision"

Risk of Rupture
~~Risk of Rupture~~

* Low transverse incision \Rightarrow 0.2-0.9%

* Low vertical incision \Rightarrow 1-7%

* Classical incision \Rightarrow 4-9%
↳ weakest incision

* ~~⑤~~ classical incision [scar is already given

↳ it is Absolute indication for Repeat caesarean section

but Not an indication for Repeat classical incision

* Indication of classical \Rightarrow ① ⊕ Caes;

② Dense Adhesion b/w bladder & Uterus (May injured bladder)

③ Repaired vagino-vesical fistula

④ Past Martem C.S.

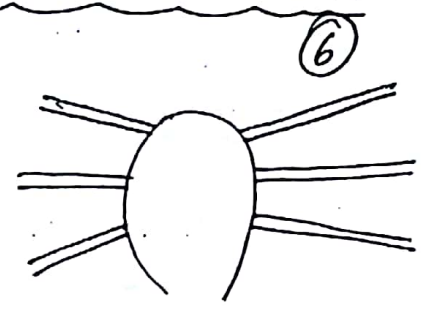
N.B. Anterior Located Placenta previa (only if a Not trained enough doctor)

↳ So; for Anterior placenta previa \Rightarrow LSCS do

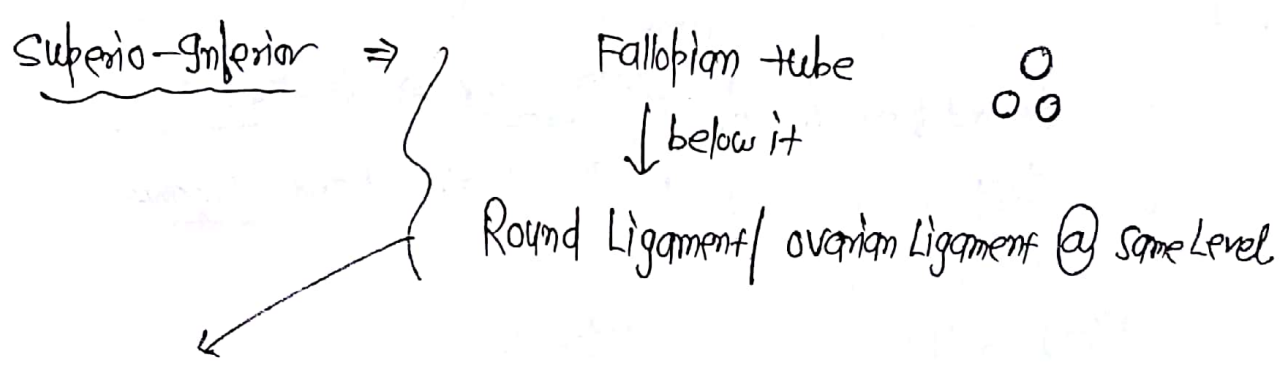
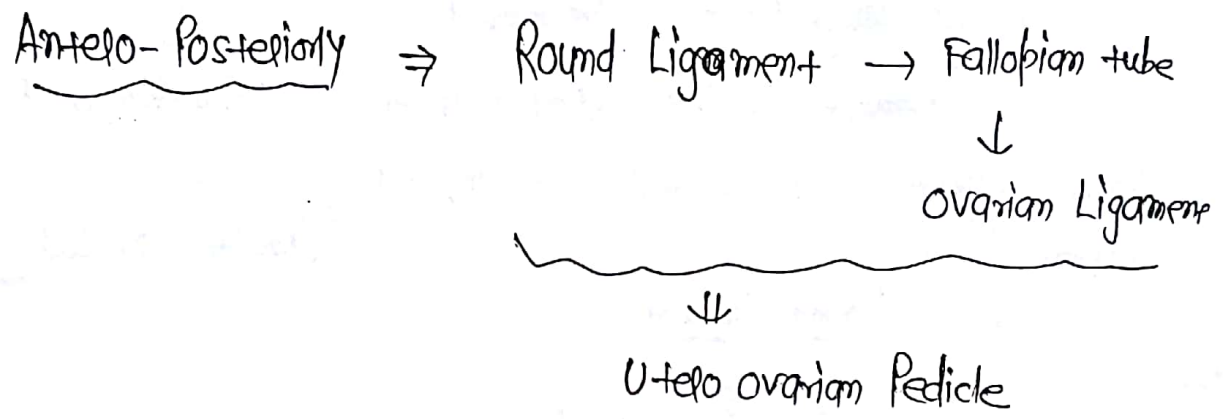
* on other side of uterus 3 structures are attached

Mnemonic
↓
RMO

- Round Ligament
- Fallopian Tube
- Ovarian Ligament



Adnexa ⇒ Fallopian tube + ovary



They assist you in tubal Ligation Surgery

↳ M/c cause of failure of tubal Ligation Sx ⇒ Ligation of wrong structure

FUNGUS \Rightarrow Part of uterus lies above the attachment of Fallopian tube

Round Ligament Path \Rightarrow Upper Uterus \rightarrow deep Inguinal Ring

Pulling Uterus Anteriorly i.e. in Anteverted position.

\downarrow
Inguinal canal

\downarrow
Superficial Ring

\downarrow
Inverted on Labia Majora

* CANAL OF NUCC (\oplus in Fetus only) :-
Fold of Peritoneum in fetus that contains Round Ligament & extends into Inguinal canal.

* It carries Round Ligament.

* Round Ligament
Ovarian Ligament
 \downarrow
Not derived from Mullerian duct.

developed from Gubernaculum

\downarrow
Proximal \Rightarrow Ovarian Ligament

Distal \Rightarrow Round Ligament

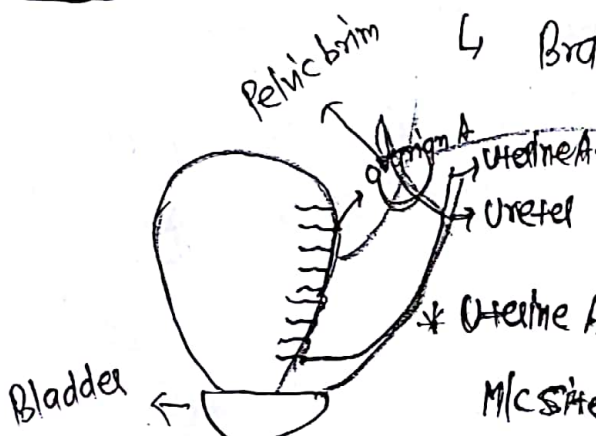
Blood supply \Rightarrow Uterine A.

\hookrightarrow Branch of Anterior division of Internal iliac Artery.

2nd M/C site of ovarian injury

"Water under bridge" \Rightarrow Danger Area
is crossing over the ureter

\downarrow
M/C site for ureteric injuries



* Location of "water under bridge" Area \Rightarrow 2 cm Lateral to cervix¹³
OR
1.5 cm Lateral to fornix,

* Ureter is Posterior to Ovarian & Uterine Artery; ⁽⁷⁾
but it is Anterior to Internal iliac A

\Rightarrow Branches of Uterine A \Rightarrow

U \Rightarrow Uterine A.

A \Rightarrow Arcuate branches \Rightarrow Supply outer 1/3rd of Myometrium

R \Rightarrow Radial branches \Rightarrow Supply inner 2/3rd of Myometrium

B \Rightarrow Basal \Rightarrow Supply Basal endometrium

S \Rightarrow Spiral \Rightarrow Supplies the Superficial / Functional endometrium

- Always do BL Ligation & do (a) the Level of Internal os.
- * Uterine A gives a special branch \Rightarrow Sampsons A \Rightarrow for Round Ligament
- Nerve supply \Rightarrow T₁₀, T₁₁, T₁₂, L₁

$\left\{ \begin{array}{l} \text{L Pain during uterine contraction} \\ \text{travels via this root} \\ \text{Pain Relay via } \Rightarrow \text{ "Frankenhauser ganglion."} \end{array} \right.$

We give "Labour Analgesia" (Level of block for vaginal delivery

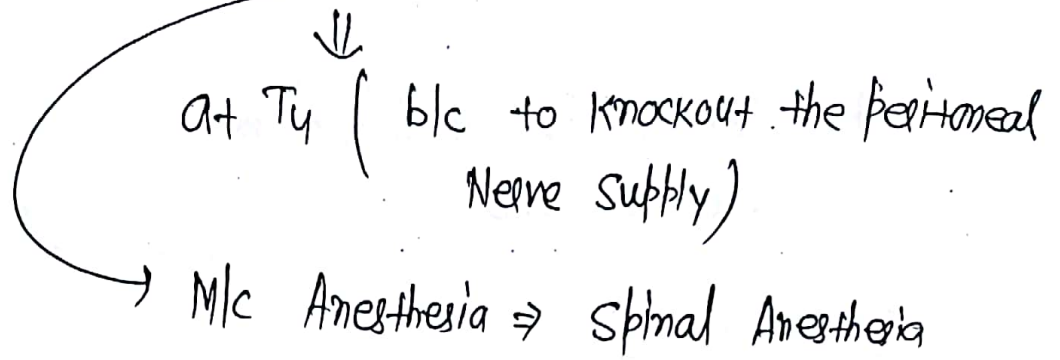
$\left\{ \begin{array}{l} \text{L via Epidural Anesthesia} \\ \text{give "Bupivacaine"} \end{array} \right.$

$\left\{ \begin{array}{l} \text{give "Bupivacaine"} \\ \text{L "0.125 - 0.25\%"} \end{array} \right.$

$\left\{ \begin{array}{l} \text{L "0.125 - 0.25\%"} \end{array} \right.$

$\left\{ \begin{array}{l} \text{L Sensory block; if given more} \\ \text{do motor block.} \end{array} \right.$

* Level of block for caesarean section



* Labour Analgesia may Prolong Labour (Active phase by 1hr)

↳ but doesn't ↑ Incidence of caesarean section,

* When we apply forceps (outlet/Low) - Pudendal N. Block

done only when complete
dilatation head @ +3.
to block if we

pierces Sacrospinous Ligament

↓
S₂, S₃, S₄

↓
Previously K/4

Direction of Needle (Posterior Medial) "Saddle block"

* Lymphatic Drainage ⇒ Internal iliac + Ext. iliac L.N

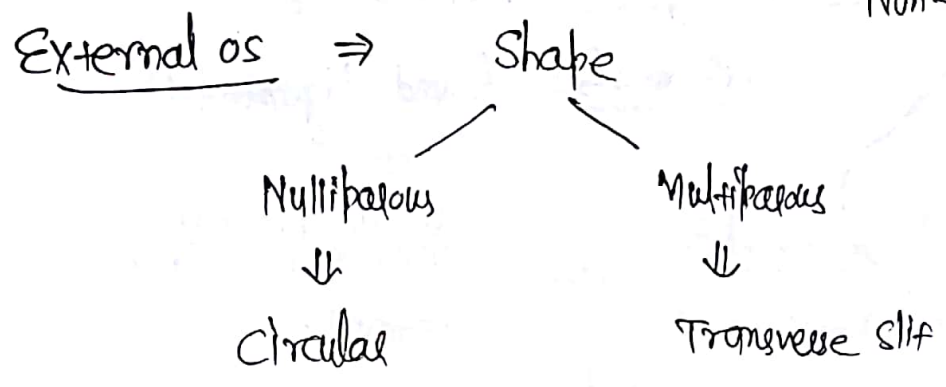
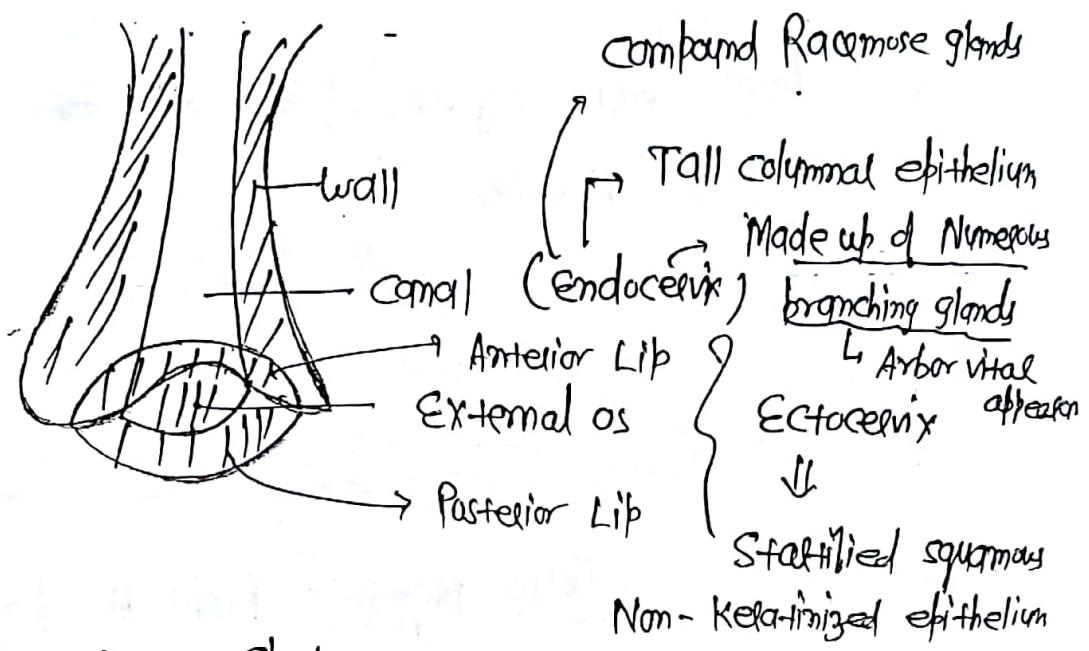
Fundus → Para-aortic L.N

ostia → Superficial Inguinal L.N

CERVIX ⇒ gt opens into vagina*

- Conical in shape
- 3cm Long
- cavity ⇒ spindle shape / fusiform

⑧
 ↓
 (a) external os**



- wall of cervix ⇒ Made up of connective tissue (collagen)

↓

10-15% Smooth Muscle

Result in effacement (there will be less in collagen & less in hyaluronic acid & less water content & less in dermatan sulfate)

Softening & tearing up ⇐

* Broad Ligament is a potential space; contains blood vessels etc.

* Angle b/w cervix & vagina \Rightarrow Anteversión
↓
90°.

* Angle b/w Long axis of body of uterus & cervix \Rightarrow Anteflexion
↓
120°
↳ @ Internal os

* In 80% women Anteverted & Antiflexed uterus ⊕
↓

Two Ligament Responsible for it
↳ Round Ligament
+
Uterosacral Ligament.



* If Fundus is More towards Bladder \Rightarrow Anteflexion

If Fundus is More towards Rectum \Rightarrow Retroflexion.

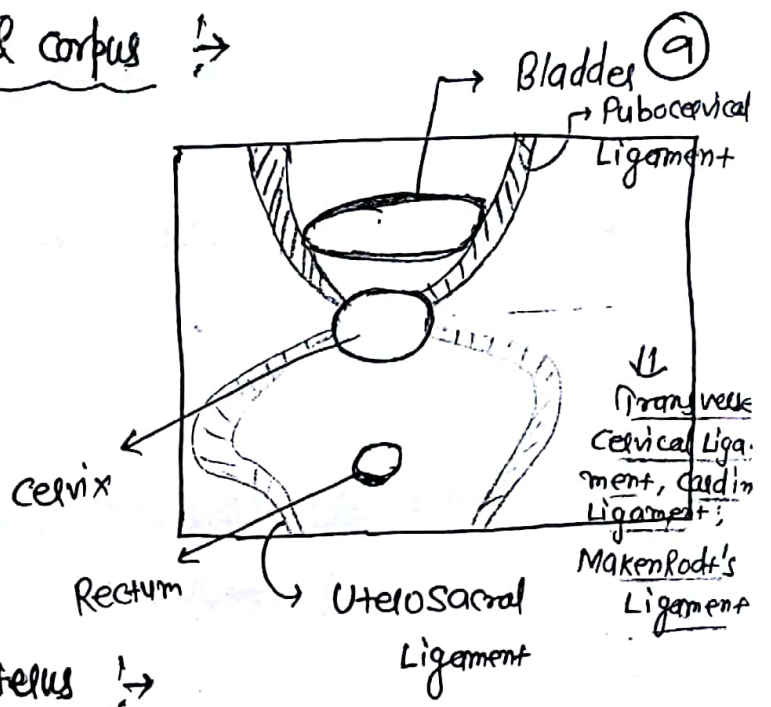
* In Plv examination \Rightarrow Which Lip you hitting 1st

Anterior Lip
↓
Anteversión

Posterior Lip
↓
Retroversión.

* Ratio of cervix & corpus →

- At birth = 1:1
- Prepubertal = 2:1
- after puberty = 1:2
- Reproductive life = 1:3
- Post Menopausal = 1:1



* Main Support of Uterus →

- Anterior ⇒ Pubocervical Ligament
- Posterior ⇒ Uterosacral Ligament
- Lateral ⇒ Transverse / cardinal / MackenRods Ligament
- Inferior ⇒ Levator Ani

Hammock (an) Radiate Ligament = Pubocervical Ligament
Uterosacral Ligament
Cardinal Ligament.

☹☹ All are Main Support except

↳ Round Ligament (support, but not main)

← Broad Ligament (Not a support)

↳ False Name

It is Nothing, but fold of Pelvic peritoneum

* Broad Ligament is a potential space; contains blood vessels etc.

* Angle b/w cervix & vagina \Rightarrow Anteversion
↓
90°.

* Angle b/w Long axis of body of Uterus & cervix \Rightarrow Anteflexion
↓
120°
↳ @ Internals

* In 80% women Anteverted & Antiflexed uterus ⊕
↓

Two Ligament Responsible for it

↳ Round Ligament

+

Uterosacral Ligament.



* If Fundus is More towards Bladder \Rightarrow Anteflexion

If Fundus is More towards Rectum \Rightarrow Retroflexion.

* In P/v examination \Rightarrow Which Lip you hitting 1st

Anterior Lip

↓

Anteversion

Posterior Lip

↓

Reproversion.

FALLOPIAN TUBE **

(10)

- Unfused part of Mullerian duct*
- Length = 10cm (10-12cm)*

* Part of Fallopian tube (Medial - Lateral) ⇒

- Intramural (Interstitial part) (1-2cm) = Narrowest part of Fallopian tube
↳ 0.7mm diameter
- Isthmus ; (3cm) = 1mm diameter

- Ampulla ; (5cm) ⇒ widest part of Fallopian tube = 6mm
- Infundibulum
↳ Fimbrial end



Site of fertilization (M/c site of ectopic ♀)

↓
b/c here fertilization takes place & dth Mucosal fold
① + ② Ampulla (k/w "plicae")

* M/c site of Ligation ⇒ Isthmus.

* Conceptus Remains in Fallopian tubes ⇒ 3 days

* Conceptus enter the uterine cavity on the 4th day.

↳ Post-fertilisation

* Anatomical Sphincter of Fallopian tube ⇒ Intramural part.

Physiological sphincter of FT ⇒ Isthmus

Q9 Main Reason for transport of conceptus

↳ Tubal peristalsis *

Anything that ↓ Tubal Motility Leads to "Ectopic @" ⁺⁺⁺

In Pelvic Inflammatory disease;
Tubal Surgery;
Progestelone only pills;

- epithelium of Fallopian tubes :-

↳ Single Layer ciliated columnal epithelium.

3 cells - Secretory

ciliated

Peg cells → Resting cells of Fallopian tubes.

* Direction of ciliary Mucle is towards Uterus.*

- Blood supply ⇒ dual blood supply *

Medial 2/3rd ⇒ Uterine A.

Lateral 1/3rd ⇒ Ovarian A.

↳ dilates 3 times in ♀
↳ site of Ligation for Management of PPV.

- Lymphatic drainage ⇒ Para-aortic Lymph Node*

(11)

Intramural + ostia

↳ Superficial Inguinal Lymph Node*

extra edge*

N. Supply ⇒

T₁₁ T₁₂ L₁

⇓

Pain Sensation from Unruptured ectopic (tubal stretch)

* Ectopic is vascular accident
↓
die dlt

VAGINA**

Embryological development →

Upper 1/3rd of va from Mullerian duct (Mesoderm)

Lower 2/3rd of vagina ⇒ from Urogenital sinus (Endoderm)

↳ from sinovaginal bulb

Hymen is Remnant of this ←

* Mucous Membrane of vagina ⇒ from Endoderm of urogenital sinus

* Muscles of vagina ⇒ from Mesoderm of Mullerian duct.

Vagina has four walls \Rightarrow Anterior
 ↓ Posterior
7-10cm Long Lateral - 2 in No.

• Posterior wall is longer than Anterior wall by 2cm*

• A-P wall opposed together

↳ after cut "H" shaped

* cervix comes inside vagina & space blw them klas



Recess = Fornix
 ↓
 Total 4 in No.

"Fornix"

↓

4 in No. (Ant, Post; 2 Lateral)

— Posterior fornix is deepest*

if More than 100ml collection in Pouch of Douglas ⊕

↳ it is significant**

Pouch of Douglas / Cul-de-sac \Rightarrow Culdocentesis

↓
 Rectouterine fold posterior
 to Vagina

↓
 if we get blood — which doesn't clot

↳ Hemoperitoneum.

→ used in Ruptured ectopic.

if blood clot \Rightarrow then it comes from blood vessels.

Colpotomy \Rightarrow Opening of the Pouch of Douglas to drain Pelvic Pus. (Abscess) (12)

Enterocele \Rightarrow Prolapse of bowel wall (Pouch of Douglas herni-ater)
 \hookrightarrow Upper 1/3rd of Posterior wall

Q9. Which of the following is Cystocele??

a) Upper 2/3rd of wall;

b) Lower 2/3rd of wall;

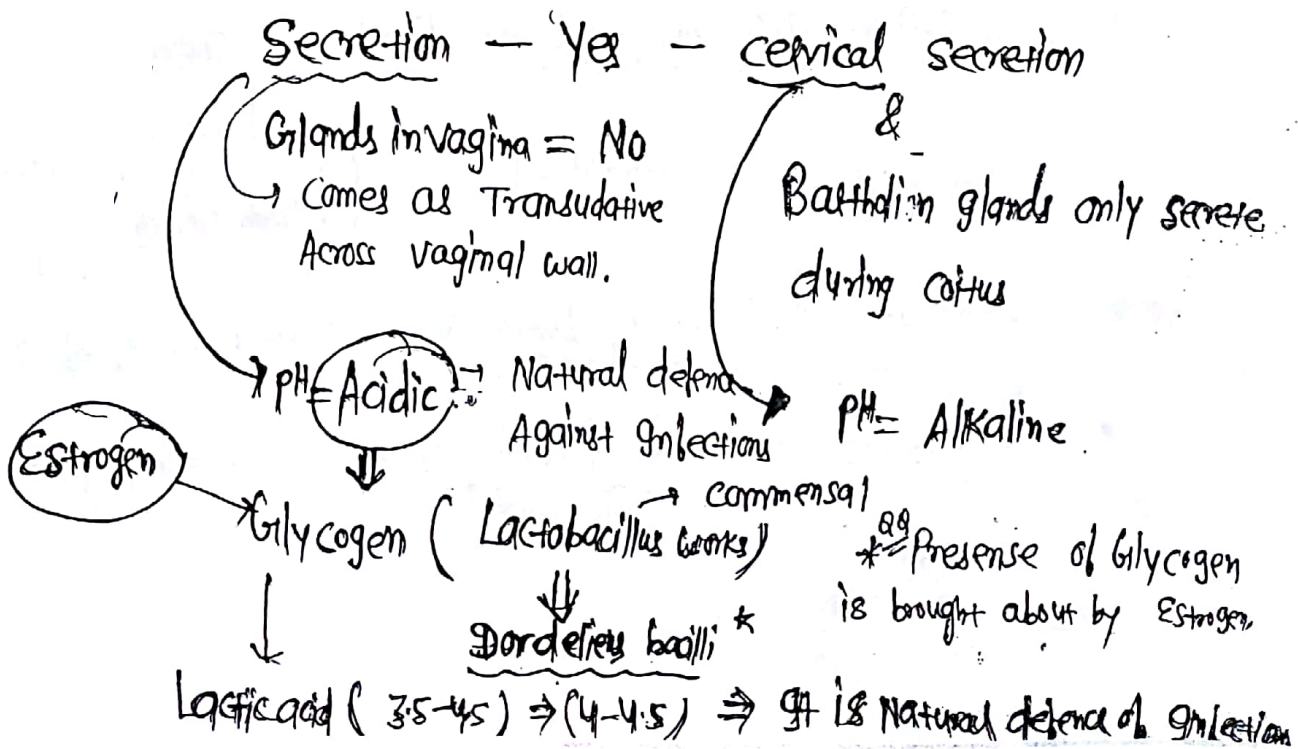
c) Upper 1/3rd of wall; * Rectocele \Rightarrow Prolapse into Middle 1/3rd of Posterior vaginal wall

d) Lower 1/3rd of wall \Rightarrow Urethrocele

\hookrightarrow Protrusion into Lower one-third of Anterior vaginal wall.

* \bar{c} the H₃-line passing through Pelvis, vagina forms \Rightarrow 45°

* Epithelium of vagina \Rightarrow Stratified Squamous Non-Keratinized



Q. Q.

Deficiency of 5 α Reductase \rightarrow

Male Pseudohermaphrodite (Genotype - Male
Phenotype - Female)

* by looking external genitalia we differentiate Male & female by 12 weeks.

*

	<u>Female</u>		<u>Male</u>
*	Bartholin's gland	<u>Homologous</u>	Bulbourethral gland (Cowper's gland)

*

		<u>Homologous</u>	
*	Glands of Skene (Para-urethral)		Prostate.

* Lymphatic drainage of clitoris \Rightarrow Superficial Inguinal Lymph Node

* Lymphatic drainage of Glans clitoris \Rightarrow Deep Inguinal Lymph Nodes
(Lymph Nodes of Cloquet)

* Lymphatic drainage of Labia Minora glands \Rightarrow Deep Inguinal LN.

* Lymphatic drainage of Labia Majora glands \Rightarrow Superficial Inguinal LN.

Vaginal part

Blood supply

Lymphatic drainage

Upper 1/3rd

Descending Uterine Artery

(13)
External + Internal iliac

Middle 1/3rd

Inferior vesicle Artery

Internal iliac

Lower 1/3rd

Middle Rectal Artery

Superficial inguinal

EXTERNAL GENITALIA

Genital tubercle → @ 6 weeks

Female

Male (Homologous structure in Male)

Clitoris

Penis

" Fold

L. Minora

Penile urethra

" Swelling

L. Majora

Scrotum

Absence of
DHT Require for
Female like organs.

Do convert in Male form

Testosterone

↓ 5 α -Reductase

Di-hydrotestosterone
↳ Pubertal Fold/Swelling.

99 Deficiency of 5 α Reductase \rightarrow

Male Pseudohermaphrodite (Genotype - Male
Phenotype - Female)

* by looking external genitalia we differentiate Male & female by 12 weeks.

* Female Bartholin's gland Homologous Male Bulbourethral gland (Cowper's gland)

* Glands of Skene (Para-urethral) Homologous Prostate.

* Lymphatic drainage of clitoris \Rightarrow Superficial Inguinal Lymph Node

* Lymphatic drainage of Glans clitoris \Rightarrow Deep Inguinal Lymph Nodes
(Lymph Nodes of Cloquet)

* Lymphatic drainage of Labia Minora glands \Rightarrow Deep Inguinal LN

* Lymphatic drainage of Labia Majora glands \Rightarrow Superficial Inguinal LN.

VESTIBULE

⇒ Anteriorly ⇒ Clitoris

(14)

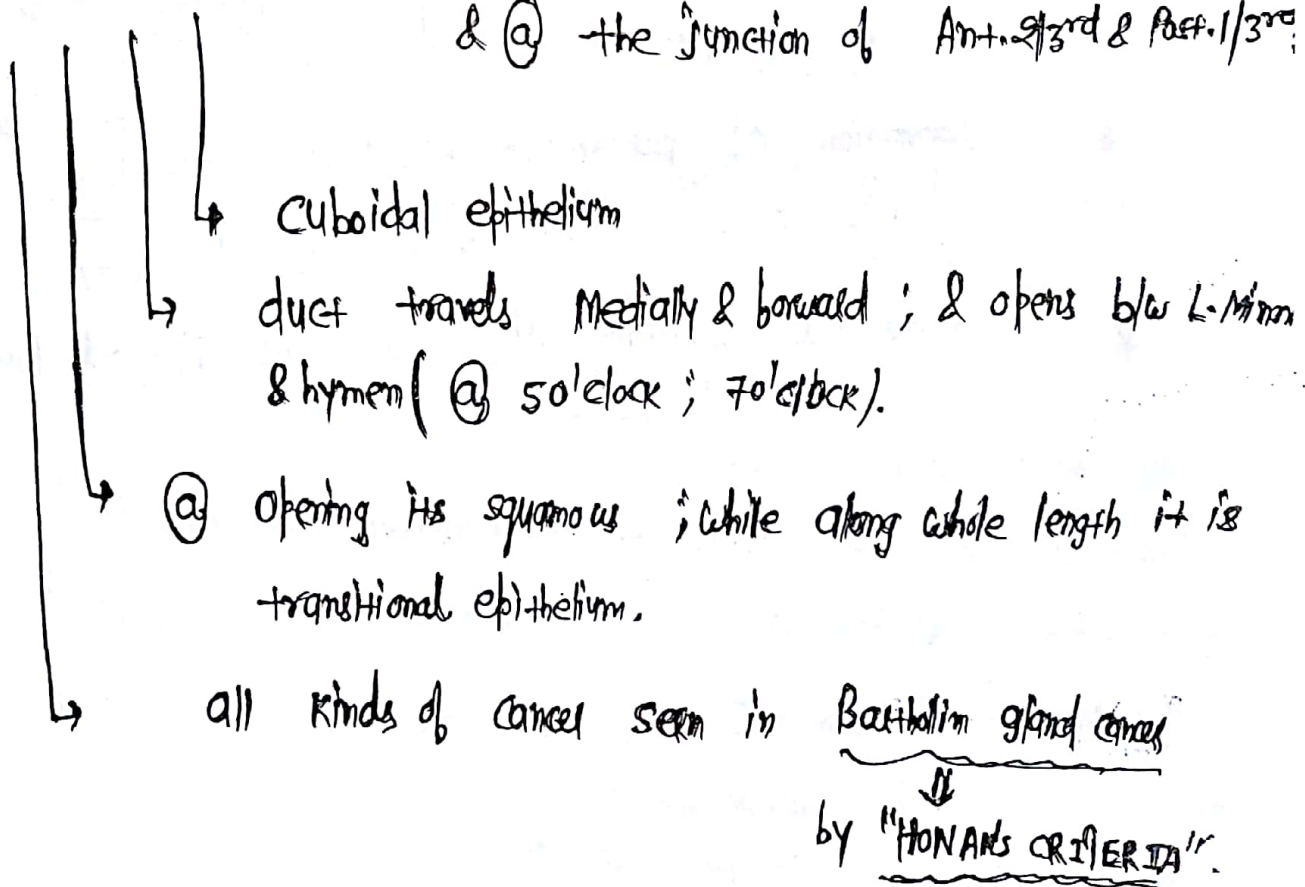
Posteriorly ⇒ Fourchette

Lateral boundary ⇒ Harts line

⇒ Structures opening in vestibule ⇒ Urethral opening;
Vaginal opening;
& Bartholin's gland opening;

* Bartholin's gland cyst ⇒ Pea size;

Bartholin's gland ⇒ Located b/w L. Minora & L. Majora groove
& @ the junction of Ant. 2/3rd & Post. 1/3rd



i) Asymptomatic Bartholin's cyst $\oplus \Rightarrow$ No Rx*

ii) Symptomatic / Recurrent cyst $\oplus \Rightarrow$ Marsupialization

↓
Exteriorization of the duct to prevent recurrence.

iii) ≥ 40 yr & have Bartholin's cyst $\oplus \Rightarrow$ Excision
↓
Tes Ca Risk*

Bartholin's Abscess \Rightarrow i) E. coli > Gonorrhoea;

ii) Initial Tx = I&D



Marsupialization on later date

* Secretion of Bartholin's gland \Rightarrow Alkaline; \rightarrow Released during coitus
b/c Acidic environment is spermicidal.

* they are content of Superficial Perineal Pouch;

RELEVANT EMBRYOLOGY

(15)

* Gonads 21 ⇒ develop by Genital Ridge - 5 weeks ⁹⁹

Ovary (8 weeks) *

Testis

• Absence of Y chromosome

• Y chromosome

In XO genotype ⇒ ovaries appear normally

• SRY functional part of Y chromosome

SRY ⇒ For testis

WNT4 ⇒ For ovary

AbN functional

① 6-7 weeks

Distal segment

b/c "XX" genotype of short arm
Require for function of Y-chromosome

Streak ovaries

↳ Seen in Turner Sx. *

if Nothing Mention;
differentiation b/w ♂ & ♀

① 12 weeks OF P.O.G.

* Gonads can be differentiated into ♂ & ♀ by 7 weeks

* Internal genitalia differentiates @ 10 weeks;

* External genitalia differentiates @ 12 weeks;

- Ovary part in "Ovarian Fossa"

↳ part in Lateral Pelvic wall

Posterior to ovarian fossa ⇒ Ureter; Internal iliac vessels;

Anterior to ovarian fossa ⇒ Obliterated Umbilical Ar.; Mesovarium

Lateral to ovarian fossa ⇒ Obturator Nerve & vessels

Pain from ovaries Related to Medial aspect of thigh b/c of Cutaneous br of Obturator Nerve.

Medial ⇒ Ovarian Ligament

Superior ⇒ External iliac vessels.

Inferior ⇒ Levator Ani

Ovaries ⇒ 3 Supports

→ ~~Ovarian Ligament~~ Ovarian ligament

→ ~~Infundibulo pelvic Ligament~~ Infundibulum pelvis ligament
(Suspensory Ligament)

- Ovarian vessels (gt carries ovarian A. & veins)

- to Lateral Pelvic wall (Attaches ovary to Lateral Pelvic wall)

- Cut - for oophorectomy

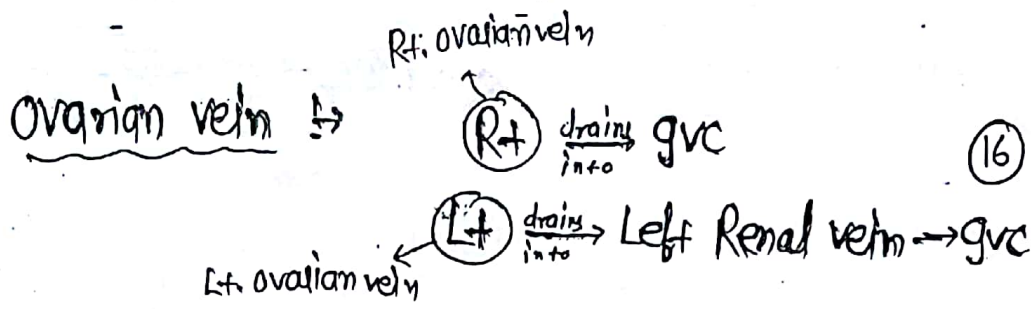
→ ~~Mesovarium~~ - ~~False Ligament~~ False Ligament
Mesovarium

↳ Fold of Peritoneum

to posterior leaf of Broad Ligament

OVARY ⇒ In Reproductive age Average 7-8 cc upto 20cc Normal
in Postmenopausal age average 2-3cc upto 10cc Normal

Blood supply ⇒ * Ovarian Artery ⇒ br of Abdominal Artery
@ L2



Varicocele \Rightarrow M/c on left side; b/c ~~it makes 90°~~ Make 90°

\hookrightarrow M/c Reversible cause of Male Infertility

\hookrightarrow ~~Left testicular vein joins @~~ Left renal vein

(Lymphatic drainage \Rightarrow Para-aortic group of \wedge Lymph Node)

Epithelium \Rightarrow Germinal epithelium

(Single layer cuboidal)

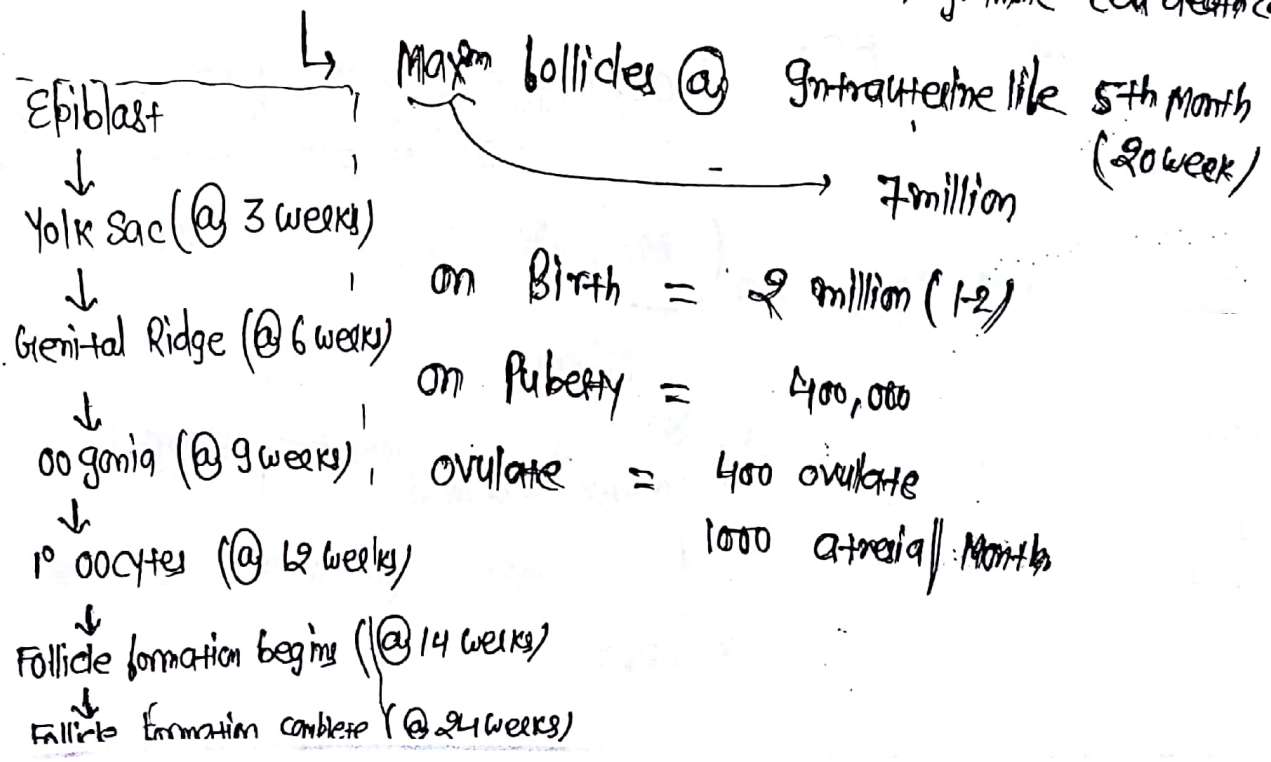
\hookrightarrow Germ cells are formed here

1° Germ cells (Primordial Germ cell)

\hookrightarrow Epiblast (Ectoderm) \Rightarrow older days from Yolk sacs

* Ovary has $\begin{matrix} \text{Cortex} - \text{Follicles} \\ \text{Medulla} - \text{vasculas} \end{matrix}$

* Menopause \Rightarrow Follicles goes into Programme cell death cause it



Follicle / oocyte

Folliculogenesis

Folliculogenesis

Oogenesis

Oogenesis



What is happening
in surrounding cells

What is happening
in Germ cells

1° Follicle - Flattened
Granulosa cell



1° Follicle - cuboidal
Granulosa cell.



2° Follicle - Theca cells
⊕



Antral Follicle - Cavity ⊕

* all follicles have 1° oocyte @ centre; but ovum have 2° oocyte.

* oogenesis begins IUL ⇒ @ 9 weeks

2n Oogonia

Mitosis

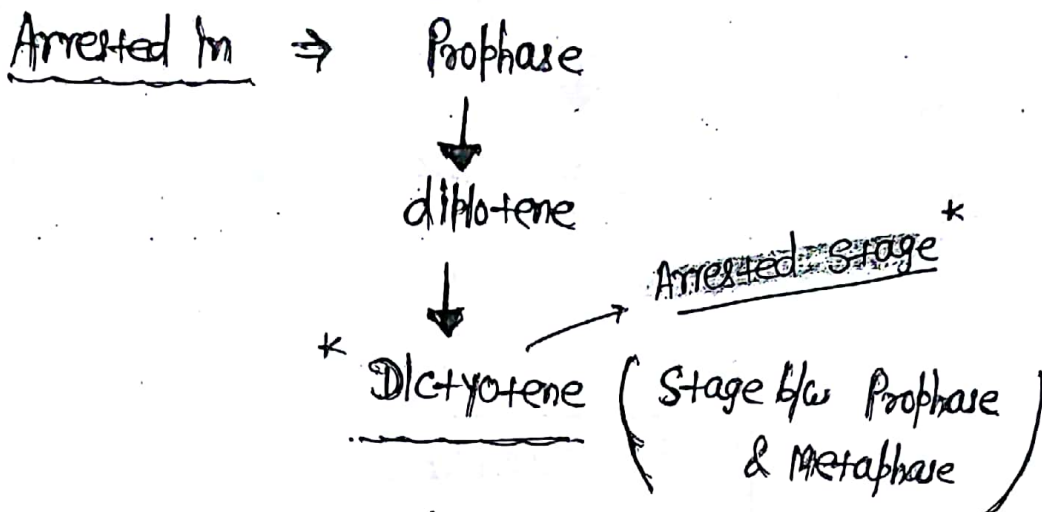
1° oocytes - diploid



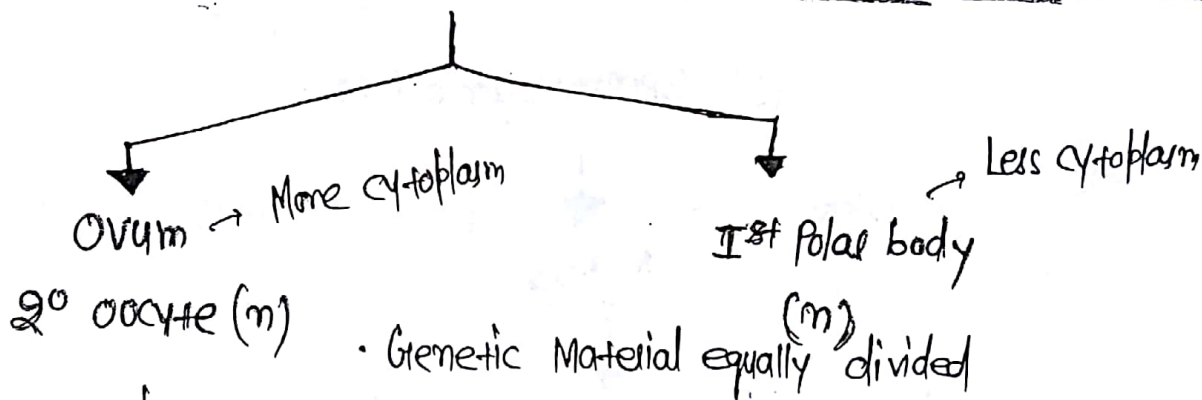
Meiosis I

(17)

Meiosis - I



When completed?? - just before ovulation
 @ ovulation (3-4 hours before ovulation)



Arrested - 21 - Metaphase*
 completed - A/b tel fertilization



Fig. Fertilized ovum*

* Life span of ovum = 24 hr.

* Size of Mature ovum = 120 μ m in diameter

Size of Mature follicle = 18-20mm in diameter

Size @ which follicle Rupture = 18-20mm *

SPERMATOGENESIS

* Begins — Puberty in seminiferous tubules.

* duration — 72 day

① Spermatogonia (2n)

↓
Mitosis

② 1^o Spermatocyte (2n)

Meiosis-I | Each (dictyotene absent) *

2^o Spermatocyte

2^o Spermatocyte

Meiosis-II

Meiosis-II

Spermatids

Spermatids

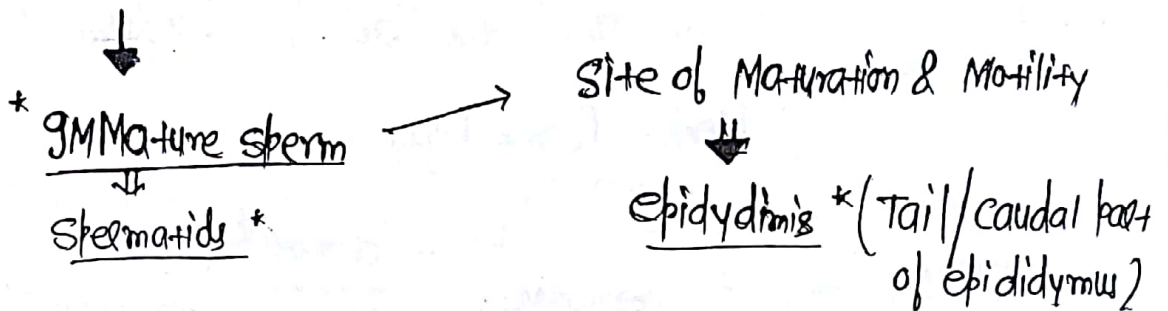
Spermatids

Spermatids

- * One 10^6 spermatocytes gives \Rightarrow 4 sperm ~~(=) spermatids.~~
- * one spermatogonia gives \Rightarrow 16 ~~10⁶ spermatocytes~~ = 64 sperm
- * Fertilizable Life span \Rightarrow 3 days; (18)
- * Mature sperm \Rightarrow ~~5.5 μ m in length (smaller than ovum)~~

* No. of sperm produced in one day = 100 million*
(Average sperm count)

* Spermiogenesis \Rightarrow 12-14 days



\Rightarrow Acrosomal cap \rightarrow Golgi apparatus

Head \rightarrow Nucleus

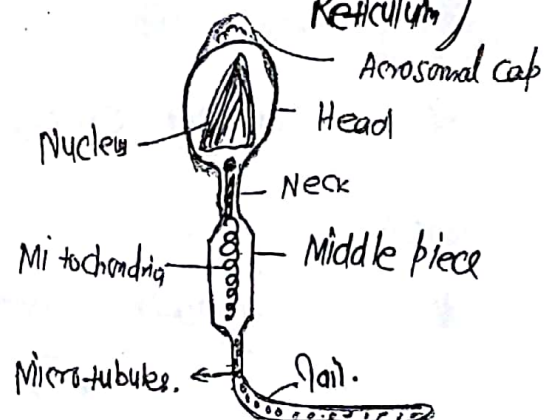
* Middle piece \rightarrow Mitochondria
 (energy house of sperm)

Tail \rightarrow Microtubules

Sperm don't have ^{aa} endoplasmic Reticulum (Rough endoplasmic Reticulum)

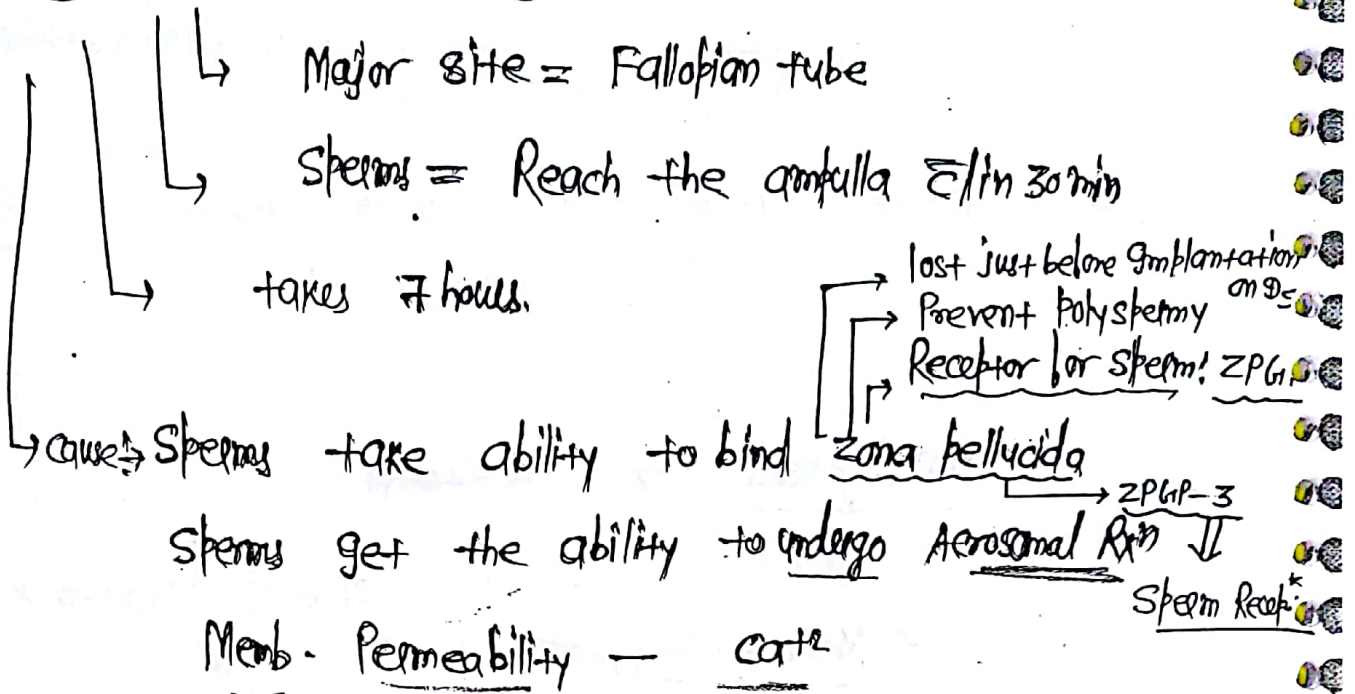
* Ion Responsible for Motility of sperm \Rightarrow Calcium ^{aa}

* Gene for Motility \Rightarrow CATSPER



FERTILIZATION

Capacitation → begins in cervix*



* Acrosomal Rxn ⇒ hypermotility
Main Enzyme! Hyaluronidase⁹⁸

* For all Embryological events ⇒ Days ⇒ from Fertilization
⇒ Weeks ⇒ from LMP (1st day)

- 3 Rules ⇒
- ① 28 day;
 - ② 14th day - ovulation
 - ③ days of ovulation = days of Fertilization

* 1st cleavage occurs ⇒ 20-30 hour after fertilization

* Conceptus enters in uterus ⇒ Morula stage @ 4 days
↓
16 cell stage > 8 cell stage

* Implantation occurs ⇒ in Blatocyst form
↳ on 96 begins (96-97) ↳ on 95 it form

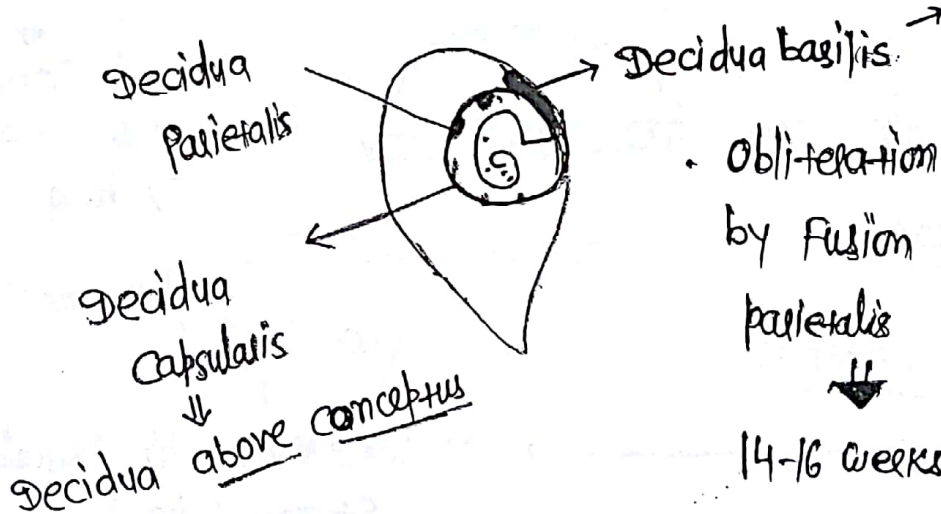
* Implantation occurs in 3 Phases ! →

- 1. Apposition → Selectins
- 2. Adhesion → Integrins =
- 3. Invasion → Matrix Metalloproteinase.

* Implantation completed @ D10.

* M/c site = upper posterior wall (Eccentric)
↳ one half bigger than other side

* Endometrium of ♀ → klas "decidua" *



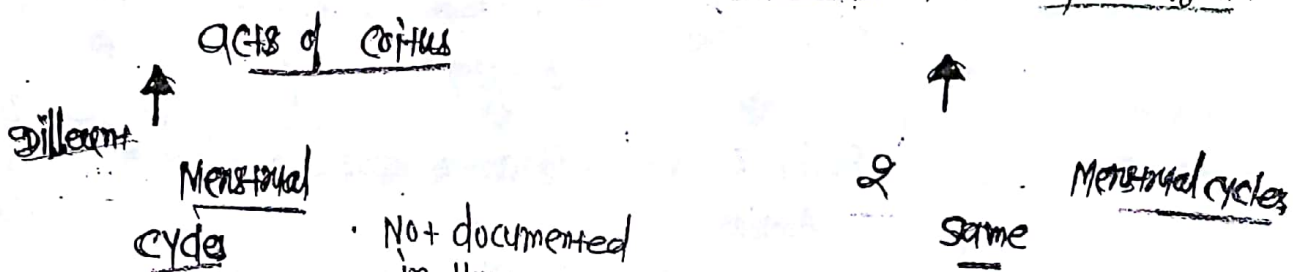
• Obliteration of uterine cavity by fusion of capsularis / parietalis

↓
14-16 weeks ** (After fusion klas "Decidua Vera")

SUPERFETATION

SUPERFECUNDATION *

- Fertilization of 2 ova by 2 different sperms by 2 different

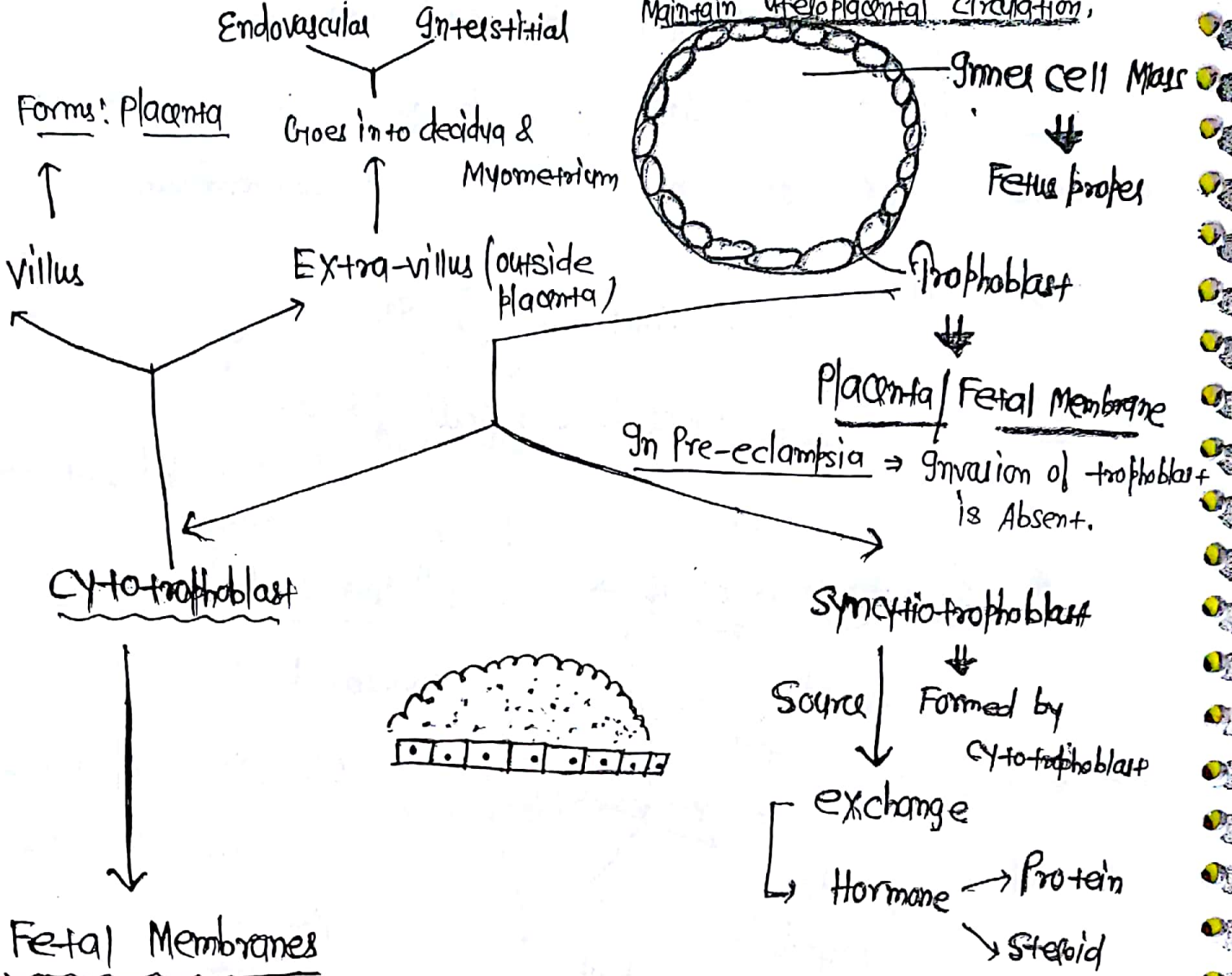


- Theoretically twinning can happen up to 16 weeks.

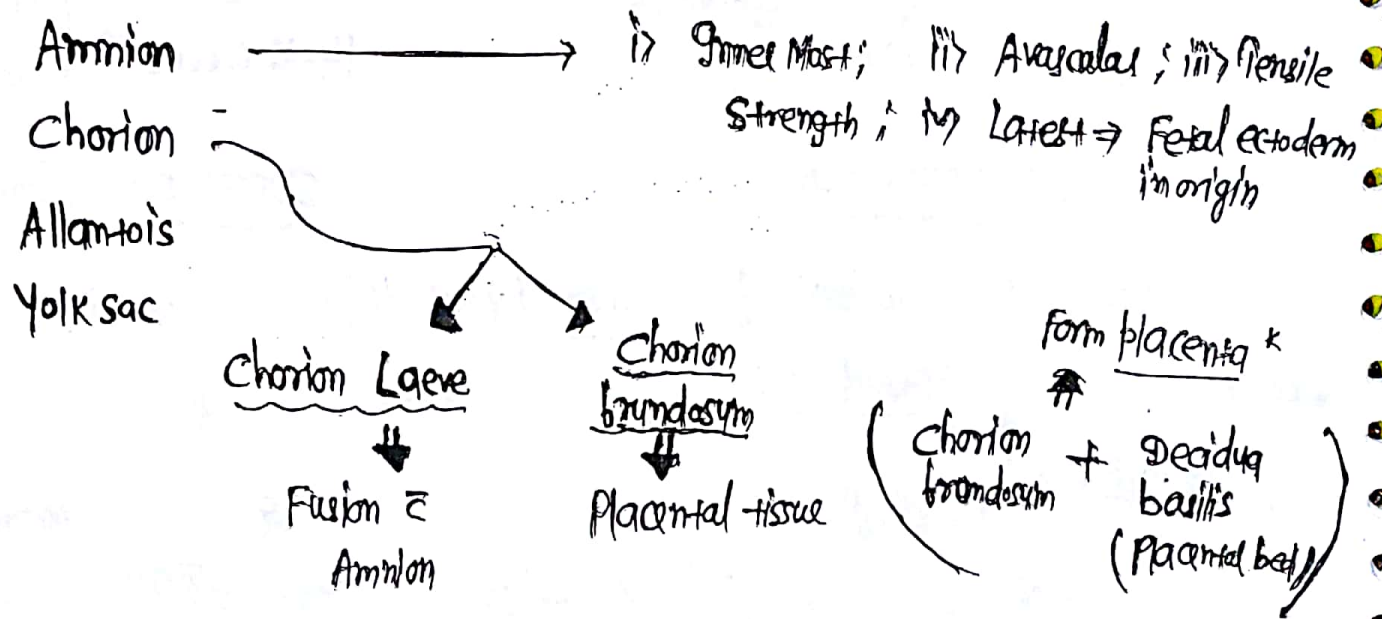
* Embryonic phase lasts upto 8 weeks post fertilization & 10 weeks from L.M.P.

Implanted embryo ⇒

Invasive Blood Vessels on placental bed,
 Make blood vessels Resistant to Vasopressin
 Maintain uteroplacental circulation,



Fetal Membranes

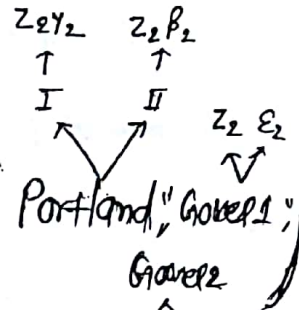


Allantois ⇒ Diverticulum from hindgut grows into connective tissue stalk. (20)

↳ Umbilical vessels (It gives rise Umbilical vessels).

Yolk sac ⇒ 1st site of hematopoiesis

↳ 3rd - 6 weeks (Portland, "Gowers 1")
Gowers 2



> 6 weeks - Linear - **HbF** ⇒ $\alpha_2\gamma_2$

> 20 weeks - Long bones

↳ higher affinity for O_2

• Bigger in size & shorter in life span.

• less 2,3 DPG & carbonic Anhydrase; Hb- O_2 curve shift to left.

* Fetal Hb is resistant to both alkali & Acid denaturation

ALKALI DENATURATION TEST

APTT (test)

↓

1% NaOH used

Bed side test

Qualitative test

differentiate

Maternal blood & Fetal blood from each other

Mom's ⇒ \ominus ve (colour change)

Baby's ⇒ \oplus ve (colour resistant)

• Singer's test (other kind of alkali test)

ACID DENATURATION TEST

• KB (Kleihauer Betke)

• citric acid & Ph buffer used.

• Laboratory test

• Quantitative test

Fetal **RBC** from Maternal **RBC** → count

Used in Rh \ominus ve ♀ to calculate dose of Anti-Rh.

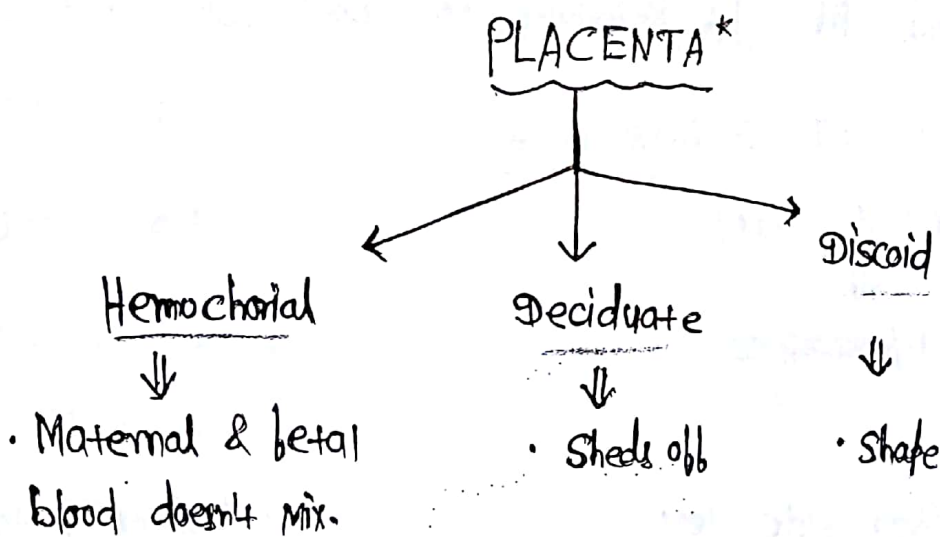
In Antepartum hemorrhage we can do "APT test". *

* Fetal RBC \Rightarrow Larger in size
Shorter Life \Rightarrow 90 days *

- Hb of baby @ birth \Rightarrow 18 gm%.

\downarrow
75-80% of HbF & Rest Adult Hemoglobin.

- @ 6 Month $<$ 1% HbF pres. out of total.



Wt of term placenta \approx 500 gm

Volume of term placenta \approx 500 mL (volume)

Diameter \approx 20 cm

Thickness \approx 2.5 cm

At term; Placenta: Fetal wt = 1:6

Maternal side

Fetal side

↓
Facing decidua

↓ (2)
Facing the fetus

↓
Lobes (divided into 15-20 lobes)

Identify ⇒ Smooth / Shiny
(b/c of Membrane)

↓ each lobe divided into 3-5 lobules
Lobules (Functional Unit)
↳ klas "cotyledon"

Umbilical cord attached

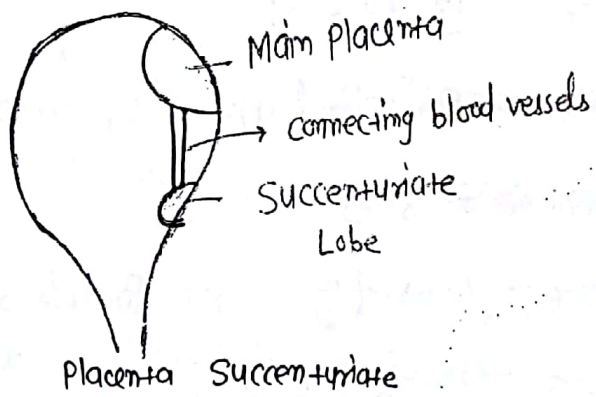
* Placenta bilobata ⇒ Placenta is separated into lobes;

• Division is incomplete & the vessels of fetal origin extends from one lobe to the other before uniting to form umbilical cord.

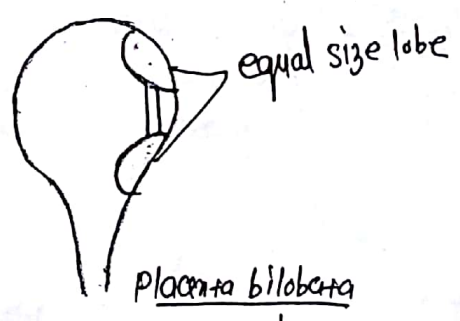
• ilb cord attached to the periphery ⇒ Battledore Placenta

* Placenta Succenturiate ⇒
↳ having Accessory Lobe; which is connected to the main part of placenta by blood vessels.

It can cause Post partum Hemorrhage



• Velamentous Placenta ⇒ ilb fetal vessels travel outside the cord for some distance before reaching placenta (∴ More chance to injury).



• Vasa previa ⇒ vessels are travelling over the internal os.
type of velamentous placenta;
Rare type of APH (Fetal blood loss).

C.S. do ← Obstetric emergency ← Result in severe Fetal stress

* Intervillous space \Rightarrow Maternal blood*

Inside villi \Rightarrow Fetal blood*

1^o villous

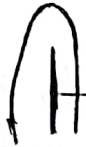
\Rightarrow



\rightarrow Solid - D13

2^o villous

\Rightarrow



\rightarrow Mesodermal core - D16

3^o villous

\Rightarrow



\rightarrow Blood vessel



* Fetal blood flow through placenta \Rightarrow 400 ml/min.

Fetal circulation Established @ D21

Uteroplacental circulation = D12

@ term = 450-650 ml/min.

* Intervillous space \Rightarrow 140 ml blood

\downarrow
Po₂ in Intervillous space

\downarrow
- 35-40 mm of Hg.

O₂ Saturation = 65-75%

Low pressure = 10 mm of Hg

\rightarrow Invade spiral A

Klas "endovascular"

* Cytotrophoblast



\downarrow
Permanent vasodilatation

\downarrow
Good uteroplacental circulation

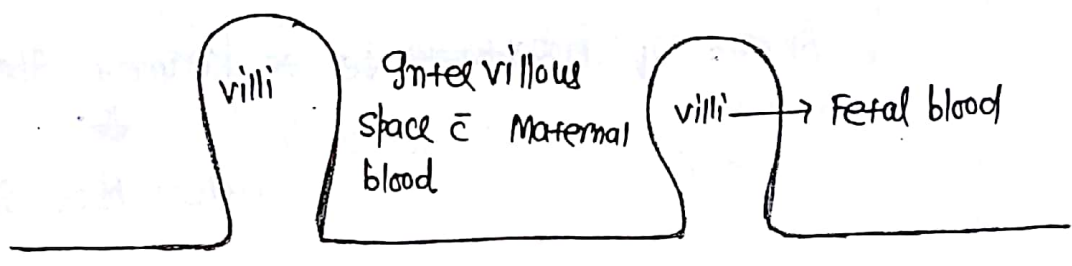
\downarrow
Vascular Remodelling

* Vascular Remodelling is controlled by decidual Natural Killer cells. (22)

* Completed 2 phases -> 12 weeks
16 weeks

* Absent Vascular Remodelling => Pre-eclampsia
JUGR

* (ABN) Vascular Remodelling => Adherent Placenta*



PLACENTAL FUNCTIONING*

• Placental formation begins @ 6 weeks.

Anatomically - Placenta completely formed by 16 weeks.

Physiologically - Maturation continues

- ↑ POG => • Cytotrophoblast ↓ ^{term} (X) No cytotrophoblast @ term
- Syncytiotrophoblast thickness ↓ (thin)
- ↓ Stroma
- ↑ Fetal blood vessels
- also keep moving towards periphery of villus
- Hofbauer cells ⊕
↳ Fetal Macrophages

Function of Placenta →

- Nutritional
- Excretory
- Respiratory
- Endocrinal (Most imp)

Progesterone | Estrogen | HPL | HCG
↳ Human Placental Lactogen

Progesterone ⇒ Maintenance of ♀

↳ Smooth Muscle contracts

↳ Amount of progesterone ↓ ⇒ Recurrent Abortion

↳ Luteal phase defect (L.P.D.)

also do decidualization (Hypersecretory change)

On HPE ⇒ "Arias stella Reaction"

↳ No Progesterone

↳ No Arias stella Reaction.

* Source of Progesterone in early ♀ ⇒ Corpus Luteum

Reserve the corpus luteum from Luteolysis

↳ Pregnancy

HCG

Maintain

Pregnancy

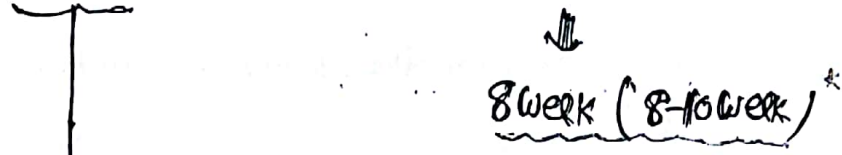
↳ Corpus Luteum survive.

Naturally late

Luteolysis*

(23)

* Placenta will take over the function of corpus luteum.



Make Progesterone by precursor \Rightarrow Maternal LDL cholesterol

* corpus luteum of pregnancy will Regress

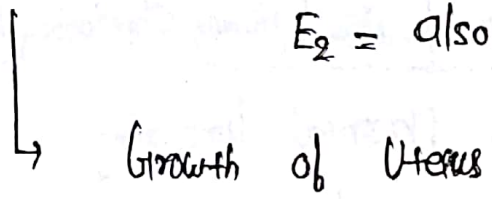
\hookrightarrow 12 wks

* Ovarian cyst of 1st Trimester \Rightarrow May be enlarged corpus luteum

\hookrightarrow Observation & Resolve after 12 weeks.

Estrogen \Rightarrow Specific to ♀ = E_3 Estriol*

E_2 = also formed; but Not specific



\rightarrow Mask of ♀ = Melasma
Hyperpigmentation
(stimulates Melanocytes)

\rightarrow Retain salt/water

\rightarrow obstetric cholestasis

\rightarrow Thyroxin binding globulin \uparrow

* Placenta can't synthesise estrogen on its own

↳ b/c 17 α hydroxylase Lacks
↳ dependent on the fetus for synthesis of estrogen.

↳ Fetal DHEAS (from Adrenal glands)



Placenta



Sulphatase
Aromatase



Estrogen (E_3)



Tells about fetal well being

HPL (Human Placental Lactogen) | HCS (Human Chorionic Somatomammotropin)

⇒ GT tells about i) Placental functioning



as p.o.h. rises ⇒ Matures ⇒ ↑HPL

Peak = 36 wks.

Q9 Which hormone is produced by placenta in max^m Amount

at term = HPL (1gm/day).

ii) endocrinal function ⇒ Main function

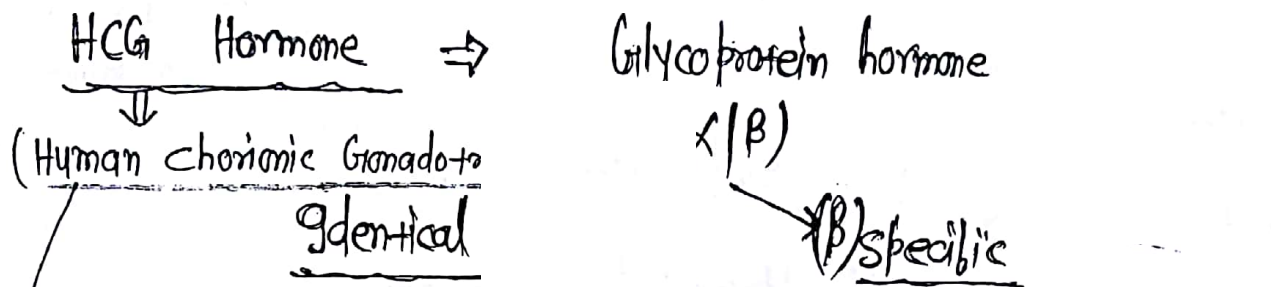
↳ Insulin Resistance in ♀
(Plasma cortisol: Growth hormone)

Q. Which hormone is responsible for fetal growth

(24)

Insulin like growth factor/ IGF

* Promotes Maternal Lipolysis - Levels of free fatty acids are low which mother utilizes as a source of energy; sparing glucose for fetus.



α Subunit \Rightarrow LH / FSH / TSH

Non-specific

Syncytiotrophoblast

as early as 8 days post fertilization it secretes "HCG" (8-9)

aa \rightarrow This hormone has highest carbohydrate content of any human hormone

we want to take hCG before 8 days

\hookrightarrow do serum hCG \Rightarrow Quantitative

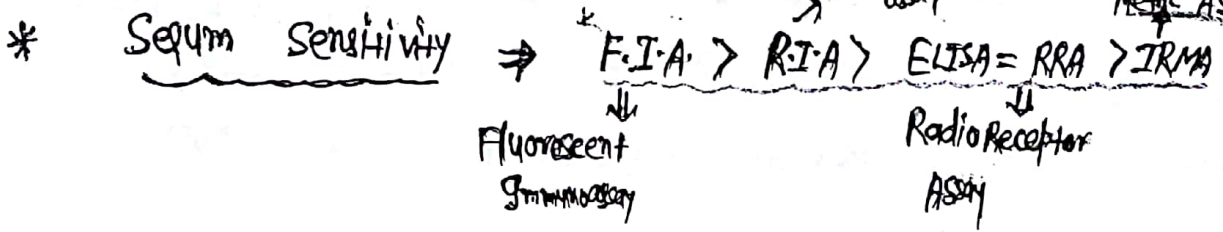
Very sensitive

\hookrightarrow detect 1 IU/L

Urine hCG \Rightarrow Qualitative

\hookrightarrow 20 IU/L \Rightarrow \oplus ve after missed period.

based on "Sandwich ELISA"



⇒ hcg Value ↑ as P.O.G. ↑

Max^m = @ 10 wks.*

After 10 weeks = ↓

Min^m = @ 16 weeks

After 16 weeks = Plateau

→ normal ↑
Doubling time of β-hcg

↓
48 hrs.

↳ it means ↑ in 55% - 66%
Min^m after 48 hrs.

↳ it does N't Mean 100%
or double

v.v.g.
Q9

1st D_1 / D_3 - ↑ by 55% ?

(N) Intrauterine Pregnancy

2nd D_1 / D_3 - ↑ by < 55% ?

Ectopic ♀

3rd D_1 / D_3 - ↓

Non-viable ♀ / Abortion

* Critical value of hcg for TVS = 2000 IU.

TAS = 6500 IU.

⚡ If hcg is More than or equal to these value & we don't see
Sac in the uterus ⇒ Likely to be ectopic ♀.

(25)

* Condⁿ where hCG is than Expected

- Multifetal ♀
- GTD (Gestational Trophoblastic Disease)
- Down's Syndrome
- Hyperemesis gravidarum
- Underexpected Gestational Age.

* Condⁿ where hCG is less than Expected

- Non-viable
- Ectopic ♀
- over expected gestational Age
- Trisomy 18.

- Functions of hCG

- ⇒ Maintenance of ♀
- ↓ uterine contraction
- Growth & development of umbilical cord
- 1st stimulus for Release of testosterone from Male fetus - hCG
- Immunosuppressant

Qq Why the conceptus Not Rejected??

- i) Villous trophoblast Lacks HLA (MHC);
- ii) EVT (Extra villous trophoblast) - that have HLA \rightarrow only in Human species \rightarrow Immunosuppressive

Placenta decidua → NK cells have deficient cytotoxicity.

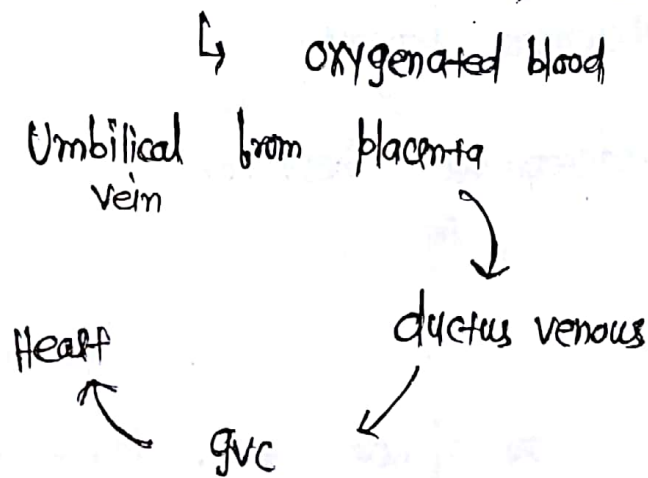
→ M/c Insertion ⇒ center.

UMBILICAL CORD (Attached to fetal side of placenta).

- Average Length = 55cm (30-70cm)

- 3 vessels ⇒ 2 Umbilical A. (deoxygenated blood)
1 Umbilical vein (Left) "Right U-vein disappear"

* Short cord < 30cm
* Long cord > 70cm



* Max^m O₂ saturation ptr. in ⇒ Umbilical vein
↓
80%.

* M/c Vascular Anomaly ⇒ SUA (2 vessel cord)
(Single Umbilical A)
↓ then check
GCA (Gross Congenital Anomaly)
↳ CVS; ↓ M/c gross Anomalies
↳ Renal ⇒ M/c Anomalies
Not gross ⇒ SUA

* if we see SUA + GCA



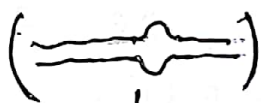
↑ Risk of Aneuploidy *

(Trisomy 18) **

(26)

* Isolated SUA ⇒ doesn't ↑ Risk of Aneuploidy *

* Incidence of SUA is highest in ⇒ Twin ♀ / Multifetal ♀



↑ Risk of true knot of Umbilical cord.

False knot ⇒ Protrusion of Wharton's Jelly containing loop of Umbilical vessels.
↳ No clinical significance

True knot ⇒ ↑ the Risk of still birth;

↳ cause ⇒ Fetal Movement.
↳ d/t Active fetal Movements
↳ ↑ in Twin.

1st Umbilical Artery



Umbilical vein



Ductus Venosus



Ductus Arteriosus



Foramen ovale

DOWN'S SYNDROME*

• Trisomy 21.

• Causes \Rightarrow Most common cause* \Rightarrow Non-dysjunction (95%)

\nearrow during Meiosis-I

\Downarrow
It is by chance; so;
Non-heritable in Nature

• Recurrence = 1%

• H/O 1st baby down's \rightarrow Antenatal testing done in Next ϕ .

• 5% \rightarrow Inheritable

4%
 \Downarrow

Translocation

1%
 \Downarrow

Mosaicism

100%

18% baby \rightarrow Down \rightarrow (Translocation 21, 21)

Robertsonian Translocation seen in Down sx.

\Downarrow
Risk = 100%

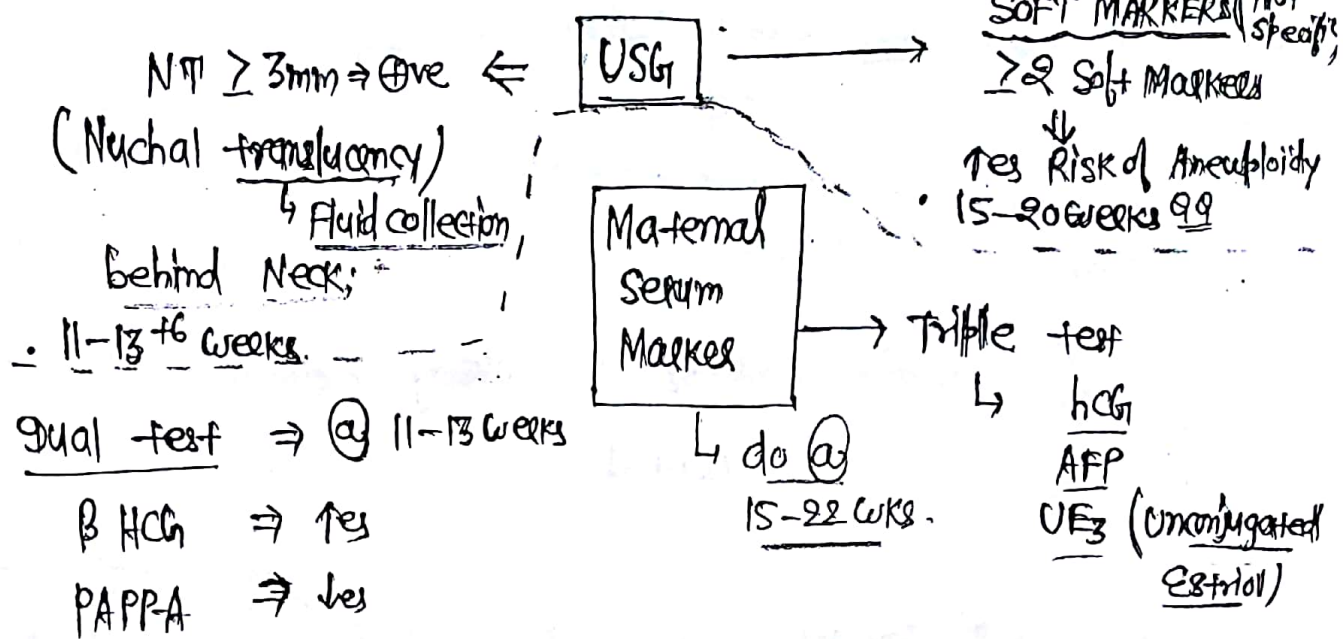
\rightarrow Abortion. (in 2nd baby)

* All ϕ Women

\rightarrow offered down's screening. (opt-out screening)

SCREENING →
1st Trimester

2nd Trimester (27)



(Pregnancy associated plasma protein A)

* USG for NT + Dual test
↓
Combined test*

→ Quadruple test
 ↳ hCG
 AFP
 UE₃
 + Inhibin A (↑)
 ↳ Produced by placenta during 2nd & corpus luteum in Non-pregnant female
 **

* Soft Markers ⇒ (Absent Nasal bone (Hypoplastic) ⇒ can be seen @ 1st trimester al.)
 Most imp. ⇒ Nuchal fold thickness ↑ (≥ 6mm) → Screening ⊕ve

- echogenic cardiac foci
- echogenic bowel foci
- Short femur
- Short humerus
- Short frontal lobe
- Short ear length
- Simian crease (single palmar crease)
- Short 4th middle phalanx (clinodactyly)
- Saddle gap
- Pyelectasis (Mild dilatation of Renal pelvis)

Q9

M/c Congenital cardiac Abnormality in Down's sx child

Endocardial cushion defect > VSD > ASD

→ Not central (More towards one side)

Q10

Gastroschisis is Not seen in Down's sx patient

Omphalocele → Covering Membrane (+)
↳ central

*

Confirmatory test

⇒

Karyotyping

M/c Method ⇒ "G Banding"

In Metaphase

Drug: Colchicine

1st Trimester

↓

Chorionic villous Sampling

⇒

≥ 10 weeks

M/c ⇒ 11-13 weeks

don't do before 9 weeks

↓

b/c it may cause

↓

Limb defects

- can result in false +ve test (Placenta se cell / Late hair)
- Risky fetal loss 1%

- early result trophoblast (48-72 hr)

2nd trimester

↓

Amniocentesis

≥ 15 weeks

M/c = 16-18 weeks

don't do 11-14 weeks

↓

early amniocentesis

↳ fetal loss

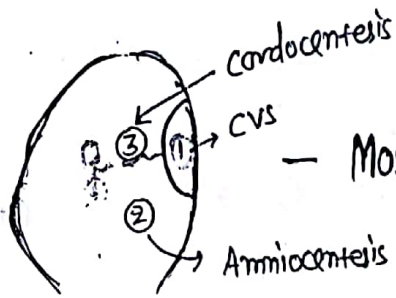
- More accurate
- More safe < 0.5%

- delayed (7-10 days) result

↓

Amniocytes fibroblast

* Cordocentesis → Umbilical cord blood cells



— Most Risky ⇒ 3% fetal Loss.

↓
from Umbilical vein (placenta end)

↓
b/c it is stable end.

* Amniocentesis all USG guided procedure; Not blind - procedure.

* NIPPT ^{→ costly} (Non-Invasive Pre-Natal test) ⇒

- Genetic Material ⇒ cell free fetal DNA
- done on Maternal blood (Some amount of cell free DNA circulating in Maternal blood)
- ≥ 10 weeks
- this test is used as 2^o screening (costly)
- has to be followed by confirmatory test;
- takes 7-10 days for Results.
- tells us about Trisomy 13, 18, 21
Monosomy - XO

PHYSIOLOGICAL CHANGES AFTER PREGNANCY

Presumptive sign



Women experience

Reusell (eg ⇒ Amenorrhoea;
Morning sickness
etc)

Probable sign



Seen by doctors

Positive sign



Confirmatory

earliest = hCG ^{serum} / _{urine}

USG

O/E ⇒ Fetal parts

↳ Fetal heart rate

Probable sign

⇒

i) Hegar's sign

⇒

Softening of Isthmus

(on Bimanual examination)



Seen on 6th week

ii)

Good sign

⇒

Softening of cervix



Seen on 6th week

@ 8th weeks

iii) Chadwick's (Jaquimier's sign) ⇒

Bluish discoloration of vaginal/vulva

iv)

Osiander's sign

⇒

Lateral vaginal fornix
Pulsations

v)

Piskacek's sign

⇒

one half of uterine keels
more enlarged (Asymmetrical)

vii) Palmer's sign ⇒ Regular Rhythmic Uterine ⁽²⁹⁾ contraction
@ 8th week (No Pain; only felt by examiner)

viii) Haltman's sign ⇒ Implantation bleeding (Not a problem)
↳ @ 8th week

On USG ⇒ 1st sign of pregnancy on USG*

↓
Gestational Sac**

On TVS ⇒ 4-5 weeks (4-3 day) ← empty bladder
On TAS ⇒ 5 weeks ← Full bladder
TVS is higher frequency (25Hz)
TAS is lower frequency (3Hz).

Intradecidual sign ⇒ Marker of an Intra-uterine ♀
early

Yolk Sac appear ⇒ TVS = 5-5 week
TAS = 5 week

Fetal pole ⇒ Cardiac activity
→ TVS ⇒ 5-6 weeks
→ TAS ⇒ 6-7 weeks

* Double-decidual sac sign ⇒ Marker of early Intra-uterine ♀.
↳ Decidua capsularis
↳ Decidua parietalis

* Double bleb sign \Rightarrow Sign of early Intrauterine ♀
 ↙ ↘
 Amniotic Yolk sac

Q. Q. earliest to see Gestational sac \Rightarrow 5th week on USG

Q. Q. Earliest time Gestational sac can be identified on TVS from LMP ??

Q. Q. Earliest time Gestational sac can be identified on TVS from fertilization ??

\hookrightarrow LMP - 15 days = 30 - 15 = 15 days

\Rightarrow What ever seen on TAS can be seen in TVS (vice versa is not true)

\Rightarrow In Ectopic : Ring in Uterus \Rightarrow Single Ring in Uterus

\downarrow
 "Pseudosac" \Rightarrow prt. in cornu of uterus.

General changes \Rightarrow ① Additional caloric Requirement

In ♀ ; everywhere Progesterone excret \Rightarrow Obstetric cholestasis } \Rightarrow estrogen
 salt & water Retention
 \uparrow

\downarrow
 350 Kcal/day
 \hookrightarrow In 1st trimester = No additional caloric Requirement.

Brought about by
 \uparrow
 Retain salt/water

② Average wt. gain
 \hookrightarrow 12 kg (10-14 kg)

③ BMR \Rightarrow 1st by 20%

④ salt/water \Rightarrow 65L

④ Plasma osmolality \Rightarrow \downarrow ves (10 mosm/kg)

⑤ Plasma volume \rightarrow \uparrow 40%

Red cell Mass \rightarrow \uparrow 20%

Hemodilution condition \Rightarrow Anemia of Pregnancy

Anemia \Rightarrow Hb $<$ 11 gm% ; Hematocrit $<$ 33%

\hookrightarrow M/C = Iron deficiency Anemia*

Total Fe Requirement = 1000mg
300mg (Fetus)

Best test = S Ferritin

Prophylaxis = GFA (100mg Fe + 500 Mg FA) ^{In Fetus form}

\rightarrow 1 tab

\rightarrow 6 month during pregnancy

6 month after delivery

T/t of Anemia \Rightarrow IFA tab (2 tabs) ^{iv} ^{Acute blood loss} & Hg $<$ 6 gm%

Parenteral

Blood transfusion

i) if the patient is Not compliant by oral tab

ii) Not tolerating oral tab

iii) Malabsorption sx.

i) if $>$ 34 weeks \odot & Hg $<$ 7 gm (even in Heart failure No sign/symptom)

ii) if $<$ 34 weeks \odot & Hg $<$ 5 gm (even No sign/symptom of Heart failure)

iii) Anytime sign & symptom of CHF.

⑥ TLC \Rightarrow \uparrow es (It doesn't Mean Infection)
 \hookrightarrow 15,000 during Primetel
25,000 after Postpartum.

⑦ DLC \Rightarrow Neutrophilia
ESR \uparrow
CRP \uparrow

⑧ Platelet count \Rightarrow Average platelet count \downarrow es
 \hookrightarrow Not causes Thrombocytopenia

⑨ Clotting factors \Rightarrow all \uparrow es except \Rightarrow Factor II & III.

⑩ Insulin Resistance \Rightarrow Hyperinsulinemia
 \uparrow as \uparrow OGTTes
Significant $>$ 24 weeks
 \rightarrow Fasting = Hypoglycemia
Post-prandial = Hyperglycemia

aa

* Anemia : $<$ 11 gm/dl

Severe Anemia : $<$ 7 gm/dl

Very severe Anemia : $<$ 4 gm/dl

CVS **

(31)

- ↳ Plasma Volume $\Rightarrow \uparrow 40\%$
- Red cell Mass $\Rightarrow \uparrow 20\%$
- Cardiac output $\Rightarrow \uparrow 40\%$

O_2 demand of tissue $= \uparrow 20\%$

feature of $\leftarrow O_2$ carrying capacity = ↓ *
 Hb; Not of
 Red cell Mass

A-V O_2 gradient of tissue = ↓

- all heart sounds are loud = Loud S_1
- S_3 (Gallop Rhythm)
- Systolic Murmur (Ejection systolic Murmur)
 ↳ Physiological up to grade 2.

Diastolic Murmur

↳ almost/ Always Pathological

- Heart Rate ↑ (by 16-18 beats/min above baseline)
 (<100)
- split S_1

- Apex beat \Rightarrow heard @ 4th ICS (b/c heart is rotated Anteriorly & Pushed up).

on CXR \Rightarrow cardiac silhouette (appears big)

cardiomegaly \Rightarrow always pathological

on ECG \Rightarrow LAD \Rightarrow Physiological
 \hookrightarrow Left Axis deviation **

- Blood pressure = DBP $>$ SBP \downarrow (Both Fall)
(10 mm Hg) (all vaso pressure tes)

E/P (Estrogen / Progesterone)

- \rightarrow i) vasodilation
ii) Resistance against vasopress

begins - 5 weeks (\downarrow in DBP begins @ 5 weeks)

Maxim = 24-26 weeks

after 26 weeks = begins tes
(come back to pre-pregnancy value)

* ≥ 20 weeks \Rightarrow Supine hypotension syndrome
 \downarrow
Gravid uterus compresses IVC

changes position to Left Lateral position

\hookrightarrow tes utero-placental circulation

Fetal O₂ saturation by ICF

* Preload \Rightarrow \uparrow

afterload \Rightarrow \downarrow (d/t fall in systemic vascular Resistance)

* Ejection fraction = No change

Central venous pressure = No change

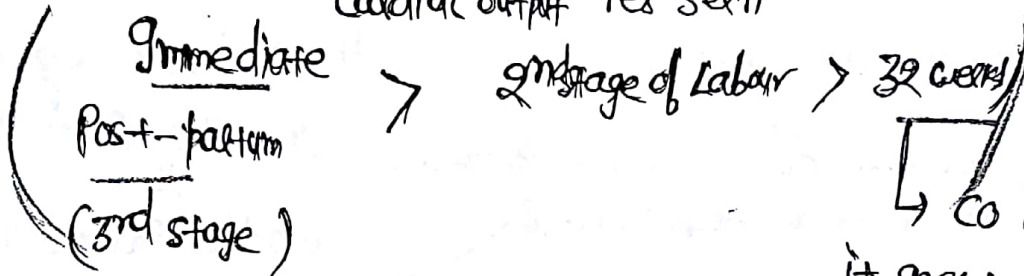
(32)

↓
Persistently distended Neck veins

↓
Pathological Always *

CHF (Highest Risk)

↓
cardiac output Tes seen



Q9 CO is Max^m @ →

a) 28 week; ~~b) 32 weeks~~; c) @ term; d) 36 weeks

Q9 Pre-eclampsia Not commonly dx in

↳ 3rd trimester.

26/4/18

KIDNEY

⇒ Tes Renal blood flow by 80%.

GFR Tes by 50%.

⇒ S Creatinine ↓; BUN ↓

S Urine acid Level ⇒ No change b/c Reabsorption.

* S. Creatinine ↑
S. Uric acid ↑ } Pre-eclampsia*

* Kidney - enlarge by 1cm

* Hydronephrosis - b/c of Progesterone
↳ Smooth Muscle Relaxant

↳ R+ side > Lt. side; Why??

↳ In @ uterus becomes dextrorotation towards R+ side

↳ Compresses Right Ureter

@ the Pelvic Brtm.

but still doesn't become incon-tinent
as urethral pressure also ↑

↳ Bladder Pressure ↑

↳ From 8cm H₂O

↳ TO 20cm H₂O.

* Urinary Stasis ⇔

Urine — Routine — Asymptomatic Bacteriuria > 10⁵
— Microscopic

↓
Treat; if we don't treat than
chances of Pyelonephritis becomes high.

* Glycosuria - b/c Renal threshold ↓.

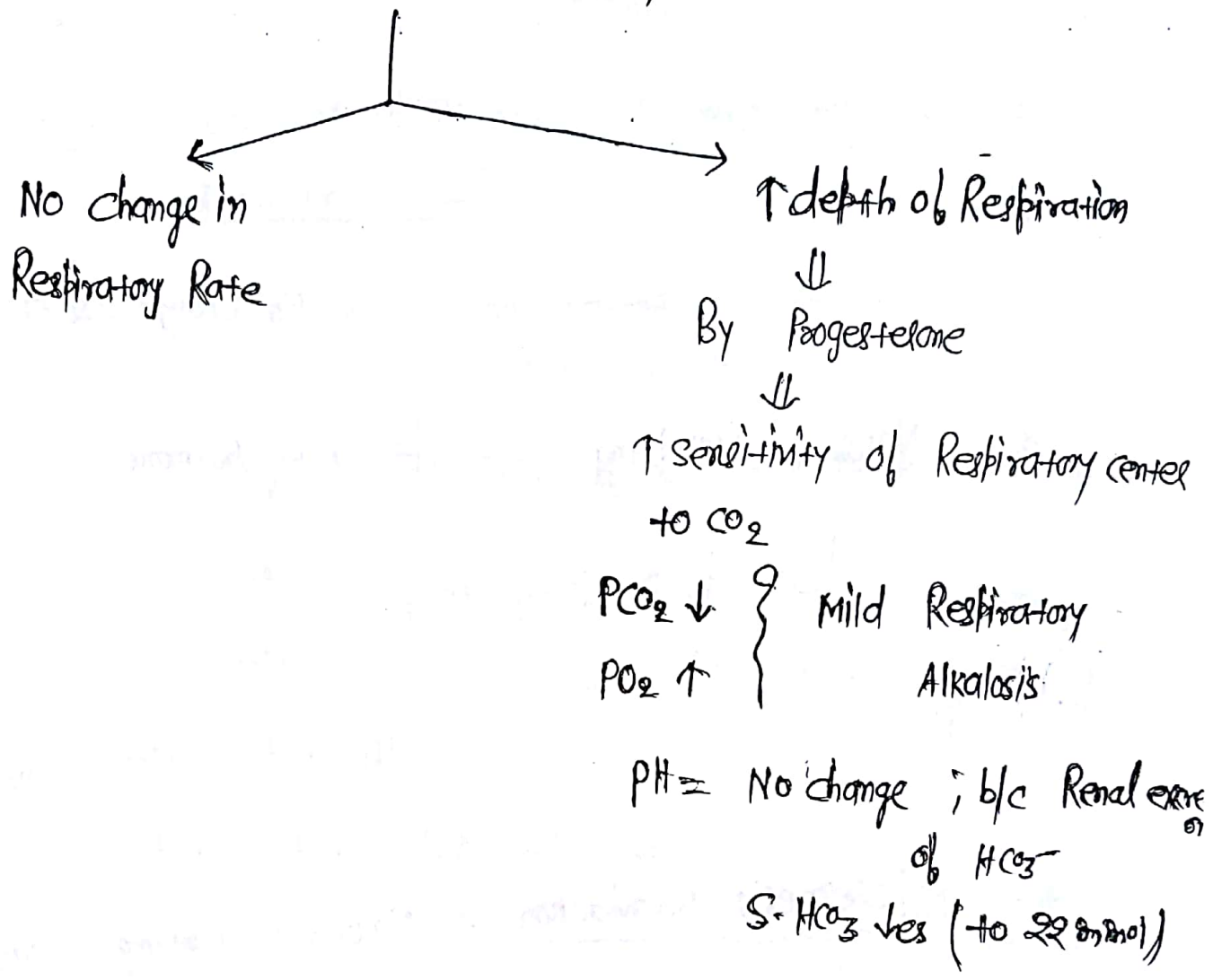
* Proteinuria - ≥ 0.3 gm/24 hr of urine sample

↳ Not Physiological; it's Pathological

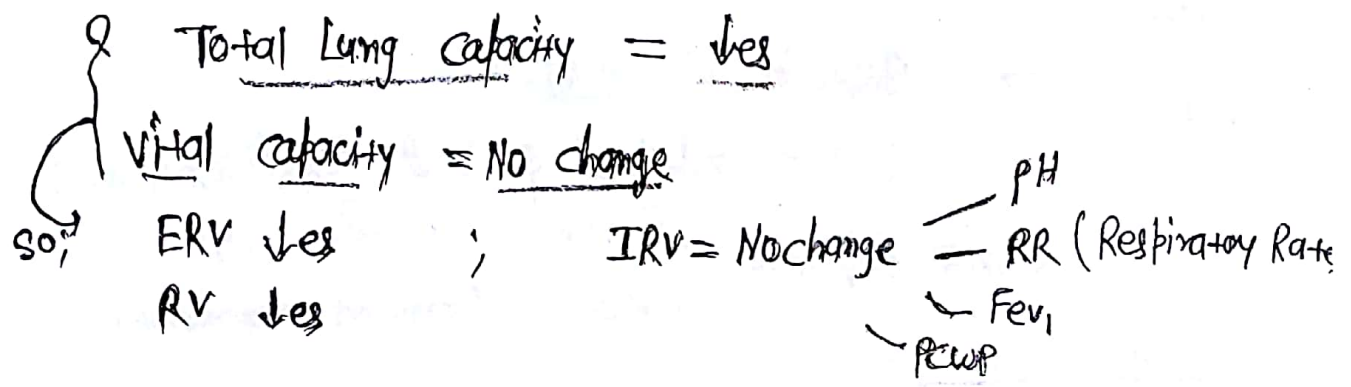
Respiratory system

(33)

- * Tidal volume T_{es}
- * Minute ventilation \uparrow (40%)



- * Diaphragm Rises by 4cm
- ↑ transverse thoracic diameter = 2cm



* Diaphragmatic Excursions \Rightarrow ↑ in Pregnancy.

GIIT

* GERD seen \Rightarrow b/c LES tone ↓ by Progesterone & Gastric Pressure ↑.

* Gastric emptying time shows No change in all trimesters.

* Nausea, vomiting = d/t hCG hormone

↳ 1st line drug in ♀ = Pyridoxine

↓ No Response

Doxylamine + Pyridoxine

* Hyperemesis Gravidarum \Rightarrow Nausea & vomiting causes either $\geq 5\%$ wt. loss &/or Ketonuria.

↳ d/t hCG

↳ Infection of H. Pylori

Seen in Multifetal ♀; & female sex fetus;

* 2 vit. deficiency \rightarrow vit. K - Coagulation defect
 \rightarrow vit. B₁ - Wernicke's encephalopathy,

- Can cause Esophageal tear (Mallory-Weiss tear);
- Renal failure - (Acute Renal failure). (34)

Rx \rightarrow Stop all oral Intake
give ivr Fluids + ivr Antiemetics.

- * Liver \Rightarrow ALP \approx Gross Test
 ↳ b/c Placental - ALP
 ↳ Not a Marker of cholestasis (it is Physiological Test)
- Test Production of Albumin/Globulin
 - S. proteins \rightarrow Fall (b/c Production is less than Volume Test)
 - AST, ALT both rise

Endocrine system*

Pituitary \Rightarrow N In size 135x

b/c Lactotroph TT (vascular supply TT)

In severe PPH — Vascular supply — \downarrow to Pituitary
 \downarrow
 Injuration
 \downarrow
 "Sheehan's syndrome"

Sheehan's Sx \Rightarrow M/c Presentation \Rightarrow Failure to Lactate

2nd M/c \Rightarrow Amenorrhoea

Usually - Ant. Pituitary affected

\downarrow
Post. Pituitary spared.

\Rightarrow if a ♀ doesn't Lactate - Menses by 6-8 weeks.

S. Prolactin Level - Highest

Pregnancy

Lactation

\downarrow
After delivery S. Prolactin ↓ by 50%

Prolactin - Milk Synthesizing hormone

Oxytocin - Milk Letdown / Ejection hormone

1st Stimulus \Rightarrow Initiation of Lactation
(\downarrow $\bar{E} + \bar{P}$)

Fall of E & P also causes \Rightarrow Post-partum depression
(Blues)

THYROID ⇒

- Thyroid binding globulin ↑ (Estrogen) ⁶⁰
- Total T_3 & T_4 ↑
- Free T_3 T_4 ↑ (slightly) } ⇒ ↑ Production ⁽³⁵⁾ from the gland
- TSH ↓ (slightly) Why? ⇒ b/c of hCG
- I_2 requirement ↑
- (♀ & Lactation) both have RDA = 250 μ g/day. α = TSH (same as TSH)

Pregnancy is Euthyroid condition

I_2 excretion tes.

- * M/c of hypothyroidism in ♀ ⇒ Hashimoto's ds
- M/c of hyperthyroidism in ♀ ⇒ Graves ds.

Q K/c/o hypothyroidism ; L. thyroxine 25 μ g

Dx = Pregnancy ⇒ Tes ^ by 50% (b/c some part of thyroid doses become degenerated)

Hypothyroidism May cause abortion.

Maternal Nerve Injury ⇒ M/c in Lithotomy position

↓

Common Peroneal Nerve

* M/c in Postpartum / Peripartum / Intrapartum

↓

Lateral cutaneous N. of thigh } Femoral N. extended Lithotomy position

* Foot drop in ♀ is d/t "Lumbosacral Plexus Compression."

* -

Fetal Swallowing ⇒ 10 weeks

Fetal breathing Movement ⇒ 11 weeks

Fetal Urine production ⇒ 12 weeks

Fetal Meconium production ⇒ 16 weeks

IgM production in baby ⇒ 20 weeks

by mother ← IgG transfer in baby ⇒ 16 weeks

Surfactant synthesis begins ⇒ 20 weeks

Surfactant appears in Amniotic Fluid ⇒ 28 weeks

Glucagon production ⇒ 8 weeks

Insulin production ⇒ 12 weeks

H-P (circulation) ⇒ 12 weeks*

AMNIOTIC FLUID

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Major Source \Rightarrow Fetal Urine

Major Removal \Rightarrow Fetal Swallowing

Major Source \rightarrow in 1st 12 weeks \Rightarrow Ultrafiltrate of Maternal Plasma
 In 12-20 weeks \Rightarrow Transudate across fetal skin
 in >20 weeks \Rightarrow Urine.

* 98% Amniotic fluid \Rightarrow water (Nutrition)
 \hookrightarrow Not help in Nutrition⁹⁹

* Colour of Amniotic fluid \Rightarrow Straw coloured

Green colour = Meconium-stained

Dark = Abrasion

Golden = Rh Incompatibility

Tobacco Juice (dark brown) = GUD

Greenish Yellow (saffron) = Post Maturity

* pH \approx 7-7.5

* Osmolality \approx 260 mosm/l.

* Water is replaced in every 3 hrs.

Normal

AFI (Amniotic Fluid Index)
5-24cm

DVP (Deep Vertical Pocket)
2-8cm

Polyhydramnios

≥ 25

≥ 8

Oligohydramnios

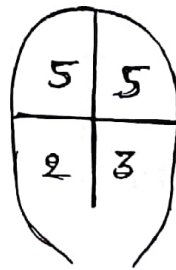
≤ 5

≤ 2

More Common Method

Better (No quadrant so; Less error)

AFI \Rightarrow



\Rightarrow divide in 4 quadrant Arbitrarily

15 = Amniotic fluid

DVP \rightarrow Not divided in quadrant; only pocket division.

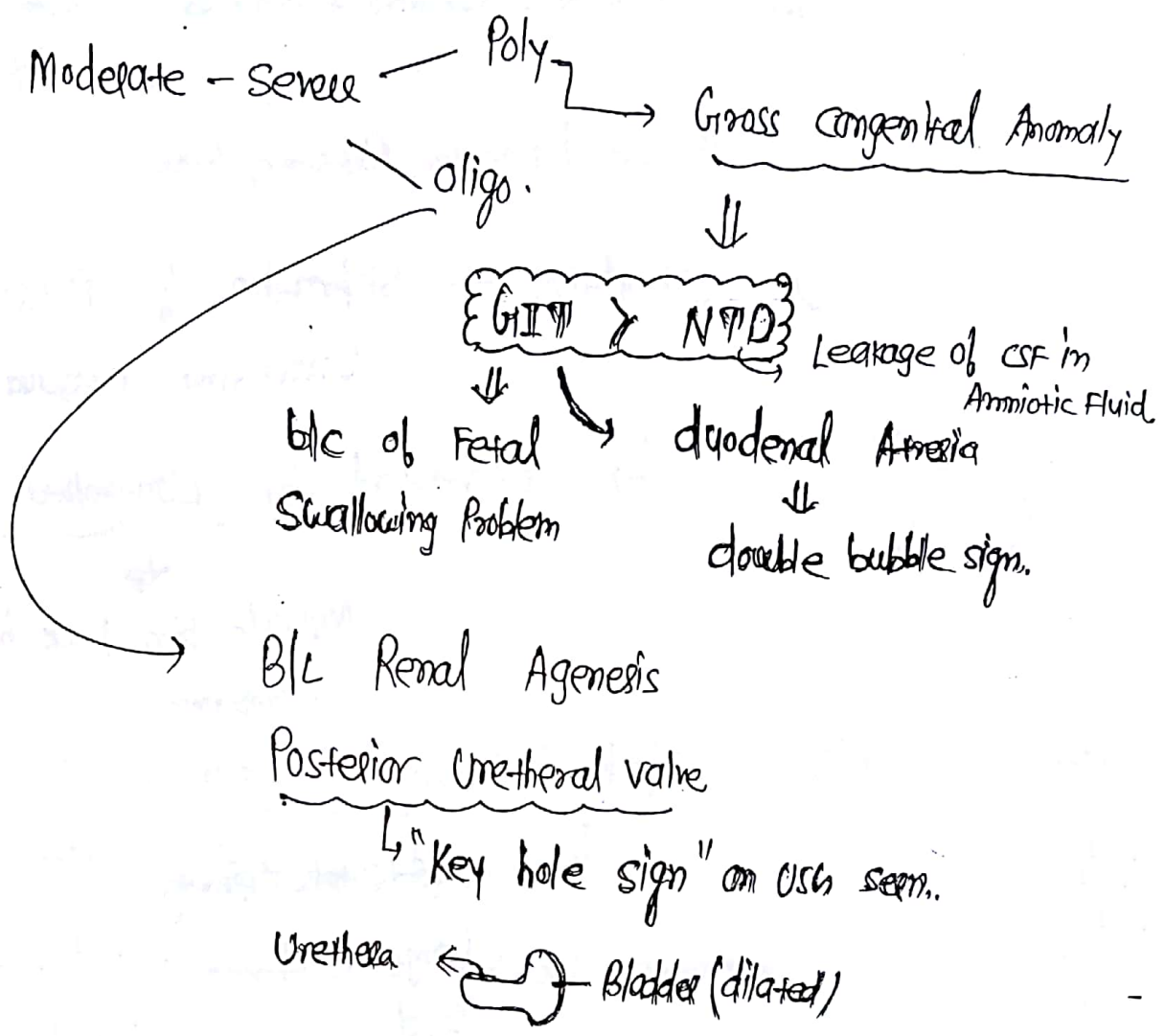
* Absolute value \Rightarrow Polyhydramnios = $\geq 2L$
Oligohydramnios = $\leq 200mL$

* In twin \odot ; we can't use AFI; only use DVP;
 \hookrightarrow b/c we don't know about quadrant of both fetus.

(37)

*

Mild — Poly — idiopathic
 — oligo.



Lady comes w/ Poly/oligo (Moderate-severe)
 ↓
 diabetes → Leaking Amniotic fluid
 ↓
 See by Per speculum examination.

* Polyhydramnios have high Risk of \rightarrow (Moderate - severe)

- ① Maternal Respiratory distress;
- ② Pre-term Labour - < 37 weeks
- ③ Premature Rupture of Membrane - Membrane Rupture before Labour.

Preterm premature Rupture of Membrane - < 37 weeks & PROM.

- ④ Abruptio - Separation of Placenta from underlying decidua.

\downarrow
 \rightarrow prevented by Controlled ARM

\Downarrow
Multiple pin prick holes into the Membrane

- ⑤ Cord prolapse

\rightarrow obstetric emergency b/c of temperature change to \rightarrow vasospasm
Cord \rightarrow severe fetal distress.

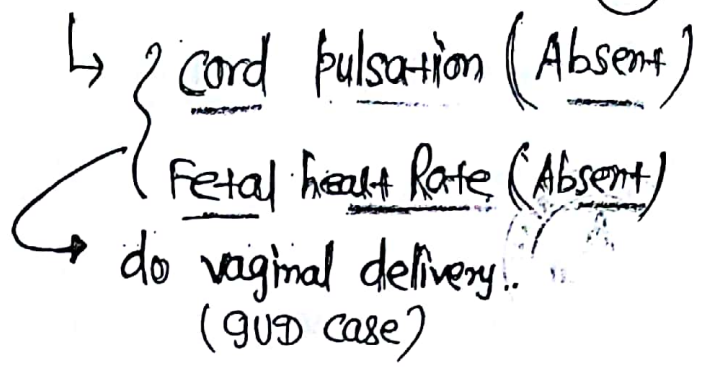


do emergency C.S.

- i) Reposit the cord above the presenting part;
- ii) Fill the bladder;
- iii) elevate the buttock,
- iv) O₂ by mask v) iv fluids given.

* We do emergency c.s. ; depends on

(38)



- ⑥ Malpresentation;
- ⑦ PPH;
- ⑧ GICA;
- ⑨ Diabetes;
- ⑩ Amniotic fluid embolism

* Oligohydramnios have high Risk of →

- ① Pulmonary hypoplasia;
- ② Fetal distress (cord compression)
- ③ Malpresentation
- ④ GUR
- ⑤ PE (Pre-e) association \bar{c} oligo. b/c of Uteroplacental Insufficiency (UPI)
- ⑥ Early in pregnancy oligo.
 - ↳ viral Infection (TORCH/zika)

⑦

Amniotic band sequence ⇒

tears in Amniotic Membrane



Severe oligohydramnios (d/t leakage)



Bands & tight wrap around fetus.

M/C Anomaly in Amniotic band sequence ⇒

Limb Anomalies > Craniofacial Anomalies

⑧

Amnion Nodosum

↳ S. oligo + yellowish nodules on the Membrane
↳ severe

⑨

Compression defect ⇒ CTEV (club foot)

QA

Diagnostic Amniocentesis ⇒ ① Karyotyping;

② Neural tube defect → AchE / AFP*

Best screening test for NTD ⇒ USG

Best test for NTD ⇒ AchE / AFP (Amniocentesis)

Other screening test for NTD ⇒ Serum Alpha-feto protein

16-18 wk

> 9-0 ← ... (unintelligible)

* AFP Peak — In Fetus — 13 weeks
 — In Mother — 32 weeks
 ↳ $t_{1/2} = 5-7$ days.

(39)

* ↓ Level of AFP Serum ⇒ Down's Sx;
 Diabetes;
 obesity;
 Molar ♀;
 GUG.

③ Lung Maturity ⇒ $M/C = L/S$ Ratio ⇒ > 2 ⇒ Mature

↳ Best test ⇒ PG (Phosphatidyl Glycerol)

↳ Not affected by presence of
 Contaminants.

↳ in Diabetes Mothers.

④ Hemolytic Anemia ⇒ In Fetus

⑤ ASKE of acute viral Infection in fetus

↳ Amniotic fluid Polymerase chain reaction. 99

In Acute Maternal Infection



4 fold Rise in Ab titre in Paired sera.

— Avidity test.

⑥ Chorioamnionitis

*

<u>P.O.G.</u> -	<u>Amount of Amniotic Fluid</u>
12 WK →	50mL
16 WK →	250mL
20WK →	400mL
32 WK →	1 Litre (Max ^m)
36WK →	900mL
Term 40WK →	800mL
42WK →	200mL

POST-PARTUM HEMORRHAGE

40

3rd stage ⇒ Placenta expulsion

↳ Avg. time - 15-20 min

Prolonged > 30min.

Signs ⇒ ① Gush of blood;

Best sign ←

② Lengthening of cord;

③ Subpubic bulge;

④ Fundal height test

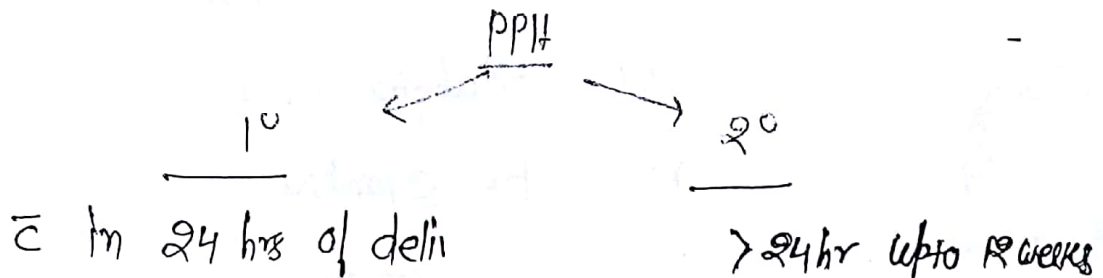
Apparent

Permanent

Best sign ⇒ Placenta lying in vagina > Lengthening of cord

④ Avg. Blood Loss

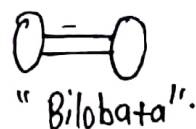
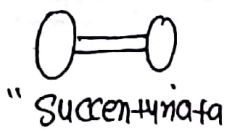
⇒ Vaginal delivery = 500ml
 C.S. = 1000ml
 Twin vaginal delivery = 1000ml



M/c cause ⇒ Uterine Atony

Retained Placental tissue

Placental Abnormality which is RIF for PPH ⇒



4 Ts — Tone (Uterine Atony)

Trauma

Tissue (Retained Placental tissue)

Thrombosis (defect)

* RIF for Atonicity ⇒ i) Multifetal ♀

ii) Macrosomia

iii) Polyhydramnios

iv) Induction of Labour

v) Augmentation of Labour

vi) Precipitate Labour

(onset - expulsion \bar{c}/in 3 hours)

vii) Any kind of APH

viii) Multiparity

ix) Diabetes Mellitus

x) Pre-eclampsia

xi) Chorio amnionitis

* Most Important things for placental separation

↳ Uterine contraction.

Plane of Placental separation ⇒

** 80
Spongiosum

(41)

Methods by which placenta separates ⇒

↳ Layer of decidua*



Placental separation

↳ starting @ centre

starts @ Periphery

Schultz Method*

Duncan's Method*

• external — after complete separation
bleeding

begins @ onset of separation

• Blood loss ↓ (Total)

Total blood loss ↑

• side presents @ vulva = fetal side

Maternal side



Shiny side

Shiny

Shiny

↳ Schultz*

↳ Duncan's Method*

80% cases (M/c separation)

20% cases

• Retroplacental clot or is formed

• Absence of Retroplacental clot.

* M/c of PPH ⇒ Atomy > Genital tract Trauma > Retained tissue > Inversion > Rupture Uterus > Amniotic fluid embolism.

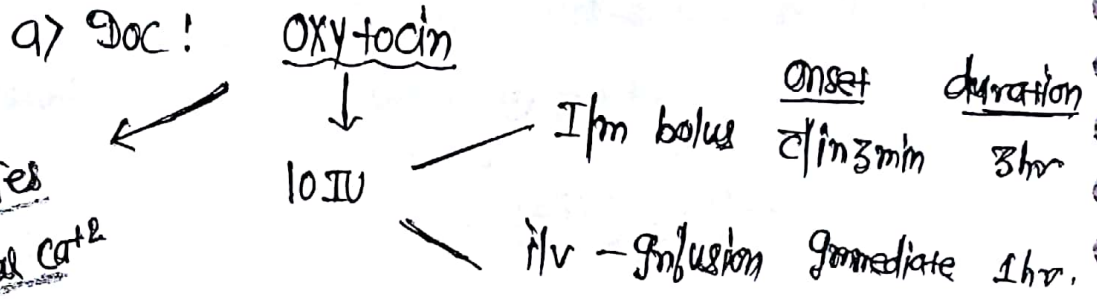
Q9

Prevention of PPH (AMTSL)

↳ by WHO
Active Management of Third stage of Labour.

Components ⇒

1) Give uterotonic Agent Immediately after delivery
↓
2/1m 1min.



MOA ⇒ by Tes
Intracellular cat²

• Oxytocin causes
Release of PUF₂s
from decidua.

R/F for = Hypotension
↓

Reflux Tachy. | Arrhythmia | MI | Cardiac Arrest Q9

- Naturally synthesis oxytocin ⇒ Nonapeptide
- Artificially synthesis oxytocin ⇒ Octapeptide

- Synthesized from - hypothalamus
↓
Paraventricular Nucleus

- t_{1/2} = 3min (3-5min)
- Stored in cold chain (2-8°C)

↓

(42)

b) Methergin (ergometrine)

↳ 0.2 mg i/m

↓
don't give i/v → causes transient severe hypertension

So; CI'm ⇒ Pre-eclampsia

Eclampsia

CVS disease

Peripheral vascular disease

tetanic contraction (Acts More on LUS; while Oxytocin on all uterus).

Brown colour - b/c of photosensitive nature.



c) Syntometrine (5 U Oxytocin + 0.5 mg Methergin)

• Very Potent

• Not doc → expensive

→ Not available



d) Carbetocin = Synthetic oxytocin

↓
Octapeptide / Longer t/2

↳ 100 mg slow i/v over 10 min.



e) Misoprostol = PGE₁ analogue



Prophylaxis = 600mg (per oral)

Route = oral (In India ⇒ ~~per~~ Rectal)

Asthma is Not a C/I.

M/c side effects ⇒ ^{**} Hyperpyrexia (fever + chills).

↳ directly proportional to dose

Other side effects ⇒ Nausea; Vomiting; Abd. Pain; Hypotension.

II) Delivery of placenta by controlled cord traction ⇒

eg Klaus "Modified Brandt-Andrew Method"

Rt. hand ⇒ Hold cord ↳ do only when trained birth staff present.
Lt. hand ⇒ Push Fundus up.

III) Delayed cord clamping ⇒

≥ 60 sec. (1-3min)

↳ Goes More blood to fetus (80ml)

↑ Hb by 2gml. ← 50mg Fe

In HIV patient ⇒ delay cord clamping done
transmission happen during Labour.

↳ Never done due to
AMTSL in Normal Labour.
early cord clamping

↳ \bar{c} in 60sec.

Indications ⇒

- Baby Needs Resuscitation
- Rh Incompatibility
- Baby is known case of Heart disease

IV) Intermittent Uterine tone assessment ⇒

Uterine Massage = Not done

Q9 Most Imp. component?? ↳ Not a component of AMTSL.
(A) I; (B) II; (C) III; (D) IV ↳ 1st component hypotension & tachycardia blood loss by 20%.

Management of PPH (Shock Index = $\frac{\text{Heart Rate}}{\text{SBP}}$)

Symptomatic T/t

- i) 2 Large bore ilv
Cannula
(14/16 Gauge)
- ii) ilv fluids - crystalloids
- iii) Arrange blood & blood products;
- iv) Catheterize - Urine output
- v) Blood group / Rh / CBC / Coagulation Profile

to decrease Morbidity & Mortality Rate.

↳ (N) = 0.5-0.7
if > 0.9 ⇒ Immediate Resuscitation

Specific T/t (Algorithm)

- BiManual T
-) B/M Uterine Massage
- + call for help
- + Uterotonics given
- ↳ DOC P/t
- ↳ Oxytocin
- ↳ 20IU — 40IU / 500ml of NS/RL
- ↳ Not in 5+ dext.

If we give Large doses } in Large duration } in electrolyte deficient mediq

Result in uterine intoxication.

↓ ib Not Responding

Inj. Tranexa (Tranexamic Acid)

↓

↳ Anti fibrinolytic drugs

Anti fibrinolytic
1gm slow

↓

ib Not Responding

Methergin
0.2 mg (i/m)

carboprost (Methyl PGE₂)

↓

0.25 mg (i/m)

↳ H/O Asthma ⇒ Not given; Pt. dies due to bronchoconstriction

↓

Diarhoea (M/c side effect of carboprost)
ib Not Responding.

Misoprostal → to be used when i/m; i/v can't be given.

↳ Ht = 800mcg (sublingual)

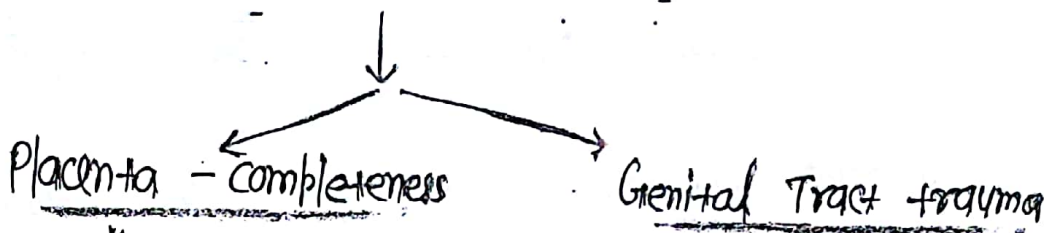
Not given P/V (Per vagina)

can give P/R (Per Rectal)

* All drugs total to happen upto 30min.

↓ ib Not Responding.

(44)



↓
 Uterus feel Like Intermittent atonicity
 ↓
 i.e Not tonically contracted b/c of Retain Placental tissue

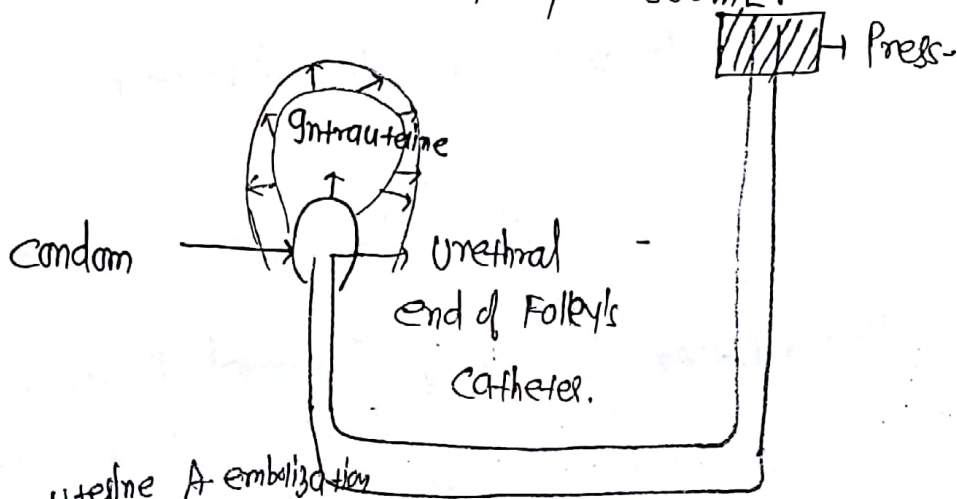
↓
 PPH ⇒ Uterus tonically contracted then Reason is

No trauma
 Placenta complete

Balloon Tamponade

Bakri Balloon tamponade
 Sengstaken Blakemore

Max^m Fluid capacity = 500 mL.



uterine Arterial embolization
 do UAE ← Minimally Invasive Method if Not Responding
 ↓
 do in hemodynamically stable Pt. Surgical method
 • Availability.

Surgical Management

- Uterine compression suture (B-Lynch suture) → Brace



↓
Applied on uterus & helpful
only in Atonic PPH

↓ if Not Responding

B/L Uterine A. Ligation

↓ if Not Responding

B/L - Anterior division of Internal iliac

↳ 5 cm distal to bifurcation of common iliac
It ↓ ves Pelvic pulse pressure by 80%

↓ if Not Responding

Hysterectomy (Possible $\frac{to}{do}$ Sub total Hysterectomy
(Remove uterus - spares the
Klas "Supracervical Cervix
Hysterectomy")

↓ if bleeding continues think about "D.I.C."
G-CU.
↳ DIC**

↳ to improve sexual life of female

TRAUMA → Perineal tear

differs from episiotomy ⁽⁴⁵⁾



to prevent

It is surgically planned incision.

① do Routine episiotomy ⇒ No

② one hand = Support the perineum

done in special cases



Forceps; Breech condⁿ

③ other hand = Maintain flexion of head

MedioLateral

Median

④ tell the Mother Not to push @ the time of head delivery.

• ↑ Pain

• ↑ Dyspareunia

• breaks down easily

• ↑ Blood loss

• Poor Cosmetics

↓
extends

↓
Rectum sphincter & Mucosa

⑤ NICE ⇒ application of warm Guidelines Perineal compresses

degree of Perineal tear ⇒

Repaired In Labour Room. 1st degree — vaginal Mucosa & Skin

2nd degree — Perineal Muscle

3rd degree — A — < 50% EAS torn

complete Perineal tears B — > 50% EAS torn

C — Both IAS & EAS torn

4th degree — Upto Rectal mucosa

Complete Perineal tears ⇒ obstetric emergencies
 ⌊ In 24 hrs
 ↳ 3 weeks

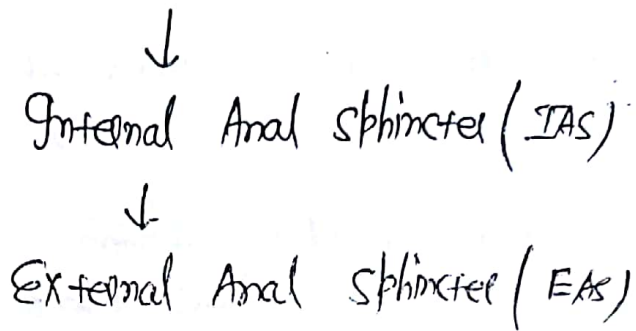
1st to be Rejoined ⇒ Rectal Mucosa

Sphincter ⇒ End-End Anastomosis

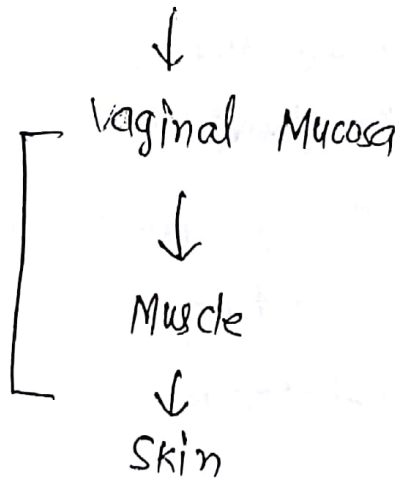
Mucosa = Continuous suture

Muscle = Interoblique suture

Skin = Mattress suture

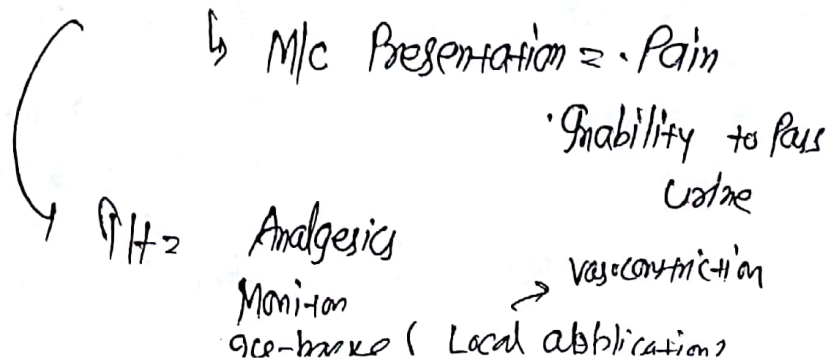


Episiotomy



M/c used suture = vicryl (Polygalactin-910)

* Another Tear ⇒ Hematoma



- Looks like bluish tender swelling
- Surgical Mx =
 - ① Shock
 - ② expanding in size
 - ③ excruciating Pain (hematoma expanding internally)
 - ↳ Muscle.

46

* In Inversion ⇒

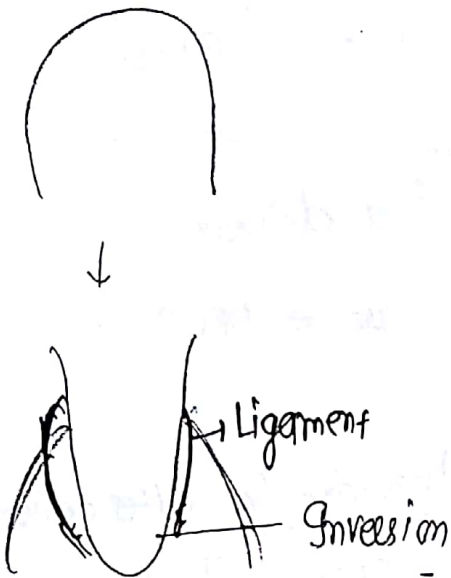
Stretch on the Ligament



Neurogenic Shock (1st shock to develop)

↳ Hemorrhagic shock

↳ death.



Rx ⇒ do Manual Repositioning (Johnson's Method)

Part which comes out last; has to be Reposited first.

↓ ib balls

Hydrostatic Method (O Sullivian Method)

↓ ib balls

surgical management

Surgical Management
for Inversion

Haultain
Huntington
Spinelli

— emergency; give general Anesthesia; Not spinal Anesthesia
↓
Relaxes uterus
also in Inversion
Manual Removal of Placenta
Hemodynamically Unstable patient

QA. Pt. goes into shock after delivery ^{or death}
Most probable cause ⇒ PPH

QA Pt. goes into shock immediately after delivery
- Most probable cause ⇒ Inversion

QA Pt. goes into shock (unexplained) after delivery
Most probable cause ⇒ Amniotic fluid embolism
(Very Rare / diagnosis of exclusion)
↳ "Klas" Anaphylactoid syndrome of Pregnancy

→ No Lab test to confirm or Rebutte diagnosis.

* Clinical diagnosis of AFE ⇨

(47)

1st phase ⇒

Sudden onset of breathlessness



Highly Mortality



hypotension



Cardiac arrest



Coma

2nd phase ⇒

Pt. goes into DIC + Hemorrhage



death seen

Test we can do ⇒

Sample from Pulmonary vessels



Lamugo | vomit | amniotic fluid seen

DIC

M/cc = - Abruption

also by = AFE

Massive Hemorrhage (APH/PPH)

Sepsis (septic abortion)

and (2 weeks risk of DIC)

Management of GUD \Rightarrow wait & watch



Must go into spont. Labour \bar{c} in 2 weeks

\hookrightarrow signs \Rightarrow Robert's sign \Rightarrow Comes @ 12 days
 \hookrightarrow gas in Major blood vessels

Spalding sign \Rightarrow comes @ 7 days

Ball sign \Rightarrow Hyperflexion of spine

Buddha \Rightarrow subcutaneous edema

sign

\hookrightarrow seen in hydrops fetalis

\hookrightarrow Not a sign of GUD.

PRE ECLAMPSIA

(48)

- * Gestational HTN \Rightarrow i) BP \geq 140/90 on 2 occasions;
4-6 hr apart
- ii) BP high $>$ 20 weeks;
- iii) Return to (N) \bar{c} in 12 weeks Post-partum.
iv) No evidence of Proteinuria

* Blood Pressure should taken in sitting position.

* sth kontroll sound heard

* Pre-eclampsia \Rightarrow i) BP \geq 140/90 on 2 occasions; 4-6 hr apart

ii) BP high $>$ 20 weeks

\bar{c} Any of the following

Proteinuria

\geq 0.3 gm / 300mg

in a 24 hr Urine Sample

or

Primary protein \geq 0.3
creatinine

OR

Dipstick (1+)

End-organ damage

(It could be Any of the following).

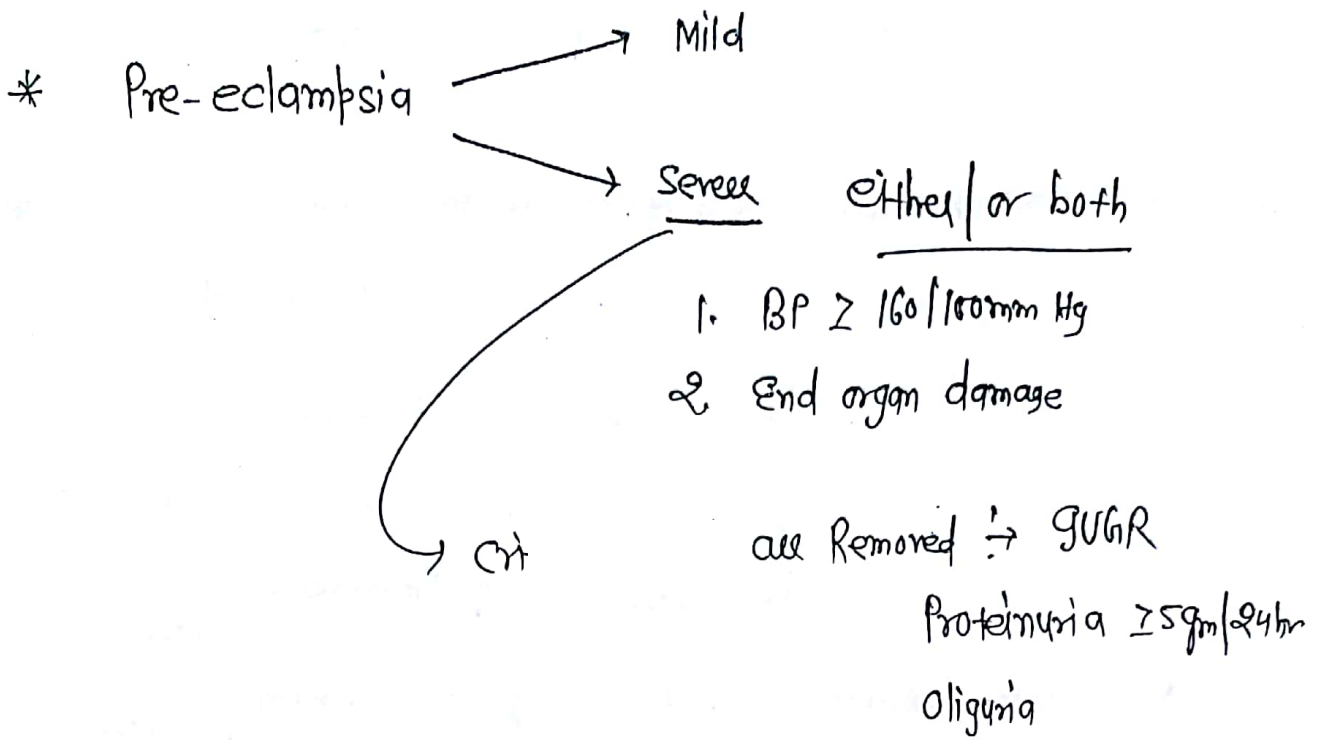
i) platelet count $<$ 1 Lakh;

ii) Serum creatinine $>$ 1.1

iii) Liver enzymes all More than
twice the (N) value

iv) Pulmonary edema

v) cerebral / visual symptoms



* Eclampsia \Rightarrow Pre-eclampsia + Seizures

* Chronic HTN in ♀ \Rightarrow

- BP is high before conception;
- BP is high in 1st 20wk;
- BP Remains high $>$ 62 week post partum period.

* Chronic HTN \bar{e} Superimposed Pre-eclampsia \Rightarrow

- New onset proteinuria beyond 20 weeks.
- End organ damage $>$ 20 weeks
- Uncontrolled HTN $>$ 20 weeks

- Impending eclampsia \Rightarrow if she has Any of the following sign & symptoms \Rightarrow $\text{in epigastric pain (49)}$
 \hookrightarrow stretching of Liver capsule.
 Spontaneous subcapsular hematoma

ii) Headache / dizziness
 \hookrightarrow cerebral hypoxia

iii) Blurring / diplopia / Blindness
 \hookrightarrow ~~Blindness~~ ^{central} ~~central~~ ^{retinal} ~~retinal~~ ^{Lobe hypoxia} ~~Lobe hypoxia~~
 Scotoma \hookrightarrow Peripheral = Retinal detachment
 \hookrightarrow Hypertensive Retinopathy

iv) HELLP Syndrome
 \hookrightarrow Mostly - Feature of severe Pre-eclampsia
 \hookrightarrow Blood pressure in 1st of patient is (N)

Criteria \Rightarrow H = Hemolysis (in Peripheral Smear)
 \hookrightarrow Schistocytes (Helmet cells);
 \hookrightarrow S. Bilirubin ≥ 12
 E \Rightarrow Elevated Liver enzyme (ALT ≥ 70 IU)
 L \Rightarrow Low Platelet count (Plt. Count $< 125,000$)

all should be in patient \Leftarrow

* M/c Presentation \Rightarrow Pain \rightarrow epigastric pain
 (Rt. upper quadrant)
 \Downarrow
 Seen in 3rd trimester

D/D \Rightarrow ~~i~~ Acute Fatty Liver of $\textcircled{+}$ (closest to HELLP)
 of HELLP Sx ii) Hepatitis
 iii) obstetric cholestasis

differentiated by presence of

- ① Hypoglycemia
- ② Hepato Renal Sx
- ③ Coagulation defect
- ④ Pancreatitis

Pathophysiology \Rightarrow i) $\textcircled{\text{ABN}}$ of β -oxidation of Fatty acids — Mother (Mitochondrial)

Mainly in 3rd trimester
 (More severe form Liver injury) ii) LCHAD enzyme deficiency — Fetus
 by Postmortem Liver Biopsy confirmed.
 M/c cause of Acute Liver failure in $\textcircled{+}$ = AFLP

— High Mortality Rate

* Acute hepatitis

↳ have Prodromal symptom.

(50)

- Liver enzyme Raised

- Bilirubin - Markedly Raised

M/c Acute hepatitis in ♀ ⇒ Hepatitis E

↳ high Mortality Rate

* Obstetric cholestasis

- M/c Symptom ⇒ Pruritis

- seen in 3rd trimester

- Estrogen

- Mutation in genes ABCB4
ABCB11

- Serum bile acids become accumulating

↳ Diagnostic test

- IT ⇒ Urso deoxycholic Acid

Most Risky for fetus = Pre-term Labour;

↑ Irritate the bile acids; ⇐ Sudden still birth;

Result in the all of
three.

Meconium Abstraction syndrome

Termination of \odot \Rightarrow (≡) 37 weeks \rightarrow (≡) 38 weeks
 In obstetric cholestasis

* * Recurrence Risk of HELLP = 4-7%
 Obstetric cholestasis = 70%

* Pathophysiology of Pre-eclampsia \rightarrow

Vascular Remodelling

Absent

\Downarrow

Vessels in Decidua Basalis

\downarrow

\hookrightarrow it means remain sensitive to vasopressin

Result in Utero Placental Insufficiency

\rightarrow Vasodilators
 PLGF, VEGF etc

\hookrightarrow Pl. Ischemic

\rightarrow Inflammatory Mediators Release

SFlt-1; s-Eng both tes

Pale & Small Placenta seen in Pre-eclampsia,

\rightarrow Soluble FMs like tyrosine kinase
 S-Flt-1 } both are vasoconstrictors
 s-Eng }

\rightarrow Soluble-endoglin

\downarrow

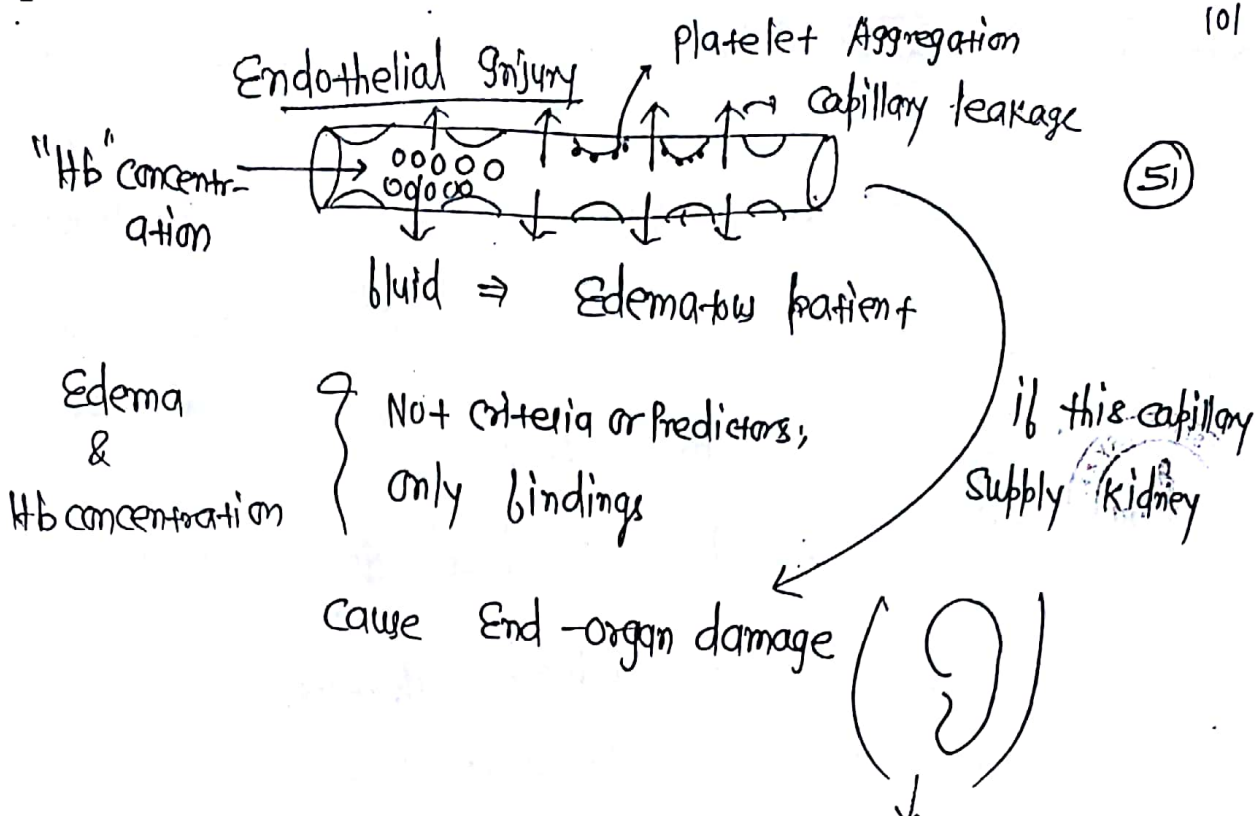
Endothelial activation

\downarrow No availability

\rightarrow \uparrow Ris, PAF

HN

1st sign & symptom in Pre-eclampsia



1st & M/c organ affected in Pre-eclampsia

Kidney shows "Glomerular Endotheliosis" on HPE.

↑ Serum Uric acid => also a ~~criteria~~^{finding} not criteria

* Immune theory :-

Normally => ↓ TH₁ → ↑ TH₂ Response

In pre-eclampsia - this shift doesn't happen

↓
So, ↑ TH₁

↓
Altered Immune Response to paternally derived Antigen.

Risk factor

i) Primigravida; (b/c 1st time expose to Antigen)

ii) Molar pregnancy (Extra paternal chromosome)

↳ can develop early onset pre-eclampsia;

iii) APLA

iv) Multifetal @

v) Chronic Hypertension

vi) Renal disease;

vii) Diabetes Mellitus;

viii) obesity

ix) extremes of age < 18; > 35 years

x) Previous H/o Pre-eclampsia

Protective factor

i) Smoking

ii) It has \ominus ve association \bar{c} Placenta previa;

It has \oplus ve association \bar{c} Abruptio.

Predictors of Pre-eclampsia

i) \uparrow sFlt-1

ii) \uparrow s-eng

iii) \downarrow PLGF

iv) \downarrow VEGF

v) Uterine & Dobbles

vi) \downarrow Urinary cate excretion,

* Uterine A. Doppler

(N) → Notching — disappear by 22 weeks

(52)

Persistent + Notching — beyond 22 wk — Predictor for Pre-eclampsia

Prevention of Pre-eclampsia

⇒ • Aspirin

↓
150 mg — once in a day
— start in 1st trimester

↳ Continue throughout pregnancy
↳ only indicated for high Risk patient for Pre-eclampsia

• Ca Supplementation

↳ Prevent Pre-eclampsia only in women who have Ca²⁺ deficient

- Salt Restriction
- Omega 3 Fatty acids
- Vit. C, E

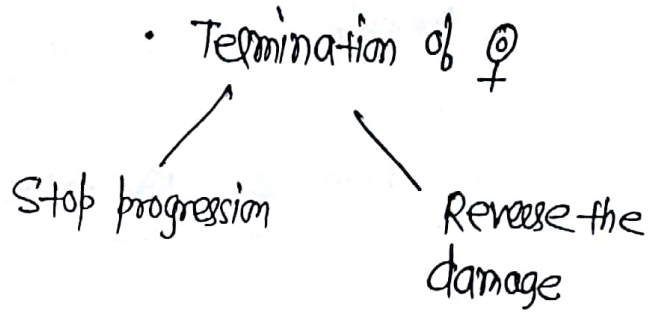
} No Role in prevention.

* Management of Pre-eclampsia !⇒

Symptomatic

- Anti-hypertensive
- Anti-seizure

Specific



Anti hypertensive

i) When do you start Anti-hypertensive

↳ if BD persistently $\geq 150/100$ mm of Hg

ii) Which drug — doc

↳ Labetalol ($\alpha + \beta$ β -Blocker)

but; in Chronic Hypertension

↳ Methyl dopa (safest Antihypertensive in Pre-eclampsia)

Doc for Acute HTN in ♀ ($\geq 160/110$)
mm of Hg

↳ acc. to ACOG, Any of these we as 1st line drug

①
iv Labetalol

②
iv hydralazine

③
Sustained Release
Nifedipine

iv Labetalol



20 mg iv

↓ after 10 min

40 mg after 10 min → 80 mg → 80 mg upto total 300 mg/24 hr.

(53)

(4) iv Nitroglycerine ⇒ used in Pulmonary edema

(5) Lat + Resist Refractory ⇒ Sodium Nitropruside
Anti hypertensive drugs cause (cyanide toxicity)

* Methyl dopa is Not given in Acute HTN
↳ b/c

ii) drugs not to be used as Antihypertensive →

i) ACEi ;

ii) ARBs ;

iii) Diuretics ;

iv) β Blockers ;

v) Diazoxide

↳ Target Blood Pressure ⇒

Systolic blood pressure = 120-130 mm of Hg

Diastolic blood pressure = 80-90 mm of Hg

TERMINATION OF PREGNANCY

Gestational Hypertension / Mild Pre-eclampsia ; well controlled BP

↳ @ 37 weeks

Severe Pre-eclampsia — well controlled — POP — @ 34 weeks

* Indication of Termination of ♀ Irrespective of Gestational age →

i) Impending Eclampsia;

ii) Eclampsia

iii) HELLP Syndrome;

iv) Abruption / Fetal distress

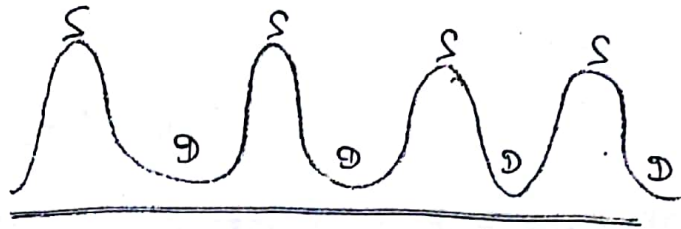
v) Uncontrolled HTN / Rising serum creatinine

vi) REDF — Reversal of End diastolic flow

↳ doppler of Umbilical Artery.

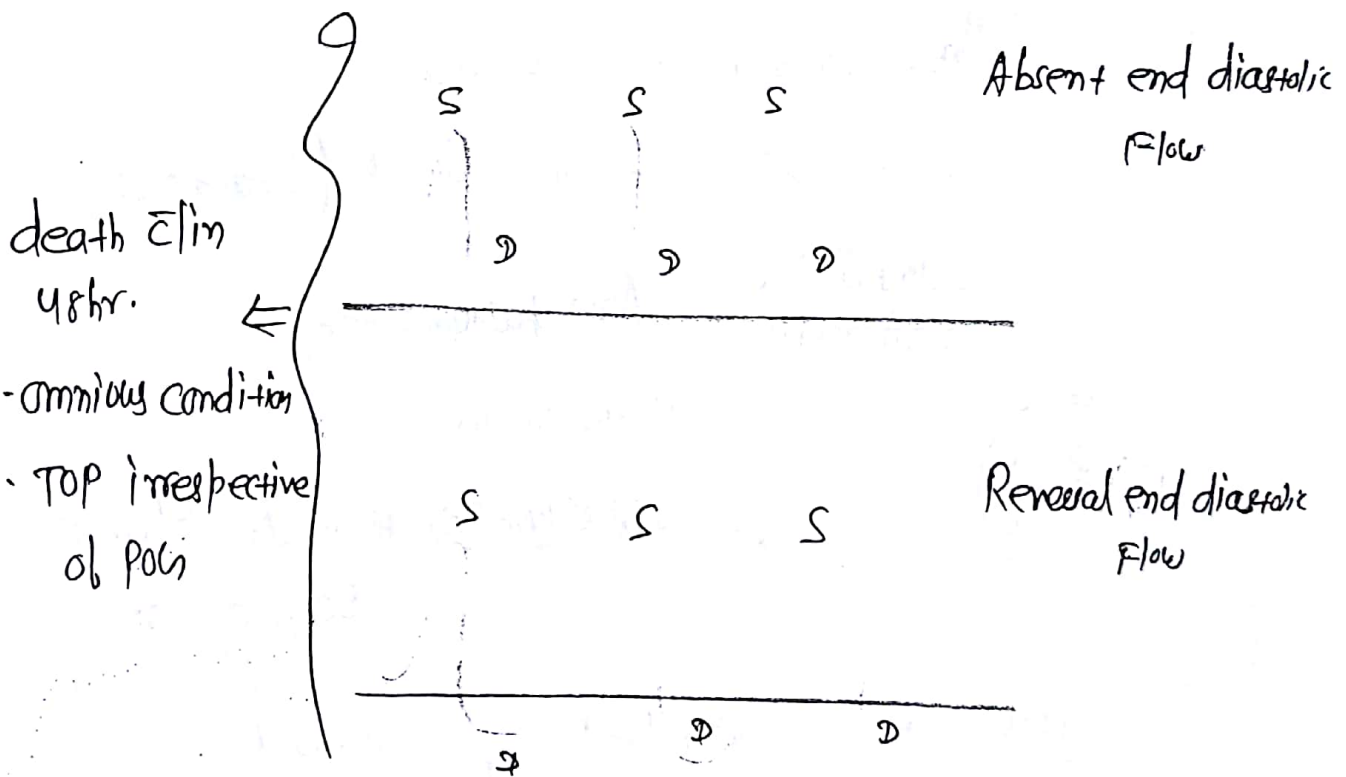
(N) Umbilical Artery Flow $\Rightarrow \frac{S}{D}$ Ratio

(54)



In (N) Pregnancy $\Rightarrow \frac{S}{D}$ Ratio \downarrow as pos tes

In UPI $\Rightarrow \frac{S}{D}$ Ratio tes ($\geq 30 \frac{S}{D} \geq 3$)



* Whenever possible do vaginal delivery \gg cesarian section
 (High Risk)
 Anesthetic complications
 blood loss

*

In C.S.

↳ epidural Anesthesia given

↳ if Not given then

Mark Neuraxial Anesthesia

Never give General Anesthesia

ECLAMPSIA

Preeclampsia + Seizures

↳ GTCS (Grand mal)

1st seizure ⇒

Antepartum (M/C)

Intrapartum

Postpartum ⇒ It Means Clin 48hrs seizure comes

Causes ⇒

Cerebral hypoxia

+

Cerebral edema

Rx ⇒

doesn't do any peripheral

vasodilation

MgSO₄

⇒

central Action

NMDA Receptor

↳ cerebral vasodilation

* In Periphery $MgSO_4 \rightarrow Ca$ channels

(55)

So; don't club Ca^{2+} -channel blockers (for HFN) & $MgSO_4$

Causes "Neuromuscular Paralysis"
So; $MgSO_4$ gives & Labetalol (anti-HFN)

* 1st ly - looks for vitals

↓
Stabilize the position (by tie her legs & hands)

↓
Secure Airway — Mouth GAG
Suction
Oxygen by Mask

↓
Inj. $MgSO_4$ + Anti-HFN

Pritchard's Regimen

↙
Loading dose

↓
14 gm
↓
4 gm - Slow i/v @ 1 gm/min
12 gm - i/m (can be over 24 hrs)

Monitor by Heart Rate

↘
Maintenance dose given every 4 hourly until 24 hr after delivery or sub. after Latex seizure
↓
before giving dose check \Rightarrow Patellar Reflex \oplus RR > 12 /min
↓
then give 5 gm i/m dose (at alternate site) Urine output > 30 ml/hr to check absorption

16 Any of the Patellar Reflex \oplus or RR $> 12/\text{min}$ or Urine output $> 30\text{ml/hr}$ } Any Absent
 ↓
 Omit the dose
 ↓

Send serum Mg^{+2}

↓
 Therapeutic Level = 4-7 meq

① 9-12 meq }
 ① 10 meq = Patellar Reflex absent } warmth, diaphoresis, slurring of speech
 ① 12 meq = Respiratory distress }

Antidote for $\text{MgSO}_4 \Rightarrow 10\text{ml of } 10\% \text{ 1hr Ca gluconate}$

* Indication of Prophylactic $\text{MgSO}_4 \Rightarrow$ In Impending Eclampsia;
 In HELLP syndrome;
 In severe preeclampsia

* oliguria is Not a toxicity symptom of MgSO_4 .

Q. Q. Primigravida 34 weeks gestation; Casualty with headache x 4hrs; 2 episodes of vomiting of E BP=160/110 mm Hg; FHR-(N); Most appropriate step of Mx (56)
 Fundal height = 34 weeks; Urine output Protein (2+).

(Firstly) MgSO₄ + Anti HFN + TOP
 (MgSO₄) → then (Anti HFN) → then

* Eclampsia is indication for TOP
 ↓
 vaginal delivery > C.S.
 delivered within 24 hours

DIABETES IN PREGNANCY MELLITUS

Over+

Pregestational

Known case of "Diabetes Mellitus"

Gestational

1st time deranged
sugars are in pregnancy
diagnosed

- Most common

Single Step

↳ both screening & diagnostic

2hr OGTT

- National guideline GIPSI criteria

- done @ 24-28 weeks

- glucose load 75 gm (oral)

- ± Fasting

↳ after 2 hrs
75 gm oral
glucose

Blood sample

> 140 - GDM

≥ 200 - overt GDM

120-140 = Impaired glucose tolerance
of pregnancy

* All @ Women Must undergo & have OGTT

↳ Universal Screening (57)

* Early testing — 1st Antenatal visit

① if she gives H/o baby \in GCA

② if she gives H/o Still birth

③ if she gives H/o Macrosomic baby

④ if she gives H/o Diabetes — 1st degree

⑤ if she is obese

HbA1c

FBS
or

HbA1c

Fasting $\geq 126 \Rightarrow$ overt diabetes.

* Diabetes \Rightarrow High Risk

For Foetus

↳ ↑ Risk of Gross Congenital Anomaly by
four fold in compair to @ ♀

↓

dist hyperglycemia (feto-toxic)

↓

Free Radical Injury

* Risk of GCA Tes in over-
all gestational Tes after
24-28 weeks

* M/c GCA \Rightarrow CVS > NTD

* Most specific \Rightarrow Caudal Regression Syndrome
(Santal Agensis)

* M/c CVS Anomaly in fetus \Rightarrow VSD
of diabetic Mother

* M/c Specific Anomaly in fetus \Rightarrow TGA
of diabetic Mother

* M/c finding in fetus of diabetic \Rightarrow HOCM
Mother

li>NFB = Amnencephaly

Frog eye sign / Mickey Mouse sign on USG

Amnencephaly — earliest \Rightarrow 10 weeks

— diagnosed \Rightarrow 14 weeks

* Screening / diagnosis \Rightarrow USG

* Recurrence Risk = 4% ; Recurrence Risk = 10%
in previous 1 amnencephaly baby ; in previous 2 amnencephaly baby

(58)

* Max^m complication seen in female foetus except ⇒
 Mamosomia } In Male
 Respiratory complication }

* Anencephaly seen in Polyhydramnios ♀
 ↳ Post-term Labour > Pre-term Labour
 (Initiation of Parturition ⇒ CRP)
 ↳ Mainly Face Maldevelopment seen.

* Banana sign } seen in spinal
 Lemon sign } bilida & Arnold-Chiari syndrome.

- Test - low Risk of Anomalies
 HbA1c ⇒
 @ 6.5 ⇒ Risk tes by 3%
 @ 7.5 ⇒ Risk tes by 4%
 @ 8.5 ⇒ Risk tes by 8%
 > 9 ⇒ Risk tes by 15%

* USG ⇒ low Anomalies diagnosis → Fetal imaging for fetal anomalies
 ↳ @ 18-20 weeks ↳ Level-II (TIFFA)

Diagnostic for Macrosomia



EBW \geq 4kg



on US

↳ single - Fetal growth



Abdominal circumference (Macrosomia IUGR)

* USH also used to calculate gestational age

Best In 1st Trimester for gestational age = CROWN-RUMP Length

2nd " = Biparietal diameter

3rd " = Femur Length

Best for Gestational Age = CRL

* Best for gestational Age →

(A) CRL for 16 weeks

~~(B) BPD @ 16 weeks~~

(C) Femur Length 30 weeks

(D) Abdominal circumference 30 weeks

* Macrosomia Patient presents \bar{c} Shoulder dystocia

↓
> 1mm delay in the delivery of
Shoulders after delivery of head

↓
TURTLE SIGN



↳ Sudden pulling back of head towards
Maternal Pelvis

* Mx of Shoulder dystocia ⇒

H — Call for Help

E — empty bladder [episiotomy do]

L — 1st — Legs — Mc Robert's Manoeuvre

↳ Sudden flexion & Abduction of Maternal
thigh on the Maternal Abdomen

P — Suprapubic pressure
Not fundus pressure

↳ It ↑ space in A-P
diameters

E — Enter — Wood's screw Manoeuvre
OR

R — Removal of Posterior Arm

R — Roll over on 4 limbs



gaskin all boys

↓
We injured Lateral
cutaneous Nerve of thigh



Metalgia Paresthetica

Last Manoeuvre ⇒

Zavanelli Manoeuvre

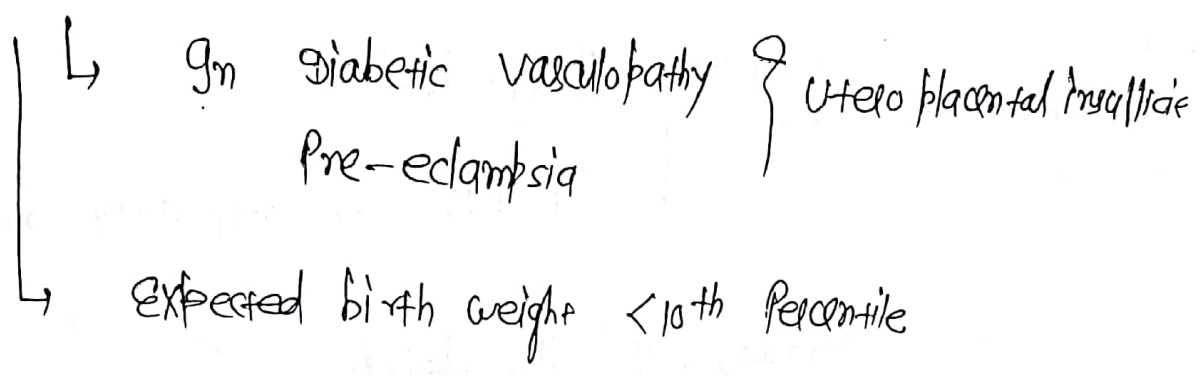
↳ push head back & do C.S.
into mother

* Theoretically - destructive procedure - Symphysiotomy
 - clodotomy (60)
 - Gatrogenic # clavicle

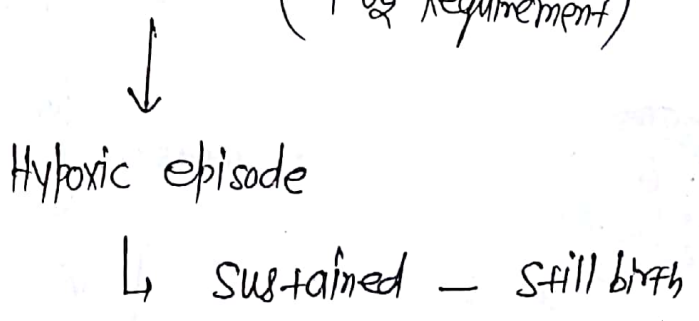
* M/c fetal injury in shoulder dystocia ⇒ Brachial plexus

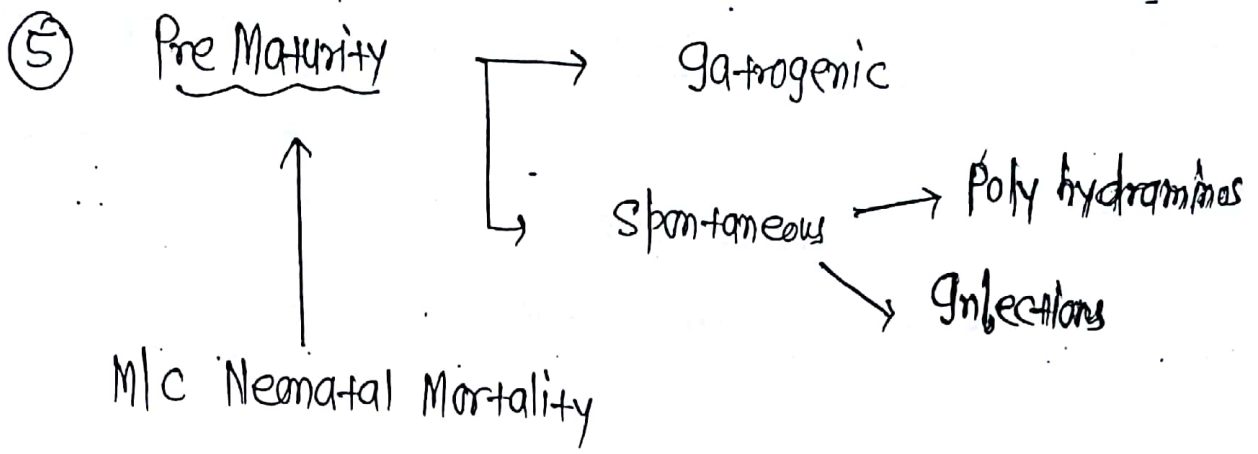
* M/c Maternal complication in shoulder dystocia ⇒ PPH

3) GUR ⇒ Rare (common)



4) Still birth ⇒ Seen in Macrosomic baby, Male baby
 (↑ O₂ Requirement)





⑥ Lung Maturation is delayed

↳ Phosphatidyl glycerol =
Not L/S Ratio

⑦ operative delivery

↳ ↑ Risk of Respiratory Problem

⑧ Immediately after delivery → i) Hypoglycemia

ii) Polycythemia

iii) Hyperbilirubinemia

iv) Hypocalcemia } Prematurity

v) Hypomagnesemia }

vi) RDS

vii) HOCM

* Maternal complication of Diabetic Mother! →

i) Abortion ⇒ Uncontrolled diabetes (61)

ii) Polyhydramnios ^{causes} → GICA
 → ↑ glucose in Amniotic fluid
 → Hyperglycemia in fetus (Polyuria)

iii) oligohydramnios (uncommon)

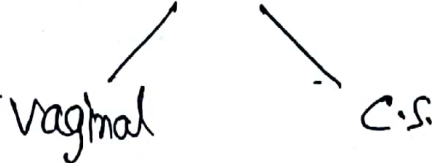
seen in → Diabetic vasculopathy } in utero-placental
 Pre-eclampsia } Insufficiency

iv) Pre eclampsia ⇒ 1st Risk Tes

v) Infections — UTI
 — Candidiasis

vi) complication of diabetes — Vasculopathy
 — Retinopathy
 — Nephropathy
 → pre existing = worsen in pregnancy

vii) operative deliveries



viii) PPH (over distension)

ix) P.f. can develop later overt diabetes (in 50% cases)

follow up \rightarrow 6 week (Post partum visit) | 12wk

by OGTT test

* Management of Diabetes Mellitus in ♀

Maternal Monitoring

- Blood Sugar Level

overt diabetes



Pregnancy



Insulin

GDM



only diabetes diet

40% = Carbohydrate

40% = Fats

20% = Proteins

↓ * after 72 hr

do sugar profile

Fetal Monitoring

- Start to Monitor at 32wk

DFMC (daily fetal Movement Count);

NST

BPP (Biophysical Profile)

USG

; Doppler Never use

only in

GUR

UTI

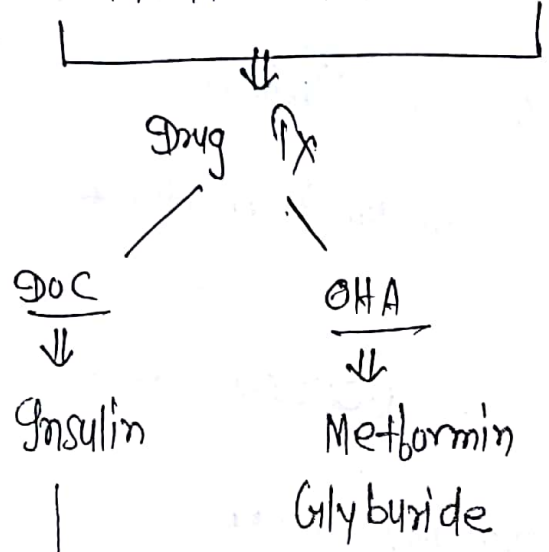
Sugar Tolerance

Fasting ≥ 95 or

1 hr PP ≥ 140 or

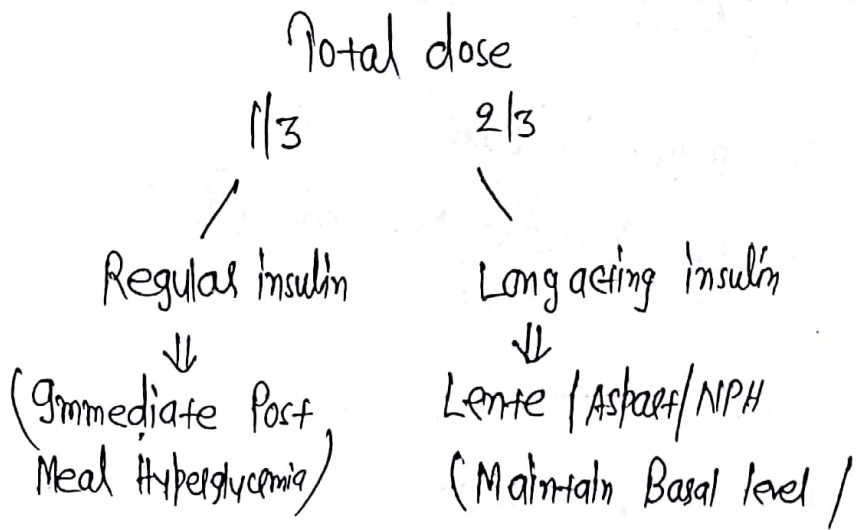
2 hr PP ≥ 120

(62)



↳ It doesn't cross placenta.

M/c Insulin use \Rightarrow Regular Insulin



- 1-12 wk = 0.7 U/kg
- 12-28 wk = 0.8 U/kg
- 28-34 wk = 0.9 U/kg
- ≥ 35 wk = 1.0 U/kg

Target Levels

- F < 95
- 1 hr PP < 140
- 2 hr PP < 120
- Avg. glucose = 100
- HbA1c $\leq 6.5\%$

* TOP = Gestational diabetes on diet alone = EDD = 40 weeks

over + GDM on Insulin = 39 weeks

* Diabetes (Alone) is Not an Indication for C.S

* Expected birth weight ≥ 4.5 kg in a diabetic pregnancy

↓

Cesarean section

* In Non diabetic pregnancy ≥ 5 kg

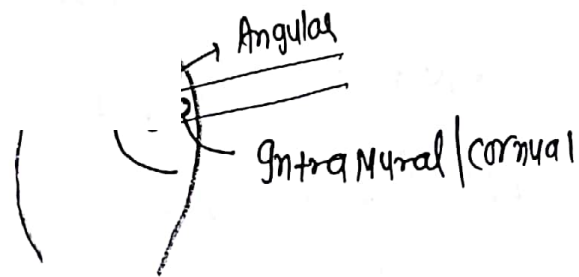
* Insulin Requirement in Labour \rightarrow ↓

↓
Stop Insulin in Labour

& do Intensive Monitoring @ 2hrly.

ECTOPIC PREGNANCY

- Any pregnancy is implanted outside the uterine cavity. (63)
- Cornual Pregnancy → ectopic ♀
Intramural part of Fallopian tube
- Angular Pregnancy → Intramural ♀
↓
gle of uterus



* Round Ligament is attached lateral to the growth of uterus

↳ Angular ♀

* Round Ligament is medial to growth of uterus

↳ Cornual ♀

* Heterotopic ♀ ⇒ 2 pregnancy simultaneously @ different sites of implantation.

Most common ⇒ Intramural + Intrafallopian tube (diff site)

- Risk factors -

- i) Highest Risk of ectopic - H/O Previous Ectopic
 - 1 Previous History $\xrightarrow{\uparrow \text{Risk by}}$ 15%
 - 2 Previous History $\xrightarrow{\text{"}}$ 30%
- ii) 2nd highest Risk - H/O Tubal Surgery
- iii) M/c Risk factor - PID
- iv) Cervicitis
- v) Infertility
- vi) ART (GVF)
- vii) Smoking
- viii) Previous C.S
- ix) Contraception & Ectopic

L All contraceptive \downarrow Absolute Risk of Ectopic;

but

\uparrow Relative Risk of Ectopic

L Tubal Ligation > IUD > POP

doesn't inhibit ovulation

so: More Risk of ♀ (ectopic)

Intrauterine Device
Progestin
terane
Oral pills

Order of gus in vesing order of Tes Relative Risk
 of ectopic ♀ ⇒ Progesterone > Mirena > Cu IUD (64)
 ↓
 It has gus & Progesterone both.

* M/c site of ectopic ⇒ Fallopian tube
 M/c = Ampulla site
 L/c site = Intramural

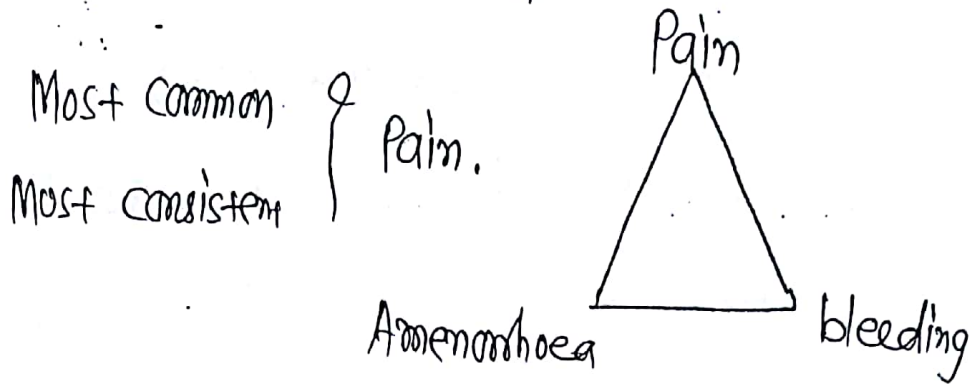
* L/c site of ectopic ⇒ C.S. Scar > Cervical > Abdominal
 <17

* M/c outcome of Ampullary ectopic ♀ = Tubal Abortion
 2nd M/c outcome of Ampullary ectopic ♀ = Rupture @ 8th week

* M/c outcome of Isthmic ectopic ♀ = Rupture @ 6th week

* M/c time of Intestinal ectopic ♀ Rupture = 12th week
 OR
 Intramural
 ↓
 Most Life threatening
 ↓
 Hemoperitoneum ↑↑

* Trial of: ectopic ♀ ⇒ seen in soft patient



- H/o Nausea; vomiting

Shoulder tip pain

↳ Hemoperitoneum

↳ Diaphragm

↓

Referred Pain via Phrenic Nerve

- Syncopal Attack

- Per Abdomen examination ⇒ Tenderness in Lower Abdomen

↳

Rigidity/ Guarding

d/+ Peritonitis

↓

i.e Ruptured ectopic

Per vaginal examination ⇒ Uterus - enlarged; less Period of gestation.

Cx Motion tenderness

Salpingitis

↓

dilate by Pils ⇐ PID ⇒ close to for ectopic.

Adnexal tenderness

Adnexal Mass QA

65

Investigation \Rightarrow UPT \oplus ve @ 99% times

Investigation of Choice \Rightarrow Trans vaginal ultrasound



1st finding ; which raises suspicion

\hookrightarrow empty uterus

finding ; which raises suspicion against

\hookrightarrow ^{complex} Adnexal Mass.

\hookrightarrow Ring of fire sign

on Doppler study

Not diagnostic of ectopic



diagnosis finding in ectopic \oplus = Extra uterine gestation sac + cardiac activity.

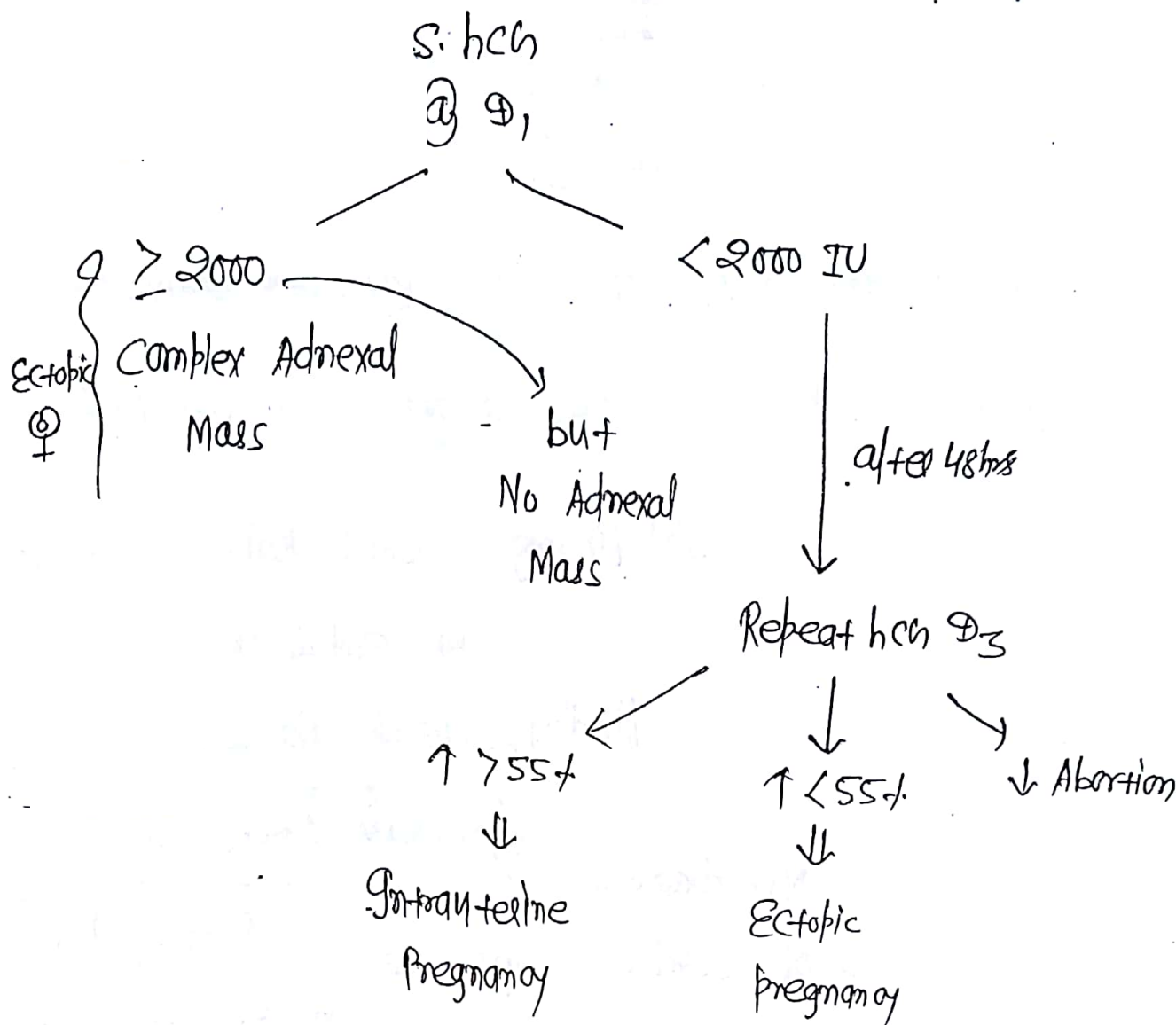
QA

No Intra Uterine gestation sac ; BL Adnexa \oplus ;

Please correlate clinically.



Pregnancy of Unknown origin ; check s. bet on \oplus ,



* Repeat hcg till it crosses the critical value

* goc = $\frac{TVS}{(=)}$ + Serial β hcg $(=)$

* Gold standard test = Diagnostic Laparoscopy

* Serum Progesterone = $> 25ng$ — Live Intrauterine ♀
 $< 5ng$ — Abortion

* culdocentesis
 ↳ i) small collection \Rightarrow USG; ii) large collection \Rightarrow Pt. variable
 ↳ Ectopic $\left(\begin{array}{l} > 100ml \\ \hookrightarrow \text{significant} \end{array} \right)$

* Which test has No Role In Ectopic @ →

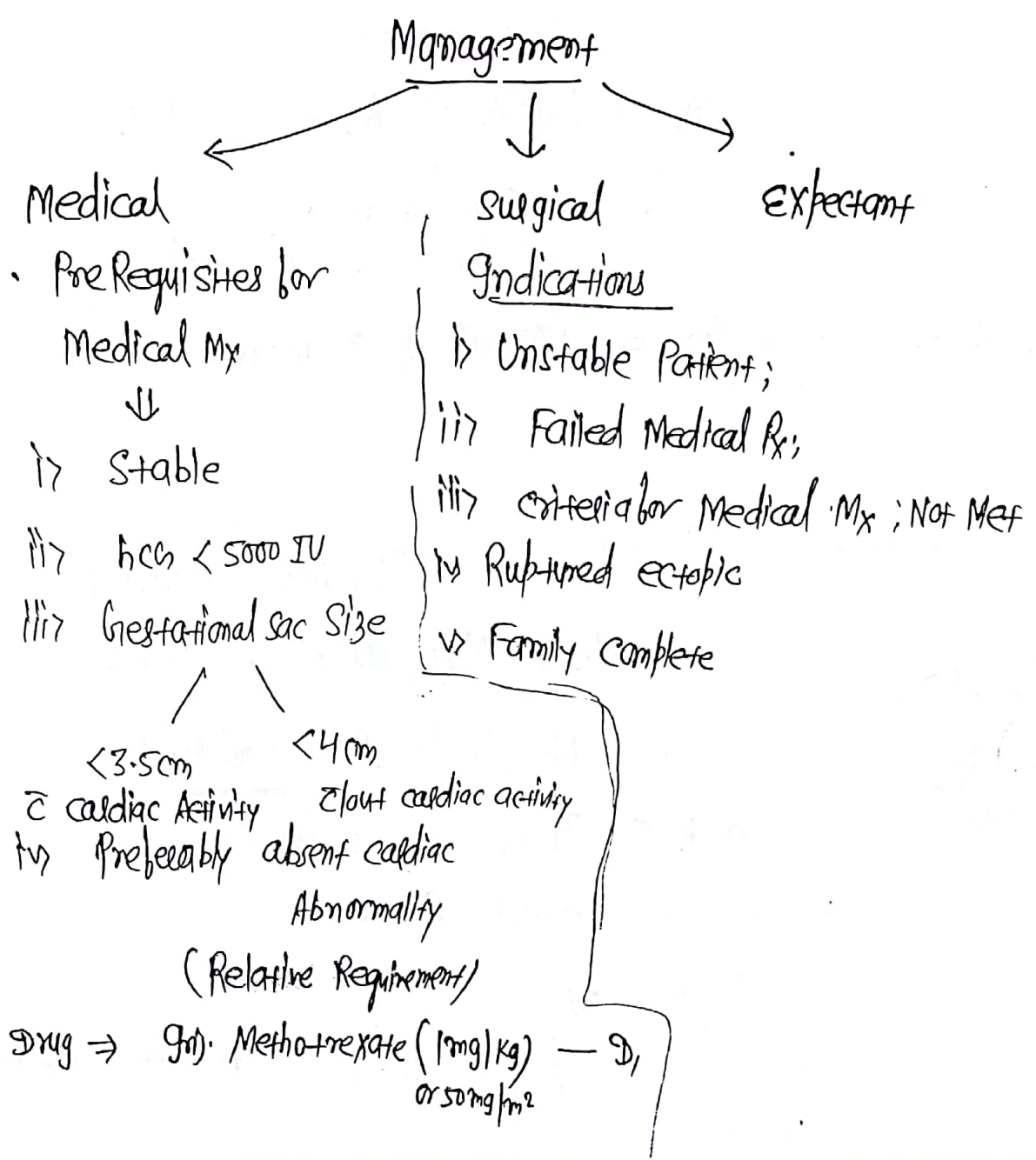
i) Colotomy

ii) HSG

iii) Hysteroscopy

} All in UP ⊕ve Patient

is all



D₄ S. hch

Management is Successful if

D₇ S. hch

S. hch level fall by 15% from D₄ value

if D₇ value fall but less than 15% from D₄ value



give Inj. Methotrexate (D₁)



D₄



D₇ (if again less than 15% of D₄ value)

↳ give Inj. Methotrexate

Failed Medical Mx ⇒ Total of 3 Methotrexate Injections

Surgical Mx ⇒

Unstable vitals ft.

Next Most appropriate step ⇒

a) i/v fluids & Medical Mx *

b) Immediate Laprotomy

c) Serial B hch

d) FAST

if localizing sign ⊕ eg ⇒ peritonitis — do Immediate Laprotomy

if No localizing sign ⊖ — do FAST

i) In general Laparoscopy is preferred over Laprotomy. (67)



but if Unstable vitals ⊕ ⇒ Go for Laprotomy.

ii) Salpingostomy - is preferred over Salpingectomy



Small Incision (~1cm)
on Anti-Mesenteric border
(No closure of Incision)
↳ b/c chance of ectopic

↑es by suturing; suturing heal
by fibrosis & Fibrosis ↑es chance
of ectopic ⊕

i) Family complete

ii) Ruptured ectopic

iii) ≥ 5cm

iv) if you can't achieve hemostasis

Q. Gyn of Marriage — Infertility — Conceived. Rupture ectopic

a) Medical Mx

b) Salpingostomy

~~c) Salpingectomy~~

d) Expectant Mx

Partial



Later date

↳ Tubal Reanastomosis.

* Expectant Management ⇒

Pt. is stable
 β hCG < 200 IU & falling trend
No visible gestational sac
Monitor serial β -hCG

* Heterotopic Ectopic ⇒

Risk factor = ARM (GVF)
Missed on USG

↓

late diagnosed ⇒ by 16 weeks

↳ Big size sac
↳ Impending Rupture / Ruptured

TOC: Medical Mx is CI; -

Sx ⇒ Laproscopic salpingectomy

If we want to give drug in clinical trials ⇒ $\text{Mn} \cdot \text{KCl}$

Under USG guidance ← cardiotoxic
give to Ectopic child & kill.

* cervical ectopic

- (68)
- ↳ Painless bleeding
 - ↳ Medical Mx (as long as Pt. is stable)
 - ↳ Criteria for Dx of cervical ectopic
 - ↳ Paalmans (Rubins criteria)

* Abdominal ectopic ~~criteria~~ ⇒ Painless; No Bleeding

- Studdiford criteria
- Late -3rd trimester (Abd. ♀)

- 32 week Abd. ♀



Immediate Laprotomy

- ↳ deliver the baby + Placenta in situ
 - ↳ Leads to Autolytic digestion

* Ovarian ectopic ⇒ Speigelberg criteria

- ↳ Surgical Mx

ABORTION **

weight of baby @ 20 weeks
 ~ 300gms

< 20 weeks

< 500gms (wtk)

Spontaneous Abortion
 (Sporadic)

Recurrent Abortion
 (Habitual)

→ Means Spontaneous

> 3 consecutive Preg. losses
 < 20wk.

Most common cause of Abortion

New guidelines
 ≥ 2 losses documented
 ↓ confirmed ♀
 Start evaluation

↓
 Chromosomal

also in 1st & 2nd trimester Most common.

* 50% of total abortion in 1st trimester } Chromosomal
 35% of total abortion in 2nd trimester }

* Chromosomal
 ↓
 M/c chromosomal Anomalies cause Abortion
 Aneuploidy (alteration in No. of chromosome)

90% - M/c cause of Abortion ⇒

M/c Aneuploidies cause Abortion ↑

Trisomy (13, 16, 18, 21)

- a) Trisomy 16
- b) Monosomy X
- c) ~~Aneuploidy~~
- d) Tetraploidy

Single M/c/c

Monosomy X - 20% Abortions

↓
 Trisomy 16 - 16% Abortions

* Most viable Aneuploidy = Trisomy 21

Most Lethal Aneuploidy = Trisomy 16 (69)

* M/C cause of Recurrent ♀ Loss ⇒ Idiopathic (75% cases)
(RPL)

↓
APLA - 16% RPL

↓
Uterine Anomalies — Congenital / Acquired

↓
Endocrinopathies

M/C ⇒ Balanced Robertsonian Translocations

↓
Chromosomal - only 4% cases of RPL

* Which of the following doesn't cause RPL?

i) Infections — sporadic

↓
TORCH Infections

Can't cause early RPL

Which Infection can cause RPL ⇒ "Syphilis"

↓
Kassowitz's Law
Mainly cause still birth, Not Abortion

i) 1st child → 1st loss
2nd " → 2nd loss
3rd " → 3rd loss
4th " → Live born child (Stigmata syphilis)

Every Successive ♀ in syphilis keeps Testing

Q. G15 M4 @ 12 weeks present 2 Missed Abortion.
all her previous early losses; all the following investigation
for her evaluation except ⇒

- ① Karyotyping
- ② LAE
- ③ AEA
- ~~④ VDRL~~

* ~~Karyotyping~~ should be done in patient of RPL

* APLA (Anti Phospholipid Antibody Syndrome) ⇒

↳ Single most common cause of RPL.

Diagnosis of APLA ⇒ 1 clinical + 1 Lab criteria

↳ Any one of the following

i) ≥ 3 ♀ losses of < 10 wks

ii) ≥ 1 ♀ losses > 10 wks of a Morphologically
Normal fetus

iii) At least 1 preterm delivery (< 34 wks)

↳ Secondary to severe pre-eclampsia
or Heliochental Insufficiency

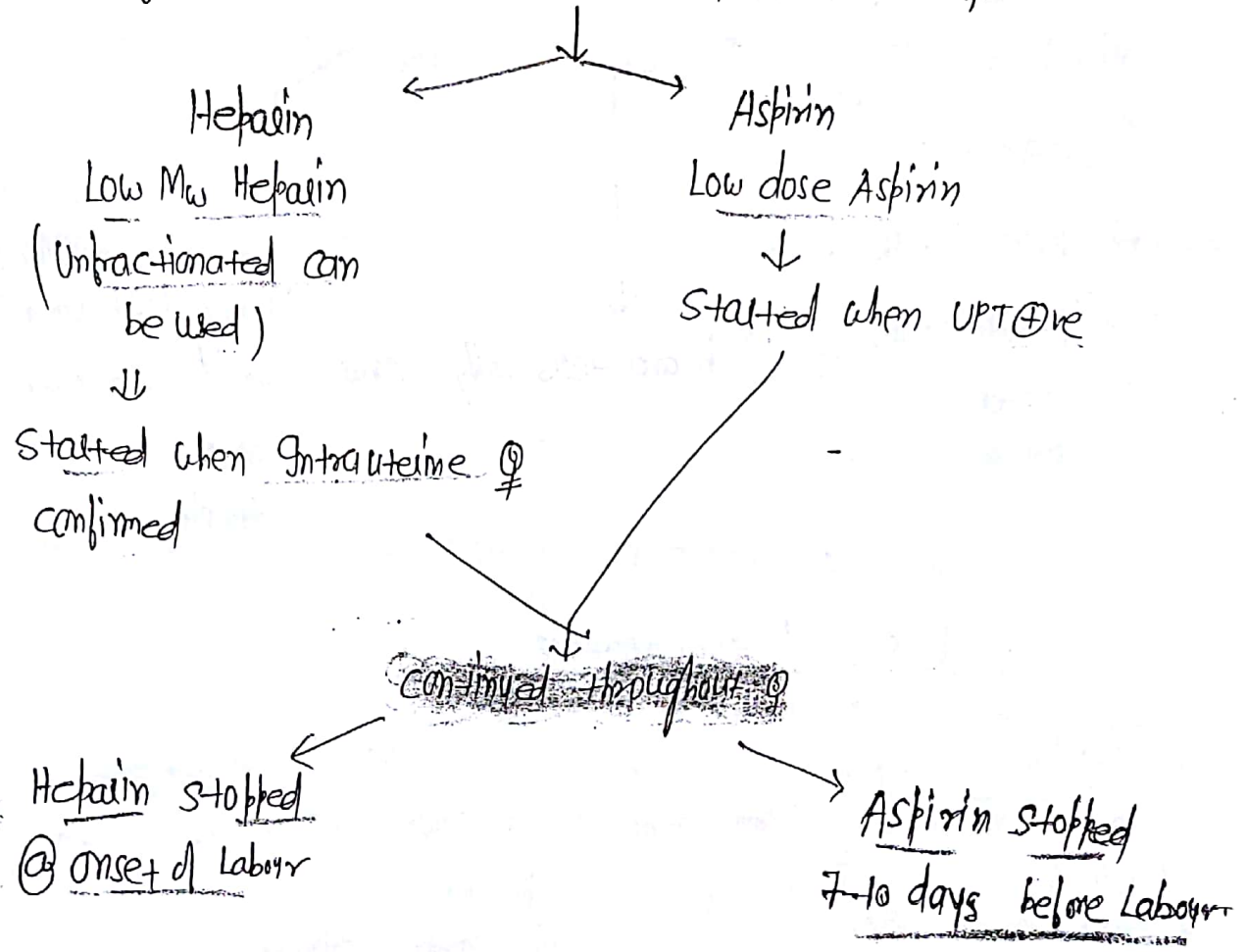
iv) Venous & arterial thrombosis

Lab criteria \Rightarrow Antibodies

- i) LAC \Rightarrow Lupus Anticoagulant
 - ii) ACA \Rightarrow Anti: cardiolipin Antibodies
 - iii) Anti β_2 GP₁ Antibodies \Rightarrow Most specific
 \hookrightarrow current disease activity
- test to look \Rightarrow Russel viper venom test for LAC
- IgM & IgG^{ACA} \oplus ve in Medium to high titres on 2 occasions done 12 wks apart.

(70)

* if the pt. is K/clo APLA syndrome & \oplus also



2nd line \Rightarrow should be used when only 1st line fails.

\Rightarrow Plasmapheresis
Iv Ig

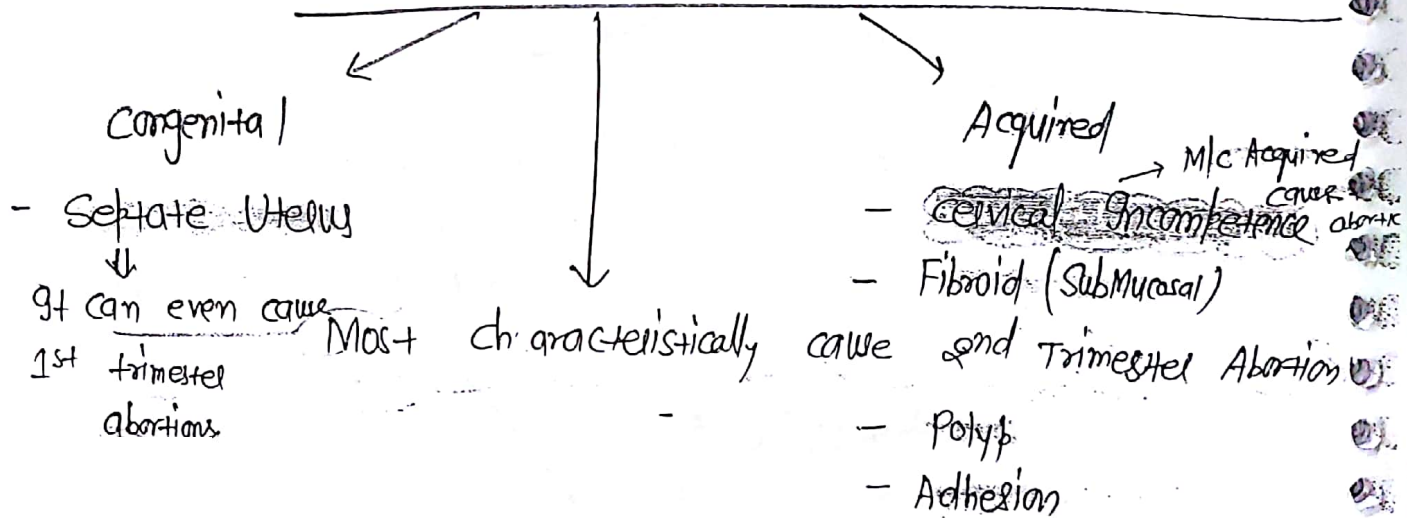
Doc for APLA \Rightarrow ~~Warfarin (Not given in ♀)~~
↳ (cause embolopathy)

Doc for APLA in ♀ \Rightarrow Low Mw Heparin.

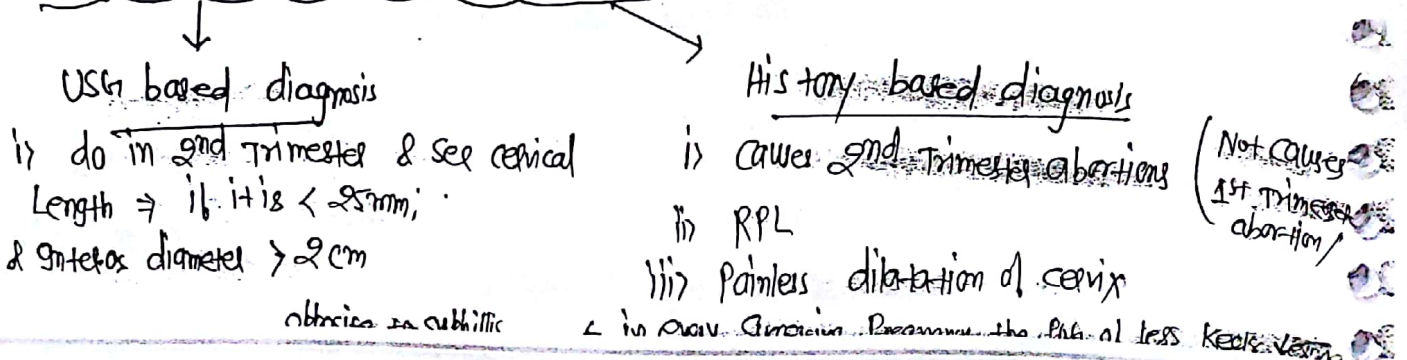
* Routine test Antibodies for APLA - In women \bar{c} RPL

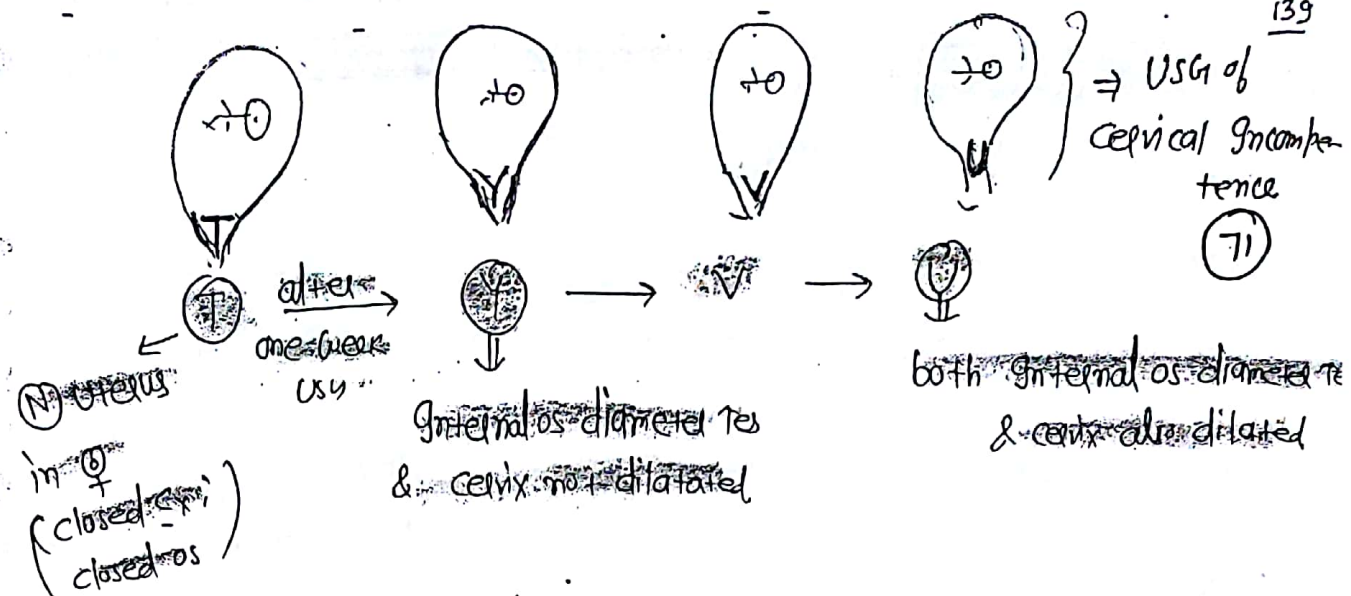
* Most characteristic trimester for APLA - 2nd trimester

* UTERINE STRUCTURAL ANOMALIES CAUSES ABORTION



Diagnosis of cervical incompetence





* Diagnosis of cervical Incompetence in Non pregnancy state :-

i) Passage of No 8 Hager's dilator through the Internal os without Resistance

↳ done in premenstrual period

ii) No 16 Foley catheter → fill the balloon & see Normal saline

↓
Pull it out without Resistance from the Internal os

↓
- Cervical Incompetence (+)

* Screening for Uterine Anomalies (Mullerian Anomalies)

HSG

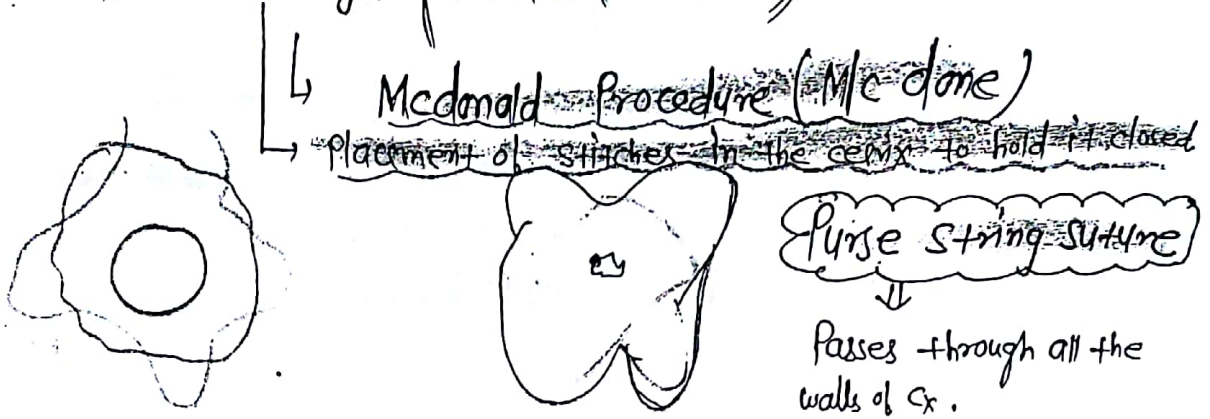
Sonohysterography

↓
do MRI / Hysteroscopy / Laparoscopy

↳ for diagnosis

ii Pt. has Cx Incompetence ??

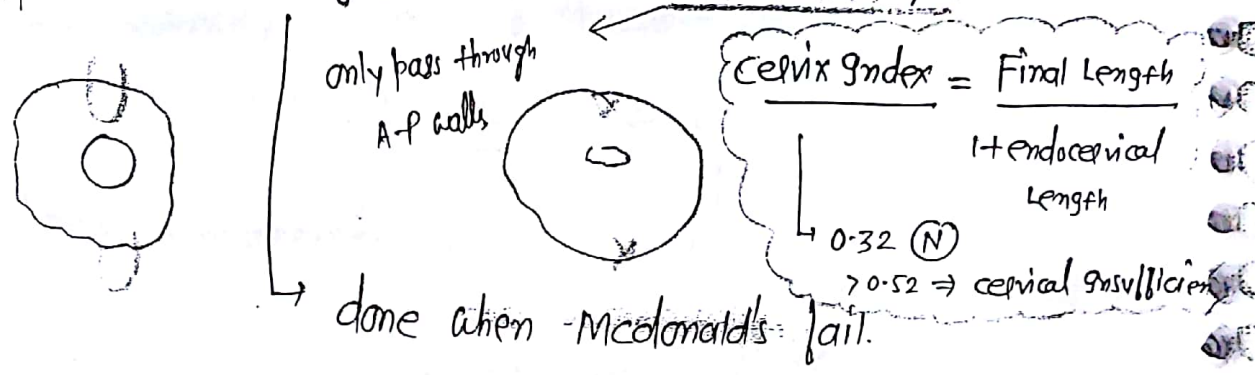
do cerclage (cervical stitch)



↳ McDonald Procedure (Mc done)

↳ placement of stitches in the cervix to hold it closed

Shirodkar cerclage ⇒ use Merselene tape*



* Ideal time to do ⇒ 12-14 weeks (14 weeks)

Up to what time we put the cerclage ⇒ up to 24 weeks

At what level we put the cerclage ⇒ as close to Internal os

- Contraindication of cerclage ⇒
- i) GTC
 - ii) Current pelvic Infection
 - iii) Ruptured Membrane
 - iv) Placenta previa

Removal of Suture ⇒ @ 37 weeks

So if any of the following develop ⇒ Ruptured Membrane
↳ Necessary Chorioamnionitis
Pressure on labour

* In Non-♀ State ⇒ ~~Lash & Lash Surgery~~
Rx of cervical incompetence ⇒ Lash & Lash Surgery (72)

Qa Should we do Sonohystelography/USG in Recurrent Preg
nancy loss

⇓
Yes (~~Should be done as a routine in Non-♀~~)
⇓
In ♀ ⇒ do USG

ENDOCRINOPATHIES CAUSES RPL

* Thyroid !! - Hypothyroidism
⇓
do TSH → as Routine Screening for RPL
↳ Subclinical Hypothyroidism → Abortions

* Diabetes → Uncontrolled diabetes
↳ Blood sugar is ~~Not~~ a Routine procedure
↳ do when pt. is symptomatic / significant Family History

* Prolactin → Hyperprolactinemia
S. Prolactin → ~~Not a Routine test~~
M/c presentation in ♀ ⇒ Amenorrhea & Infertility

* L.P.D (Luteal Phase defect) → corpus Luteum is formed by secreting Less Progesterone

↳ Not a established cause of RPL
↳ only when Progesterone < 15 ng/ml.
↳ Not a Routine test

* only 4 established cause of RPL

↳ APLA
Uterine Anomalies
Chromosomal
Hypothyroidism

* How does women \bar{c} Abortion presents ⇒ Spontaneous Abortion

Missed

all presents \bar{c}

Threatened

Amenorrhoea + Pain + Bleeding

Inevitable

* P/S examination

↳ Os closed

os closed

os open

* Size of uterus

POG

POG

POG

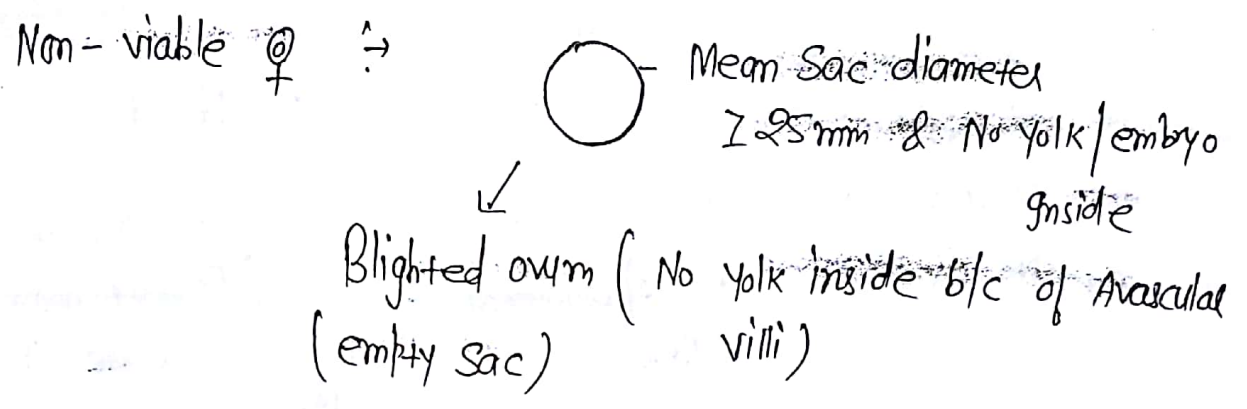
* In UGS examination
Cardiac activity is absent
(missing)

Cardiac activity is
present

Q. Lady is 6 week Amenorrhoeic; Pain + Spotting;
USG = Intrauterine gestation Sac & embryo; \bar{c} No cardiac activity.

* if CRL \geq 7mm & absent cardiac activity (73)
↳ Missed Abortion. ↳ Non viable pregnancy.

but if CRL 5mm & absent cardiac activity
↳ Repeat USG after some times (Min^m 1 week)



Q. Missed Abortion is missed b/c Symptoms presented after later date of Abortion; so; we missed to diagnosis

Mx ⇒ Induce Abortion (to open the os)
↳ In Missed Abortion

- Empirical Rx (Bed Rest; Progesterone Supplement, i/m -weekly injections, Micronised oral tab)
- avoid intercourse
- avoid Lifting heavy weight
- ↳ In Threatened abortion ⇒ abort d/t chromosomal abnormality
- No specific Rx ⇒ In Inevitable Abortion.

~~** Complete Abortion~~

~~Amenorrhoea + Pain + Bleeding + H/o expulsion of foetus~~

~~PE examination~~

~~L US closed~~

~~↓~~

~~Symptoms improve~~

~~altered exfoliation~~

~~USG~~

~~↓~~

~~For confirmation~~

~~↓~~

~~Uterine cavity empty~~

~~No Retained Product of Conception (RPOC)~~

~~** Incomplete Abortion~~

~~OS open~~

~~↓ Period of conception (POC) can be seen in the cervical canal~~

~~Uterine size > POC~~

~~Bleed a lot~~

~~↓ Unstable vitals~~

~~(Patient may present c~~

~~Shock)~~

~~Mx~~

~~Symptomatic Rx~~

~~Mx~~

~~Specific~~

~~complete the process~~

~~↓~~

~~do Suction & evacuation (digital evacuation could be life saving)~~

~~* Induced Abortion~~

~~⇒~~

~~MTP Act~~

~~↓~~

~~Up to 20 weeks~~

~~Pt. Asked for Abortion for~~

~~⇒~~

~~Rape~~

~~⇒~~

~~Humanitarian~~

~~Contraceptive~~

~~⇒~~

~~Social~~

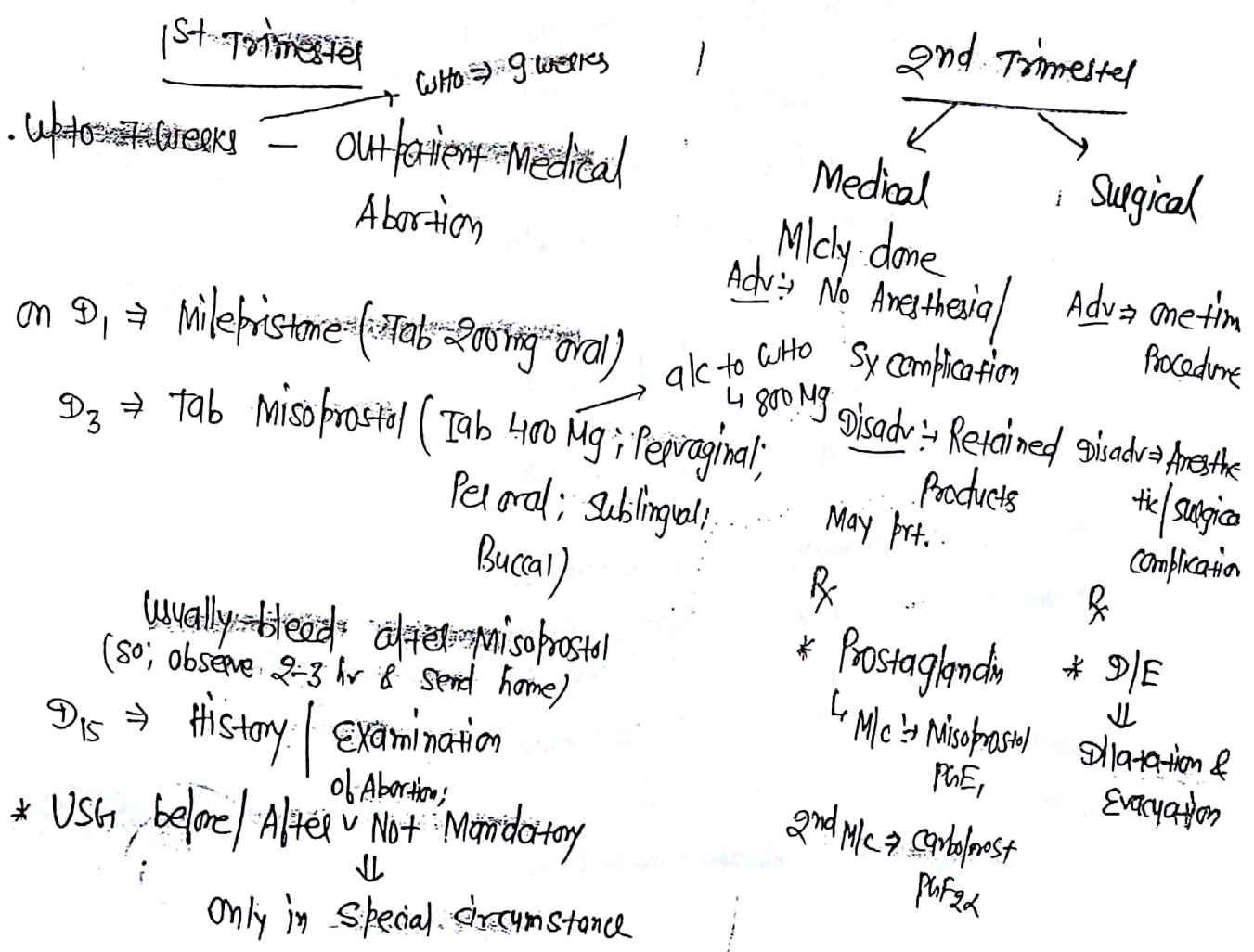
~~Failure~~

~~ABCA~~ → Eugenic
Medical/surgical disease → Therapeutic

(74)

Who do Abortions ⇒ RMP's (Registered Medical Practitioner)
↓
i) degree/diploma in field of OBG ;
ii) 6 months of ~~lowe~~ ~~surgenst~~ in department of OBG ;
iii) assisted in ~~25~~ procedure of abortion in govt. hospital (out of 5 done independent)

< 12 week ⇒ 1 RMP
12-28 week ⇒ 2 RMP



• Beyond 7 weeks & upto 12 weeks

↳ do "Suction & evacuation"

↓

• Hegar's dilator ⇒ graduated

blunt devices (if we perforate)

it heals itself; wait & watch

↓
Monitor the vitals

• Karman's cannula ⇒ 600 mm of Hg pressure

↳ white plastic device

generated

↳ if we perforate by Karman's

cannula ⇒ immediate lapro-

scopy

↳ on P/V examination

↳ size of cannula = size of uterus

on P/V examination to check

Avascular

Necrosis of

bowel loop

↳ usually 1 less than P/G's

• End point of Suction & evacuation

→ i) ↓ bleeding

ii) Air bubbles in cannula

iii) Gripping sensation on cannula

• Check curettage ⇒ Sharp devices

↳ give grating sensations

On village; alternative to Suction & evacuation ⇒ MVA ⇒ Manual Vacuum Aspiration

camera in
bore of syringe (sterile)

↳ upto 12 weeks

Syringe = 60 cc

Pressure 600 mm of Hg ⇒ 600 mm of Hg

Medical Rx

Extracannal

Ethacridine

Intracannal hyperosmolar saline

Oxytocin

Dilatation & Evacuation

75

↳ ~~Overm~~ Forceps ⇒ Spoon shaped forceps
No Locks ⊕
Piecemeal the products

* MOBIUS SYNDROME ⇒ ~~diff~~ ~~Misoprostol~~

↳ Palalysis of Facial Muscles
(Facial Nerve affected)

* if Cx is Not open after giving Misoprostol & all drugs in Under 20 weeks

⇓

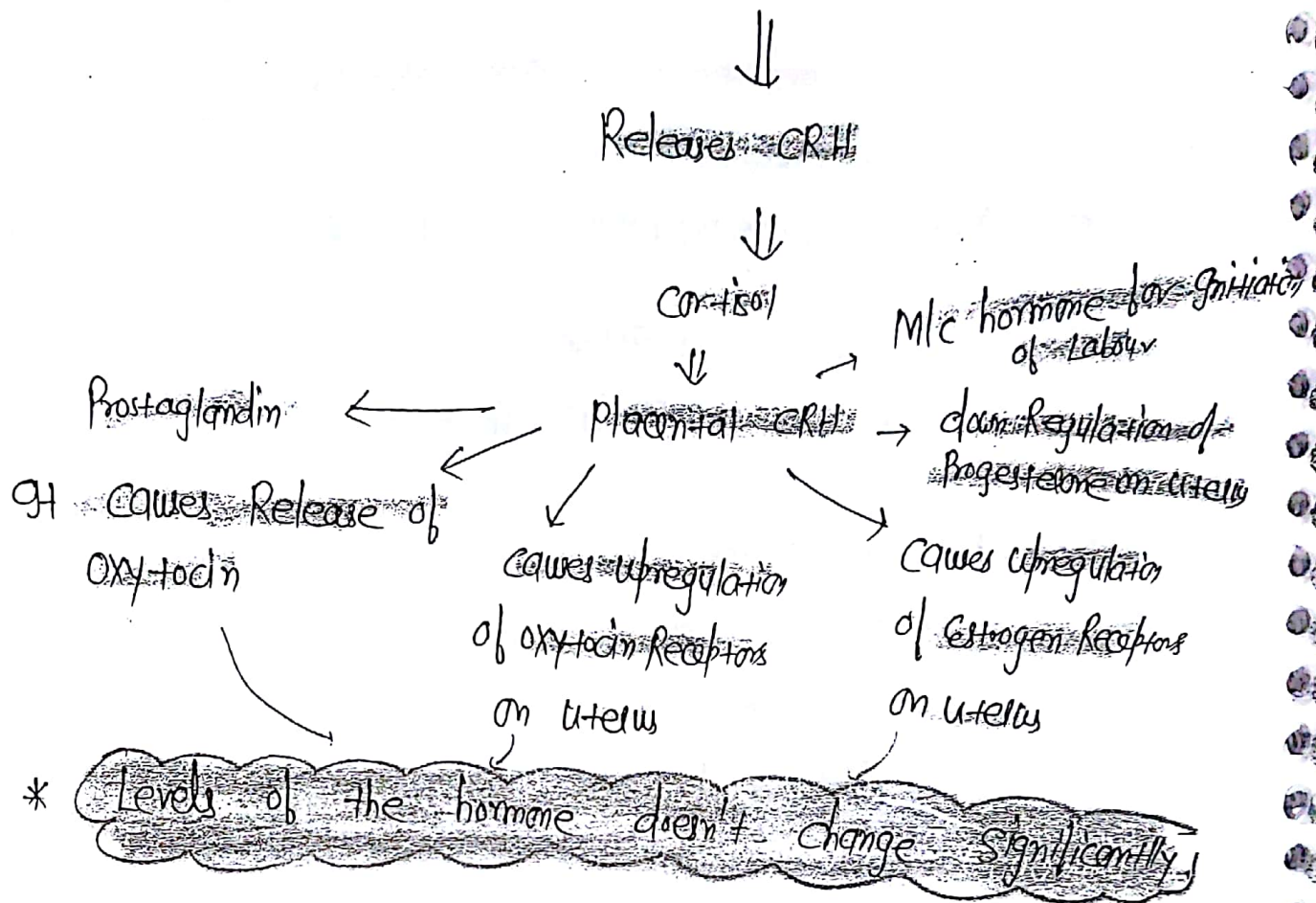
~~Opening of uterus~~ — In early Gestation

⇓

Hysterotomy
(So) it gives Classical Scar; so, separate
alter it always do cesarian section in Next ♀
b/c @ 20 weeks we don't dilate are the Lower uterine segm

LABOUR

Initiation of Labour \Rightarrow ~~Functional fetus Hypothalamic - Pituitary Axis~~



Oxytocin comes via \Rightarrow Ca²⁺ influx

↳ PlF_{2α} from the decidua

↳ Max^m Oxytocin Receptors ⊕ ⊖ 2nd stage of Labour

Expected date of delivery = 40 weeks (Naegels formula)

- ↳ on EDD \Rightarrow 47% delivery occur
- FDD + 1 week \Rightarrow 50% delivery occur
- EDD + 2 week \Rightarrow 80% delivery occur

* Preterm Labour \Rightarrow before ~~37 weeks~~

(76)

Term \Rightarrow ~~Early term~~ \Rightarrow 37-38⁶

Term \Rightarrow 39-40⁶

~~Late term~~ \Rightarrow 41-42⁶

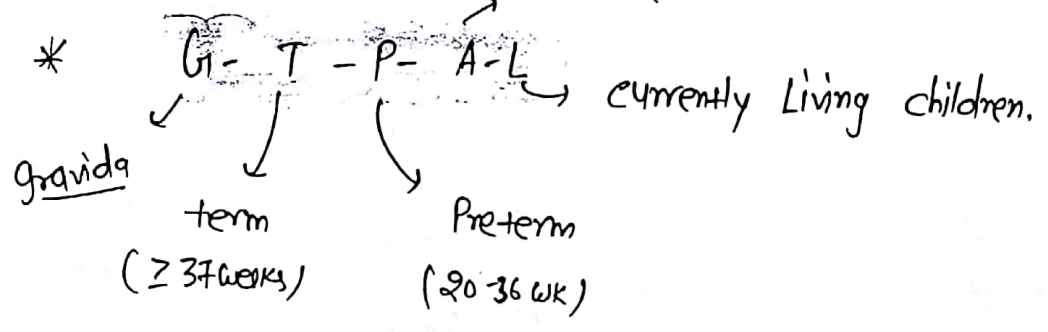
~~Post term~~ \Rightarrow \geq 42 weeks

* Non-Medically Indicated C.S. \Rightarrow don't do before 39 weeks

* Gravida \Rightarrow No. of ~~times~~ the women has been \odot .

Parity \Rightarrow beyond ~~period of~~ viability.

Abortion (< 20 weeks)



Q. In twin \odot None of the Parameters change; except \Downarrow Currently living children

Q. 2nd pregnancy; 1st was twins 34 wk delivery; Both alive

G₂ T₀ P₁ A₀ L₂

* Patient comes c Uterine contraction

check

False Labour

True Labour

- Uterine contraction
⇒ Not ↑ in frequency
Intensity
Duration

- Uterine contraction ⇒ ↑ in frequency
Intensity
Duration

- No passage of blood mixed cervical mucus
- No Progressive ex dilatation & effacement

- It shows ⇒ Passage of blood mixed cervical mucus

- Progressive ex dilatation & effacement
↓
in-1.
↳ 9cm
↳ Fully dilated
↳ 10cm

In Primigravida → effacement → dilatation

In Multigravida → both all simultaneously

- No Rupture of Membrane
- Sedatives ⇒ Uterine contraction will subside

- Rupture of Membrane
- Sedatives → Pain perception vs. Labour will progress

Best sign ⇒

Progressive ex dilatation

Rupture of Membrane

↳ ble of PROM ⇒ Rupture of Membrane before onset of Labour

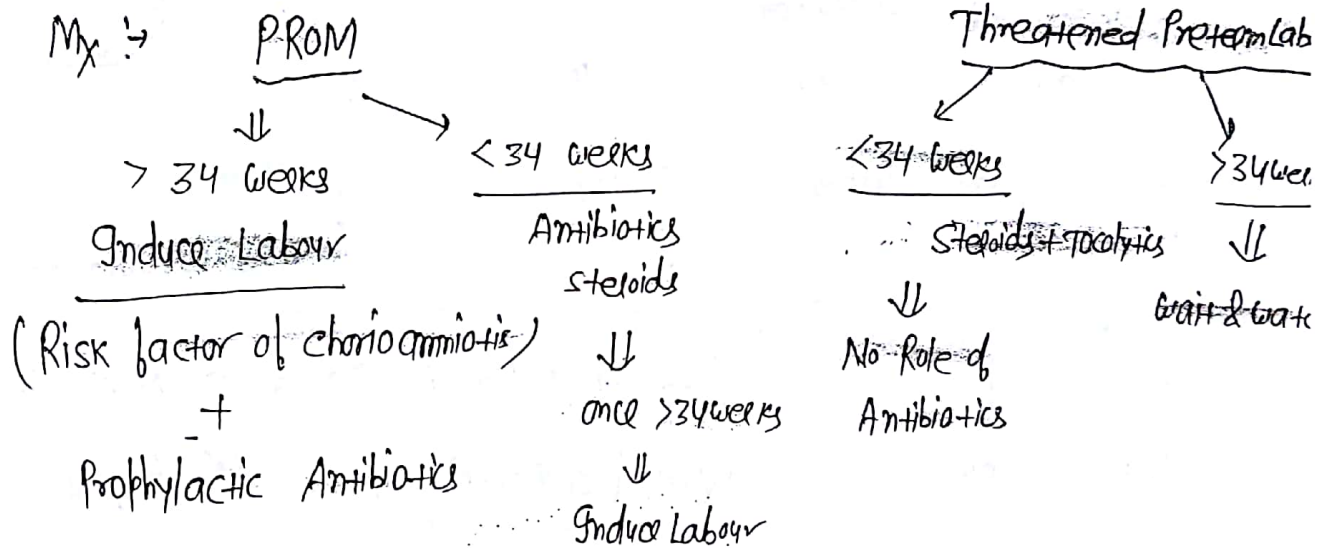
~~Best to know~~ PROM \Rightarrow Pel. speculum examination (5)

\Downarrow

- Leaking of Fluid from the OS. (77)
- pH \Rightarrow Nitrazine paper test
- USG \Rightarrow oligohydramnios
- dye test.
- FFN (fetal fibronectin)
 \hookrightarrow Marker of pre-term labour also.

PPROM \Rightarrow Rupture of Membrane before 37 weeks

Preterm Premature
Rupture of Membrane



* Most Imp. R/F for Pre-term Labour

\hookrightarrow Previous H/o of Pre-term Labour
Infection

Prophylaxis in high Risk previous pre-term Labour Patients

↳ Progesterone

* if patient is in True Labour; we know about! →

1st stage

Onset of True Labour Pain
to full dilatation of cervix

2nd stage

From full dilatation to
expulsion of fetus

Latent Phase

Active Phase

0-5cm	Cervical dilatation (AcoG)	≥ 6cm
0-3cm	(WHO)	≥ 4cm

↓
duration of Latent Phase

	Normal	Prolonged
Primi	24 hrs	20 hrs
Mult+	8 hr	14 hr

↓
Rate of cervical dilatation
Rate of descent of head

↳ In Primigravida (1.2 cm/hr)
 ↳ In Multigravida (1.5 cm/hr)
 (N) Progress = 1 cm/hr

↳ Primigravida 1 cm/hr
 ↳ Multigravida 2 cm/hr

3rd Stage of Labour ⇒

From the birth of child to complete expulsion of placenta

4th stage of Labour ⇒

Duration of observation of Mother : 1hr

PARTOGRAM

153 B, B, B ⇒ Blood stained
M, M ⇒ Meconium stained
C, C, C, C, C

Top Most part ⇒ tells about fetus (clear Amniotic fluid)

Middle part ⇒ Progress of Labour (Cx dilatation) (78)

Lower part ⇒ tell about Mother

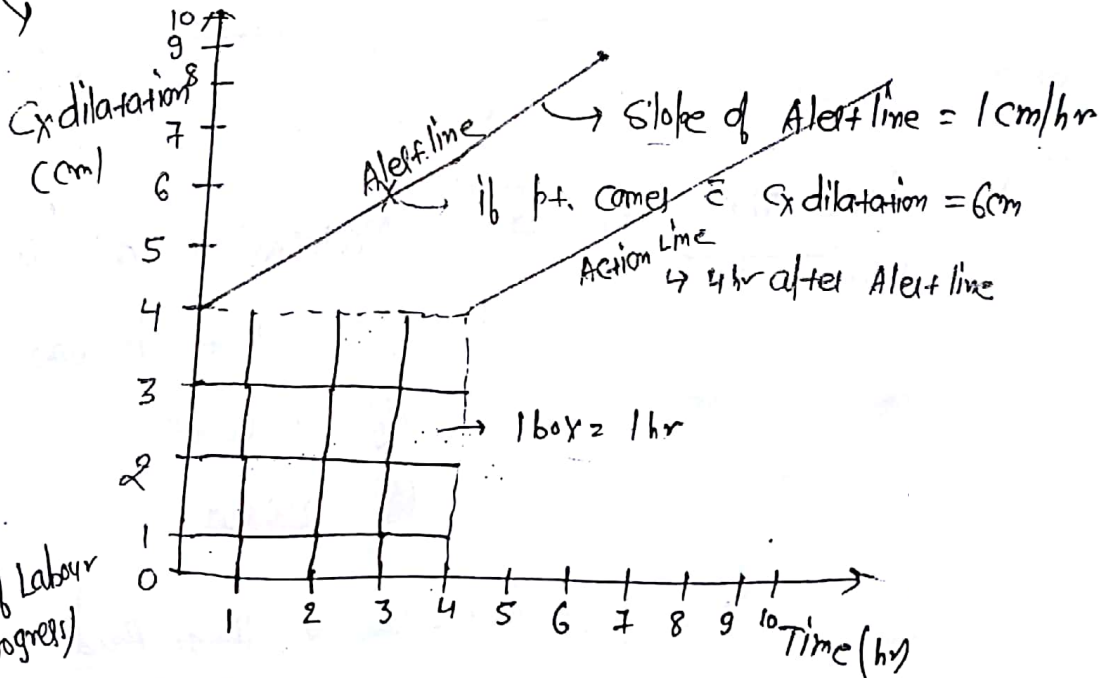
tells about Moulding

↳ Grade 1 ⇒ Parietal bones touch each other

Grade 2 ⇒ overlap; can be separated

Grade 3 ⇒ overlap; can't be separated

Plotting always done on Alert Line



↑
Dystocia of Labour
(Alert Progress)

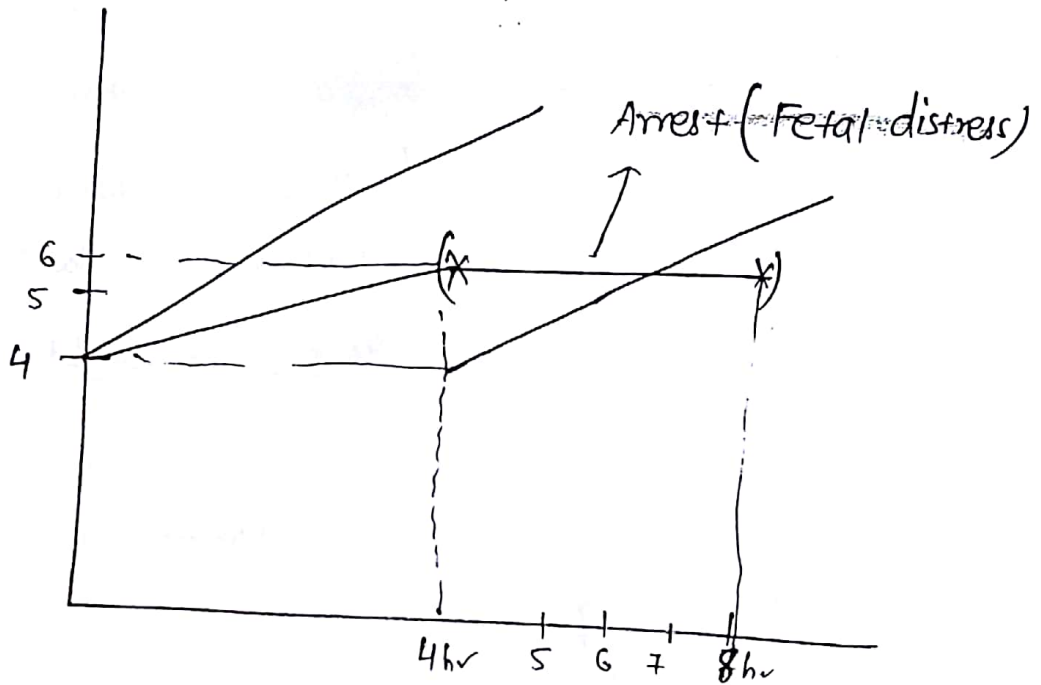
Not plot Latent phase in Partogram (plot after ≥ 4cm)

Partogram is Right to Alert Line ⇒ Referral to higher center


Partogram goes to Right to Action Line ⇒ Intervention

Arrest of Active Phase \Rightarrow No change in C_x dilatation even after 4 hrs of Adequate uterine contraction \rightarrow only if FHR \Rightarrow (N)


Intervention? ~~Cesarean Section~~



* Lower Most Part \Rightarrow Maternal condⁿ (uterine contraction)

 < 20 sec in one contraction

 20-40 sec

 > 40 sec

\rightarrow also tells about \Rightarrow Heart Rate
BP
temp
Urine output
drugs to be given

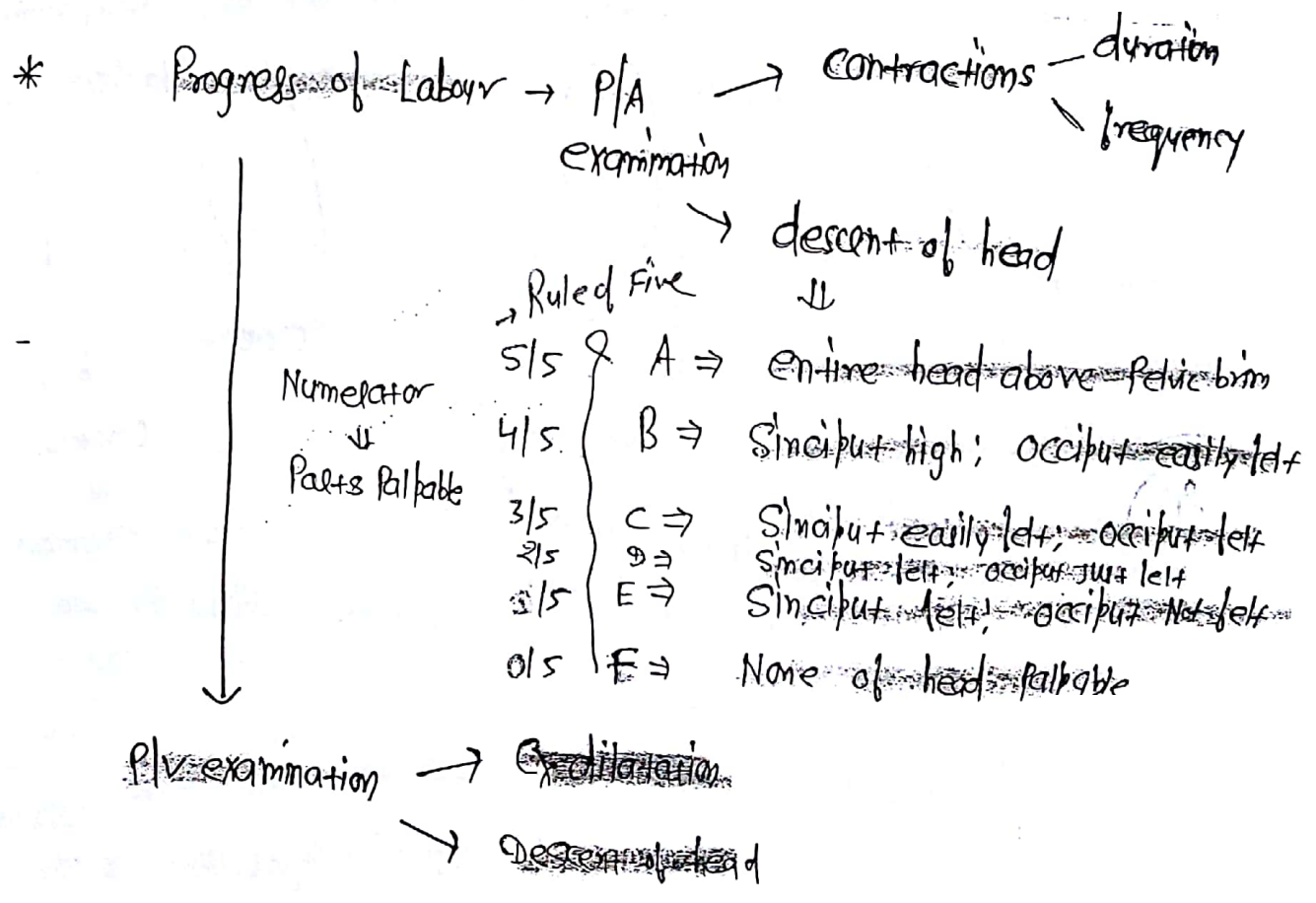
2nd stage of Labour ⇒ Av. duration
 ↳ 1 hr = Primigravida
 30 min = Multigravida

Arrest of 2nd stage

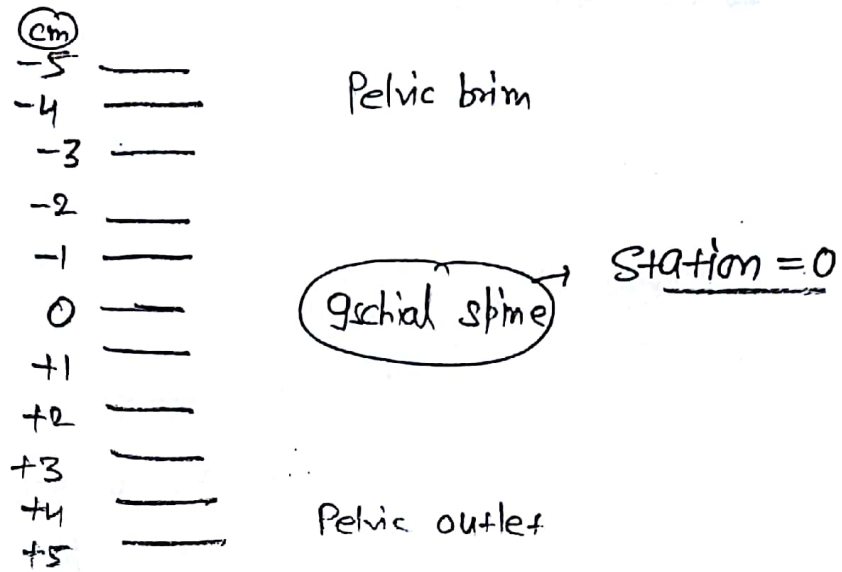
↳ No descent of head in the presence of Adequate uterine contraction
 for 3 hrs → Primigravida
 2 hrs → Multigravida

do G.S.

• ~~Arrest of 2nd stage~~ = epidural Analgesia
 4 hr ⇒ Primigravida
 3 hr ⇒ Multigravida



Descent of head in p/v examination ⇒

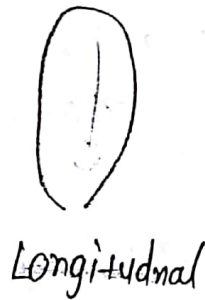
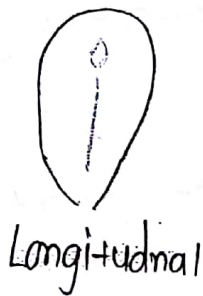


Station -2 ⇒ baby's head 2 cm above ischial spine

Station +3 ⇒ baby's head 3 cm below ischial spine

* LIE ⇒ Relationship of Fetus & Long axis of Uterus

↓
 firstly correct the ~~deformation~~ & empty the bladder



Unstable lie

↓
 keeps changing even after 37 weeks

Mildly abnormal location of placenta ⇒

1st M/c ⇒ placenta previa (75%)

2nd M/c ⇒ Placenta previa

Polyhydramnios

Oligohydramnios never cause unstable lie

Presentation ⇒

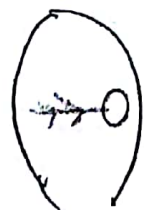
Part of fetus which is foremost in the birth canal. (80)



Breech



Cephalic



Shoulder

Leopold Maneuvre ⇒

~~Soft broad part~~ ⇒ ~~buttock~~

~~Smooth curve~~ ⇒ ~~Back~~

~~Limbs~~ ⇒ ~~knobby feel~~

(A) ~~Fundal grip~~ (1st Leopold)

(B) ~~Lateral grip~~ (2nd Leopold) → is with ~~single hand use~~

(C) ~~Pauline's grip~~ (3rd Leopold) → ~~2nd pelvic grip~~

(D) ~~Pelvic/rectal pelvic grip~~ (4th Leopold) → ~~1st pelvic grip~~
Both hand use

- * if head is in complete flexion ⇒ ~~Vertex presentation~~
- Partial flexion ⇒ ~~Similit presentation~~
- Partial extension ⇒ ~~Brow presentation~~
- Complete extension ⇒ ~~Face presentation~~

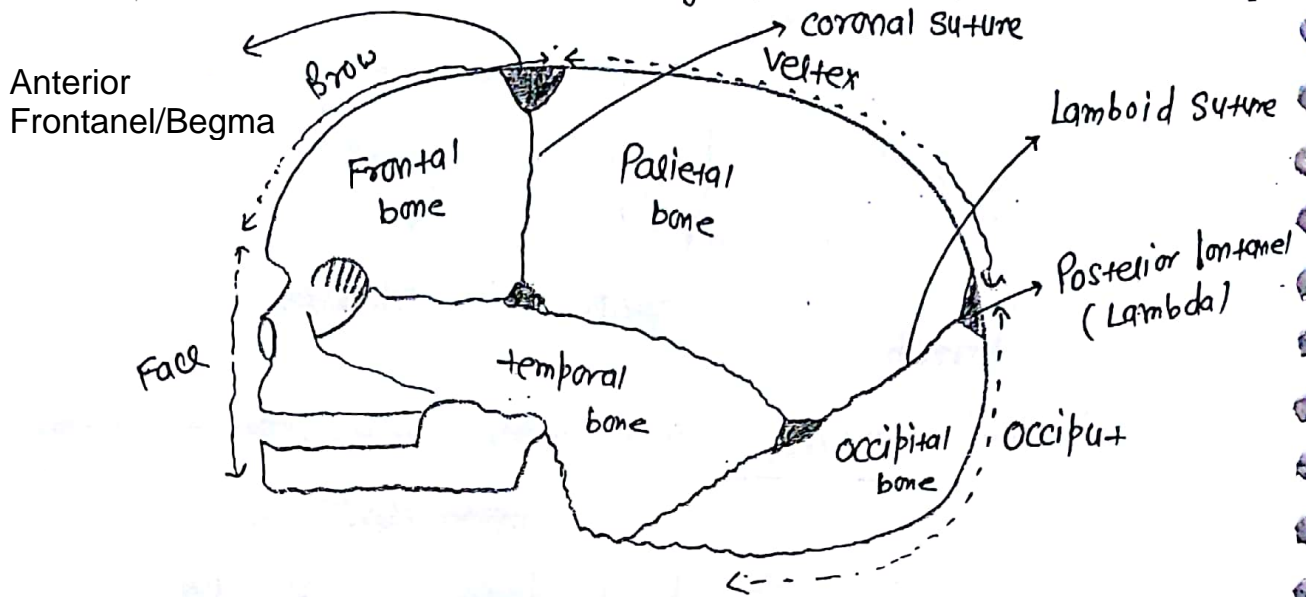
* ~~Anterior fontanel~~ ⇒ ~~Bregma~~

~~Posterior fontanel~~ ⇒ ~~Lambda~~

~~Posterior boundary of Brow~~ ⇒ ~~Anterior boundary of vertex~~

* Anterior fontanel \Rightarrow diamond in shape

Posterior fontanel \Rightarrow triangular in shape



Engagement \Rightarrow When the Largest diameter of the presentation crosses the pelvic brim.

Largest Transverse diameter \Rightarrow Biparietal diameter
 $\approx 9.5\text{cm}$

engaging dm in different presentation \Rightarrow

	Vertex	Sinciput (Parietalexion/seller)	complete extension/ Face	Brow
Engaging diameter (AP)	Suboccipito bregmatic (9.5cm)	Occipito frontal (occiput to anterior end of Anterior fontanel) 11.5 cm	Submento-bregmatic (9.5cm)	Mentovertical (14cm)
\hookrightarrow Antero-posterior		Sub-occipito frontal (10.5 cm)		\Downarrow Largest dm of fetal head

- * In Most of the Primigravida
↳ engagement @ 37 weeks
- * bree bloating head @ 37 weeks in Primigravida
↳ d/t deflected head:
CPD;
Placenta previa;
Polyhydramnios.
(CPD)
- * Engagement Rule out cephalo pelvic disproportion at the inlet.
- * when the head is Engaged; station = 0.
↳ on P/A exam; 2/5 is palpable
- * DENOMINATOR - Bony point on the presentation used to describe the position of head

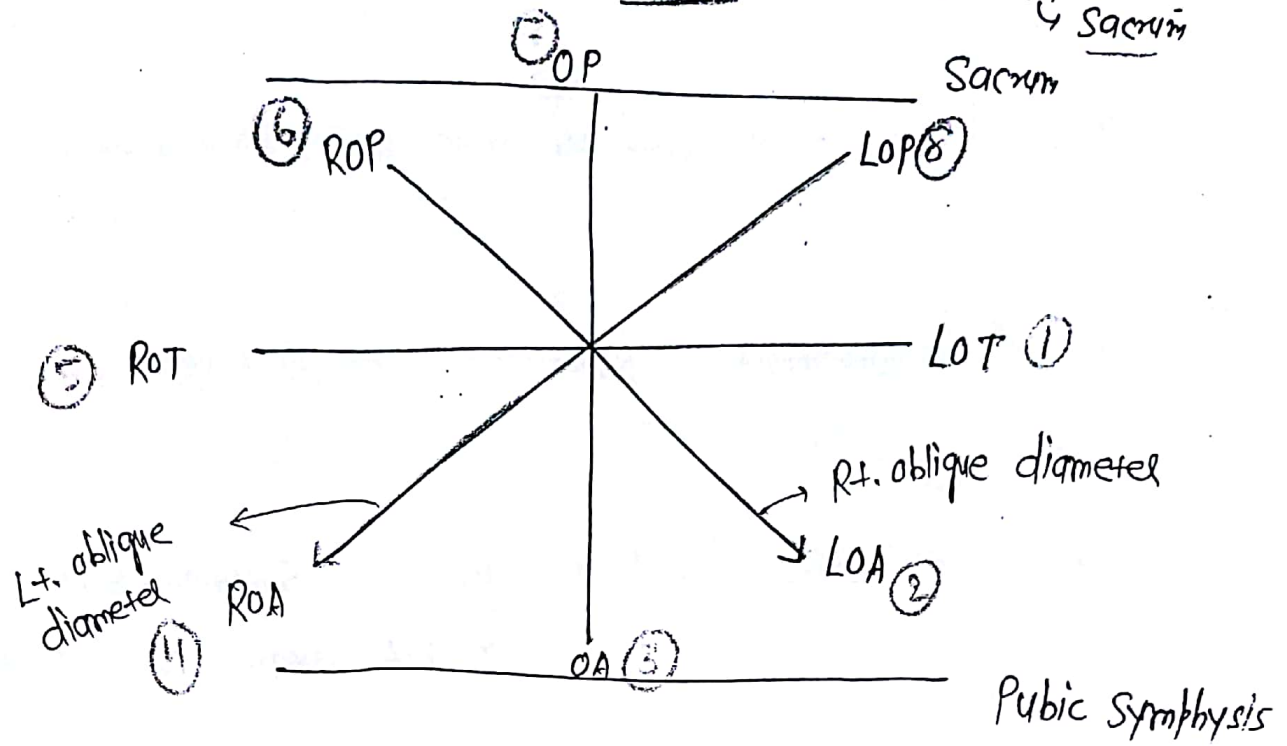
<u>Presentation</u>		<u>Denominator</u>
<u>Vertex</u>	→	<u>Occiput</u>
<u>Breech</u>	→	<u>Sacrum</u>
<u>Brow</u>	→	<u>Frontal bone</u>
<u>Face</u>	→	<u>Mentum</u>
<u>Shoulder</u>	→	<u>Acromion process (Scapula)</u>



Maternal Pelvis →

occiput ⇒ for vertex presentation

if breech ppt. ⇒ write "s" in place of "o"
 ↳ Sacrum

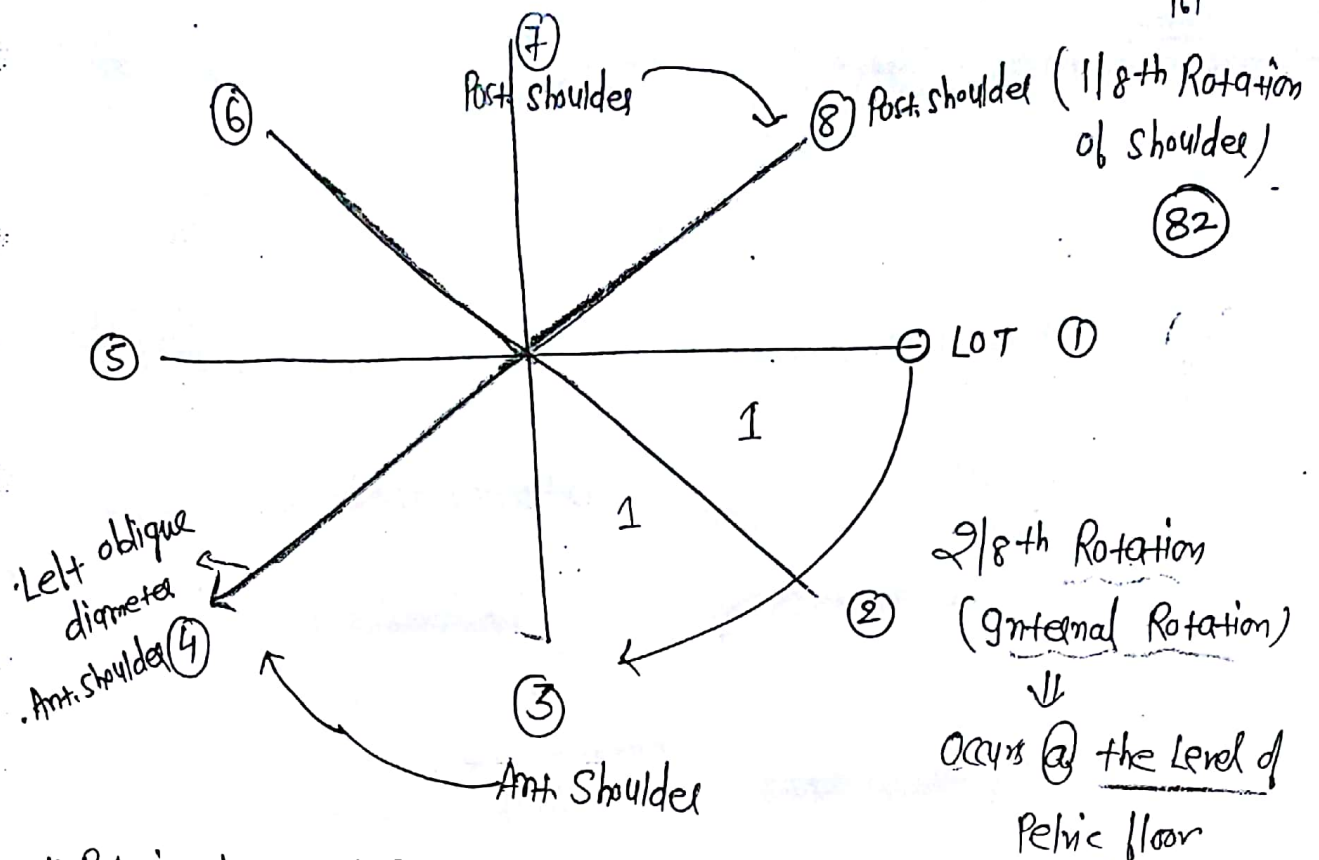


* In Routine exam; Plv exam done in active Labour 4 hourly.

* M/c position of (N) Labour ⇒ LOT**

cardinal steps of (N) Labour

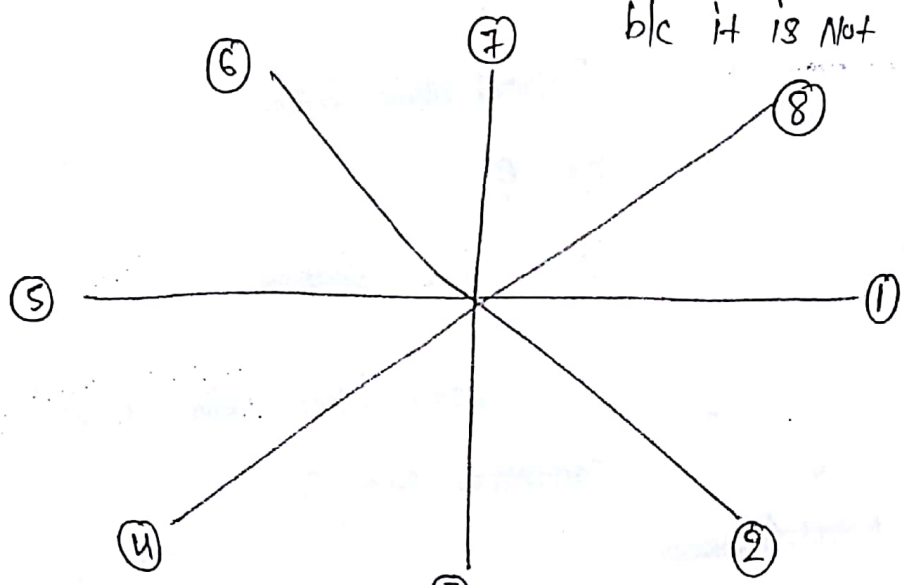
- (1) Engagement
- (2) descent
- (3) flexion
- (4) Internal Rotation
- (5) Extension ⇒ delivery of head (occiput → vertex → forehead → chin → Mouth → Glabella)
- (6) ~~external~~ rotation



- * Relation of Internal Rotation to External Rotation
↳ opposite direction.
- * Relation of Restitution to External Rotation
↳ same direction

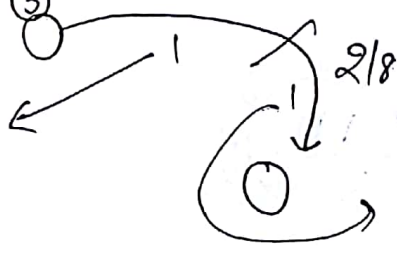
but shoulder rotate only by 1/8

* External Rotation → Restitution is not a cardinal movement b/c it is not the separate movement



after external rotation head goes back to original position (LOT)

Restitution (to remove torsion) head & shoulder



Internal Rotation of shoulder

3 "P"s" Region for (1) Labour \rightarrow

Passage

Push

Passages

Passage \Rightarrow

Maternal Pelvis \Rightarrow by Caudal Entry

~~M/c Pelvis in ♀~~ = ~~Gynecoid Pelvis~~ (Circuta Pelvis)
4 in 50 ♀



Transverse diameter \geq AP diameter

= ~~Anthropoid Pelvis~~



~~Antero-posteriorly oval Pelvis~~

in 25 ♀



AP diameter

Transverse diameter

~~2nd M/c Pelvis in ♀~~

Other

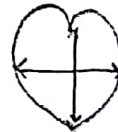
=

~~Android Pelvis~~



~~Typical Male Pelvis~~

20 ♀



Transverse diameter

AP diameter

~~Inlet = Heart shaped~~

Least common Pelvis in ♀ = 5+ Cases (Platy Pelloid Pelvis)



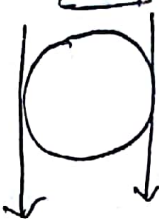
~~Transversely Oval Inlet~~

~~Flat Gynecoid~~



Transverse diameter \gggg AP diameter

Gynecoid



Side wall - Parallel

Android (Male Pelvis)



convergent side wall

M/c of Occipito-posterior \Rightarrow Android Pelvis

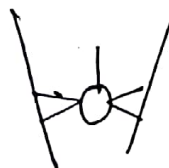
(Posterior space is more than Anterior)

Sharp

(83)

Ischial spine \Rightarrow Blunt

• DTA Not seen



Deep transverse Arc (DTA)

L M/c in Android

Subpubic Angle \Rightarrow obtuse
(90-100°)

Acute

(85°)

Shallow Pelvis

Deep Pelvis

(* Deepest Pelvis \Rightarrow Anthropoid)

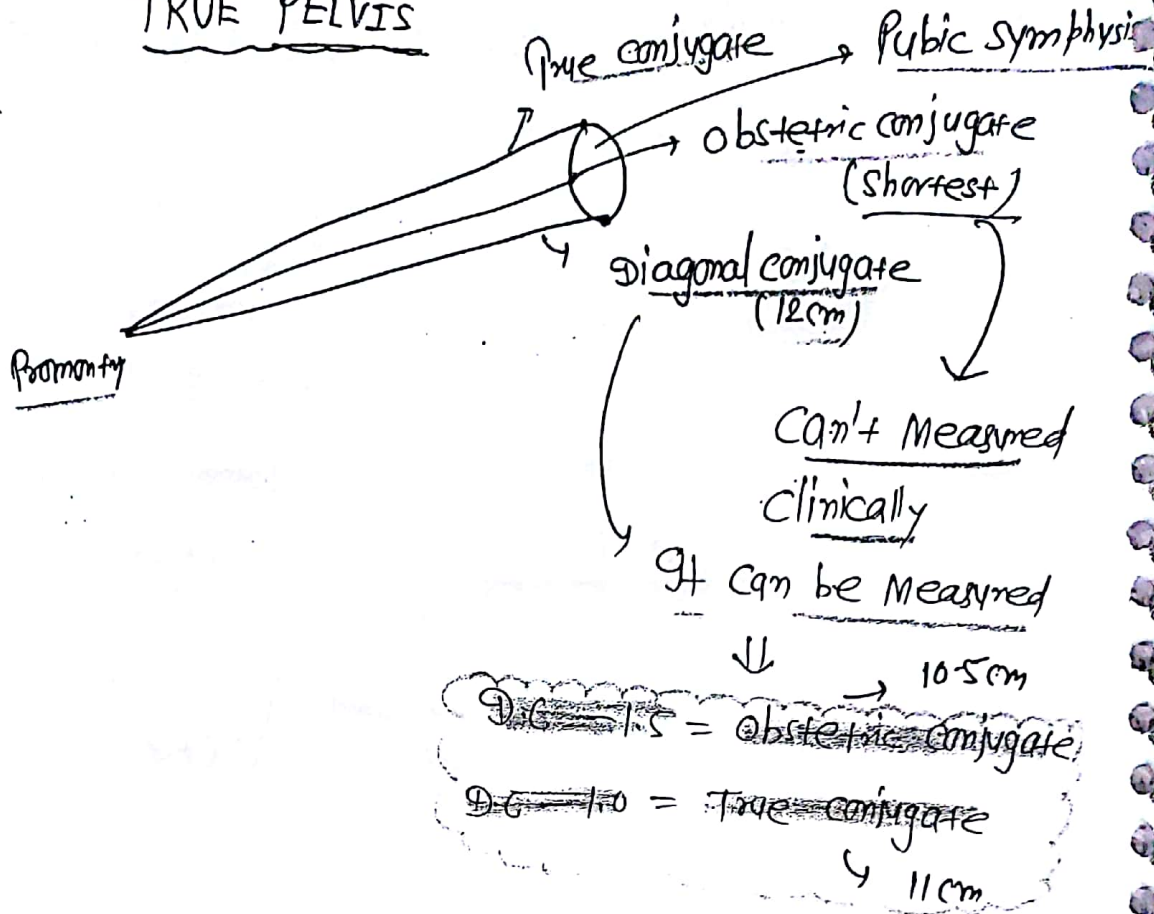
occipito-posterior \Rightarrow Android

Persistent occipito-posterior \Rightarrow Anthropoid

Direct occipito-posterior

TRUE PELVIS

INLET →



Cavity:

→ distance b/w ischial spine
~~Inter ischial spine diameter (IID)~~

↳ 10.5cm

↳ we shouldn't able to touch the both ischial spine simultaneously. Normally

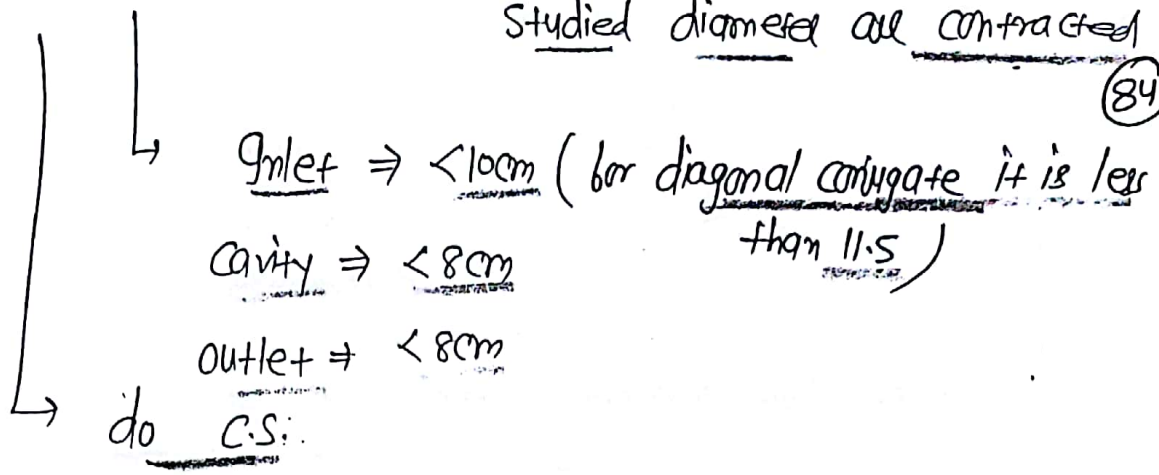
~~Contracted cavity~~ ! IID < 8cm

OUTLET →

~~Intertuberous diameter = 11cm~~
 (ischial tuberosity)

↳ "Four knuckle test" is do to measure it.

Contracted Pelvis ⇒ Any 1 or More of the Previously Studied diameters are contracted (84)



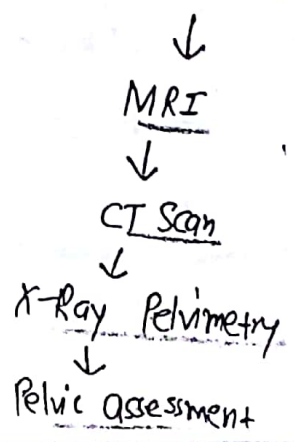
to know about the contracted pelvis; we will do "Pelvic Assessment"

↓
 In Primigravida; it do @ 37 weeks
 In Multigravida; it do; when she goes in Labour

Q. G2 P, L1 ; Previous 1 C.S.; done for contracted Pelvis; comes @ 37 week ANC ⇒ Recurrent Indication for C.S. (Never do VBAC)

* Cephalo Pelvic Disproportion (C.P.D.) ⇒

↳ Best way to know ⇒ Trial of Labour



a G₂P₁L₁; Previous 1 G.S. done for C.P.D.; she presents \bar{c}
 Labour & Pain 37 wk; OS 1 cm dilate; 70% effaced vertex.
 At $\Rightarrow -3$; What is Mx?

~~do VBAC~~

↓

~~Trial for VBAC~~

CPD is Non-Recurrent Indication for G.S.

Forms Roof of obs. outlet

↑

* ~~Plane of Least Pelvic dimensions = obstetric outlet~~

= ~~ischial spine~~
 (Passes laterally to ischial spine)

* ~~Anatomic outlet = ischial tuberosities~~
 (Passes laterally to ischial tuberosity)

* ~~Plane of Greatest Pelvic dimensions = Disc space b/w S₂-S₃~~

II. PUSH

Uterine contraction; Pacemaker \Rightarrow Rt. cornu

Strongest \Rightarrow Fundus

Rate of speed \Rightarrow 2 cm/sec; depolarise entire uterus in 15 sec

At what Intrauterine pressure



~~contraction became palpable~~ \Rightarrow 10 mm Hg

At what grip contraction became painful \Rightarrow 15 mm Hg

\hookrightarrow Min Pressure Required to initiate Cx dilatation

* Fundus can't be intended \Rightarrow Moderate contraction

\Downarrow
40 mm Hg

(85)

* end of 1st stage \Rightarrow 50 mm Hg

2nd stage \Rightarrow 80 mm Hg

* Adequate uterine contraction \Rightarrow

i) 3 contraction in a span of 10 min & each is lasting for 45 sec;

ii) If contraction generates pressure of 200-250 Montevideo Unit.

Montevideo Units \Rightarrow No. of contraction in 10 min
" P₁ generated

Tachysystole \Rightarrow > 5 contractions / 10 min

good or bad?
 \Downarrow
Bad

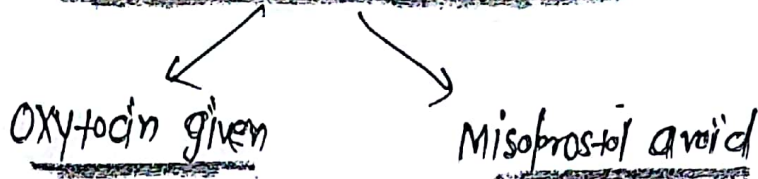
\swarrow UPI \rightarrow Fetal distress

Tachysystole + Fetal distress Hypersimulation

\rightarrow b/c Most of blood supply goes to baby in diastole

- Rx \rightarrow 1stly Stop the Inbusion
- \rightarrow Left Lateral Posture
- \rightarrow O₂ by Mask
- \rightarrow IV fluids
- \rightarrow Tocolysis (Stop the contraction)

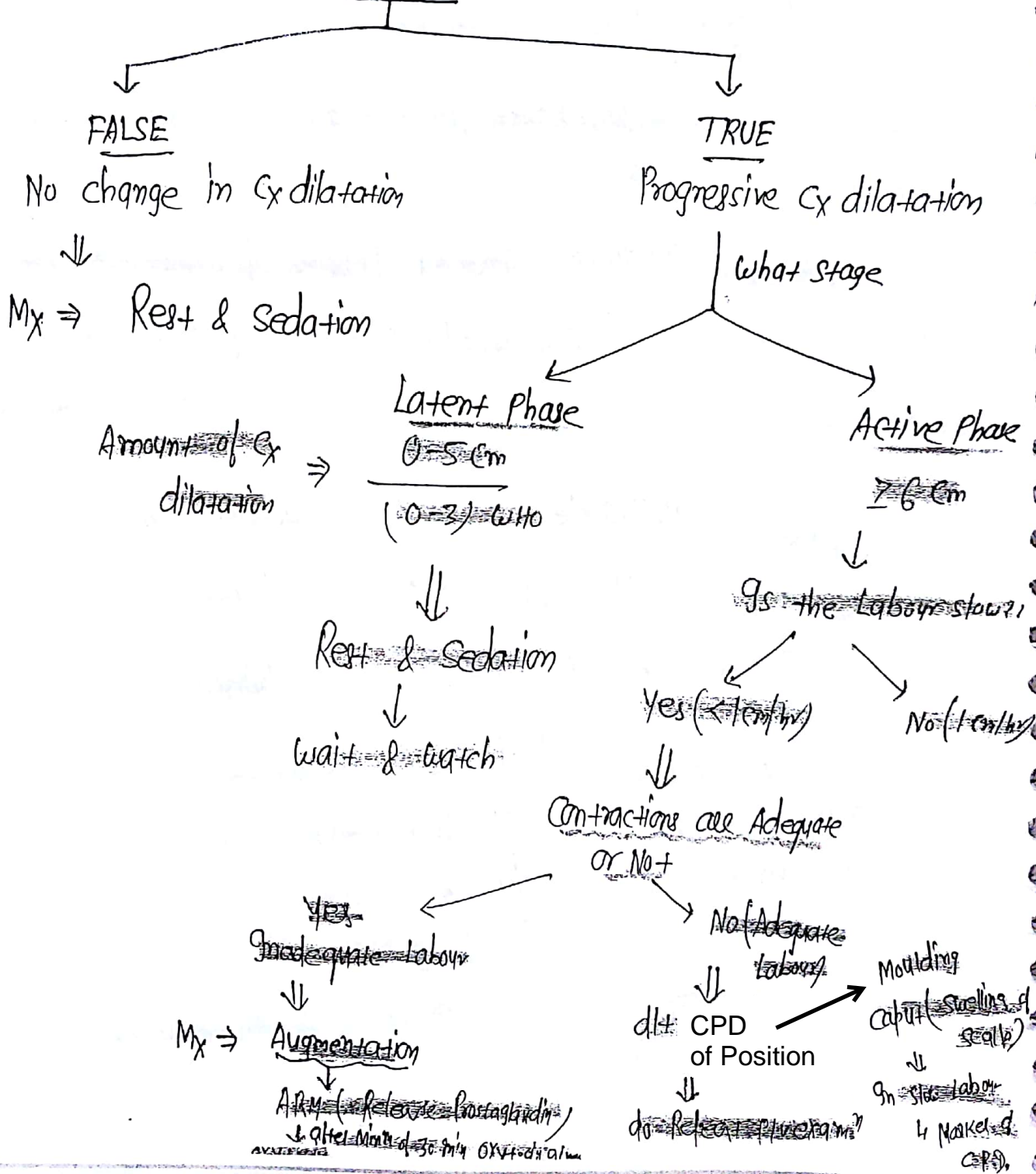
* In Augmentation of Labour



↳ May cause hyperstimulation
Rupture uterus

Management of

⇒ LABOUR



Q. Pt. comes @ 2 PM. 6cm dilated ex; 70% effaced;
 Vertex @ -1; Membrane absent, Liquor clear, contracted.
 220mv (Montevideo Units) (Rhythmic) (86)

@ 6 PM; 7cm 80% effaced; Vx @ -1; caput ⊕;
 Moulding ⊕

↓ ~~do Trial of Labour~~

@ 10 PM; Finding absolutely same

↳ i.e Arrest of Labour

↳ ~~do C.S.~~

OBSTRUCTED LABOUR

On General Physical examination ⇒ Exhausted
 dehydrated
 Tachycardia
 Tachypnea
 Acidotic breath

On P/A examination ⇒ upper segment ⇒ Tonically contracted

Lower uterine segment = stretched / thinned out

Blw U.S. & L.U.S. = depression (Ring)

Bandl's Ring / pathological Retraction Ri

Subpubic bulge (Blad)

FHR = ~~Severe distress~~

~~Absent~~

P/v examination \Rightarrow

~~Swollen vagina~~

* If we put Foley's we can not be able to enter as it is

~~Capitulum molding~~

Compressed by head we can Only able to put feeding tube
8 can see hematuria

~~Hematuria~~

Doesn't pass urine

is the earliest marker of obstructed labour

* obstruction is the absolute indication for CS, even if the fetus is dead

* Destructive procedures \rightarrow ~~Not in Modern obstetrics~~

L do craniotomy

Symphysiotomy

a) Not done CS \Rightarrow ~~Rupture uterus~~

* ~~if patient present~~ \bar{c} ~~Not passing urine and told us about bladder injury then we prove it as not bladder injury by~~

L VVF (vesicovaginal fistula) in developing countries

L ~~obstructed labour~~

In developed countries

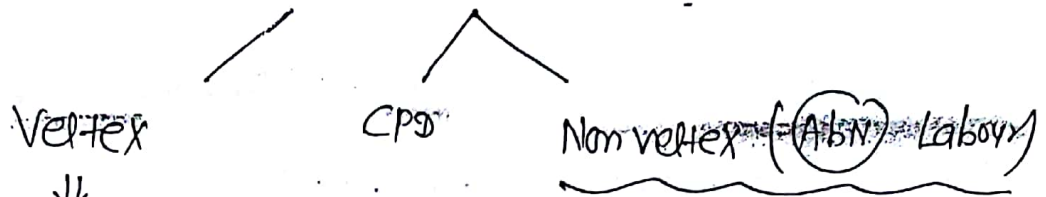
L \rightarrow

Gynaecology
surgeries

(Malignancy)

III. Passenger

Fetus convert (N) into (ABN) Labour (87)



Vertex
↓
(N)

Non vertex (ABN) Labour

Malpresentation

M/c ⇒ Breech

- R/F ⇒
- i) Multiparity;
 - ii) (ABN) Pelvis;
 - iii) Prematurity;
 - iv) Poly/oligohydramnios
 - v) GICA
 - vi) Placenta previa

* M/c Malposition

↳ OP (occipito posterior)

BREECH

M/c cause ⇒ Prematurity

POG

Breech

~~2/10K~~ → 25%

~~3/10K~~ → 7-11%

~~Term~~ → 3%

M/c type of breech ⇒ Frank breech (extended breech)

↓
Hip joint flexed, knee joint extended

But we don't feel heel here



Complete breech (Flexed breech) ⇒ Least common type of breech.


↓
Both knee & Hip are flexed; p/v ⇒ -Gschial tuberosity; Anal opening; Heel pads



* Primigravida \Rightarrow Frank breech

Multigravida \Rightarrow Complete breech

* Footling breech \Rightarrow Incomplete breech

Queeee  \hookrightarrow baby may have cord prolapse
Plv \Rightarrow can see foot of baby

~~Cord prolapse~~ \Rightarrow ~~Exposed to temp.~~

\hookrightarrow ~~Intense cord vasoconstriction~~
 \hookrightarrow ~~death of baby.~~

\Downarrow
Highest Risk in Footling breech

Least Risk in Frank breech

* Indication of C.S. in breech \Rightarrow

Absolute Indication \Rightarrow

① Footling breech;

② Staggeer breech;

\hookrightarrow Hyperextended head

Relative Indication of C.S.

\hookrightarrow Vaginal is Not C/I; but C.S. is preferred over vaginal delivery.

① Primigravida \bar{c} Breech;

② Previous C.S. \bar{c} Breech;

③ Macrosomia \bar{c} Breech;

④ Hydrocephalus

\hookrightarrow do ventriculo-peritoneal shunt by Pediatric Surgeon.

⑤ Prematurity

Q. Primigravida ; 37 week ; ANC checkup; o/E = Breech presentation; FHR (N); Liq (N); Placental fundal; Pelvis adequate; Mx = ? (88)

do ~~ECV (External Cephalic Version)~~
 can be done in Latent Phase of Labour
 ↳ for all ~~Non Cephalic presentation.~~
 if Requirements Met.

done for (a) Single term ♀ ;

(b) ≥ 37 wk / 36 wk baby ;

(c) Liquor Adequate ;

(d) Membrane should be Intact ;

(e) FHR (N)

(f) No c/I for vaginal delivery,

Relative c/I for ECV \Rightarrow Risk More than Benefit

↳ avoid ECV \Rightarrow (a) Previous CS

(b) GUR

(c) Pre-eclampsia

* ~~ECV is always done under continuous fetal monitoring;~~

* ~~It is done under tocolysis (terbutaline ifm);~~

* during ECV \rightarrow if fetal distress \oplus

↓
 Revert baby to original position

↳ do CS.

Q. G2P1 L1 39 weeks Breech presentation



first try ECV



but if any CI of ECV ⊕

→ Primigravida ⇒ C.S.

→ Multigravida ⇒ vaginal delivery
↓
(Assisted) Breech vaginal delivery

→ if fetus has extended Arms ⇒ Lovset Maneuvres

extended Legs ⇒ Pinard's Maneuvres

After coming head ⇒ Burn-Maushall

Maneuver



Grab foot & goes 180° towards Pelvis of Mom; after head comes out by

spine of fetus is Anterior & towards the obstetricians

In this delivery of head by "Flexion", Not by extension.

After coming head ⇒ Mauriceau Smellie vlet (MSV)

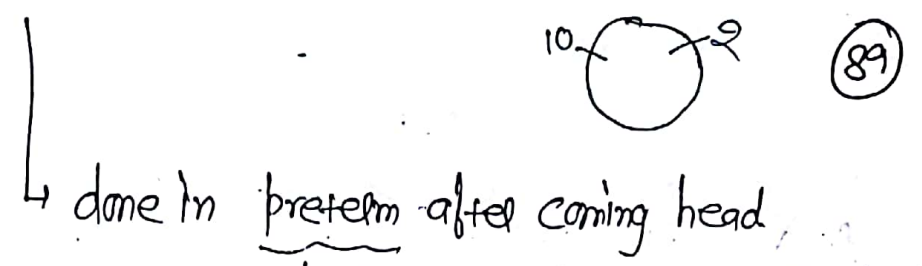
* Burn Marshall & MSV

↓
only when baby is DorsoAnterior
DorsoAnterior

↓
do Mallebone flexion & Shoulder traction & delivery of head by "Flexion"

In dorso-posterior breech; after coming head ⇒ Prague's Maneuver

* Dührssen's Incision → 2 Small Incision on Cx



↳ done in preterm after coming head
↳ if most of the part of baby is deliver & only head is inside

* Forceps delivery ⇒ Piper's Maneuver

* Last Resort ⇒ Zavanelli Maneuver

* M/c cause of Fetal death ⇒ cerebral Hemorrhage (ICH) in breech

* O.P. (occipito Posterior) ⇒ Not Malpresentation; Malposition

↳ M/c cause ⇒ Android Pelvis

2nd M/c cause ⇒ Deformed head

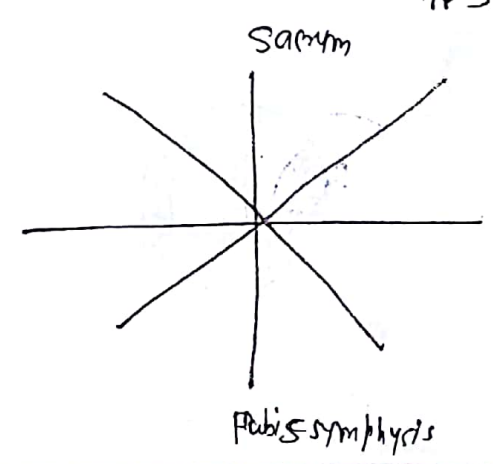


Engaged diameter ⇒ occipito frontal

↳ 10.5

M/c in Primigravida

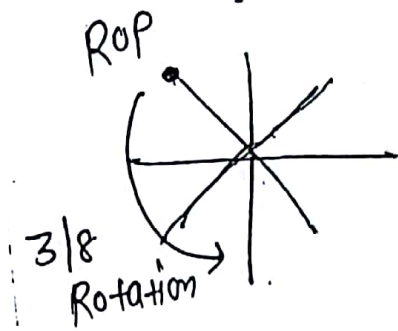
M/c Position - R.O.P.



1st outcome \Rightarrow all favourable — Push

Passenger

Passage



\hookrightarrow so take much time; Slow progress of labour.

\Downarrow

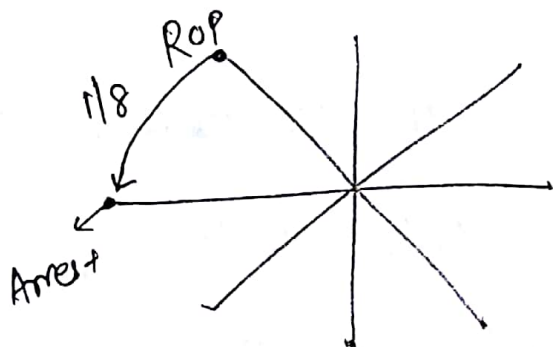
$R_x \hookrightarrow$ wait & watch; when baby is Anterior then delivers

2nd outcome \Rightarrow Deep transverse arrest

\hookrightarrow d/t android pelvis

\Downarrow

do the c.s.



but if pelvis is (N); then

i) if inadequate contraction seen

\hookrightarrow give oxytocin

ii) if contraction is Adequate

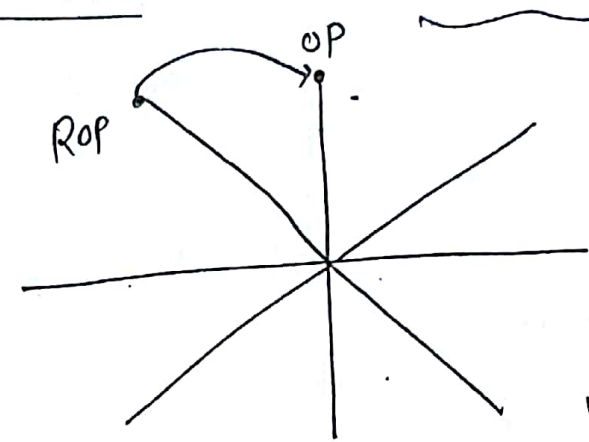
\hookrightarrow Manual Rotation

Forceps Rotation

Vacuum device extraction

3rd outcome

Persistent OP / Direct OP

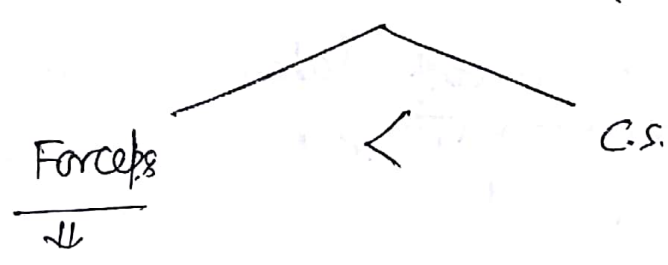


Li seen in Anthropoid Pelvis
(90)

⇓
Mechⁿ of Labours

⇓
"Face to Pubes" delivery

* In case spontaneous delivery (Not happening)



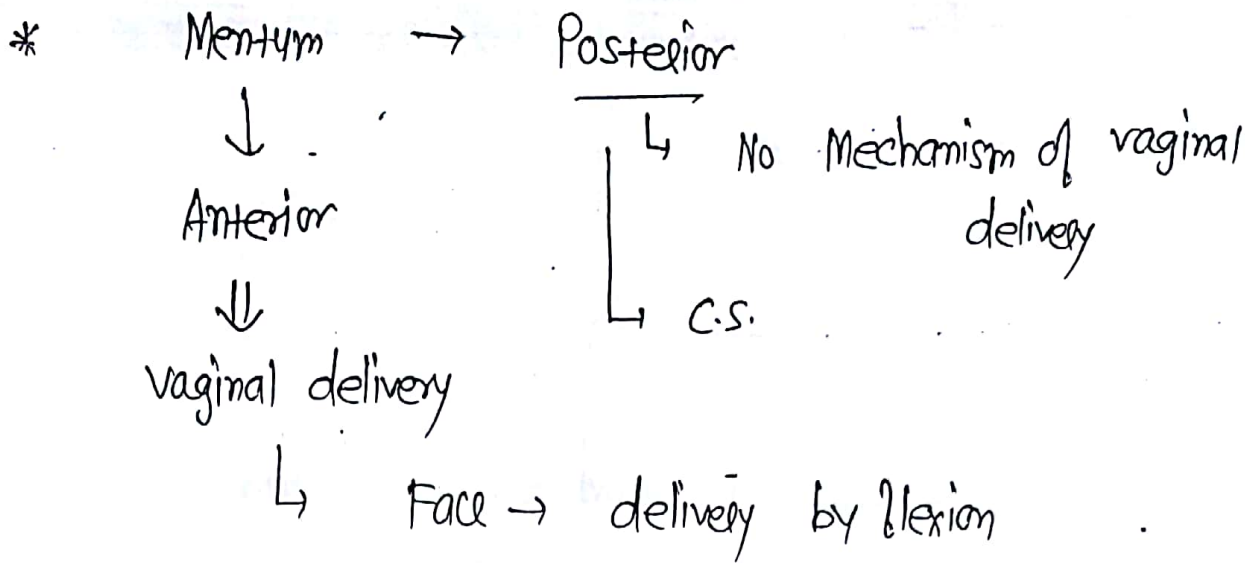
In Posterior Position ⇒ Incidence of Maternal Injuries & severity ↑

* f. of babies are in O.P. at onset of Labour = 20%
at the end of Labour = 5%

* FACE / BROW PRESENTATION

M/c cause of Face presentation ⇒ Anencephaly;
Pelvis type; which favour it ⇒ Platypelloid

Anything that prevents flexion of head ⇒ Risk Factors (> 1 Loop of cord around Neck; tight loops / ...)



* Mentoposterior in Labour ⇒ C.S.

Mentoposterior in early Labour ⇒ wait & watch

↳ to Rotate gives time

* Brow presentation

↳ engaging Diameter ⇒ Mentovertebral = 14cm

↳ No Mechanism of Labour

↳ Brow in Labour → C.S.

Brow in early Labour → wait & watch

TRANSVERSE LIE

M/C cause ⇒ Prematurity

M/C cause at term ⇒ Placenta previa

- Most Commonly Seen in "Platypelloid" Pelvis.

- Highest Risk of cord prolapse

* Transverse Lie — In Labour — do C.S.

↳ Pries ECV
↳ if Requirement Mets. (91)

* Neglected Shoulder

↳ Upper segment ⇒ Tonically contracted

↳ Lower segment ⇒ Stretch

Bend's Ring ⊕ in b/w US & LUS

↓

Case of obstructed Labour

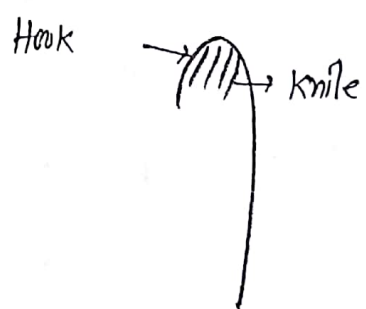
↳ FHR Absent

↳ do C.S.

Destructive procedure ⇒ i) Evisceration;

ii) Decapitation;

↓



Q: Lady pr. ♂ Neglected shoulder presentation ♂ dead baby.

all the following can be done except ⇒

a) C.S.

b) Evisceration

~~c) Craniotomy~~ (bc for it vertex presentation require &

d) Decapitation here transverse lie pr.

Q:

Confusion of Breech presentation on P/V examination \Rightarrow

Face presentation

- \hookrightarrow Frank breech Most commonly confuse.
- \hookrightarrow In it ~~three~~^{two} bony prominences & one opening (Mouth) Makes a triangle; while in breech @ it forms straight line

INSTRUMENTAL DELIVERY

- do when we cut short 2nd stage of Labour

- Pre-Requisites \Rightarrow

F \rightarrow Fully dilated Cx

O \rightarrow No Obstruction in the path

R \rightarrow Ruptured Membrane

C \rightarrow Good uterine contraction

E \rightarrow Engaged head / Empty bladder / episiotomy

P \rightarrow Favourable presentation

•

Forceps delivery

Vacuum delivery

- difficult
- Maternal Exhaustion
- we're in heart disease
- In Fetal distress (Forceps > Vacuum delivery)
- * In general Forceps prefer over Vacuum
- In preterm
 - if P₀ < 34 weeks, Vacuum is absolutely C/I
- Face presentation (MentoAnterior Position)
 - In face presentation C/I.
- After coming head of breech (Piper's Forceps)
- More Maternal Injuries

More fetal Injuries

Q Which fetal Injuries are M/C in Forceps delivery??

- IVH
- Facial Nerve Injuries
- Brachial Plexus Injuries
- Cornea of the eye

Q Injuries which see in Vacuum Not in Forceps delivery??

- 6th Nerve Injuries
- Ethelhematoma
- Retina Injuries

* Classification of Forceps delivery \Rightarrow

Outlet $\geq +3$

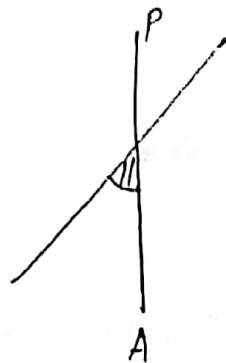
Low $+2$

Mid cavity - b/w 0 & +2 } Not in Modern days
 High - above 0

* criteria for application of outlet forceps \Rightarrow

- ① Scalp visible at Introitus
 - ② head on Pelvic floor
 - ③ skull on Perineum
- $\geq +3$
- ④ Sagittal suture should be preferably in A-P position

⑤



Rotational defect $< 45^\circ$

\hookrightarrow apply outlet forceps

How to know that Forceps is correctly applied \Rightarrow



(93)

Blades have to be Equidistant from sagittal Suture

along which fetal diameter we apply Forceps blade

\hookrightarrow Occipito Mental (13.5 cm)

* Left blade of Forceps \Rightarrow Introduced first

\hookrightarrow M/c Position = Left,

* M/c outlet forceps used \Rightarrow Wrigley's outlet forceps

\uparrow Not used in After coming head; b/c of short nature



for both outlet & Low position,

short forceps & have English Lock

* Kelland's forceps \Rightarrow for Rotational defect

\hookrightarrow Long forceps

* Piper's forceps \Rightarrow for after coming head

\hookrightarrow Pelvical curve \oplus (Baby body Rest here)
very long forceps

VACUUM DEVICES

- Use only plastic cup

↳ Bell shaped

diameter = 5-7 cm

Silastic

Pressure = 0.8 kg/cm^2
Generated

centre of the cup @ Flexion Point



3 cm Anterior to Posterior fontanel

OR

6 cm Posterior to Anterior fontanel

@ sagittal suture

- The Margin of cup touches the posterior fontanelle

Failed Vacuum ⇒ 3 Pulls - No descent of head
3 Pop offs

* if one device fails we go for C.S.

CI of Instrumental delivery ⇒

- a) Contracted Pelvis
- b) CPD.
- c) HIV ⊕ve Patient
- d) K/clo coagulation defect
- e) osteogenesis imperfecta

} in babies

ANTEPARTUM HEMORRHAGE (APH)

(94)

* Bleeding from or into the genital tract beyond the period of viability

↳ In India ≈ 28 weeks

* Causes of APH ⇒ Abruption

Placenta Previa

↳ Placenta lying in the LUS

↳ Premature separation of a normally located placenta (from underlying decidua)

ABRUPTION

- ✓
- ✓
- ✓

Previous history upto 1st. (Relative Risk)

- Pre-eclampsia ; Thrombophilia
- Trauma Folic acid deficiency
- Polyhydramnios Fibroid
- PROM
- Long standing oligohydramnios

Smoking

Multiparity

Highest Risks

High Risk

Early ♀ USG

↳ In subsequent scan ⇒ Placenta ⇒ Upper segmen.

In previous G.S.

↓ Migration abt

↳ So; RIF for Placenta previa

PLACENTA PREVIA

- ✓
- ✓

Previous history (5+)

Previous L-S.G.S (More the Number higher Risk)

Placenta May be Low Lying

d/d differential growth of uterus

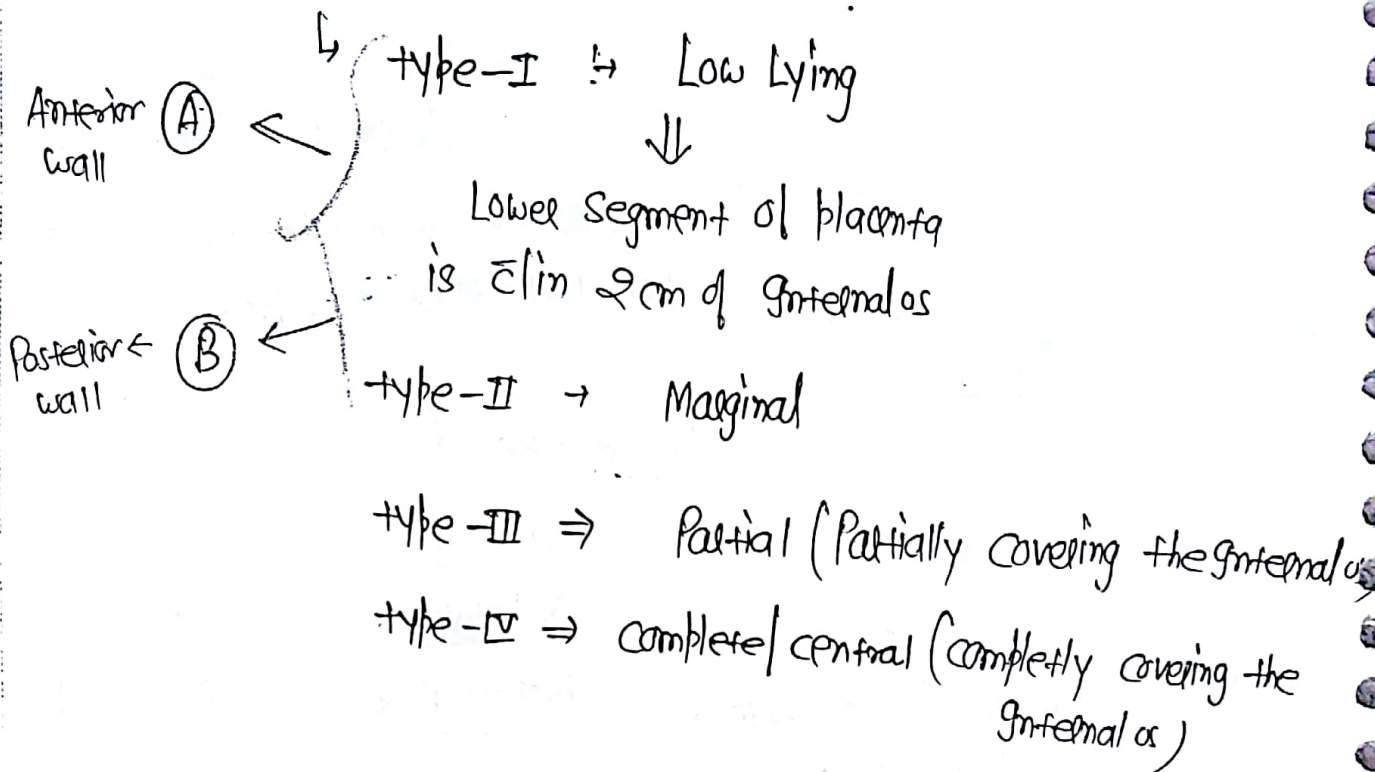
Placenta migrates

Endometritis

Mullerian Anomalies

CLASSIFICATION

Placenta PREVIA



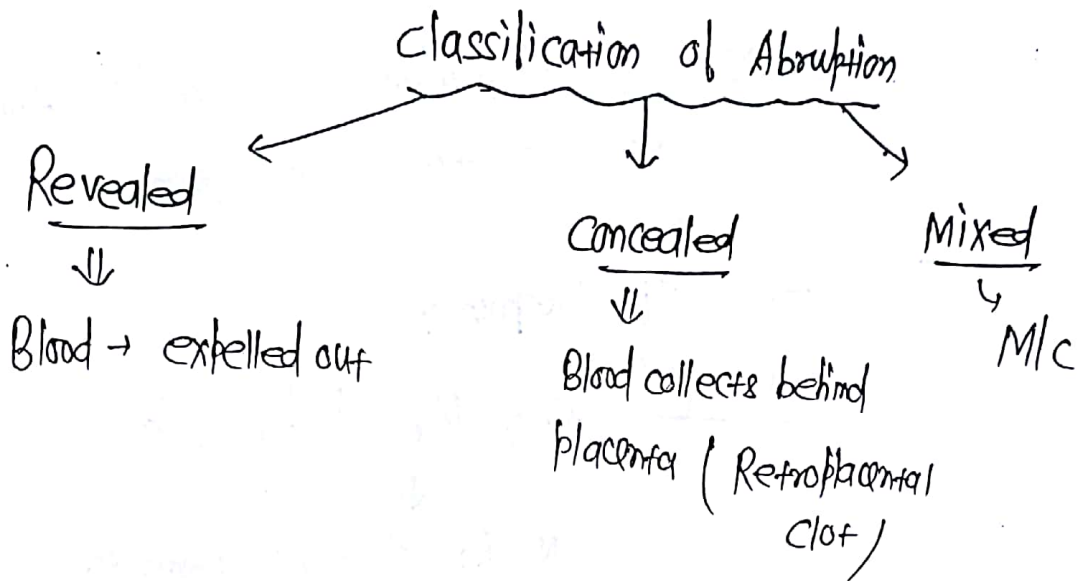
Other classification ⇒
Minor Placenta previa ⇒ Type IA; IB; 2A

Major " ⇒ Type 2B; 3; 4

↳ Mode of delivery ⇒ C-S

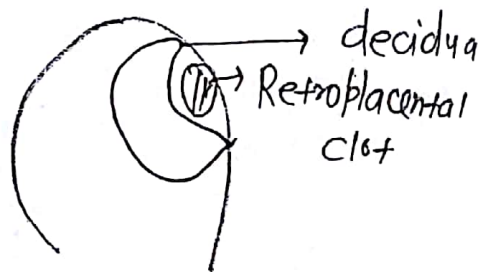
* Dangerous Placenta previa ⇒ 2B ⇒ Fetal distress

↳ on type 2B
↳ Stallworthy sign ⇒ Pushing down of head produces a dip in FHR.



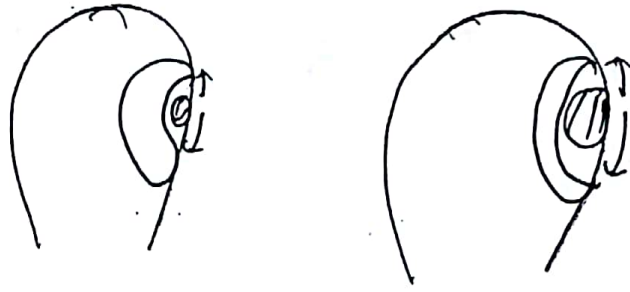
* PAGE'S classification of Abruption ⇒

- Type 0 ⇒ Retrospective diagnosis,
- Type 1 ⇒ bleeding & Pain (FHR ⇒ \odot)
- Type 2 ⇒ Bleeding + Pain + Fetal distress
- Type 3 ⇒ Bleeding + Pain + GUD + Shock
± DIC
Maternal



⇒ tissue thromboplastin
↓
Potent uterotonic
(Pain)

* Abruptio is self propagating



Risk of DIC is dependent on how long Abruptio to delivery times taken

* Termination of pregnancy

↳ only Mx of Abruptio



No Role of conservative Mx.

* Pt. comes w/ APH; how to tell about P.P. & Abruptio:

P.P.
Bleeding
↳ Painless
Bright Red (Fresh bleeding)
Causeless

M/c Presentation

Abruptio
Bleeding
↳ Painful
→ Altered (dark colour)
→ Preceding event
↳ Pre-eclampsia
Trauma

Hypotension M/c

← General Physical examination

⇒ Hypotension is Not common (bc basal BP is high)

P.P.
Uterus Relaxed
Non-tense
Non-tender

PIA
examination

Abruption
Uterus tense
tender (96)

Fundal height = P.O.G. ≡
< P.O.G. (Some times)

Fundal height > P.O.G.

Less common

Fetal
Distress

More Common

Most Common

Malpresentation

Less common

(Fundal height < P.O.G.)
→ Transverse Lie

H/O warning hemorrhages

No warning hemorrhage
(Acute event)

Should not be done in APH
Until you Rule out placenta previa
P/s And P/v examn

Transabdominal USG

TUC

* TVS is Not CI; but it
is More sensitive for Posteriorly
Located Placenta

Qe

Pt. Comes \bar{c} APH

L Pt. Not Low Lying (Fundal)

No RPC

ans:

Still a case of Abruptio

L Plv examination

L ARM

confirm

↓

Blood stained
Liquor

Induce Labour

*

Placenta praevia

L Mx \Rightarrow Are there any indications for TOP

↓

Yes (it is in order to do TOP)

↓

i) P.P. + Unstable vitals; (Mlc Indication for TOP)

ii) P.P. + Fetal distress;

iii) P.P. + > 37 weeks.

↓
do it Right Now

iv) P.P. + Continuous Bleeding Per vagina

v) P.P. + Woman goes into Active Labour

vi) P.P. + GUD

vii) P.P. + GGA (Incompatible for Life)

Q. G₁₃P₂L₂; 34 weeks APH (Bleeding p/v; Painless); on examination Fundal height = 34 weeks; Relaxed Non-tender Non-tense; Relaxed uterus; RR = 100/m; BP = 114/76. (97)

USG = type 4 PP.

Mx \Rightarrow Conservative Mx.

* Indication of CS in APH \Rightarrow

- i) Unstable vitals;
- ii) Fetal distress;
- iii) Major degree of Placenta Previa;
- iv) Far from term (< 32 wk)

* Conservative Mx in Placenta Previa

\hookrightarrow MacCallister Regimen

i) Bed Rest

ii) Maternal Monitoring : vitals;

FH (Fundal height)

AC (Abdominal circumference)

BPV (Bleeding Per vagina)

Baseline Ix

Uterine contraction

iii) Fetal Monitoring : FHR

iv) if patient < 34 weeks \Rightarrow Steroids for Fetal Lung Maturity

Dexamethasone (National guideline) \Rightarrow DoC

\Downarrow
6 mg im x 4 doses x 12 hr apart

only bleeding is Not
Indication of blood transfusion;
Anemia also a Indication

Beta Methasone - 12 mg x 2 doses 24 hrs apart.

∇ < 34 wk + Uterine Irritable (Mild contraction)

↳ give Tocolytics (to buy time for steroids cover)

↳ also for ECV

Hyperstimulation

• Doc. (Tocolytics) ⇒ Nifedipine

• Safest Tocolytic ⇒ "

• Doc. of Tocolytics in heart dx ⇒ Atosiban (Oxytocin Receptor Antagonist)

• Initially β-Agonists used as Tocolytics

↳ cause ⇒ Hyperglycemia AIIMS May 18

Hypokalemia

Tremors

Tachycardia

Arrhythmia

Cardiac arrest

Pulmonary edema

only β-Agonist used as Tocolytics

Now a days

↓

Terbutaline

Indomethacin

↳ Not given > 32 week

← Premature closure of ductus arteriosus

Halo-thane

Alcohol

Digoxin

MgSO₄

(acts as tocolytics @ 9-10 meq.) so Not given as a tocolytic agent.

↳ given 4-7 meq

gt has Neuroprotective action (prevent cerebral palsy)

↳ for preterm babies

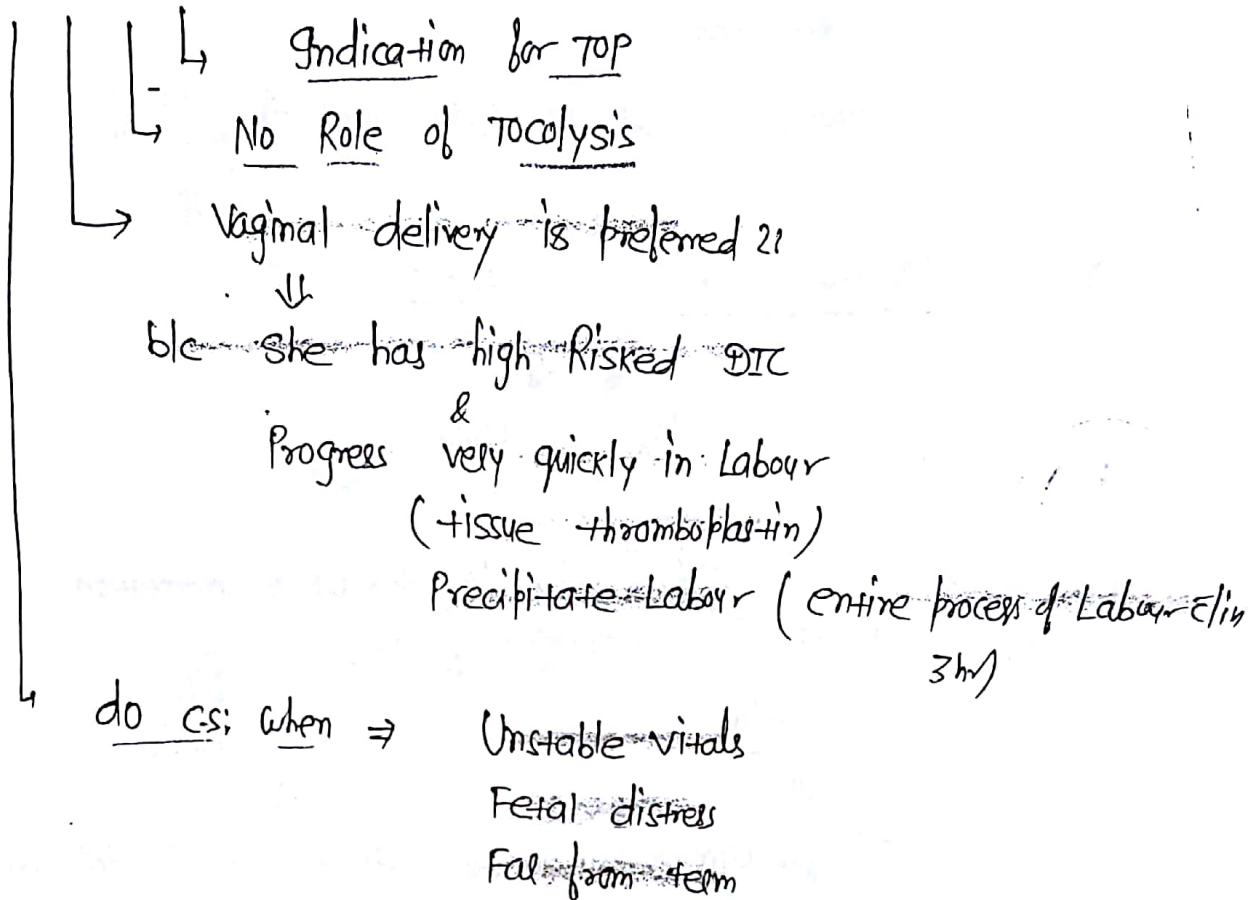
→ toxic dose for Mother

End point of conservative Mx

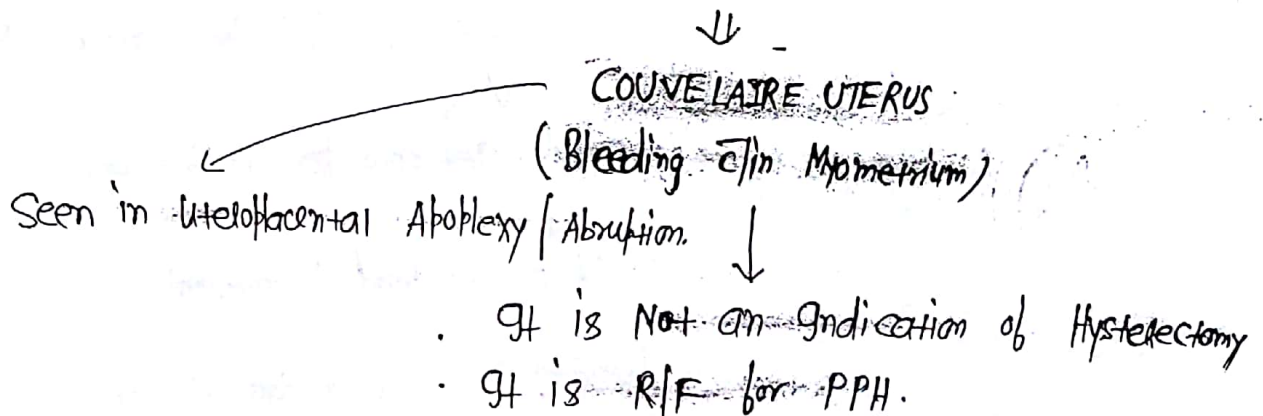
(98)

↳ 37 wks @ or any other indication of TOP.

ABRUPTION ⇒ No Conservative Mx



ib Intraoperatively we see Red Uterus



Q. 34 week ♀; Bleeding Plv & Pain; O/E uterus 36 wk; tense, tender; FHR = 100bpm; on Evaluation BT/CR/INR = deranged
 Mx ⇒ It is the case DIC

Transfuse FFP and try for vaginal delivery.
 in cesarean section; Pt. Bleed profusely from all the sites
 & dies on table.

Most cause of DIC in obstetrical ⇒ Abruption.

*

Placenta previa → term

Mx ⇒ USG

OR

do " Double set up examination "

← Plv in O.T →

1 table

↓

Ready for emergency C.S.

2nd table

↓

on Plv ⇒ Fornices boggy
 (≥ 2 fornices)

↓

We are done for Major placenta previa

if we don't get any fornices boggy

↓

Goes up to the level of Internal os

↓

if bleeding (+)
 or Placental tissue (+)
Near Internal os.

↓

do emergency C.S.

↓

if No placental tissue
Near the Internal os
 (Minor)

↓

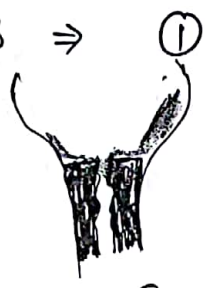
ARM → Labour Room

ADHERENT PLACENTA

(99)

Placenta is attached to Decidua; but here placenta is attached to Myometrium & Intervening decidua is absent

- Types =>



① Accreta => Attached to Myometrium
↳ Most common
↳ "Large intraplacental Lacunae" => Sonological finding

② Increta => Invades into the Myometrium



③ Percreta => Penetrates through & through the Myometrium. It comes out in the serosal surface.
↳ Least common

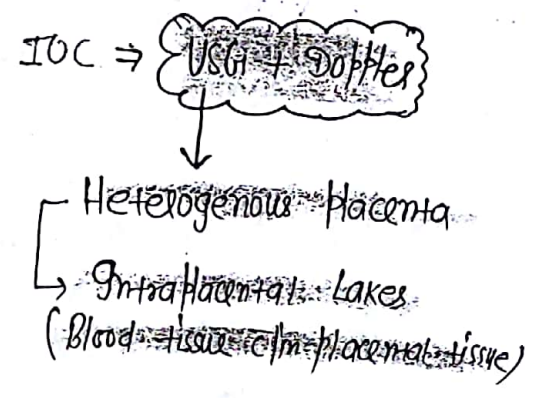


- Highest RIF => Previous L.S.G.s. + Placenta previa (current)

qa Highest RIF for Adherent placenta :-

- ① ~~Previous L.S.G.s.~~
- ② Previous P.P.

Other RIF => Curettage
Scal
Multiparity
Increasing Age



if we have any doubt / OR we ^{want to} know about depth

↓
do MRI

(N) prt. b/w placenta & decidua

on HPE ⇒

Absent / ~~Incompletely developed~~ Mitabuch's Membrane
(Layer)

↓
It is fibrinoid degeneration
b/w trophoblast & decidua

Mx ⇒ Kleio Adherent placenta

↳ Elective ~~CS~~ (classical)**

+
- Hysterectomy

↳ if we incise on LUS;
Myometrium cuts & we
damage placental tissue

• presentation in Undiagnosed case ⇒
deliver

↳ No sign of placental separation

↓
if we try to Manual Removal

↓
Pt. bleeds

↳ Refractory PPH (Pt. prt. with Refractory PPH)

↓
do hysterectomy

* if patient denies hysterectomy

↳ deliver vaginally

It will go autolytic digestion

2/m - 6 months

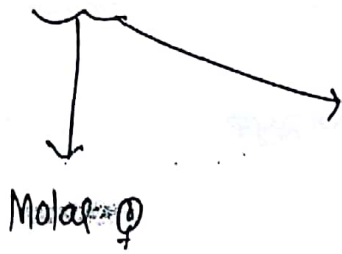
↳ cut the cord short as possible & close
to placental end

← leave the placenta
intact

MOLAR PREGNANCY

(100)

GTD ⇒ Gestational Trophoblastic disease



- Partial
- Complete

G.T. Neoplasia

- Invasive Mole
- Choriocarcinoma
- PSTT (Placental Site Trophoblastic Tumor)
- ETT (Epitheloid Trophoblastic Tumor)

PARTIAL MOLE

Chromosomal Make up of Molar ♀

↳ Triploid (9at)
 ↳ 69 XXY

10+ Tetraploid
 Extra set - Paternal
 Dispermic

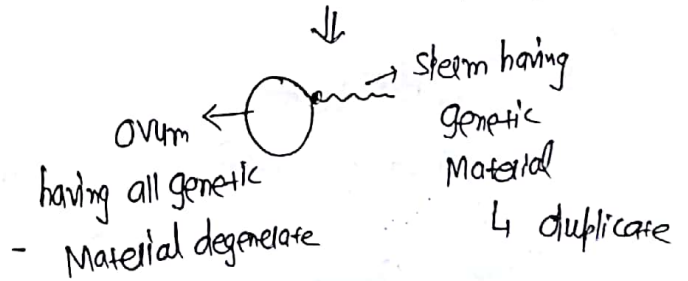


COMPLETE MOLE

Diploid (46XX) ^{9at}

Monospermic (Dispermic) ^{10+ 46XY}

All genetic material is Paternal



Bleeding (2nd Trimester ♀)

O/E ⇒ Fundal height < POG

Misdiagnosis USG

↳ Missed Abortion

M/C presentation

FOC

Bleeding (2nd Trimester ♀) ^{Like vesicle}

O/E ⇒ Fundal height > POG

USG

↳ Snow storm appearance

PARTIAL MOLE

- Fetus seen
 - ↳ die d/+ Multiple Anomalies

*If we can see focal hydropic in Placenta

↳ Transverse diameter > AP diameter

Most commonly diagnose in 1st Trimester (also in 2nd trimester)

- HCG > than expected (but Not Markedly high)

HCG

- Markedly Raised (>105)

HPE

— Gold Standard

- i) Less Marked trophoblastic Proliferation (Confirmation of investigation)
- ii) Focal hydropic degeneration of villi (diagnosis)
- iii) Fetus ⊕
- iv) Vascular

HPE

- i) extensive trophoblastic Proliferation;
- ii) complete hydropic degeneration of villi
- iii) No Fetus
- iv) Avascular

- Trophoblastic

Scalloping ⊕

↓

Stromal Inclusion

X

X

X

X

Medical complication

i) Thyroid Storm

ii) Pulmonary embolism

iii) Early onset pre-eclampsia

iv) Hypertensive gravidity

↳ dlt high hcb
↳ dlt paternally exposed Ag,
↳ dlt hca

COMPLETE MOLE

• No Fetus on USG seen

• ovaries → Theca-lutein cyst in it.

↳ 97-98-30% cases

~~Partial~~ Partial Mole \rightarrow GTN (3-5+) ~~Complete~~ Complete Mole \rightarrow GTN (15-20+) ¹⁹⁹ (101)
~~Partial~~ Mole \rightarrow chorio.ca (<14) ~~Complete~~ Mole \rightarrow chorio.ca (4-7)

$M_x \Rightarrow$ ~~Suction & evacuation (do Hill Fundal height \leq 12wks)~~
 \Downarrow
 if pt is \geq 40yrs + Complete Mole \oplus
 \hookrightarrow do hysterectomy (\downarrow Risk of chorio-ca)

\rightarrow SE \Rightarrow Pulmonary Embolism
 Thyroid storm
 to prevent Embolism \Rightarrow Start suctioning first then followed by oxytocin.

Check curettage \Rightarrow Sharp

* Send sample for HPE

\downarrow
then follow up \bar{c} HCG

Weekly s. HCG \Rightarrow until 3 \textcircled{N} values

\downarrow
Monthly HCG
Total \Rightarrow up to 6 Months
Surveillance

* Avg. time to β -hcg to become \textcircled{N} \Rightarrow 9 weeks
 Avg. time to β -hcg to become \textcircled{N} in Partial mole \Rightarrow 7 weeks

* Say patient Not to conceive

\hookrightarrow Contraceptive of choice \rightarrow OCP
 Barrier can be used
 gus Avoid blood bleeding

* CRITERIA FOR Diagnosis of GTN (Any 1) !→

① 4 consecutive HCG values shall show of Plateau
(Less than $\pm 10\%$ values)
~~D₁ 7 14 21 ($\pm 10\%$)~~

② 3 consecutive HCG value that shows a Rise
~~D₁ 7 14 21 ($> 10\%$)~~

③ HCG Remains elevated even after 6 months of Suction & evacuation

④ HRE → GTN

* Clinical presentation of GTN !→

i> Bleeding Per vagina — Persistent

ii> Shock

iii> Persistent Theca Lutein cyst



① Theca Lutein cyst shows Spontaneous Resolution
clin 2-4 months of Suction & evacuation.

iv Uterine Subinvolution

↳ uterus doesn't go to ① Non-♀ state after Suction & evacuation

v Metastasis

* High Risk for conversion of GTN ⇒

- i) ~~45 yr~~ (Maternal Age)
- ii) ~~Fundal height~~ > 90cm
- iii) ~~HCG~~ > 105
- iv) ~~Blc Large~~ (> 6cm) theca lutein cyst

Should Receive
Prophylactically
Chemotherapy
↓
Actinomycin D
OR
Methotrexate

* M/c GTN ⇒ Invasive Mole

4c GTN ⇒ ETT

M/c GTN to develop after Full term ♀ ⇒ Choriocarcinoma

* Choriocarcinoma M/c develop after ⇒ Complete Mole
which type of ♀

Malignant

↓
Metastasis seen (Common ball Metastasis on CXR)
↳ M/c site ⇒ Lungs > vagina > Liver > Brain

2nd M/c finding on Choriocarcinoma on CXR Lung ⇒ "Snow Storm Appearance"

also seen in USA of complete mole

* In vagina

↳ Bluish Suburethral Nodules ⊕

↳ don't take biopsy b/c very vascular

* How to differentiate Invasive Mole from Choriocarcinoma

(HPE) \rightarrow Presence of villi \Rightarrow Invasive Mole
 Absent villi \Rightarrow Choriocarcinoma

Choriocarcinoma

- M/c
- M/c after complete mole

Tumor Marker \Rightarrow HCG

Malignant

Bimorphic cells

(Cytotrophoblastic + syncytiotrophoblastic)

• Hemorrhage / Necrosis +

• Chemo sensitive

• TOC \Rightarrow chemotherapy

PSTT

Rare

M/c after ETP

Full term ϕ

HPE \rightarrow PLAP

Human placental Lactogen

Not benign

Placental alkaline Phosphate

Mononuclear Monomorphic cells
 (No syncytial ones)

\downarrow

Chemo resistant

Hysterectomy

* STAGING OF GTN

Stage I \Rightarrow Tumor is confined to uterus

Stage II \Rightarrow - Outside uterus but in pelvis

Stage III \Rightarrow Metastasis to Lungs (Good Prognosis)

Metastasis

Stage IV \Rightarrow Metastasis elsewhere except \rightarrow vagina

PNI

Modified WHO Scoring \rightarrow Low Score
 (Good Prognosis)

High Score
 (Bad Prognosis)

i) Age ≤ 39 yr

≥ 40 yr

ii) HCG ($\frac{240,000}{0.4}$) $\leq 10^3$

$\geq 10^3$

iii) Type of Antecedent ♀	Molar	FTP (Full term Pregnancy)
iv) Duration of Antecedent pregnancy	4 months	7-8 months
v) Site of Metastasis	Lungs	Liver, Brain
vi) No. of Metastasis	< 4	> 8
vii) Tumor size	< 3cm	> 5cm
viii) H/o Previous Chemotherapy	Single Agent	Multi Agent

if Total WHO Score ≤ 6

≥ 7

↳ Low Risk GTN

High Risk GTN

also in stage I

→ Single agent chemotherapy

Multi Agent chemotherapy

↓ Methotrexate

E → Etoposide - D₁

↓ if Pt. Resistant

M → Methotrexate - D₂

Actinomycin D

A → Actinomycin D - D₃

C → Cyclophosphamide - D₄

O → Oncovin (vinorelbine) - D₅

↓ if Resistant

E → D₁

M → D₂

A → D₃

E → etoposide

P → cisplatin

Follow up \Rightarrow Weekly hcg - till 3rd value

End point of chemo. \Rightarrow 1st value - 3 more cycles of chemotherapy

After 3rd value \Rightarrow Monthly follow up

Period of Surveillance for Low Risk GTM - 12 Month

" for high " " \rightarrow 24 Month

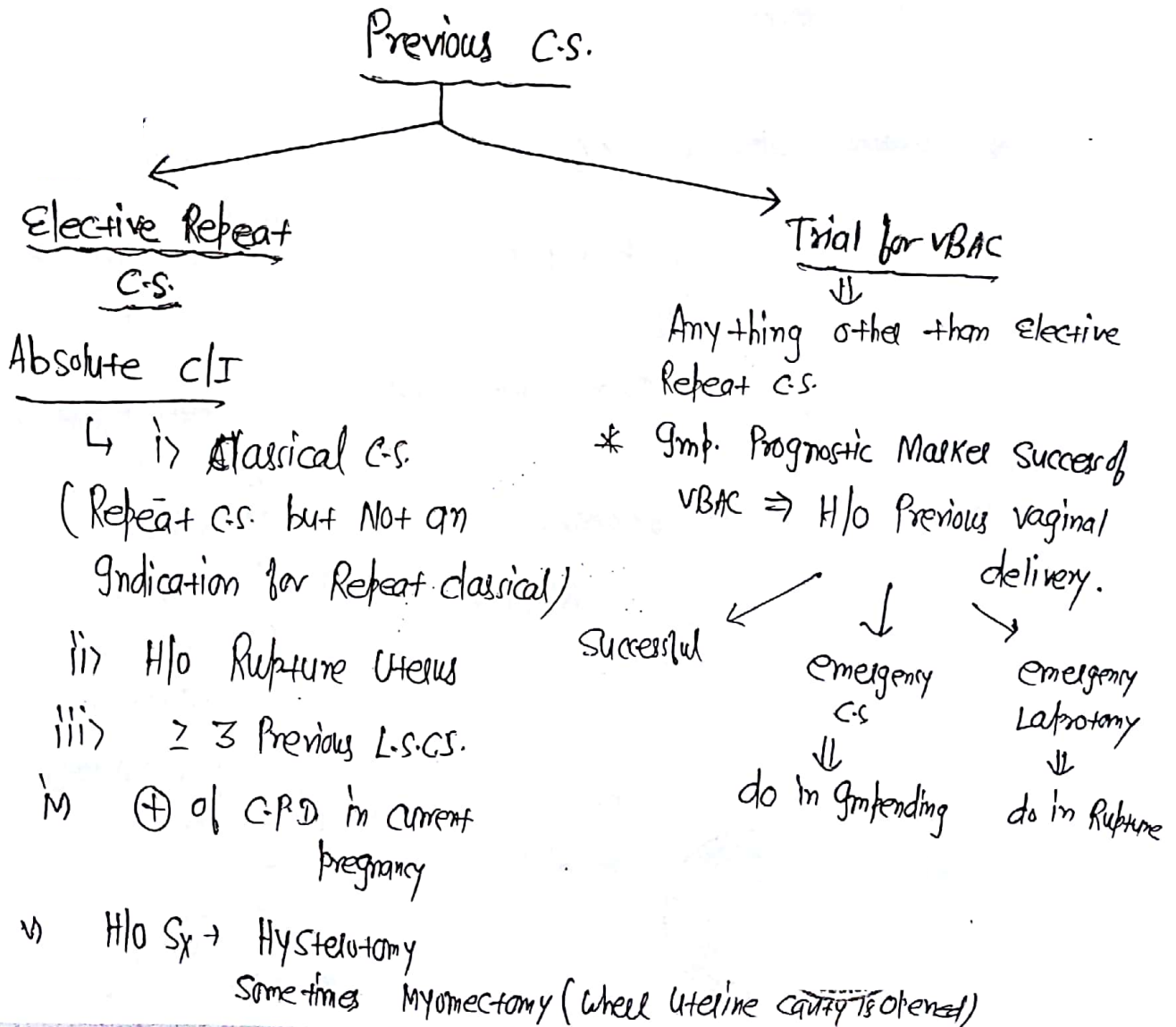
Advice & contraception of choice is same as Molar ϕ

\Downarrow

\hookrightarrow

Combined ocp's

Not to conceive



Q. $G_{12} P_{1L}$ \bar{c} previous L.S.G.S. ; done for c.p.d. 40wk; Head is breech bloating Cx os closed ^{Unellaced} \downarrow Myx (104)

\downarrow
C.S. (blc c.p.d. \oplus in current \bar{c})

vii) where vaginal delivery is c/I (Contracted Pelvis
Major degree of Placenta previa)

* Relative Indication for Repeat C.S. (C.S. $>$ V.D.) \Rightarrow

i) Previous L.S.G.S. — \bar{c} Breech in current \bar{c}

ii) " — \bar{c} Macrosomia

iii) " — \bar{c} Post-term

* Sign & Symptom of Impending Rupture \Rightarrow

i) Non Reassuring FHR

\hookrightarrow 1st FHR changes \Rightarrow Tachycardia

M/c FHR changes \Rightarrow Bradycardia

ii) evidence of Maternal Tachycardia;

iii) evidence of Scal tenderness (Pain);

become only significant if Non-Reassuring FHR \oplus .

\downarrow

ib Intraoperatively ; at the Scal site — all Layer of Myometrium have given away but overlying Serosa intact

Very Imp. for Next \bar{c}

\downarrow
 \leftarrow "Scal dehiscence"

* Sign & Symptoms of Rupture of Uterus →

- i) Maternal Tachycardia;
- ii) ± Hypotension;
- iii) Severe fetal distress / FHR absent;
- iv) Fetal parts are superficially felt; (as the fetus is expelled into Abdominal cavity)
- v) Uterine contour is lost.
- vi) Sudden stoppage of uterine contraction;
- vii) fresh bleeding per vagina;
- viii) catheterise → gross hematuria
- ix) Loss of station (Most characteristic feature of Rupture Uterus)
- x) ⇒ emergency Laprotomy (Tries to Repair)

* Can we do Induction in Labour (IOL) in previous C.S

↳ Not C/I

↓

Spontaneous Labour > IOL

↓

DOC ⇒ Oxytocin

Not be given ⇒ Misoprostol

* Can we do Augmentation of Labour

↳ Not C/I

↳ continuous fetal Monitoring (to identify 1st sign of impending Rupture)

* Can we do Ecr in Previous C.s.
 ↳ Yes (Not CLI) (105)
 ↳ Relative CLI for ECV

* Internal Podalic version (IPV) in Previous C.S.
 ↳ Absolutely CLI in Previous C.S.

* Induction of Labour is CLI in → Contracted Pelvis
 ↓
 It means we can't deliver vaginally.
 Classical C.S.
 H/o Rupture uterus;
 Transverse lie;
 Major degree of Placenta previa;
 Category 3 FHR tracing
 ↳ Ominous to baby
 ↳ Immediate delivery

* Pre-Induction Score → to Induce Labour
 (Bishop's score)

	<u>Score</u>			
<u>Cervix</u>	0	1	2	3
<u>Position (Leat-imb)</u>	Posterior	Mid-position	Anterior	
<u>Consistency</u>	Firm	Medium	Soft	
<u>Effacement</u>	0-30%	30-50%	60-70%	> 80%
<u>Dilatation (Most. Imp)</u>	closed	1-2cm	3-4cm	> 5cm
<u>Baby's station</u>	-3	-2	-1, 0	+1, +2

In Modified Bishop \Rightarrow

Cx effacement is Replaced by Cx Length

In Simplified Bishop \Rightarrow

dilatation
effacement
station

* All

Scores ≥ 9 (>8) \Rightarrow

Favourable \rightarrow

Initiate Uterine
contraction.

≤ 5 (<6) \Rightarrow

Unfavourable

Total score -13

↳ firstly do cervical Ripening

Medical
Methods

Mechanical
Methods

• Celviprimegel
(dinoprostone)
PGE₂

0.5mg

Ideal \rightarrow Intracervical

Acceptable \rightarrow Posterior fornix

• Put 6 hrly Max^m 3 doses
in 24 hrs

• Mlc used

• gt do only Ripening; so,
after Ripening; give oxytocin
+0 uterine contraction (after
6hr.)

↳

• Misoprostol
(PGE₁)

↓

25 Mgs
vaginally

- Put every 4hrly

Max^m 6 doses/24 hrs.

• Better

(do Ripening + Uterine contraction)

- after 4 hrs we give oxytocin; if
uterine contraction is Not

- Laminaria Tents
(osmotic dilators)

- Bulb of Foley catheters

TWINS PREGNANCY

* M/c type \Rightarrow Dizygotic (70+); klas "Fraternal twins"
Monozygotic (30+); klas "Identical twins"

* In ART procedure \Rightarrow M/c type \Rightarrow Dizygotic twins

* Incidence of Dizygotic twins \leftarrow Varies
Incidence of Monozygotic twins Constant

depend on \Rightarrow Race
ethnicity
Family History
ART

M/c type of Monozygotic twins
 \hookrightarrow MCDA

* Dizygotic \Rightarrow Dichorionic
Diamniotic
M/c type \nearrow

- depend on time of cell division

\bar{c} in 72 hrs of Fertilization \Rightarrow DCDA

\bar{c} in 4th-8th day " \Rightarrow MCDA

\bar{c} in 8-12th day " \Rightarrow MCMA

\bar{c} in >12th day " \Rightarrow conjoined twins (Siamese)

* Monochorionic twins has higher Risk as compared to Dichorionic

* Marker of Dichorionicity (to know chorionicity we do USG in 1st trimester (7-14 week))

i) 2 separate Placenta on USG

ii) Opposite sex twins;

iii) Twin Peak sign (Lambda sign)
(seen in 7-14 week)

\hookrightarrow ble chorion enters into

* In Monochorionic twins

↳ Inverted "T" sign seen on US

iv) 4 layers in dividing Membrane
2 Amnion 2 chorion

v) Thickness of dividing Membrane

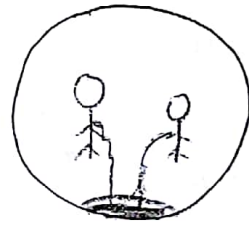
↓

$\geq 2\text{mm}$ thick \Rightarrow Dichorionic

if $< 2\text{mm}$ thick \Rightarrow Monochorionic

* Monochorionic Monoamniotic twins

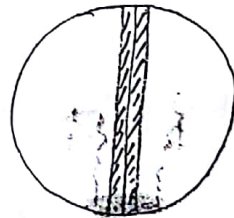
↳



* Most type of conjoined twins \Rightarrow Thoracopagus
↳ to joint

* Monochorionic Diamniotic twins

↳



* M/C Risk to fetus in twin ♀ \Rightarrow

also \Rightarrow

Pre-term Labour

GCA \uparrow

gUGR

} comparison to
single-ton ♀

* 2 things not seen in Multiletal ♀

↳ Post dated ♀
Macrosomia

* Fetal Reduction ⇒

- Ideal time to do ⇒ 10-13 week
- Under USG guided Inject KCl into thorax
- converted into 2 betw (Not less than twins)

* Monochorionic twin (specific) complication ⇒

- TTTS
- TRAP (Twin Reversed Arterial Perfusion)
 - ↳ klas "Acadiac twinning"
- TAPS (twin Anemia Polycythemia sequence)
- Selective GUR
- ↑↑ Risk of GCA

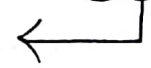
TTTS (Twin twin transfusion Syndrome)

It is seen in Monochorionic Diamniotic

↳ Reason for TTTS

↳ Vascular Anastomosis

Deep Artery of one baby anastomoses with deep vein of other baby

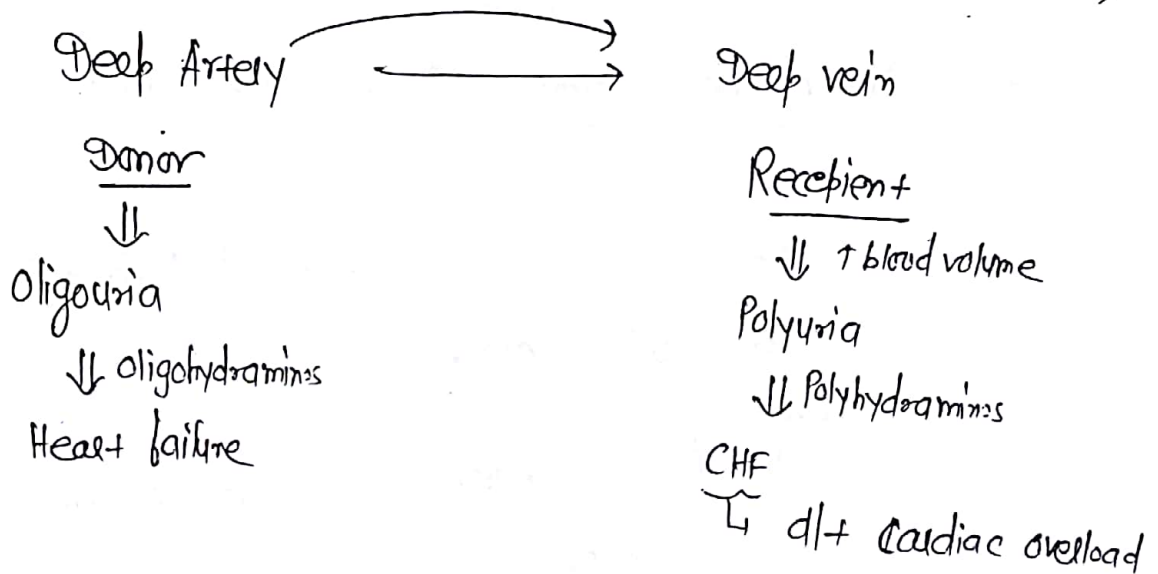


* In Monochorionic Monoamniotic twins

↳ Plenty of Superficial Artery-Artery
Anastomosis ⊕ ; so; TTTS
not seen in it

Diagnosis ⇒ USG

Simultaneously — Polyhydramnios (by DUP Method we see Amniotic fluid)
— oligohydramnios



* QUINTERO STAGING ⇒ Staging of TTTS

Stage 1 → Poly/oligo + Bladder of oligo twin is visible + Doppler (N)

Stage 2 → Poly/oligo + Bladder of oligo twin is Not visible + Doppler (N)

Stage 3 → Poly/oligo + Bladder Not visible + Doppler (ABN)

Stage 4 → Either / both hydrops fetalis

Stage 5 → Either / both GUD.

TIT \Rightarrow Fetoscopic Laser Ablation of the vascular Anastomosis. (108)

Stuck twin \Rightarrow ^{alive; only stuck} Oligo twin of twin twin transfusion sx.
 \downarrow can't move; so; Looks like stuck

Vanishing Twin \Rightarrow Spontaneous abortion of one of the twin.

Fetus Pappeyruvus \Rightarrow One twin dies & is compressed by other twins.

Impending death
 \downarrow
 do TOP
 \downarrow
 take out both the babies; as other baby may go to hypoxic injury

if one twin - already die
 \downarrow L gas
 continues pregnancy
 + Monitors the other twin
 \downarrow
 Baby may go hypoxic injury & coagulation defect both

Superfetation \Rightarrow different cycle (Menstrual)

Superfecundation \Rightarrow same cycle

OUTCOME OF TWINS \Rightarrow Lie of 1st Twin

1st - Twin - Longitudinal - vaginal delivery

Non-Longitudinal - C.S.

* M/C combination In Labour \Rightarrow Vertex - Vertex
 1st twin 2nd twin

Vertex - breech

Q. Q.

Mx = ?? ; i/b 1st twin = Breech; 2nd = vertex

Vaginal delivery is Not c/I

C.S. > vaginal delivery

Complication \Rightarrow twin interlocking

Q. Q.

Mx = ?? ; i/b 1st vertex deliver ; 2nd breech

\Downarrow

Assisted Breech vaginal delivery

Q. Q.

Mx = ?? ; i/b 1st vertex deliver ; 2nd Transverse lie

\Downarrow

do Internal Podalic Version (IPV)

Breech extraction

gives under
GA (General Anesthesia);

Mother have no effect
to push the baby; so, do
Breech extraction

\hookrightarrow i.e. No efforts by Mother at all

* The lie of 2nd baby is Not
decided until 1st baby is delivered

Q. Q.

M/c complication of Monoamniotic twins are

\hookrightarrow

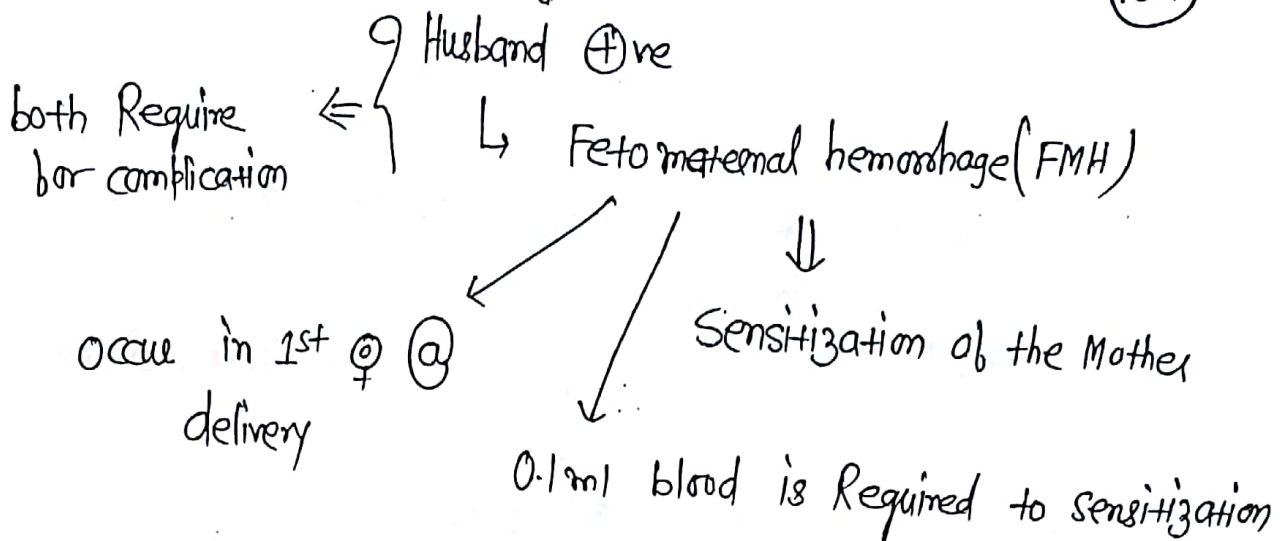
Cord Entanglement

\hookrightarrow delivered by CS:

\Downarrow

by 32-34 weeks

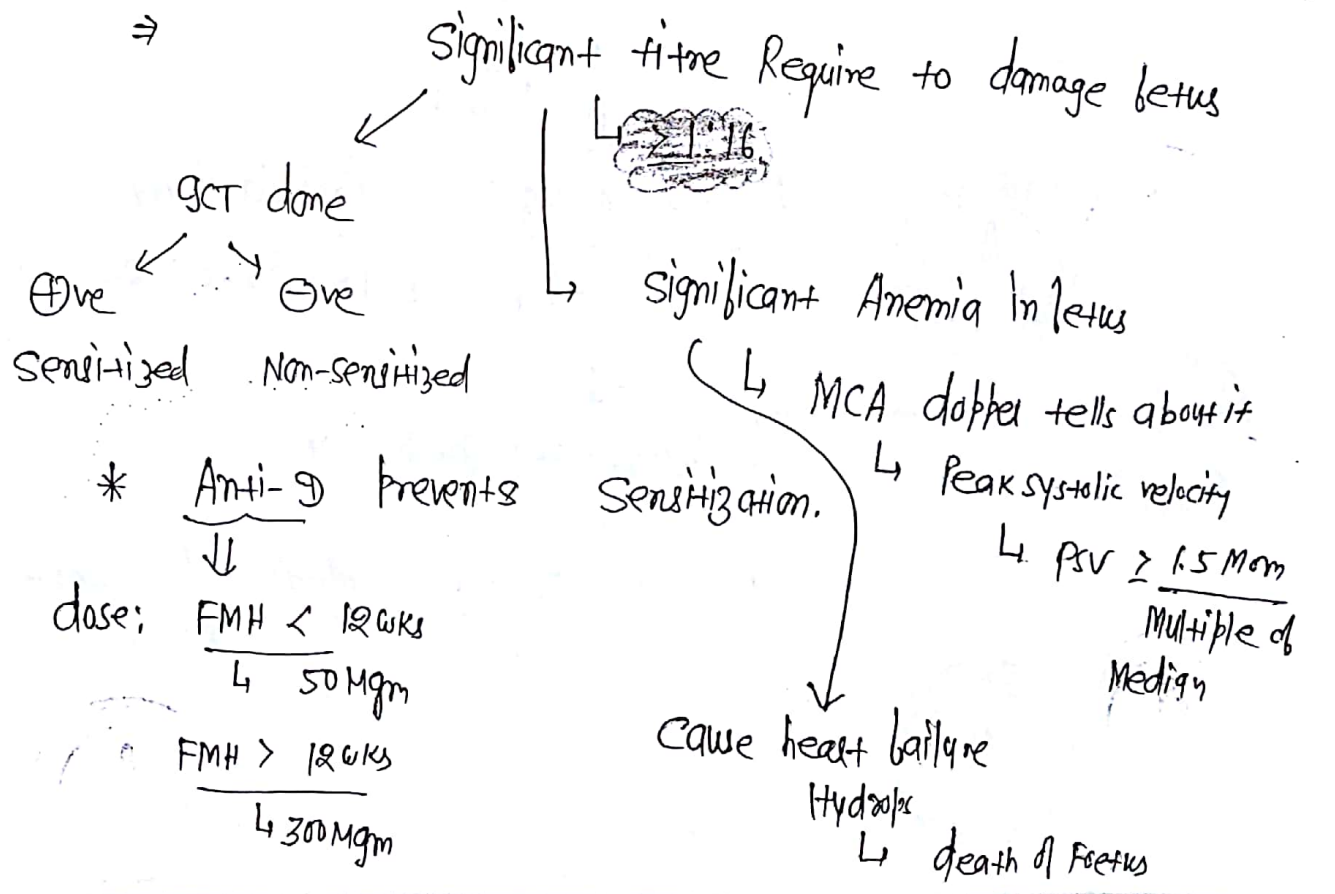
Rh \ominus ve PREGNANCY



\Rightarrow 1st Ab produced by Mother after sensitization

- \hookrightarrow IgM (doesn't cross the placenta & Not cause affect in baby in 1st $\textcircled{\ominus}$)

Later she produce IgG (cross placenta)



* 300 Mgm of Anti-D Neutralize fetal blood - 30mL
fetal RBC - 15mL

* if we suspect FMH more than usual then to calculate dose of Anti-D — Kb test
(Kleihauer-Betke)

Case I →

Rh ⊖ve
↓
Husband ⊕ve
↓
@ 12 week GCT ⊖ve
(Not sensitized)
↓
GCT (28wk)
L ⊖ve
↓
Anti-D 300-Mgm → Prophylactically
↳ this works for 12 wks; so she delivers after safely.
↓
if Baby blood group ⊕ve
↓
Anti-D 300 Mgm (ideally 2/1m 72hrs; can be given up
i/m. 28 days)

Case 2 →

Rh ⊖ve



Husband Rh ⊕ve



@ 12 weeks

GCT ⊕ve
(< 1:16)

(Anti-D has No Role in GCT ⊕ve Patient; it Means Sensitization already takes place)



Repeat GCT x 4 weekly

if Rising trend; Repeat 2 weekly



deliver at term

Case 3 →

Rh ⊖ve



Husband Rh ⊕ve



@ 28 weeks

GCT ⊕ve
(> 1:16)

if Hb level < 5 gmt. of fetus



Hydrops fetalis

↓ followed up for severe Anemia

PSV - MCA doppler

≥ 1.5 Mom

< 1.5 Mom

↳ Repeat MCA doppler

P.O.G. ≥ 34 weeks

↓
deliver

P.O.G. < 34 weeks

↓ do cordocentesis

intrauterine transfusion
if Hb < 8 gmt



Hydrops fetalis \Rightarrow Most common cause (Non-immune Mediated)

\downarrow
M/c/c

\downarrow Immune Mediated

\downarrow CVS - abnormalities

Infection that can cause hydrops fetalis \Rightarrow Parvovirus B-19

USG diagnosis \Rightarrow Any ≥ 2 of the following

- i) Pleural effusion
 - ii) Pericardial effusion
 - iii) Ascites
 - iv) Subcutaneous edema
- } criteria

Scalp edema \Rightarrow Buddha sign.

findings in hydrops fetalis \Rightarrow Placental Megaly
Polyhydramnios

* Gene for Rh factor Location \Rightarrow Short Arm of chr-1

HEART DISEASE IN PREGNANCY

219

(11)

Symptoms

- Orthopnea
- Paroxysmal Nocturnal dyspnea
- exertional dyspnea (Progressive)

↓
It is Physiological; but progressive exertional dyspnea is Pathological

Signs -

- Cyanosis / clubbing
- Diastolic Murmur
- Systolic Murmur. > grade 2
- Cardiomegaly
- Atrial fibrillation / (AbN) Rhythm
- CHF
- Persistently distended Neck veins
- Pulmonary Artery HTN
↳ Loud P₂
- wide split S₂

* M/c heart disease in ♀ ⇒ Mitral Stenosis

M/c congenital heart disease in ♀ ⇒ ASD

M/c congenital valvular heart disease ⇒ Mitral valve Prolapse

* High Mortality Rate (> 50%) in ♀ ⇒

- i) Marfan's Sy ⇔ Aortic Root Involvement
- ii) Coarctation of Aorta ⇔ Aortic valve Involvement
- iii) Eisenmenger Syndrome

→ TOP (Abortion)

In Severe MS (< 1.5 cm²)
↳ valve Area

v) NYHA grade 3/4

vi) Ejection fraction < 45%

* good outcome

↳ ASD	Corrected TOF
VSD	Mitral valve Prolapse
PDA	Ebstein Anomaly

* if a patient has severe MS

↳ ideally goes to - Valve Replacement
 ↳ if she wants to continue pregnancy

High Mortality Rate \Rightarrow 30%

Sx preconceptually

↓

Not be done during pregnancy

↳ do Balloon valvotomy

↳ ideal time

↳ 18-20 weeks

* if she has valve Replacement

Prosthetic heart valves \rightarrow Pt. is on Anti-coagulation

↓ if she wants to conceive

Antidote for heparin
 ↓
 Protamine sulphate
 Coumadin has No Antidote

1-12 weeks \rightarrow Heparin

12-36 weeks \rightarrow Warfarin (More potent)

36 - onset \rightarrow Heparin

causes embryo pathy in 1st trimester

↓
Chondrodysplasia punctata (stippled femoral epiphysis)

On close to Labour; if warfarin given; PPH happen also

* During Labour → Stop Anticoagulant (112)

However Restart the Anticoagulant In vaginal delivery ⇒ Alter 6hr
In cs ⇒ Alter 24hr

Restart = Heparin + Warfarin
With draw Heparin; core -d INR; As; Warfarin has delayed onset of Action

* GENERAL PRINCIPLES ⇨

i) Maxim Rise of CHF ⇨

Immediate Post partum > 2nd stage > 32 weeks

ii) vaginal delivery is preferred;

C.s. is Reserved for obstetric Indication

iii) = Heart disease Indication of C.s ⇒

- i) Marfan's Sy = Aortic Root Involvement
- ii) Contraction of aorta = Aortic valve Involvement
- iii) Aortic dissection
- iv) Severe AS

In all Ejection fraction is affected

* In Eisenmenger's Sy ⇒ vaginal delivery tries

* Induction of Labour (IOL) is not contraindicated
in heart disease

Lgt is safe

however; Spontaneous Labour over IOL

QA 38 wks - Heart failure; K/d/o Heart disease;

↓
Mx ⇒ Stabilize the patient

↓
Wait for Spontaneous Labour

iv Propped up | Left Lateral Posture | O₂ by Mask - Readily available

v Restrict IV fluids @ 75 mL/hr

A.S ⇒ wet side

vi Restrict the IV exams

vii ARM — Can be done

Memb. Rupture — Prophylactic Antibiotics
(AHA guidelines)

↓
Ampicillin + Gentamicin

↓
Vaginal delivery safe

viii Pain Management ⇒ Epidural Analgesia
(Neuraxial)

ix) Cut Short the 2nd Stage of Labour

(113)

↓
Forceps delivery > Vacuum delivery

x) Immediately after delivery
→ Given Grij. Lasix (to ↓ Preload)
→ Avoid Methargin

Hang down the patient leg from delivery table (to ↑ venous Return)

xi) C.S. ⇒ Anesthesia

→ S/E ⇒ Hypotension

Severe AR }
Severe AS } ⇒ Low Ejection fraction
Cyanotic H.O. }
* In Aortic dissection (emergency condⁿ) do emergency C.S. under G.A.

PERIPARTUM CARDIOMYOPATHY

- Development of heart failure Around Labour in a woman \bar{c} No underlying Heart Disease
↳ 1 Month before delivery to 5 Month after delivery
- Ejection fraction - Low.
- Left ventricle May or Mayn't be dilated
- Mainly in Pre-eclampsia patient; also in Multi-fetal ♀; advanced Maternal age
- Prolactin has some role to develop
- Mx = Same as Heart failure

Fetal Monitoring

Fetal Movements \Rightarrow Quickening (1st Fetal Movement)

\hookrightarrow 16 weeks - Primigravida
18 weeks - Multigravida

(N) ≥ 10 fetal movement in a 2 hr period of Rest
OR
 ≥ 10 fetal movement in a 12 hr period in Routine activity

Max^m fetal movement \Rightarrow @ 32 weeks Perceived by women

- earliest time for gud in Absence Fetal Movement = 12 hrs
Max^m time for gud in Absence Fetal Movement = 48 hrs

- Modified Biophysical Profile (BPP) \Rightarrow ↓ fetal movement beyond 32 weeks

also klas "cardio-tocography"

(NST)

\Downarrow
Gn Acute Injury

(AF)

\Rightarrow Amniotic fluid

\hookrightarrow Gn Chronic Injury (UPI)

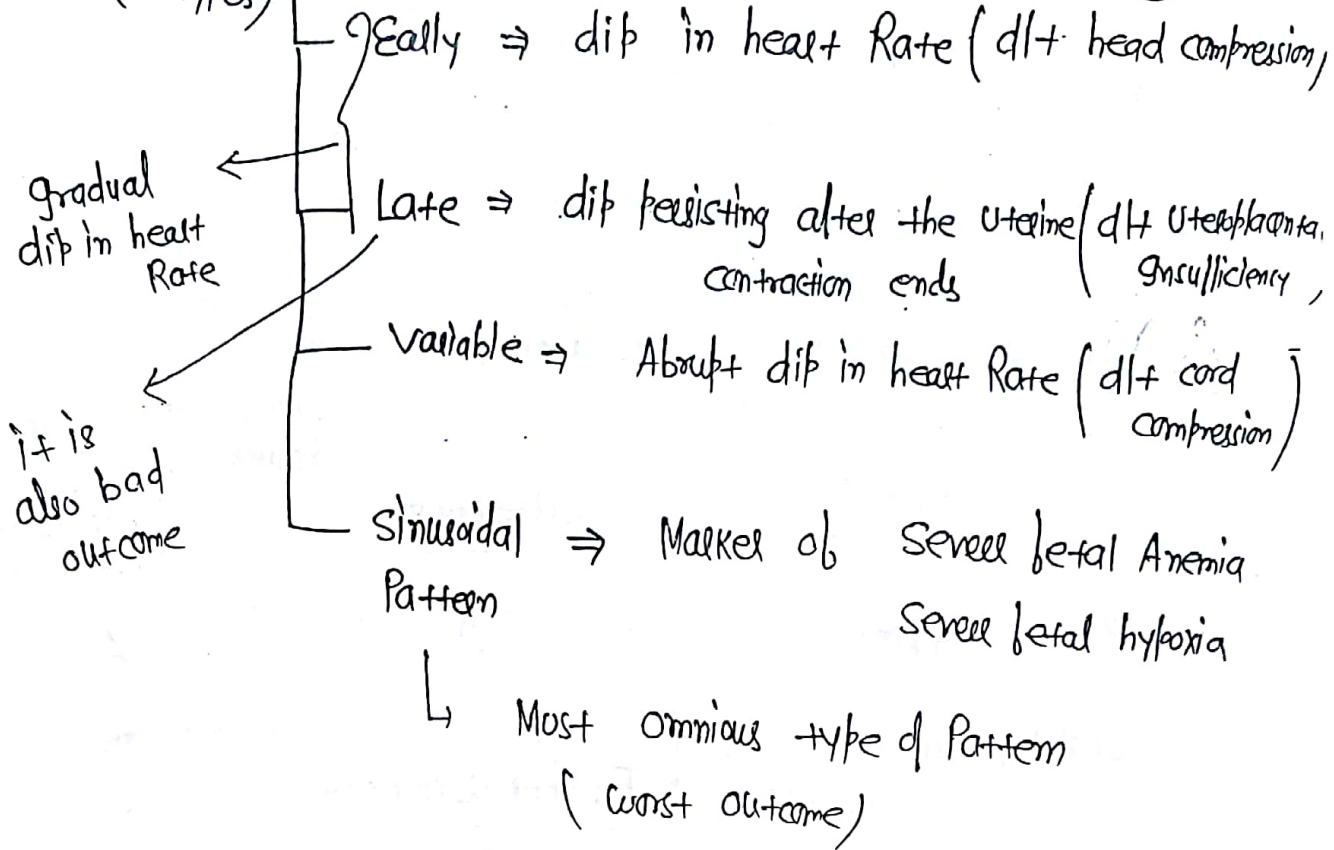
(N) Heart Rate = 110-160 beats/min.

Beat to beat variability = 5-25 beats/min

Acceleration \Rightarrow \uparrow Heart Rate by 15 beats/min above baseline for 15 sec

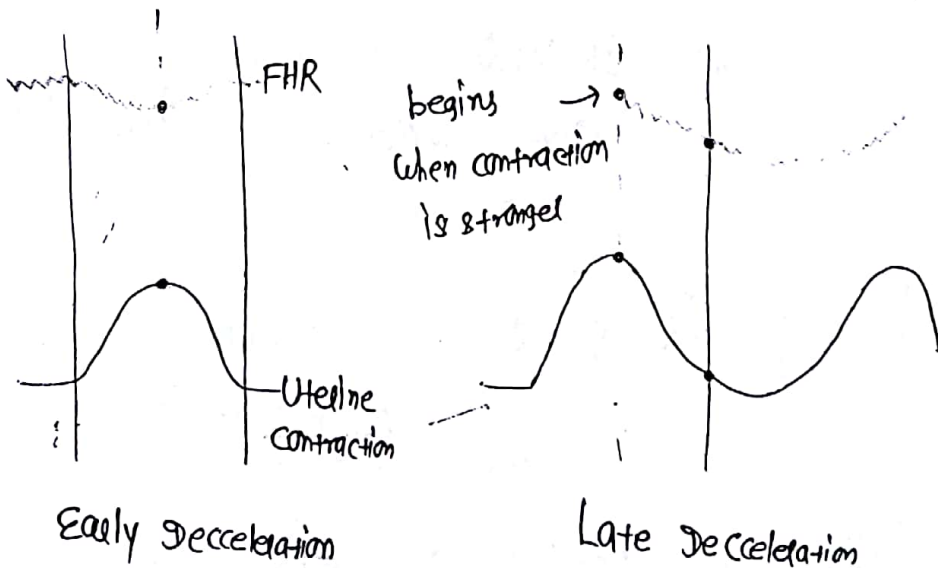
Deceleration

(3 types)



When variable deceleration is significant

- i) $HR < 70 \text{ beats/min} \times 1 \text{ min}$
- ii) Persistent Around $\geq 50\%$ of uterine contraction



Reactive NST ⇒ two or more than two ~~to~~ acceleration in a span of 20 min.

Non-Reactive NST ⇒ < 2 acceleration in a span of 40 mins
↳ Physiologically if baby is sleeping

* Category 1

Beat-
Beat
variability

(N)

Category 2

- Further assessment
- LLP (Left Lateral Posture)
- O₂ by Mask
- Stop oxytocin
- IV fluids
- Give tocolytics

Immediate delivery → C.S. ← ominous
Category 3

Absent \bar{c}

Any one of the following

↓

Bradycardia

Late deceleration

Persistent variable deceleration

• Sinusoidal

NST in high Risk ♀ ⇒ twice a week
(once in 72 hrs)

* BPP (Biophysical Profile) ⇒ Manning Score

↳ Report card of Fetus

→ USG for 30 min

(15)

↳ we use a score of +2 or zero

- It has 5 components

↓

Breathing Movement ⇒ At least one Movement Lasting 30sec +2

Gross Body Movement ⇒ 3 Movements +2

Tone ⇒ Flexion - Extension - Flexion +2

Amniotic fluid ⇒ At least 1 Pocket of 2cm +2

NST ⇒ Reactive +2

if Score is $\frac{8,10}{10} \Rightarrow (N)$

$\frac{6}{10} \Rightarrow (N)$ Liquor = Equivocal = Repeat testing on the same day

$\leq \frac{4}{10} =$ Immediate delivery

* (ABN) In Acute hypoxic

↓

Loss of Acceleration → Breathing → Gross body → tone Movement

1st to become (ABN)

* How frequently to be done in high Risk ♀
↳ once in week

DOPPLER ⇒ • M/C 1st vessel ⇒ UPEline A
↳ signs of UPE

• S/D Ratio

↳ (N) ♀ S/D ↓ing

• UPE ⇒ S/D ≥ 3

↓

> 28-30 wk

↓

≥ 3

↳ UPE

REDF → TOP

• AEDF - TOP ≥ 34 weeks

↳ (a) 34 weeks - gives steroid + further monitoring.
or less than 34 weeks

* **MCA Doppler** ⇒ Not best for UPE

↳ (N); b/c in UPE; fetus sends blood to vital organs

↓

early stage MCA doppler ⇒ (N)

↓

Brain sparing effect

* REDF — Umbilical Artery

↳ Steroids — 2 days

↓
Reversal in venous Doppler ^{Last vessel to show REDF}

⇓
Indicates impending death.

* MCA Doppler is best studied for fetal Anemia

* Best for Fetal Monitoring

↳ Fetal Scalp blood PH

(N) ⇒ 7.25 — 7.35 ^{Repeat test after 30 min}

7.20 = 7.25 = Borderline

< 7.20 = Acidosis

↳ Immediate delivery

* VAST (vibroacoustic stimulation test) ⇒

High Intensity Sound waves

Released from Artificial larynx → on Maternal Abdomen for 1 sec (to 2 sec)

(N) Response ⇒ ↑ 15 bpm from baseline by 15 bpm in 15 sec of stimulus

* M/c Method for Intrapartum Monitoring →

Intermittent - Heart Rate Auscultation.

1st stage

2nd stage

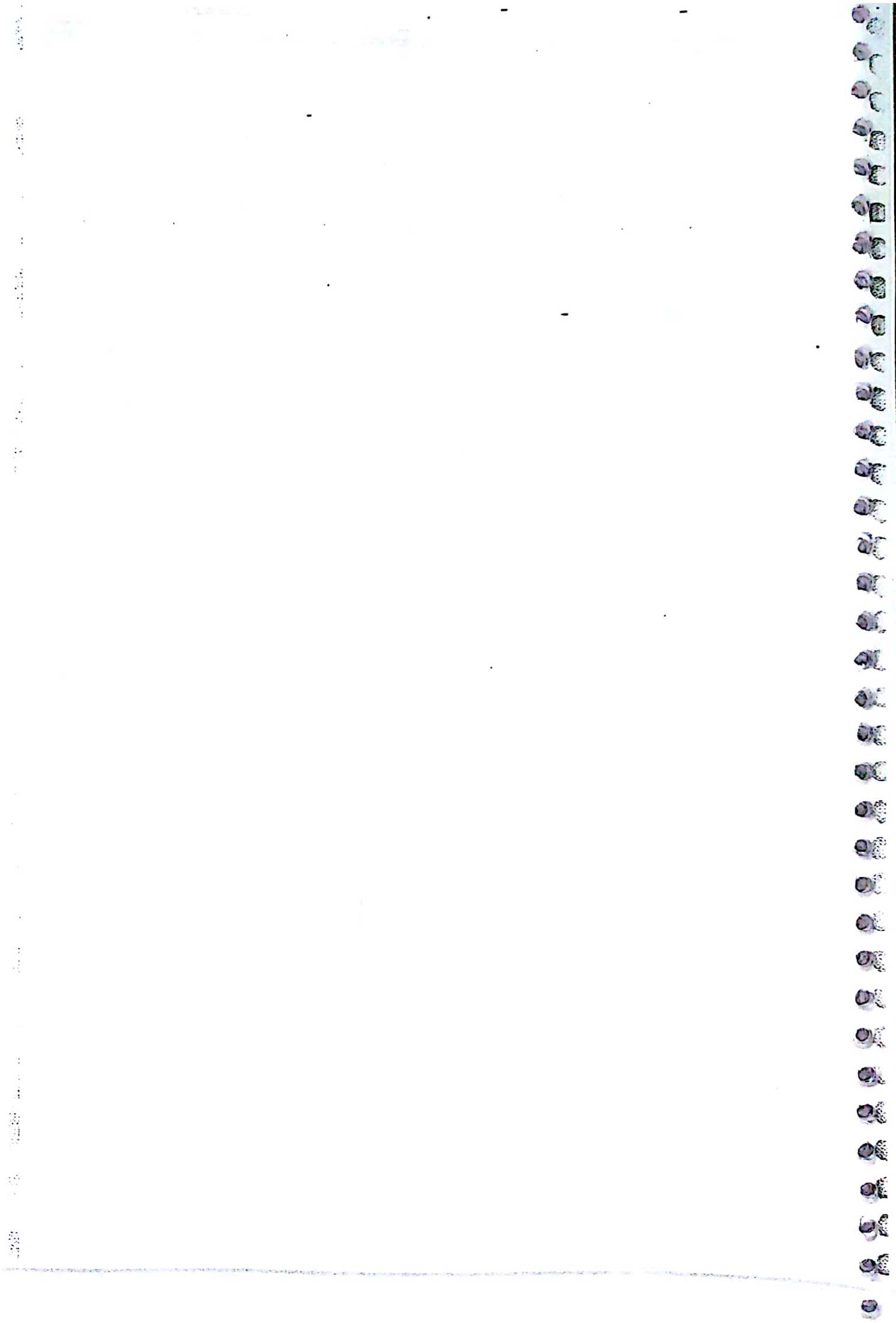
In Low Risk ♀ - every 30min

every 15min

In High Risk ♀ - every 15min

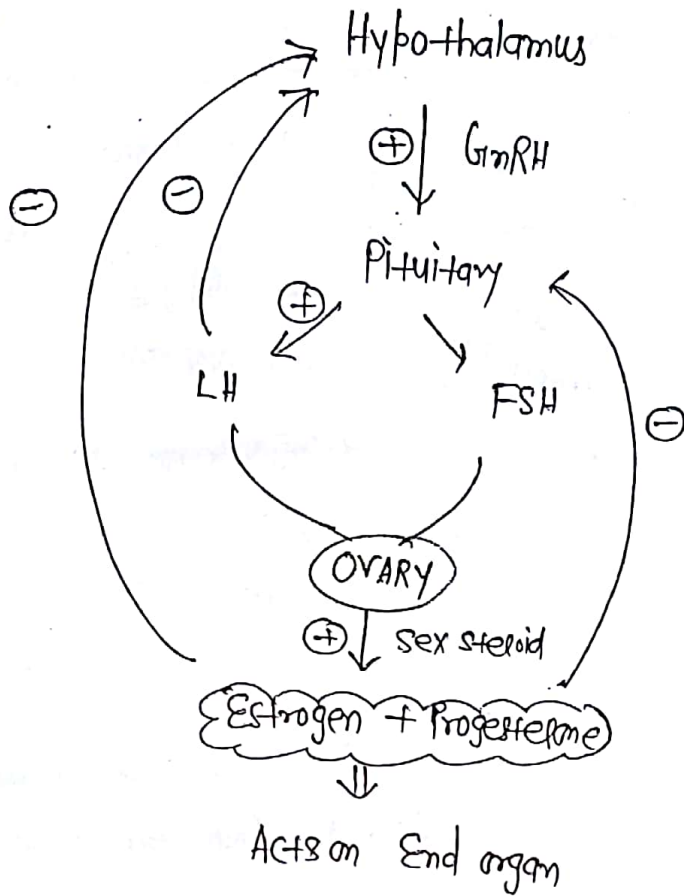
- every 5min

*Time to Heart Rate listen ⇒ Immediately after contraction
& Listen for 1 minutes
(Not 15 sec x 4)



* Hypothalamic - Pituitary - Ovarian Axis (HPO axis) ↳

↳ Not develop before puberty.
 Sensitive Around 8-12yr
 Fully established by 13-14yr



Feed forward
 Loops

* Gn abese ♀ / Pubertal change occur early, diff level of Leptin
 ↳ Hormone Made by Adipose cells that inhibit hunger.

* Estrogen

* C₁₈ steroids

Types ⇒ E₁ - Estrone - Predominant Estrogen in Post Menopausal
 E₂ - Estradiol ↳ in Reproductive Life
 E₃ - Estriol ↳ in Pregnancy

Most Potent ⇒ E₂, E₁, E₃

(Natural)

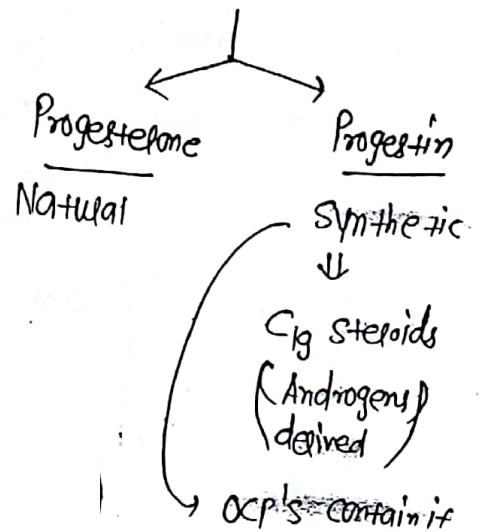
* Most Potent (Synthetic)

↳ Ethinyl-Estradiol (EE)

↳ used in combined ocp's

Progesterone

C₂₁ Steroids



* Classification of ocp's on the basis of Amount of Ethinyl

Estradiol \Rightarrow .

High dose ≥ 50 Mgm

Low dose 30-35 Mgm

Very low dose ≤ 20 Mgm.

Lowest dose 10 Mgm
(LoLoestrin)

* Classification of ocp's on the basis of Synthetic Progesterin \Rightarrow

1st generation \Rightarrow Norethindrone

2nd generation \Rightarrow Levonorgestrel (LNG)

3rd generation \Rightarrow Desogestrel (M/C used)

Gestodene

Norgestimate.

Least Androgenic

M/C ocp pills

Used in Malan, Mala D

Used in Novelon Pills

4th generation \Rightarrow Anti Androgenic

Spirolactone derivative
(Drospirenone)

Ethinogest

as generation Tes Lipid profile side effect
Tes & Androgenic side effect also tes

* Sources

$E_1 \Rightarrow$ Post Menopausal

\downarrow

Peripheral conversion

Androstenedione $\xrightarrow[\text{tissue (Aromatase)}]{\text{Adipose}}$ E_1

$E_2 \Rightarrow$ Reproductive Age

\downarrow

comes from granulosa cells of ovary \downarrow dependent on Theca cells

• corpus luteum

} 2 cell 2 gonadotropin Theory.

* Sources

• Ovary

\hookrightarrow corpus luteum

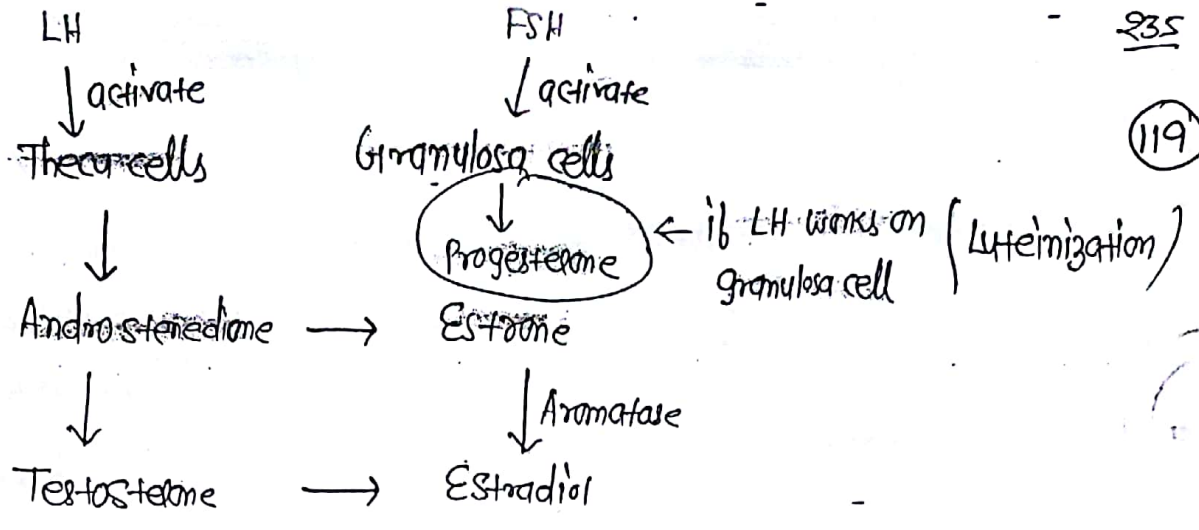
\downarrow

Formed by Luteinization of Granulosa cells.

• In $\ominus \Rightarrow$ Produced by Placenta

\downarrow

Precursor (Maternal LDL Cholesterol)



* Theca cell don't have Aromatase enzyme.

* Granulosa cells don't have 17- α Hase enzyme ; so can't make Androgen

* Placenta — E₃ (Fetal DHEA)
(Pregnancy) — E₂

* Most of estrogen → is in bound form (99%)

It is in free form

* Bound; 2% free
↳ Mainly Albumin

(cortisol binding ← CBG globulin) Not with SHBG

Mainly bound to

SHBG

Estrogen → (+ve) → SHBG (Liver) Globulin synthesis
also to Albumin → sex hormone binding

End product

↳ Pregnenedial

End products ⇒ Glyconides (sulphonides)

* Receptor ?? Location

↳ Intracellular

gonadotropin

Uterus

Non-Pregnant

(E)

Endometrial Proliferation

(P)

Protective

Stops Proliferation

↳ do secretory changes

↓
decidualisation

Growth of uterus

Growth of uterus

↓
Relaxation of Uterine

Smooth Muscle

Cervical ⇒
Mucous

Thin
Copious (Large Amount)
Watery

Thick
Scanty
highly-viscous

Elastic can be stretched
by fingers

Spinnbarkeit ()

No Spinnbarkeit

(X) Tack phenomenon

high amounts of
NaCl Required &
estrogen to secrete

ferning → Fern like pattern
on microscopic exam
↳ earliest @ 18
Disappears by 18
↳ like "Arborization"

(X) (No ferning)

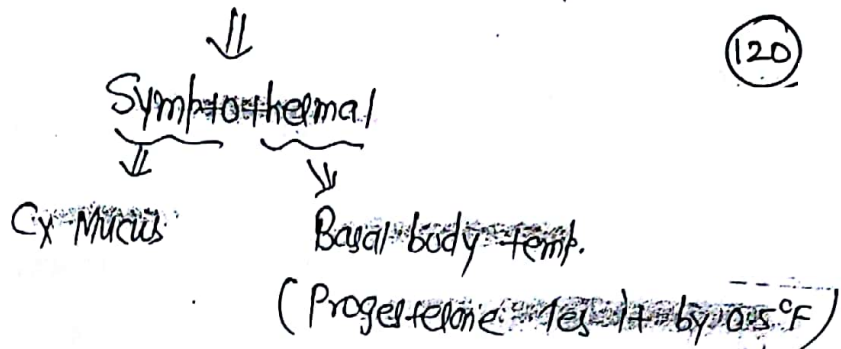
Cx Mucous breaks
on stretching

* Periovulatory Cx Mucous is (E) type ⇒ highly fertile.
↳ test of ovulation

* Mechanism of POP's (mini-pills)
↳ to make the Cx Mucous thick
(Alteration of Cx Mucous)

* Cx Mucous ⇒ Natural Method of contraception
↳ "Billings Method of Contraception"

Q8 Which Natural Method is best (least failure rate)



→ Cx Mucus is also impermeable to Micro-organism

↓
(P) + type ↓
Natural defense Mechanism

→ Mirena will ~~ves~~ the Risk of Pelvic Inflammatory disease

	(E)	(P)
Fallopian Tube	↑ Motility of Fallopian tube ↓ secretion	↑ Secretion ↓ Motility POP → RIF for ectopic P

Vaginal Cytology ⇒

→ Big cell Nuclei become small; Pyknotic & lades
 Superficial cells → Pink in colour
 Intermediate cells
 In Post-Menopausal ⇒ Parabasal/Basal cells
 ↳ Small cell; Big Nucleus
 Cytoplasm may contain the vacuoles
 High karyopyknotic Index (Small Nucleus)

tells about Hormonal Status Parabasal cell : Intermediate cell : superficial cell

Maturation Index 0 : 90 : 10 ⇒ ⊕ ♀

0 : 30 : 70 ⇒ Periovulatory ♀

100 : 0 : 0 ⇒ Postmenopausal Postpartum

(E)

(P)

effect on Salt & water

Retention

Excretion

Cholesterol

↑ HDL

↓ HDL

↓ LDL

↑ LDL

↑ Triglyceride

(Total cholesterol ↓)
↓

Cardioprotective

Bones

causes Mineralization of bone

No effect on bone

- epiphyseal closure

[Post Menopausal → Osteoporosis
[Precocious puberty → ↓ growth]

S. Ca²⁺ Level

↓

Urinary excretion

↓

Coagulation Profile - ^(E) Hypercoagulable state ^{kt} Inhibits Fibrinolysis
2, 7, 8, 10

(P) No effect, so, can be used

H/O Venous Thromboembolism, Stroke, CAD } all absolute C/I of ocp.

(121)

Estrogen → Causes upregulation of Progesterone Receptors on the Endometrium

Progesterone → Down Regulation of Estrogen Receptors on the Endometrium

Progesterone acts only on Estrogen primed Endometrium

Estrogen affects to higher centre ⇒

(E) ———— ⊖ve ———→ FSH

(E) ———— In Low Amount ⊖ve ———→ LH

————— In High Amount ⊕ve ———→ LH

Neuroendocrine phenomenon

High Amount of Estrogen ^{Initiation} → LH Surge

(P) Low Amount ⊕ve → LH/FSH
High Amount ⊖ve → LH/FSH

EIP $\xrightarrow{\ominus ve}$ GnRH

GONADOTROPINS

- Released by Anterior pituitary;
- Basophilic cells secrete
- Pulsatile
- Protein hormone

(R) \longrightarrow Trans Membrane (GPCR)
(R)

FSH $t_{1/2} = 3-4 \text{ hr}$

LH $t_{1/2} = 20 \text{ min}$

FSH (R) Gn females \longrightarrow Granulosa cells

Gn Males \longrightarrow Sertoli cells (Spermatogenesis)

LH (R) Gn Females - Theca cells

Granulosa cells - appear only in
Late Proliferative Phase

Gn Males - Leydig cells

\hookrightarrow Produce Testosterone

FSH \Rightarrow i) do selection of cohort of follicle every month

(1000)
(10000)

ii) Selection & growth of dominant follicle.

iii) Ovulation → Final Release of Ovum by collagen breakdown is brought about by FSH

(122)

LH → Function

- i) Ovulation
- ii) Formation & Maintenance of Corpus Luteum
- iii) Final growth of Follicle

LH Surge → $\frac{36 \text{ hrs}}{(24-36 \text{ hrs})}$ → Ovulation

LH Peak → 12 hrs → Ovulation

* LH Surge ⇒ Initiation by high level of Estrogen.
 (200 pg × 48 hrs)
 ↳ Amount of estrogen to cause LH surge

Maintenance of LH Surge ⇒ E + P

* When does Progesterone Synthesis begin
 ↳ Before ovulation (36 hrs) → Low in Amount

~~LH~~ → ~~Surge~~ → Luteinization of Granulosa cells

Q. Just before ovulation; which is true??

- (a) ↑ LH ; ↓ FSH
- (b) ↑ FSH ; ↓ LH
- (c) Both ↑ (LH Peak >> FSH Peak) → Small amount of Progesterone gives ⊕ feedback.
- (d) Both ↓

CORPUS LUTEUM \Rightarrow Every Month \Rightarrow die

\hookrightarrow Life span 14 days (constant luteal phase)

QA ib. 36 day Menstrual cycle ; ovulation day??

\Downarrow
on 2nd day

QA Which hormones maintain the corpus luteum

\hookrightarrow (LH)

QA Which hormones maintain the corpus luteum in ♀

\hookrightarrow (HCG)

Rescue the corpus luteum from luteolysis

Corpus Luteum

\hookrightarrow Progesterone

Estrogen

Relaxin

Inhibin A

Secreted by granulosa cell of the follicle

\rightarrow Inhibin B

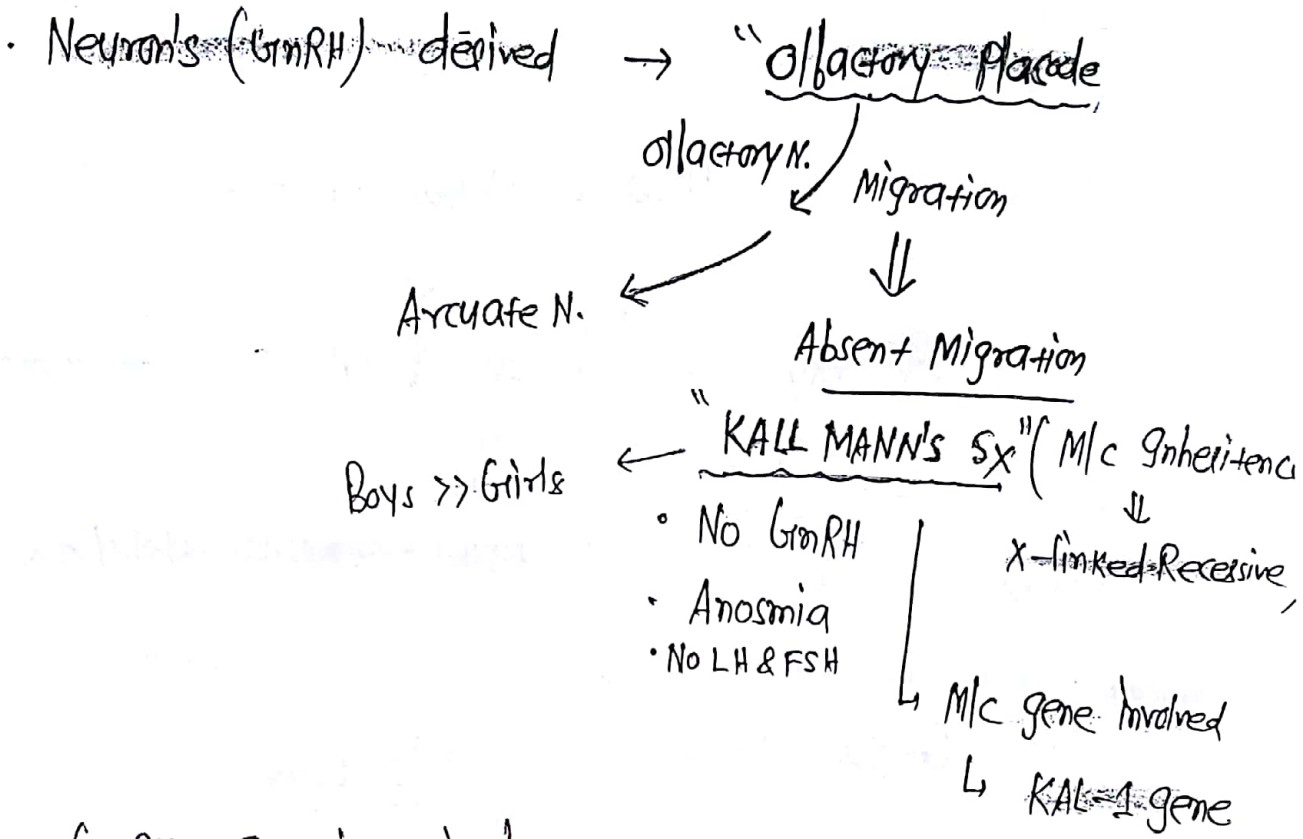
\swarrow
 \searrow
Inhibit the release of FSH

- Peak activity of corpus luteum \Rightarrow 8th day post-ovulation

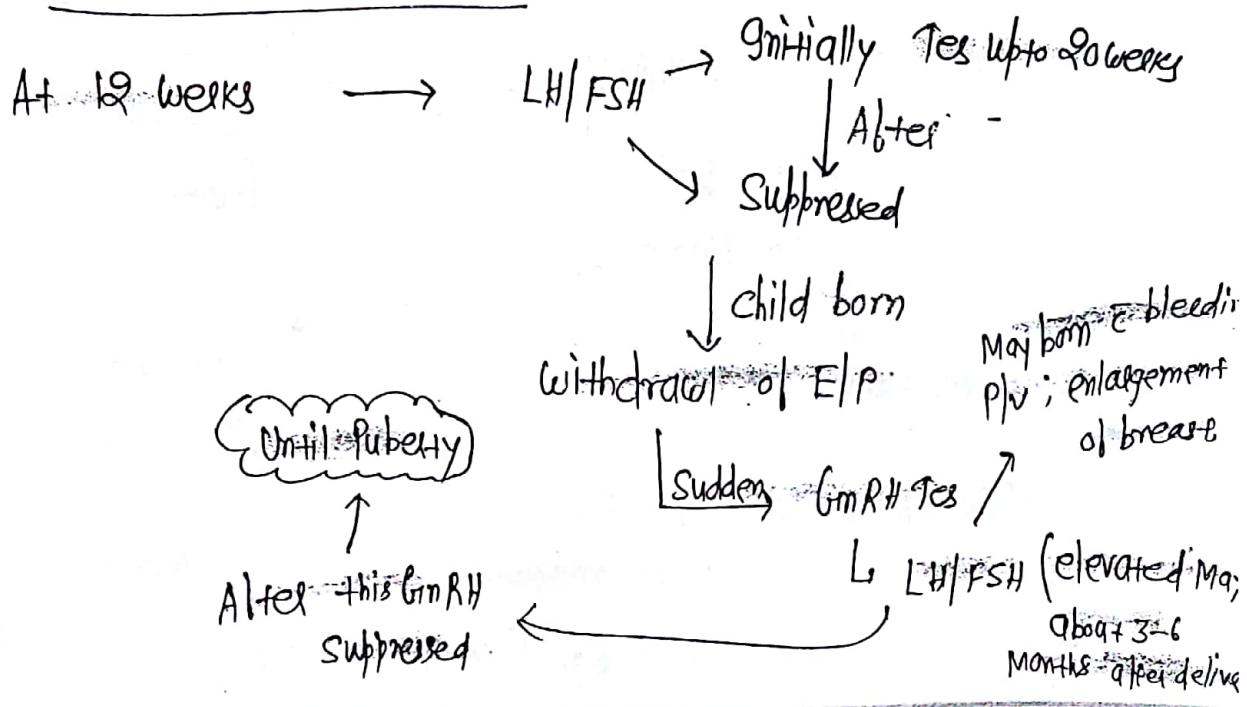
\checkmark
Maxim Progesterone production

HYPOTHALAMUS → Release GnRH

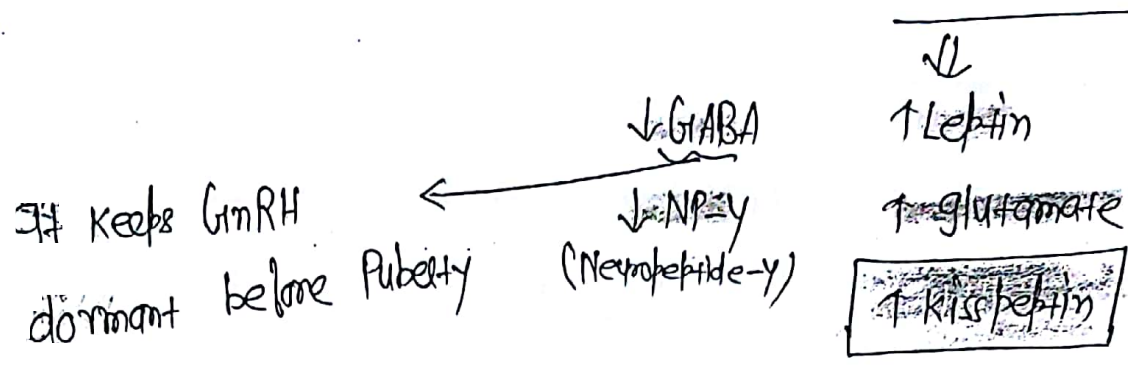
GnRH — Arcuate Nucleus Released if (in Medial hypothalamus)
↳ decapeptide
↳ t_{1/2} = 3-4min



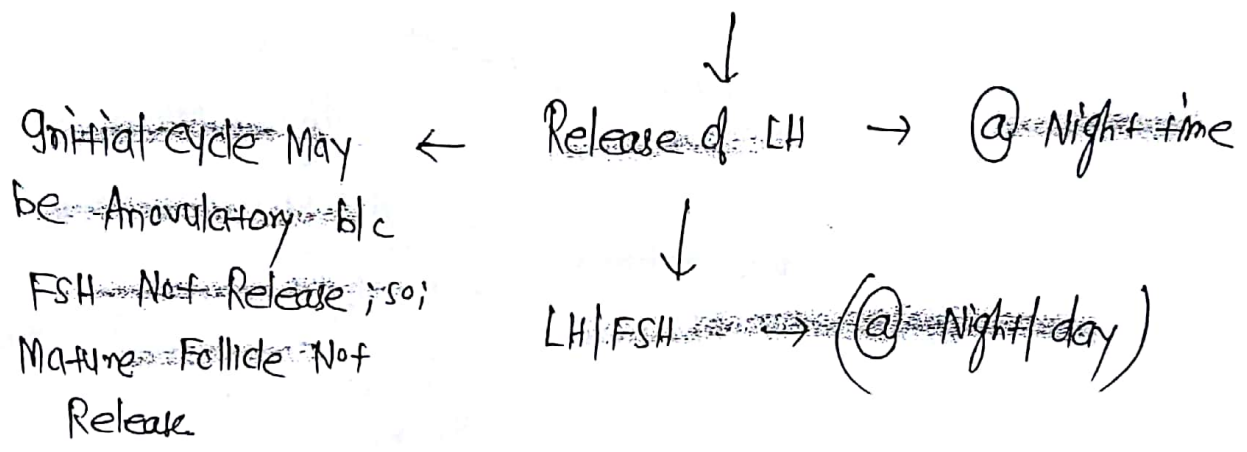
GnRH Secretion in fetus



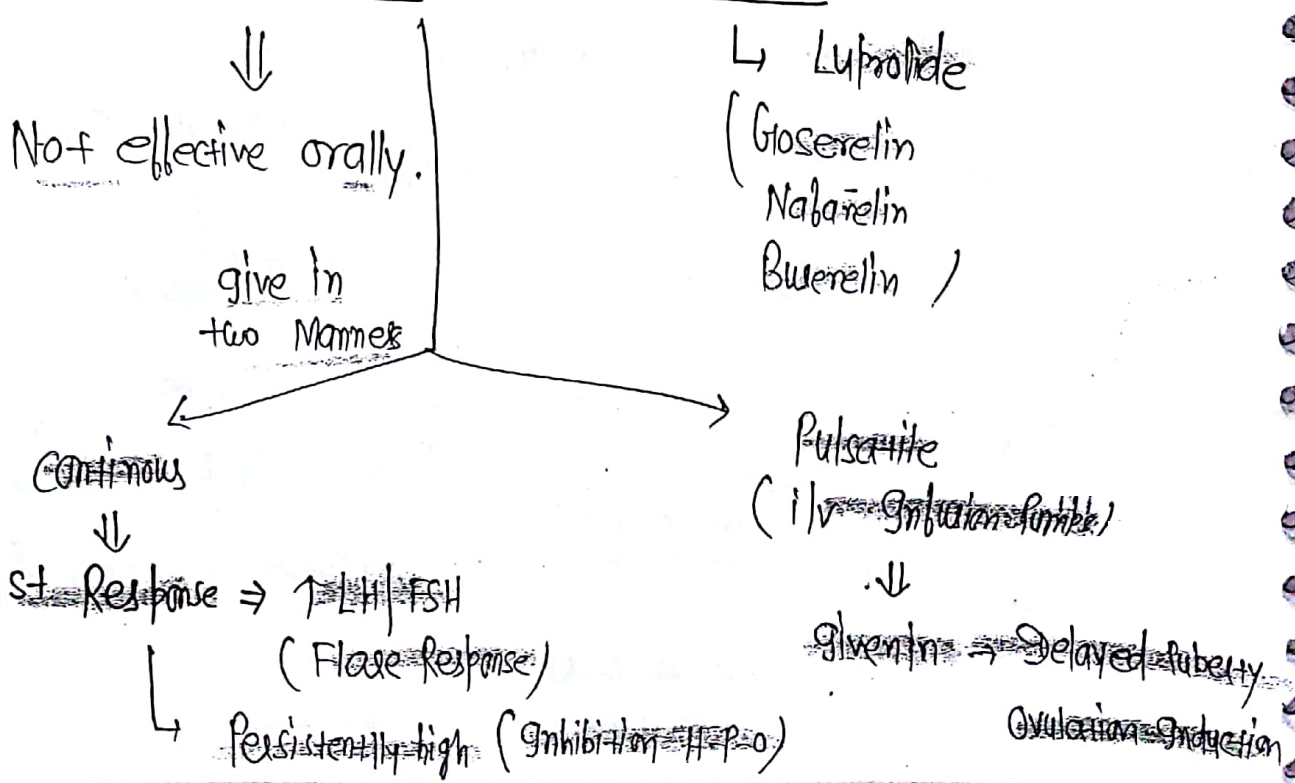
* Activation of HP axis @ Puberty \Rightarrow ~~dit Neurotransmitter~~



Pulsatile Release of GnRH \rightarrow Night-time



* GnRH Agonist \Rightarrow M/c used



Continuous

Pulsatile

(24)

Endometriosis

Kallmann's Syndrome

Fibroid Uterus

Hirsutism

Precocious Puberty

Breast Cancer

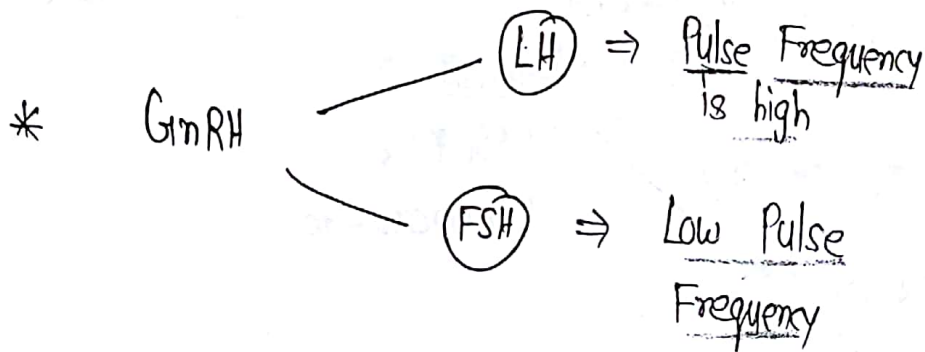
Prostate Cancer

* GnRH Antagonist \rightarrow M/c used \Rightarrow Cetrorelix*

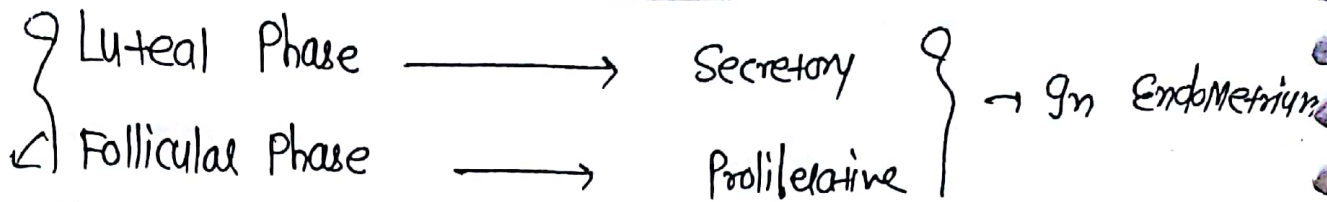
\hookrightarrow No ~~Flare~~ Response (No Initial Test)

~~Some~~ Antagonist - orally active

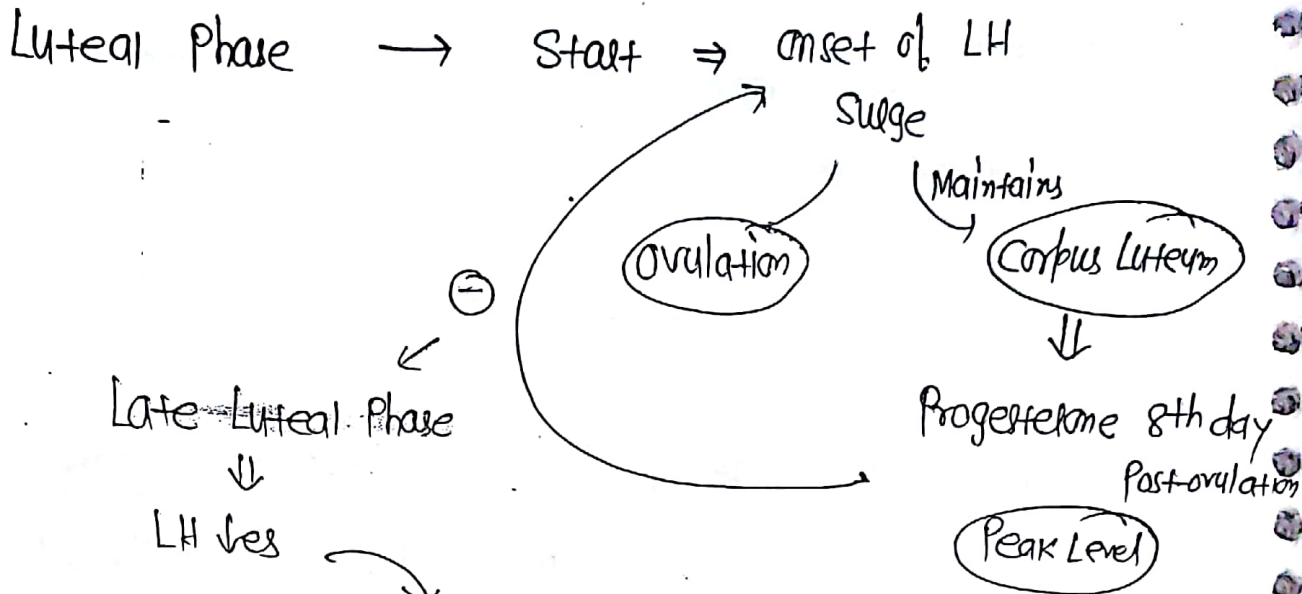
Indication \Rightarrow Same as ~~continuous GnRH Agonist~~ Indication



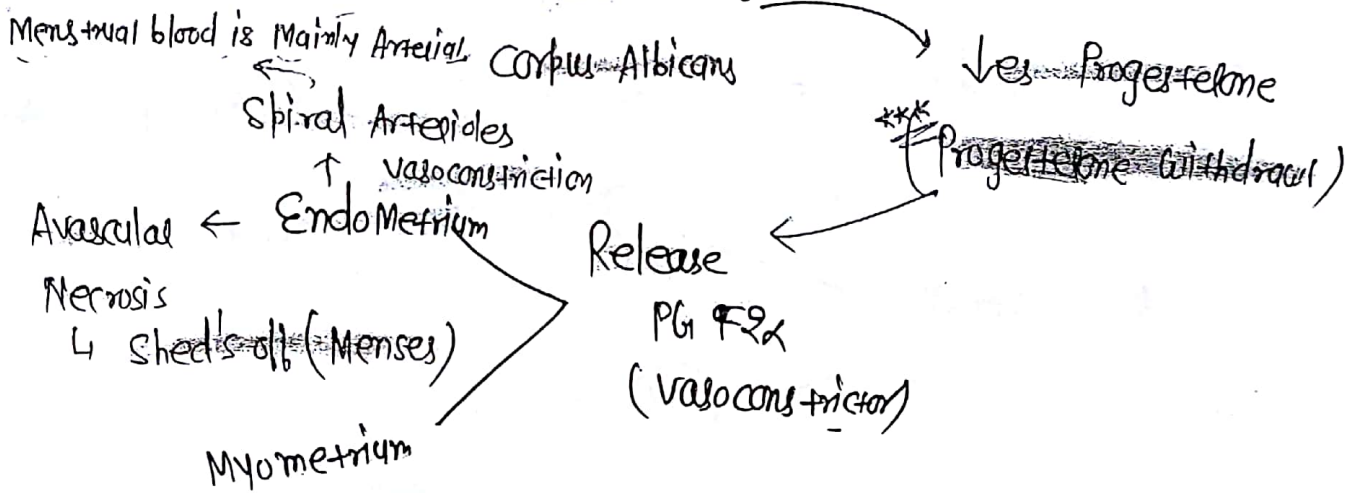
MENSTRUAL CYCLE



Gn ovary



~~CL~~ → Regress



Menstrual blood is mainly Arterial

~~Corpus Albicans~~

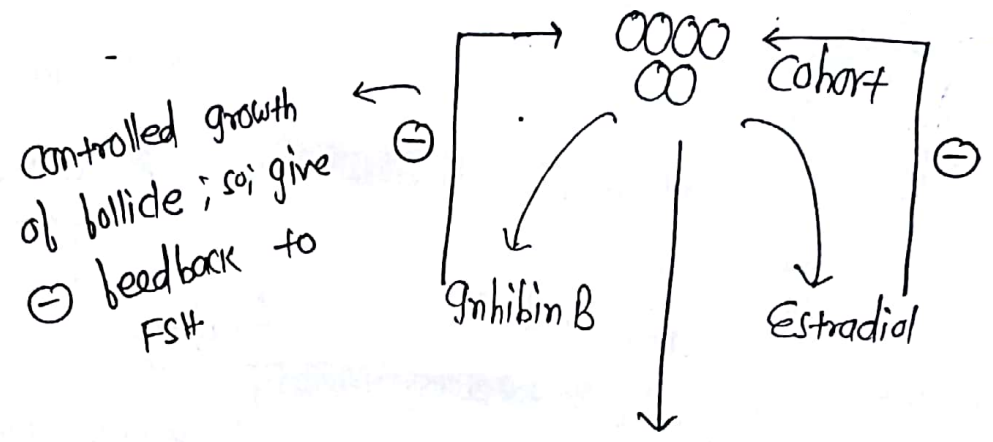
10 dysmenorrhoea (Physiological) → if present then ovulatory cycle ⊕

Anovulatory bleeding ⇒ Painless

↓ Progesterone → GnRH (Small pulses) in Pulse frequency (125)

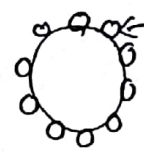
Follicular phase

↓ FSH



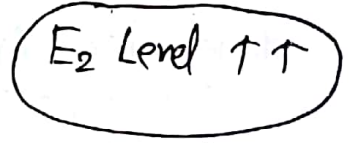
Dominant follicle selected

Middle Proliferative Phase



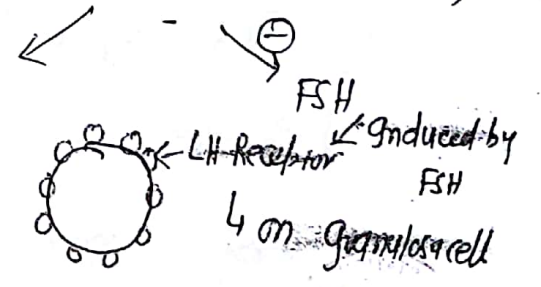
4 on 98

Late Proliferative Phase



High (200 pg x 48hrs)

LH Surge



Lutenization of Granulosa cells

Testosterone → Sex steroids

↓
Intracytoplasmic (R)
(In the absence of Ligand) C19
↓
Major Source of testosterone in ♀ ⇒ i) Peripheral Conversion of (50%)
Androstenedione

- ii) 25% adrenal gland
- iii) 25% ovary - theca cells
↓ (theca interna)

- Ovary doesn't produce DHT (5 α -hydro testosterone)
- Ovary also doesn't produce DHEAS sulfate

Androgen

↳ Which androgen is in Max^m amount

↓
Androstenedione > DHEA > Testosterone

Produced by Adrenal gland only.

* Gene for Androgen (R) Located

↳ Long Arm of X-chromosome

* Testosterone in ♀ ⇒ i) Pubarche / Adrenarche ^{depend on Androgen}

ii) Control of Libido * Estrog

iii) Intraovarian Testosterone Level

↓
Antral follicular growth
(estrogen rich environment)

* Testosterone in σ \Rightarrow Spermatogenesis (126)

\Downarrow
Intra-testicular Level of testosterone is high

\Downarrow
Sertoli cells produce TBP (Testosterone Binding Protein)

* ~~Initiation of Spermatogenesis~~

\hookrightarrow by ~~FSH~~

* ~~Spermatogenesis Require~~ \Rightarrow ~~FSH / Testosterone~~

* Sertoli cells produce \Rightarrow ~~Mullerian Inhibiting substance~~

~~TBP (Testosterone binding protein)~~

~~Relaxin~~

~~Inhibin~~

~~Estrogen~~

* Most of Testosterone is in Bound form: Binds to SHBG / Albumin
 \hookrightarrow (+ Testosterone free (Male: 2% free).

* Testosterone $\xrightarrow{\ominus ve}$ SHBG Synthesis

\hookrightarrow takes place in Liver

Qo. which has higher affinity to bind to SHBG is

~~Testosterone~~ \rightarrow Estrogen.

* End product of Testosterone \Rightarrow ~~Oxandrolone~~ (ketosteroids).

Q. Which cells form Blood-testis barrier

↳ Junction of Sertoli-Sertoli cells
↳ Physical barrier b/w blood vessels & seminiferous tubules

2 Compartments : ADLUMINAL COMPARTMENT : 1^o Spermatoocyte
2^o Spermatoocyte

Inner side of tubules;
isolated from blood & lymph

Basal compartment : Spermatogonia

↳ Outer side of tubules; in contact
w/ blood & lymph:

* Normal Menstrual cycle

↳ Length = 21-35 days

acc. to FIGO; Length = 24-38 days

Avg. Length \Rightarrow 28 days

Amount of blood loss \Rightarrow 80ml

Average of blood loss (amount) \Rightarrow 35-50 cc

No. of days = 2-7 days

average No. of days = $4\frac{1}{2}$ - 5 days

* Abnormal Uterine bleeding (AUB) \Rightarrow

Menorrhagia \Rightarrow More Amount ($>80ml$) or More Menstrual day (≥ 8 day)
Length of cycle = (N)

Hypomenorrhoea \Rightarrow < 2 day or $< 20ml$

Polymenorrhoea \Rightarrow < 21 day (< 24) Length of Menstrual cycle

Oligomenorrhoea \Rightarrow > 35 day (> 38) Length of Menstrual cycle

Metrorrhagia \Rightarrow Irregular bleeding / Intermenstrual

- classification of AUB acc. to FIGO \Rightarrow (127)

PALM - COEIN System \Rightarrow

- | | |
|-------------------------|-----------------------------------|
| AUB P - d/t Polyp | AUB C - d/t coagulation defect |
| AUB A - d/t Adenomyosis | AUB O - d/t ovulatory dysfunction |
| AUB L - d/t Leiomyoma | AUB E - d/t endometrial causes |
| AUB M - d/t Malignancy | AUB I - d/t Iatrogenic |
| | AUB N - Not yet classified |

Mx \Rightarrow acc to its cause

AUB \geq 45 yr \rightarrow evaluated for Endometrial carcinoma
(do Endometrial biopsy to Rule out Endometrial carcinoma)

PUBERTY

- development of Secondary sexual character.

	<u>Girls</u>	<u>Boys</u>
(N) Age \Rightarrow of Puberty	10 $\frac{1}{2}$ yr	11 $\frac{1}{2}$ yr
Precocious Puberty \Rightarrow <small>More common in girls</small>	< 8 yr	< 9 yr
Delayed Puberty \Rightarrow <small>More common in boys</small>	13 yr	14 yr

1st sign of Puberty

Girls
Growth Spurt

Boys
Testicular enlargement

1st visible sign of Puberty

Thelarche
(Appearance of Breast bud)

Testicular enlargement

(Tanner stage - 2)

aa Growth Spurt → Thelarche

Testicular enlargement



Pubarche
(Adrenarche)

Penile enlargement



Pubarche



Peak Height Velocity → Breast

Peak height velocity



Time gap = 6 months ←



Tanner stage 3

Menarche

* M/c cause of Precocious Puberty in girls ⇒ Idiopathic (90%)

So; girls comes c Precocious Puberty
evaluate c MRI brain

Brain tumor (10%)

↳ M/c Brain tumor



Hamatoma

Central Precocious Puberty

- Premature activation of HPO axis

LH/FSH ↑

gosexual

* M/c cause of delayed Puberty in Males ⇒ Constitutional delay

↓
Other Relatives have same history of delayed Puberty

* Other problems in adolescent/ Pubertal age group

↳ Irregular bleeding - M/c cause Anovulatory bleeding
2nd M/c ⇒ Coagulation defects

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Peripheral Precocious Puberty (128)

↓

Peripheral source of sex steroid hormones

↓

Estrogen / ~~Progesterone~~ Androgen

↳ secreting tumors

↓

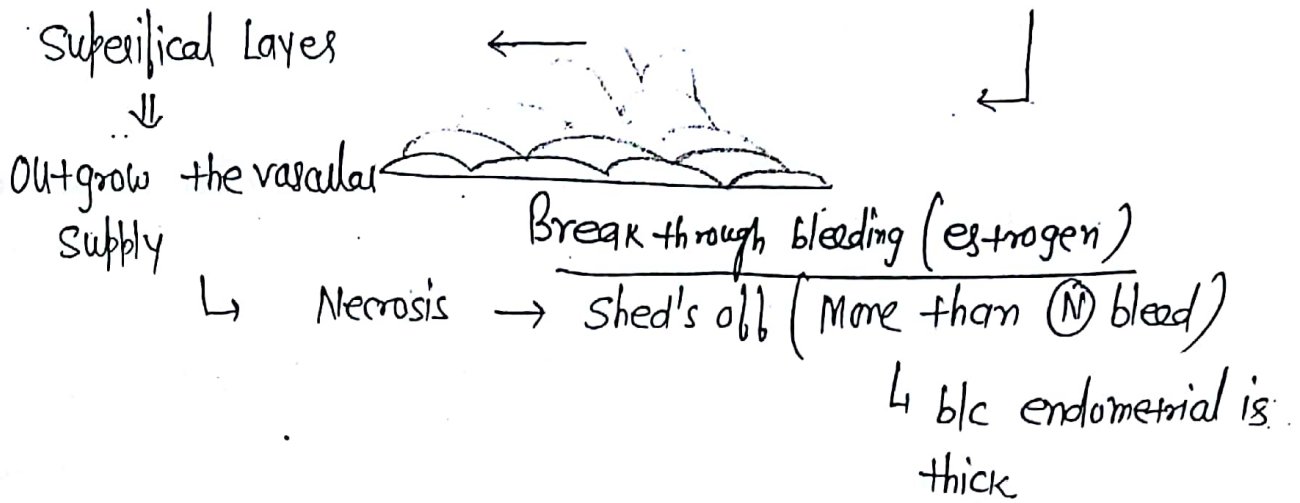
M/c a/w ⇒ McCune Albright Syndrome

(Precocious Puberty + Café-au-Lait + Polyostotic fibrous dysplasia)

↓

GT may be isosexual or may be heterosexual.

* In Anovulatory cycle → Unopposed estrogen



* DUC ⇒
for Adolescent
Irregular bleeding

OCP

↳ It Regularise the cycle

It also ves blood loss.

Protects from Unwanted Pregnancy

also ves Pelvic Inflammatory disease

also gives - only Progesterone Pills

QA.

20 days bleeding → Unstable vitals

↳ So, endometrium is thinned at time of presentation

↓
Initially give high dose iv Estrogen

↳ do endometrium Proliferation
quickly and stops bleeding by
Sealing the breaks

↓
Finally give Progesterone only Pills / Combined ocp

* High dose estrogen only given in unstable vitals; b/c in all other cases estrogen also causes closure of epiphyseal plate (stop Growth spurt). (129)

Least effective ⇒ Mefenemic Acid (NSAIDs)
↓
When hormonal therapy is Not taken by girls.
Tranexamic acid (antifibrinolytic drug)
↳ Stop bleeding

MENOPAUSE

- Cessation of Menses for 12 consecutive Months.
- Avg. Age ⇒ 51yr
- Avg. Age in India ⇒ 47yr
- It doesn't need any investigation; it is history based diagnosis

* Premature Menopause ⇒ <40yr
(Premature ovarian failure
1° ovarian Insufficiency)

↳ Confirmed by SFSH Level: if it is ≥ 40IU on 2 occasions done 1 month apart - diagnosed Premature Menopause

* Delayed Menopause ⇒ doesn't happen by 55yr of age
↓
Endometrial Evaluation (Endometrial biopsy)

FSH ↑

LH ↑

Estradiol → ↓ (< 20 pg)

Testosterone production from the ovary continues just like before

Gradual Process (climetric phase)

↳ Hormonal changes start, Perimenopausal Phase / early Menopause

M/c Symptom ⇒ Vasomotor Symptom

↳ Hot flushes

↳ d/t Estrogen withdrawal

↳ coincides w/ LH Surge

Hot Flushes ⇒

Sudden feeling of warmth followed by diaphoresis } Last for 1-5 min.

↳ More @ Night, (so; disturb sleep/wake cycle)

Moderate - severe hot flushes (disturbs daily routine)

↳ to start HRT (Hormone Replacement Therapy)

Q. 9. patient has intact uterus ~~by systemic~~

↳ give (E) + P → No Rde in Hot flushes

↳ Systemic therapy

(transdermal > oral)

Q. In Post-hysterectomy patient
↳ only (E) (1st Line) (130)

↓ if (E) is CI

give SSRI (2nd Line)

3rd Line drugs ⇒

- clonidine
- gabapentin
- Pregablin

In pre-menstrual Syndrome it is 1st Line of drug

* if patient is Not tolerating oral Progesterone

↳ give Mirena (LNG-IUD)

(E) + Bazedoxifene (SERM + SERD)

↓

protects the Endometrium Good effect on Bones

Other-SERMs ⇒

M/c side effect
↳ Hot flash

- Tamoxifene
- Raloxifene
- Clomiphene
- Ormeloxifene

Not used in Rx of hot flash

Tamoxifene ⇒ given in Breast cancer patient
↑ Risk of Endometrial cancer

Raloxifene ⇒ doesn't ↑ Risk of endometrial ca

↳ can be used for post-menopausal osteoporosis

* DOC for Post-Menopausal osteoporosis \Rightarrow Bisphosphonates

ORMELOXIFENE \Rightarrow Cenchrroman (Saheli)

↳ Indian government \Rightarrow Chhaya
↳ Non-steroidal contraceptive

It makes endometrium out of phase & prevents
Implantation.

PERIMENOPAUSAL WOMEN \Rightarrow Cycle become Anovulatory; so;
Irregular bleeding starts

DUB (dysfunctional
Uterine bleeding)

(Estrogen break through)

during the phase of
active bleeding

43yr \pm 3 Month Amenorrhoea

Bleeding x 2 days

Unstable
vitals

\Downarrow

do D&C

quickly stop

bleeding / in place
of high dose estrogen

\rightarrow On Adolescent: NO D&C b/c it alters the fertility

Stable
vitals

\Downarrow

high dose oral

estrogen \Rightarrow stop the

bleeding in 24 hrs

\rightarrow alter that give O / OCP

* Any Reproductive age women; comes to H/O Amenorrhoea
 Firstly Rule out Pregnancy.

(131)

V.V.G. ***

AMENORRHOEA

Primary

- Absence of Menses by 15yr of age in the absence of 2^o sexual character

OR

Absence of Menses by 15yr of age in the presence of 2^o sexual character

M/c cause \Rightarrow Gonadal dysgenesis

2nd M/c cause \Rightarrow Mullerian agenesis

Secondary

- Absence of Menses for 90 days in a previously Menstruating female

(In Irregular cycles \Rightarrow 6 Months Absence of Menses)

- M/c cause \Rightarrow Pregnancy

- M/c Pathological cause \Rightarrow PCOS

PRIMARY AMENORRHOEA

- Look about Thelarche

Absent

\Downarrow

d/t Gonadal dysgenesis \Rightarrow Gonads Abnormal

Kallman's syndrome } \Rightarrow Gonads Normal
 constitutional delay }

Present

Gonadal dysgenesis (Raised LH; FSH)

- Turner
- Pure gonadal dysgenesis
- Mixed gonadal dysgenesis

TURNER

Karyotype ⇒ XO

PURE GONADAL DYSGENESIS

46XY / 46XX

↳ Swyer's Sx

(Mutation in SRY gene)
Non-functional

d/f absence of Y chromosome

Functional (SRY gene)

Gonads — ovary —

Internal Genitalia — Female —

absence of MIS / testosterone

Estrogen
↓ → Absence
Growth Puberty) ↳ Hypoplastic

External Genitalia — Female —

absence of DHT (active form of testosterone)

Gonads appear ⇒ Ovary

↓
Accelerated Atresia
fibrosis (Fibrotic streaks)

Fibrotic gonads

↓
fibrotic ovary

Fibrotic / Malfunctioning
Streaks / testes

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TURNER

PURE GONADAL DYSGENESIS

MIXED GONADAL DYSGENESIS

(32)

Uterus ⇒ present
(Hypoplastic)

present

Mullerian / Wolffian
both ducts ⊕; b/c
MS
act on i/L
Mullerian duct

external genitalia ⇒ Female Like

Female Like

Ambiguous genitali
(Like of both Male & Female)

Pubic / Axillary hair ⇒ present
(Sparse / Scanty)

present

Present

Breast development ⇒ Absent

Absent

Absent

Q0. Most characteristic feature of Turner Syndrome ⇒
Short stature

Stature ⇒ Short stature

N / Tall

N / Tall

45 X 0

↓

Absent (X)

↓

Absence of (SRY) gene

↓

Responsible for growth
of Long bone

TURNER SYNDROME

↳ Short Stature

Short webbed neck

Low Posterior hair line

M/c congenital heart disease \Rightarrow Bicuspid ~~heart~~ ^{Aortic valve}

Short 4th Metacarpel

Cubitus valgus (elbow deformity)

Rudimentary ovaries

No Menstruation

True about Turner's \Rightarrow

~~(A) Uterus \oplus Breast \ominus~~

(B) Both \oplus

(C) Uterus \ominus Breast \oplus

(D) Both \ominus

* I-Q. \Rightarrow (N) in Turner Syndrome

I-Q. \Rightarrow ~~Ab~~ Sub Normal \Rightarrow when extra "X" chromosome

↳ Gn "Klinefelter Sx" (47XXY) \oplus

↳ M/c sex chromosome Abnormality.

* Libe - sperm \Rightarrow Slightly less

* LH/FSH \Rightarrow yes (so; Turner Sx is klas "Hypergonadotropic hypogonadism")

TURNER'S (45XO)

FSH ↑

Short stature

Streak ovaries

Anosmia ⊖

Uterus ⊕

Breast development ⊖

External genitalia Female Like

Pubic/Axillary Sparse

KALLMAN'S (46XX)

FSH ↓ (Hypogonadotropic hypogonadism)

(N) / tall

(N) ovaries

Anosmia ⊕

⊕

⊖

Female Like

Sparse

* constitutional delay

↳ Diagnosis of exclusion

Short @ Presentation

(N) Karyotype

* GOC for Primary Amenorrhoea ⇒ Karyotype

* Rx for Gonadal dysgenesis ⇒ give (E)/(P)

↳ She doesn't need Surrogate mother. Needs ovum donor

* Primary Amenorrhoea & Secondary sexual characters

- v/v
- i>
- ii>
- iii>
- iv>

Mullerian Agensis;

AIS (Androgen Insensitivity Syndrome);

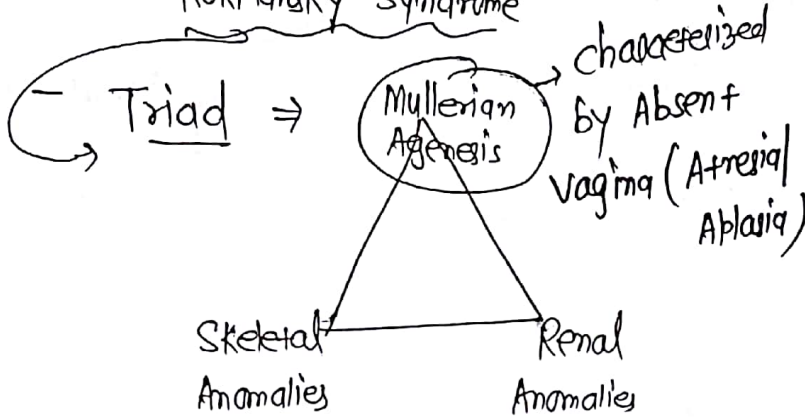
Imperforate hymen;

Transverse vaginal septum

Mullerian Agensis

Klas "MRKH Syndrome"

Rokitansky syndrome



absent vagina

absent uterus

Fallopian tube are absent - Proximally
Present - Distally

- gonads - ovary (N)

Karyotype - 46XX

AIS

- Klas "Testicular Feminization Syndrome"

- Testis is producing Androgen; but Receptor is completely insensitive

- Blind ending vagina (Small vaginal pouch)

- Uterus Absent (b/c testis produce MIS)

- Testis (+) (functional)

Karyotype => 46XY

Mullerian Agensis

Breast development → Present
external Genitalia → Female Like

AIS

265

(34)

Present @ Puberty

Female Like

Pt. May have Inguinal hernia & carrying Undescended testis

↳ May cause Malignancy

do gonadectomy ⇒ after Puberty

if done before Puberty; AIS patient Neither have female Nor Male; so; done after Puberty

- Male Pseudohermaphrodite
(M/c cause ⇒ AIS)
↳ Genotype Male; Phenotype Female
- Female Pseudohermaphrodite
M/c cause ⇒ CAH
↳ deficiency of enzyme 21-OHase

Female phenotype → Y chromosome

↓
as soon as diagnosis ← Should undergo gonadectomy is made

Pubic /
Axillary hair

Mullerian Agensis
(N)

ATS
Absent (Sparse)
Scanty

Pregnancy

Needs Surrogate Mother
Generally - the child
is of Mullerian
Agensis patient.
(by GV technique)

Physically Absolutely
(N); but they
have worst Reproductive
outcome
↓
do vaginoplasty

Best to
differentiate

⇒

Karyotyping

S-testosterone
Level

⇒

(N)

(N female Level)

Elevated

(Like of Male Level)

Q:

After doing karyotype in p Amenorrhoea; Next + testis

USG (≡)

USG + FSH (≡)

* TRUE HERMAPHRODITE ⇒ Tissue of both ovary & testes
(ovo-testes)

↳ also have Ambiguous Genitalia

↳ Mosaic karyotype

↳ 50% of Mixed gonadal dysgenesis ⇒ differentiate by
HPE of ovary & testis

* Reifenstein Syndrome \rightarrow Partial Androgen Insensitivity Syndrome

Complete AIS \rightarrow Clitoris - hypoplastic
Labia Majora - "

Partial AIS \rightarrow Clitomegaly seen

\hookrightarrow Both girl looking or Boy looking Person may have Partial AIS (depend on how much receptors are Active)

* Precocious Menarche \Rightarrow Menarche starts ^{without} ~~before~~ 10^o sexual character
 \hookrightarrow < 10yr Menarche starts

SECONDARY AMENORRHOEA

• Rule out \odot

GoC! Hormone assessment

always together \leftarrow $\left\{ \begin{array}{l} \text{TSH} \rightarrow \text{both hypo \& hypel thyroidism} \\ \text{Prolactin} - \text{Hyperprolactinemia} \end{array} \right.$

\downarrow
b/c TRH is stimulatory to Prolactin

FSH (\pm LH) / (\pm E₂)

\hookrightarrow M/C Cause
Pituitary Microadenoma (<1cm)
 \hookrightarrow M/C presentation
 \hookrightarrow Galactoria & Amenorrhoea

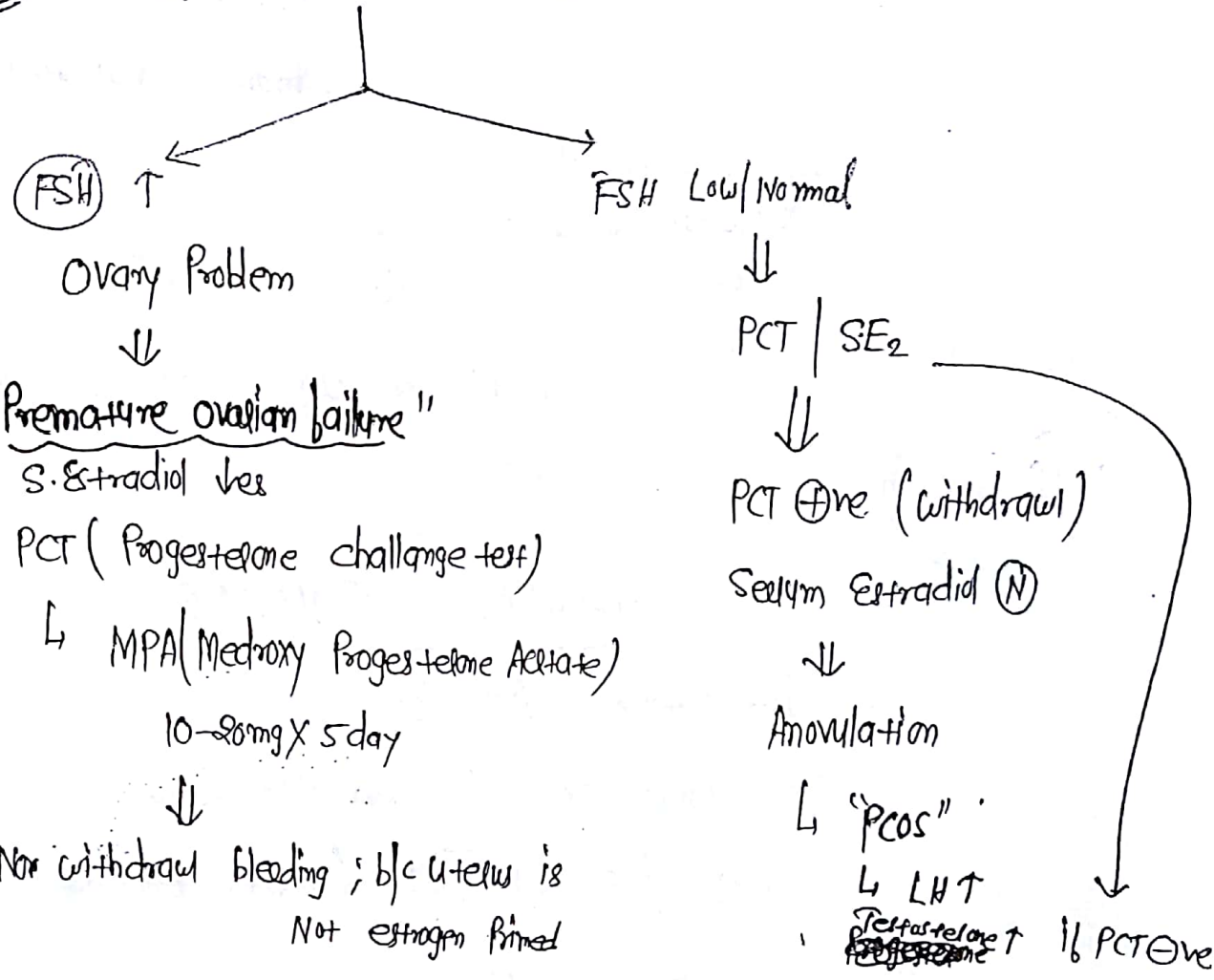
* Prolactin Feedback GnRH

(136)

↳ if < 50 ⇒ Repeat test
↳ if > 50 ⇒ MRI

DOC for Prolactin ⇒ Cabergoline > Bromocriptine
(dopamine Agonists)
↳ longer t1/2; so give twice a week only.

if TSH & Prolactin ⇒ (N)



"Premature ovarian failure"

- S. Estradiol ↓
- PCT (Progesterone challenge test)

↳ MPA (Methoxy Progesterone Acetate)

10-20mg x 5 day

No withdrawal bleeding; b/c uterus is Not estrogen primed

do karyotype also

Give E + P challenge test (by OCP & withdrawal) → S. E2 = (N) Not bleed → Endometrium Problem

↳ S. E2 = (N) Not bleed

↳ S. E2 = (N) Not bleed

↳ S. E2 ⇒ Low → Central causes → do MRI brain

↳ S. E2 ⇒ Low → Bleed → Possibility of primary: galactin & galactin → Asherman Syndrome

MRI brain

↓
(Normal)

↳ (Functional hypothalamic Amenorrhoea)

↓
Diagnosis of exclusion

↳ Seen in Anorexia Nervosa

Stress Induced Amenorrhoea

Exercise Induced Amenorrhoea

Chronic Malnutrition

Tt of Premature ovarian failure

↳ HRT

(till the age of Menopause)

ASHERMAN'S SYNDROME

- Intrauterine Adhesions
- Mlc cause ⇒ vigorous curettage
- Highest Risk ⇒ PPH (vigorous curettage)
- Mlc presentation ⇒ Menstrual Irregularities
 - ↳ Amenorrhoea > Hypomenorrhoea

* Single M/c Symptom / Presentation
↳ Infertility.

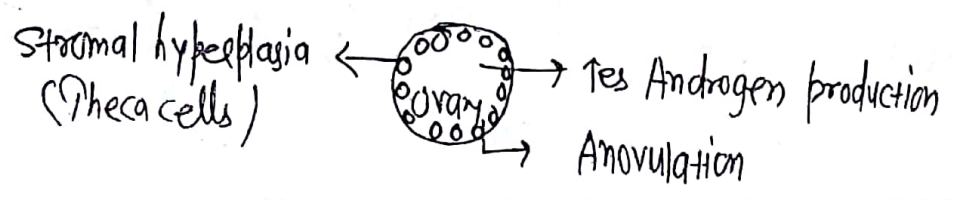
* Screening test ⇒ HSG (Not confirmatory test)
↓
Adhesion seen as filling defect
In HSG
↓
Multiple; Smooth;
Irregular Margin; Sma
in look.

IOC - Hysteroscopy (confirmatory test)
↳ both diagnostic & therapeutic

T/t ⇒ Hysteroscopic Adhesiolysis
+ CU-T Insertion + High dose Estrogen
↓ for 1 cycle
to make distance b/w Uterine wall
↓
for quickly Proliferation.

PCOS (Polycystic ovarian Syndrome)

- klas "Stein Leventhal Syndrome" *
- Primary Pathology lies in the ovary.



- ↑ Test Androgen production ; Not only inside ovary ; but also in Periphery

↳ Symptoms (Hirsutism)
↳ also cause dyslipidemia

- 50% of girls have obesity (also produces Excess Unopposed Estrogen)
↳ bc in Periphery
Androgen $\xrightarrow[\text{cell}]{\text{Adipose}}$ Estrogen

- 75% of girls have Insulin Resistance

↳ causes hyperinsulinemia

Syndromex
(Metabolic syndrome)

↳ Hyperinsulinemia
↑ Androgen production
dyslipidemia

- ↑ Test Androgen production further
- dyslipidemia

- * These patient have Resistance Test in LH

↳ Test Androgen production

* HAIR AN → Acanthosis Nigricans } ⇒ Cutaneous Marker of Insulin Resistance
 ↳ Hyper Androgenism ↳ Insulin Resistance
 ↳ Seen @ Neck
 Axilla
 Groin

Rotterdam's criteria for diagnosis \Rightarrow

if 2 or more of the following criteria are Met provisions
diagnosis of PCOS;

i) Amenorrhoea and/or oligomenorrhoea
↳ d/t Anovulation

* Menorrhagia \Rightarrow can be presentation (Mainly in obese pati)
↳ Not a criteria of Rotterdam's.

ii) Hyperandrogenemia and/or Hyperandrogenism

↓
High blood Level

↓
Clinical feature

↳ Total S. testosterone \uparrow (mildly elevated)

(N) $< 70 \text{ ng/dl}$

PCOS = $70 - 150 \text{ ng/dl}$

definitely $< 200 \text{ ng/dl}$

if $> 200 \text{ ng/dl}$ (severe) = Testosterone
secreting tumor

↳ Hirsutism \rightarrow on chin, chest
Axilla, Thigh
hair growth

Test terminal hair growth
in male pattern distribution

ii) Acne - Resistant to
usual t/t and
scarring in nature

iii) Alopecia

* Scoring System to confirm the Hirsutism

↳ Ferriman Gallway score ≥ 8

Not in PCOS \rightarrow Virilization (Hyperandrogenism)
↳ severe testosterone - seen in
Androgen tumor

Virilization presents \bar{c} \rightarrow

- i) clitoromegaly
- ii) \uparrow Muscle Mass
- iii) Hoarseness of voice
- iv) Male pattern balding
(Temporal Recession)

ii) USG criteria of Rotterdam's

a) ≥ 12 follicles in the ovary & each follicle
 < 10 mm in size

And/or

b) Ovary volume ≥ 10 cc

* (N) Female ~~also~~ May have USG pictures of PCOS;
(20-25+)

PCOS Female may have Normal USG pictures

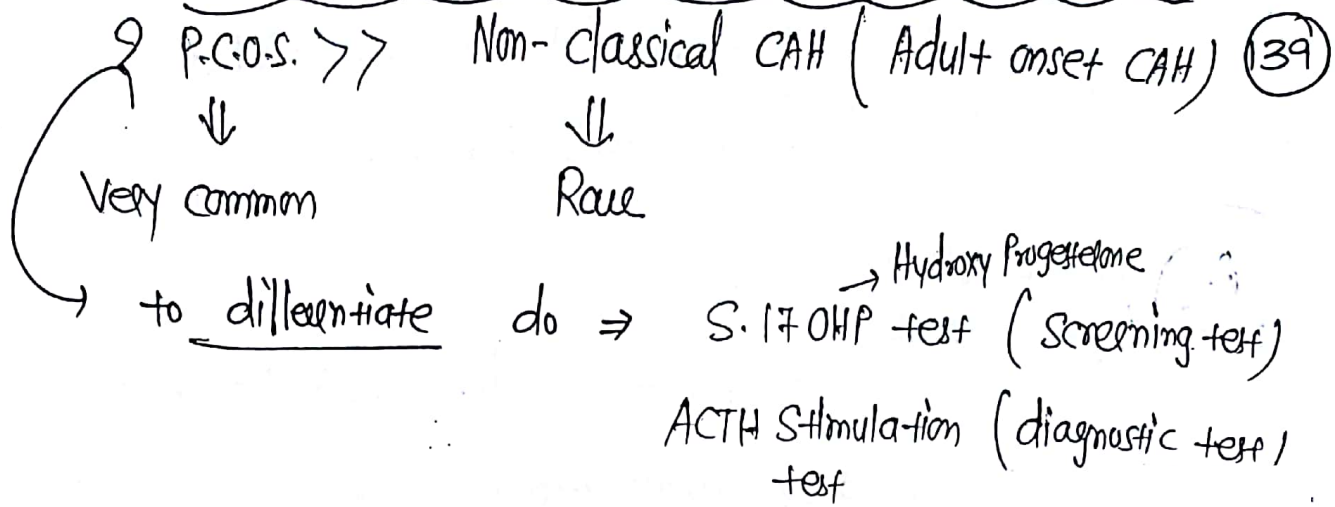
* ii) Follicles seem large in USG \Rightarrow Hyperstimulatory ovary.
(> 1 cm)

* 9m USG of PCOS \Rightarrow Necklace pattern
of ovary \downarrow
Characteristics to PCOS

Not Rotterdam's
criteria

\hookrightarrow also Stromal hyperplasia
Thick theca

* if 2 criteria are Met; then Provisional diagnosis of \Rightarrow



Other d/d of PCOS \Rightarrow Androgen tumor

- \hookrightarrow Gn it Rapid onset
- Rapid progression
- Testosterone > 200
- Virilization

Cushing syndrome

* Lab Investigation \Rightarrow

- i) S. testosterone (DHEAS - May be slightly elevated $< 700 \text{ Ngm}$)
- ii) USG
- iii) LH \uparrow / FSH (N)
 - \hookrightarrow LH / FSH Ratio \Rightarrow Test ($> 2:1$)
- iv) T. Estrogen \uparrow
 - Total $E_1 \uparrow$ / $E_2 = (N)$
 - Free $E_1 \uparrow$
 - $\frac{E_2}{E_1}$ Ratio Reverse

v) SHBG ves (So; Free E₂ Test)

↳ b/c testosterone Inhibits SHBG Synthesis
↳ Hypoinsulinemia.

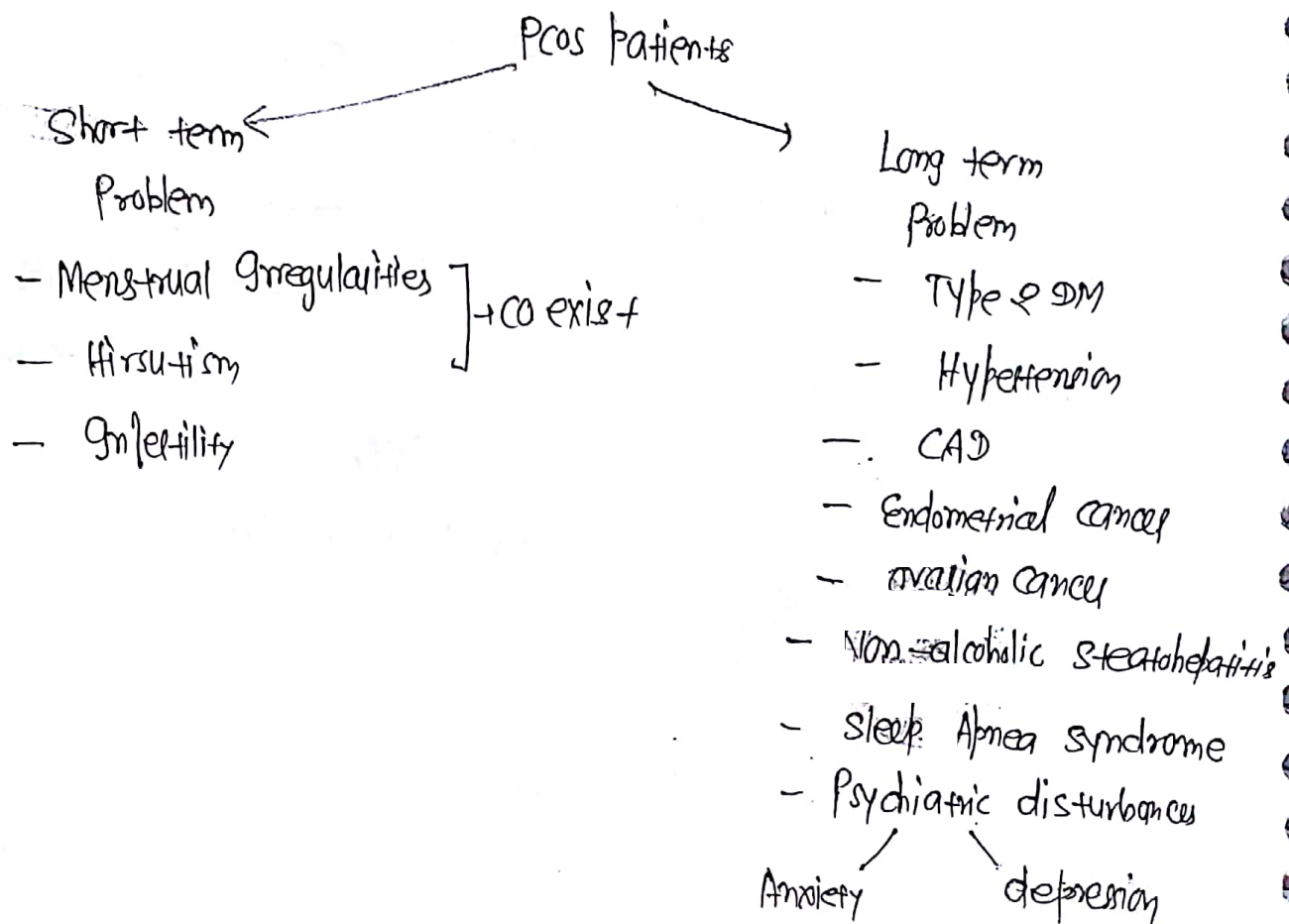
vi) Lipid Profile

vii) OGTT (to look for Insulin Resistance)

• DO $\frac{\text{Fasting Glucose} < 4.5}{\text{Fasting Insulin}}$

viii) TSH } (N)
ix) Prolactin }

x) 17 Hydroxy Progesterone test.



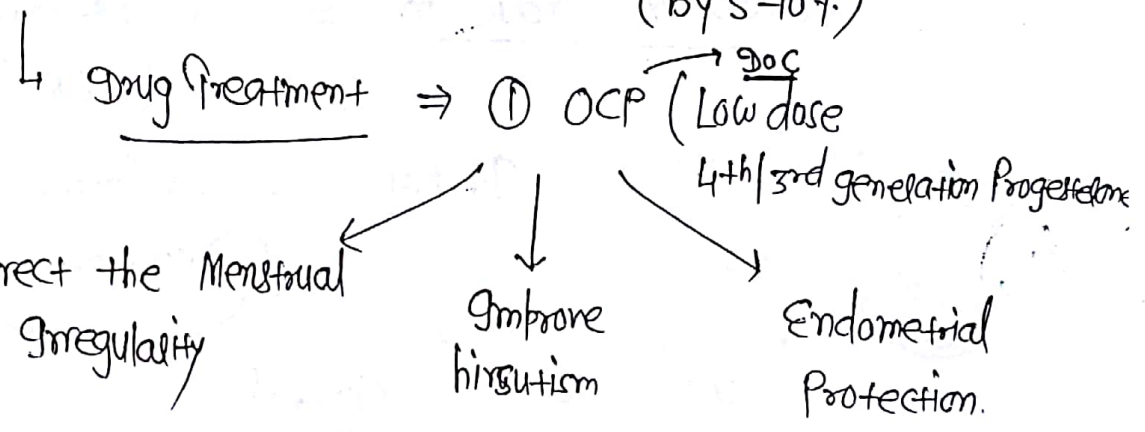
- Pregnancy Complication

(140)

- ↳ Abortion
- Gestational DM
- Pre-eclampsia
- Pre-term-Labour
- Still-birth.

* Management ⇒ Menstrual Irregularities + Hirsutism

Obese PCOS Patient → Advice weight loss (by 5-10%)



Doc for PCOS ⇒ OCP

* Only Hirsutism patient ⇒ give Spironolactone derivative

↳ Advice Not to conceive b/c it is Teratogenic drug

* Cyproterone Acetate

* Flutamide

* Finasteride

* Ketoconazole

* GnRH Agonist

* Metformin

Insulin sensitizers → weight loss (obese)

Can cause Lactic Acidosis
do LFT/KFT

Eflornithine (Topical)

all drugs are given in Hirsutism except \rightarrow Danazol
(Androgenic/E)

T/T for Infertility \rightarrow d/t Anovulatory

Obese PCOS \rightarrow weight loss

(weight gain is also advice in less weight patient)

Ovulation Induction \Rightarrow Clomiphene citrate
(SERM) does antagonistic activity

It has Zn component

(En) component

MOA \Rightarrow Central

Estrogen Receptor on pituitary prevents feedback inhibition

\downarrow

FSH \uparrow (FSH/LH)

Starting dose 50mg D₂-D₆ / D₅-D₉ of Menstrual cycle

Max^m (Approved) drug \Rightarrow 100mg.

L by FDA

Max^m approved times \Rightarrow 12 Months.

7% women ovulate after clomiphene \Rightarrow 80% (141)
 + women conceive " \Rightarrow 40%

Antagonistic effect
 [Cervix Mucus - Thick (Impermeable)
 Endometrium - growth is affected

* Poor Response (Not ovulating)

Obese patient (Insulin Resistance) - clomiphene citrate + Metformin } clomiphene citrate (CC) also

ACOG obese PCOS patient \Rightarrow DOC for infertility

2.5mg \longrightarrow 75mg (Aromatase Inhibitor)
 ↳ Letrozole (Not given for everyone; b/c Not FDA approved)

* Clomiphene citrate \rightarrow Not teratogenic

Mlc side effect of clomiphene citrate \Rightarrow Hot flushes

2nd Mlc " \Rightarrow ovarian cyst formation

Other side effect " \Rightarrow Multiletal pregnancy
 ↳ (6-8%)
 ↳ only twins

Other " \Rightarrow OHSS (ovarian hyperstimulation syndrome)
 ↳ 1% (Negligible)

2nd Line drug for ovulation induction ⇒

• Injection Gonadotropin (preferred)
(FSH/LH)



• Laparoscopic Ovarian drilling ⇒ S/E
↳ Premature ovarian failure

• Inj. HMG

(Human Menopausal Gonadotropin)

↳ taken from Urine of Post-Menopausal Women.

- Recombinant Purified preparation → Potent Inj. FSH

Highly expensive

More S/E as well

↳ Multiletal - 30% higher pregnancy order Gestation

↳ OHSS - 15%

* Step up Protocol

Start with Low dose



Monitor Response

↳ ↑ping the dose.

3rd Line drug for ovulation induction →

Doc of ovulation induction
in hypothalamic cause / Kallmann's Syndrome ⇒ GnJ, GnRH
↓
Pulsatile

* Multifetal ⇒ Gonadotropin > clomiphene > GnRH Agonist
♀ Risk Citrate

* OHSS Risk ⇒ Gonadotropin > GnRH Agonist > clomiphene > GnRH Antagonist
Citrate ↓
Prevention of OHSS.

In general ⇒

Bromocriptine → Anovulation
↳ d/f hyperprolactinemia

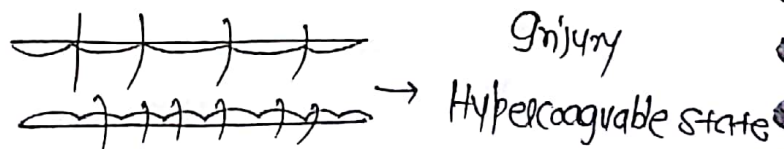
Aromatase Inhibitors → Letrozole

* OVARIAN HYPERSTIMULATION SYNDROME (OHSS)

- RIF ⇒
- i> Younger Age;
 - ii> PCOS;
 - iii> High Serum E_2 Levels (>3500 pg)
 - iv> Large size of follicle & Large No. of follicle (>20 follicles & >10 mm in size)
 - v> Pregnancy
 - vi> Gonadotropin

Causes ⇒ Injection hCG (used as ovulation trigger);

• Mediator ⇒ VEGF ⇒ Cause Endothelial



↳ Leakage of fluid in 3rd space

Early - \leq 9 days of Gnj. hcg

Late - $>$ 9 days of Gnj. hcg

↳ d/f Pregnancy Late OHSS seen

Prevention ⇒ Delay the hCG Injection;

↳ called as "coasting"

Cancel the cycle & do cryopreservation of embryo;

GnRH Antagonists

Volume expanders

Bromocriptine

INFERTILITY

- Inability to conceive with one year of Unprotected intercourse
- But; if the patient is >35 yr \Rightarrow Inability to conceive \bar{c} in 6 Months of Unprotected intercourse

Female factor	contributes	40-55%
Male factor	"	40%
Unexplained	"	10%

Female Factor \Rightarrow

- Ovulatory Factor - 30-40%
- Tubal Factor - 20-30%
- Uterine Factor - 15%
- Cervical Factor - 5%
- Unexplained - 10%

\rightarrow Ovulatory Factor \Rightarrow Most Reversible

\hookrightarrow dlt Anovulation \xrightarrow{do} ovulation Induction

Premature ovarian failure \xrightarrow{do} donor ovum

Test of ovulation \Rightarrow

- Cervical Mucus
 - vaginal cytology \rightarrow Lateral vaginal fernix (upper 1/3rd of lateral wall)
 - Basal body temp. (BBT) (Progesterone) \hookrightarrow Test BBT by $0.5-0.8^\circ F$
- if we plot Bimodal graph - cycle is ovulatory

↳ easiest test \Rightarrow S. Progesterone

\Downarrow

on D₂₁ \rightarrow $\geq 3\text{ng}$ \Rightarrow ovulatory cycle

↳ Best test — Endometrial Biopsy

\Downarrow

do in Premenstrual phase-

2 days before the expected date of Menstruation.

if endometrium Report \Rightarrow Secretory Endometrium
 \hookrightarrow ovulatory cycle

Proliferative Endometrium

\hookrightarrow Anovulatory cycle, Telescopic gland
 \hookrightarrow See Long tubular glands & Pseudostratification
 \hookrightarrow d/t estrogen hormone

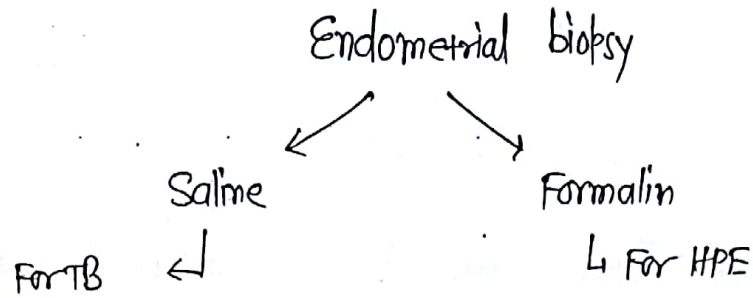
While Subnuclear vacuolation \rightarrow is the evidence of Progesterone secretion on D₁₆ on HPE

Cork-screw glands \rightarrow Seen on D₂₀ on HPE (Late secretory phase)

Max^m Stromal edema \rightarrow Seen on D₂₂ on HPE

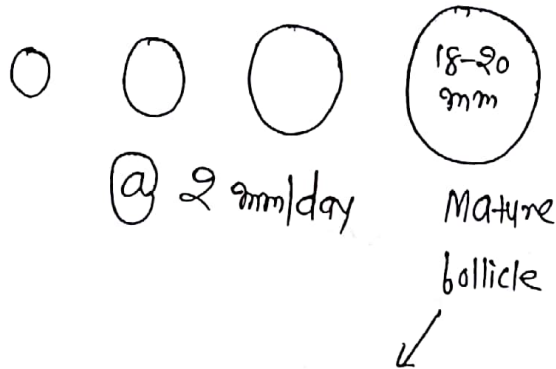
Leucocytic Infiltration of Endometrium \rightarrow (Premenstrual phase) on D₂₆ on HPE

* Endometrial biopsy at least once in evaluation of women infertility to rule out Genital TB; (144)



vi) M/c used test of ovulation in infertile patients TVs for follicular Monitoring =>

Starts on D₁₀



tells the infertile patient to Intercourse.

→ ① Sudden lvs in size and free fluid in Pouch of Douglas
↓
Suggestive of ovulation.

- also in endometrium on USG =>

Trilaminar Endometrium (Triple Layer Endometrium)

↳ Three hyperechoic lines seen in Endometrium

↳ seen in "Perioovulatory Phase".

* Single hyperechoic line = Posterior Enhancement

↳ d/t Secretion from Endometrium
↳ Suggestive of Secretory phase

vii) Urinary LH kits \rightarrow

Urinary* LH Surge \rightarrow After 24hrs
↳ ovulation takes place

OVARIAN RESERVE

M/c used test for it \Rightarrow i) S. FSH \Rightarrow D₂-D₄
↳ on D₃ (Best)
↳ (N) value of S. FSH on D₃
↳ < 10 IU

10-15 IU \Rightarrow Borderline Reserve

> 15 IU = Poor Reserve

> 20 IU = Suggestive of Premature ovarian failure

\geq 40 IU = diagnosis of Premature ovarian failure

ii) S. Inhibin B \Rightarrow on D₃ - < 45 pg - Poor Reserve

iii) AFC (Antral Follicle count) - on D₂-D₄
< 10 follicle \Rightarrow Poor Reserve

iv) Best test \Rightarrow S. AMH (Anti Mullerian hormone, equivalent to MIS)

↳ Very Small amount - Small prenatal follicle

< 0.5 ng - Poor Reserve

↳ b/c No fluctuation in menstrual cycle
(False test - less)

✓ CCCT (clomiphene citrate challenge test)

D₃ - S. FSH

D₅-D₉ - Clomiphene citrate 100mg

D₁₀ → S. FSH

high basal level which rise further on D₁₀ - poor Reserve

TUBAL FACTOR



Fallopian tube should be patent.

GoC for Patency ⇒ HSG (screening test)

↓ if abnormal

Best → Laparoscopy + Chromoperitubation (Diagnostic)

Post-Menstrual Phase ⇒ D₅-D₁₁ day (M/c on D₁₀)

↳ d/t Cervix dilation

⊕ Int @ this time

and also pregnancy Ruled out @ this time

Peritoneal spill



Cannula - Leech Wilkenson's cannula



↓
10mL dye (Water-soluble iodinated dye) used

- * CI of HSG \Rightarrow
- ① Suspected \ominus (UPT \rightarrow \ominus ve);
 - ② known case of Genital TB;
(Endometrial biopsy for Acid fast bacilli \rightarrow \ominus ve)
 - ③ Actively bleeding;
 - ④ current Pelvic Infection;
 - ⑤ K/c/o dye allergy

HSG as screening for
Uterine Pathologies

- Adhesion
- Polyp
- Submucosal fibroid
- Mullerian Anomalies

Other screening
Modality

\Rightarrow Sono hystelography
 \hookrightarrow Uses USG & Normal saline

• B/L cornual block (Proximal block)

• B/L Hydrosalpinx \Rightarrow happen d/t distal block

\hookrightarrow Represents severe injury

B/L Proximal block
Mid segment block
distal block

\Rightarrow Best prognosis
 \hookrightarrow B/L Proximal block
 \hookrightarrow very less pathologically

In Maximal case
 B/L Proximal block ⇒ Physiological
 ————
 Cornual Spasm (146)
 Mucus plug

Next Step ??

Diagnostic Laparoscopy + Chromopertubation + Hysteroscopy

OR k/as "Laparohysteroscopy"

In same sittings; it works as therapeutic by the process of "Hysteroscopic cannulation"

if it fails

TUBAL PATHOLOGY

Operate

gVF (preferred over operation; to avoid ectopic @)
 Last Resort

B/L cornual block
 Next / Best if/ +

⇒ Laparohysteroscopy

* if on HSG; B/L distal block seen

do Diagnostic Laparoscopy

Mild
 ↓ if/ + by
 Fibriolysis
 Adhesiolysis

Severe (B/L hydrosalpinx)
 ↓
 do gVF
 Fluid ⇒ embryo-toxic

So before gVF; do ⇒
 B/L salpingectomy &
 Cornual clipping

* if on HSG; Proximal & distal block both seen on both of tubes separately (severe)



do gvf.

* if on HSG; Mid segment-block seen



Tubal Ligation → Tubal Recanalization.

Good Prognostic Marker of conception after Tubal Recanalization → i) Type of Ligation

Clips > Falope Rings



Can be Reversed

can't be Reversed; cautery > Modified Pomeroy

ii) Type of Recanastomosis

Best ⇒ Isthmo-isthmic > Isthmo-Ampullary

iii) Total Length after Recanastomosis > 4cm



iv) No other cause of infertility

Test done before Anastomosis ⇒ Semen Analysis of husband

GENITAL TUBERCULOSIS

291

(147)

- Secondary Infection;
- M/c Primary - Lungs > Lymph Node
- M/c Route - Hematogenous
Direct
Lymphatics -
Ascending Infections
- M/c site \Rightarrow Fallopian tube > 90%
Endometrium 50-60%
- M/c Route \Rightarrow  Direct spread
- U/c site \Rightarrow Vagina & vulva - < 2%
- Fallopian tube - Acute - Red Inflamed edematous
Chronic - Thick walled / Adhesions
- Cobble stone appearance on HSG
Tobacco Pouch appearance on HSG
Lead pipe OR pipe stem appearance on HSG
Beads on string appearance on HSG
Golf club appearance on HSG
B/L Hydrosalpinx on HSG  \Rightarrow Genital TB

* M/c site affected \Rightarrow Ampulla (1st site to be affected on genital TB)

L/c site affected \Rightarrow Intercervical

Endometrium \rightarrow acute - Normal

\hookrightarrow affects only superficial part

Myometrium - spared

Chronic - Adhesion/Ulcers

(Asherman's Syndrome)

* M/c Presentation \Rightarrow • Infertility (World over = 10%
India = 17%)

• Pain

• Menstrual Irregularity

First Menstrual

Irregularity

\downarrow
Menorrhagia

More common

Menstrual Irregularity

\downarrow
Amenorrhoea > oligo

* M/c Finding In Reproductive Age

\hookrightarrow (N) Pelvic exam

\hookrightarrow Tenderness sometime

L/c Finding In Reproductive Age

\hookrightarrow B/L Adnexal Mass

B/L Adnexal Mass \Rightarrow M/C finding in adolescent girls = Genital TB ²⁹³
(148)

Diagnostic \Rightarrow
 \rightarrow Endometrial Biopsy (Best)
 \rightarrow Menstrual blood PCR Analysis
 (1st day sample taken to check superficial layer of endometrium)

Rx \Rightarrow ATT (Anti-tubercular therapy) X 6 Months
 \hookrightarrow Improve fertility status - Yes.

* In case there is severe Tubal Pathology (distort Normal Anatomy)
 \hookrightarrow ATT doesn't improve fertility status; go for IVF; but ATT is given for Genital TB

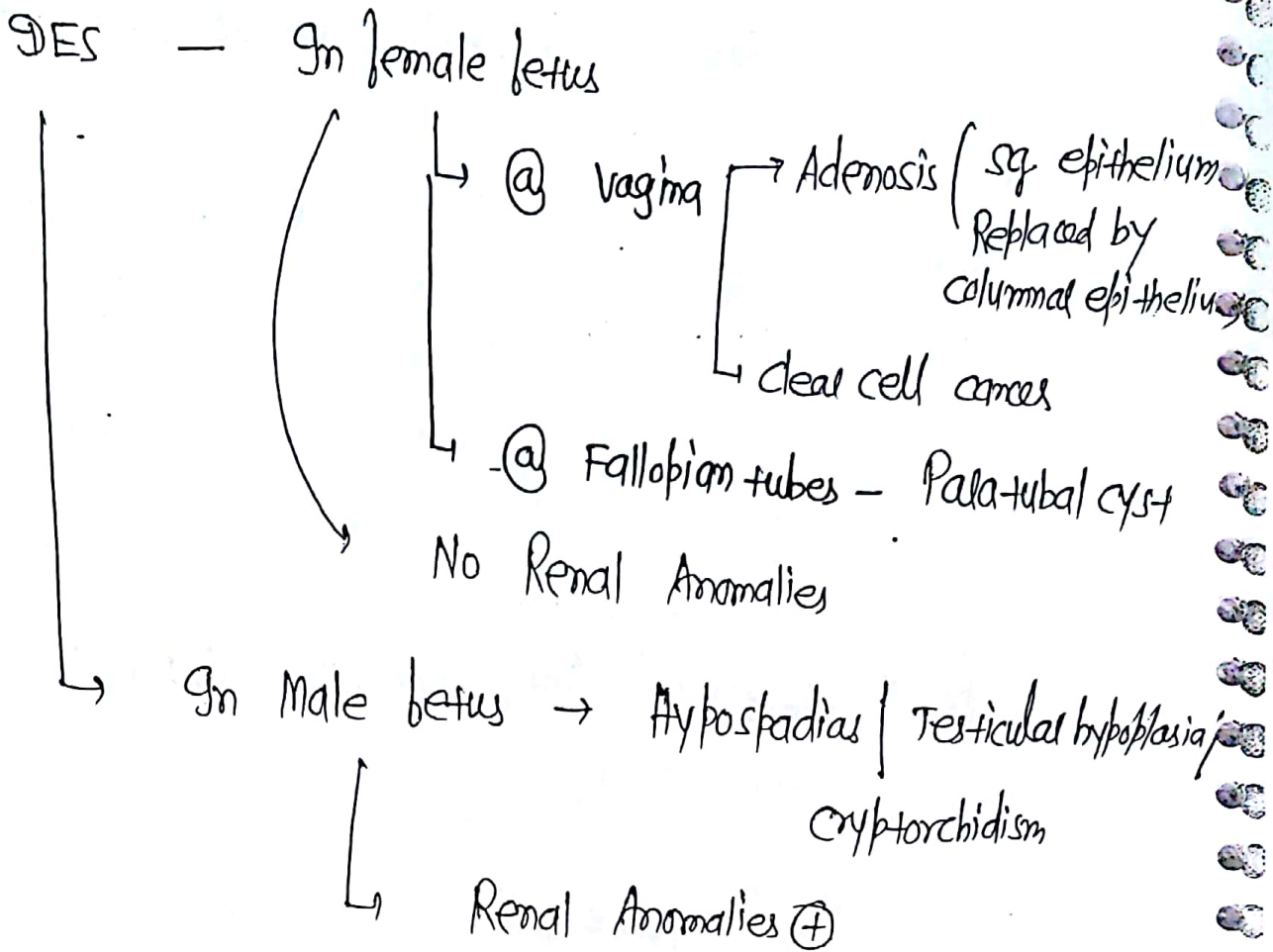
UTERINE FACTORS

Infertility caused by \Rightarrow
 i) Fibroid - Submucosal;
 ii) Polyp
 iii) Endometritis;

iv) DES (Preg. women)

\hookrightarrow Fetus Female fetus

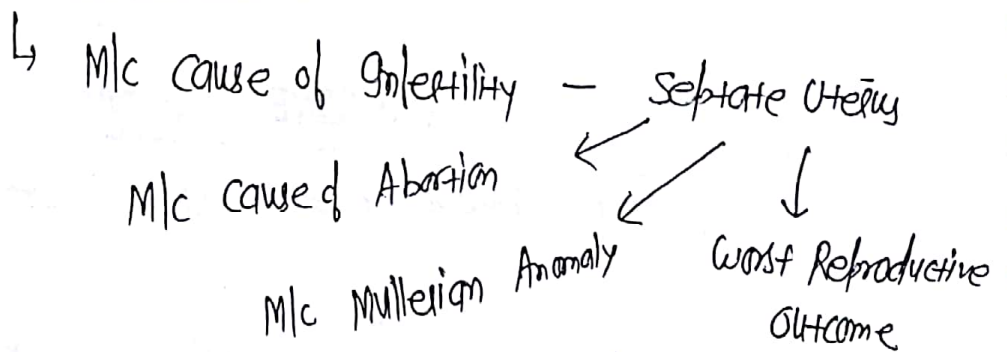
\rightarrow Uterus (Hypoplasia)
 Most characteristic T-shaped uterine cavity



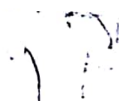
v7 Acutely Retroverted Uterus

cochleate uterus ⇒ Acutely Anterverted Uterus

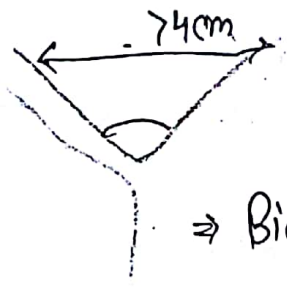
v17 Mullerian Anomalies



Best outcome ⇒ Arcuate > didelphys > Bicornuate



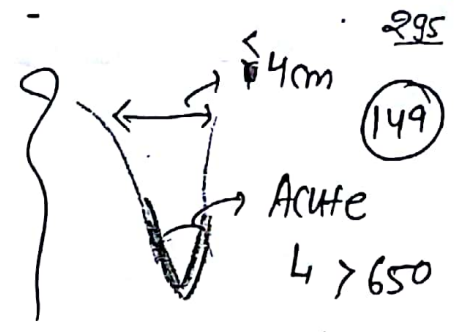
Screening test \Rightarrow



\Rightarrow Bicornuate

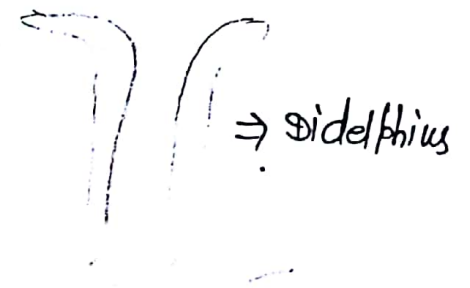
HSG
(Non-diagnostic)

Septate
uterus



Gold of septate uterus on HSG

\hookrightarrow Bicornuate uterus



\Rightarrow didelphys

IOC \Rightarrow MRI ^{***} \Rightarrow if showing fundal dip $>1cm$
 \downarrow
Bicornuate.

Sonohysterography

3D/4D HSG (Not 2D USG)
 \hookrightarrow usually do

Gold Standard Investigation \Rightarrow Laproscopy + hysteroscopy
 \downarrow
alone is Not a good Modality.

* All patients of Mullerian Anomalies Undergo evaluation for \rightarrow Urinary tract anomalies (Renal USG/gvp)
 \downarrow
High Risk

* ectopic ovary \Rightarrow ^{also} Unicornate Uterus \rightarrow U/L dysmenorrhoea
 \rightarrow highest association \bar{c} urinary tract anomalies

* TOC for Septate Uterus

\hookrightarrow Hysteroscopic Resection
 (Tompkins / Jones)

CERVICAL FACTORS

- Mucous affects fertility

\hookrightarrow characteristics — Impermeable to sperms
 \hookrightarrow Anti Sperm Antibodies — Post coital test
 \rightarrow ^{Klaus / Sims / Huhner test}

Post coital Cx Mucus — observed Under Microscope

Normal \rightarrow Forward Motility

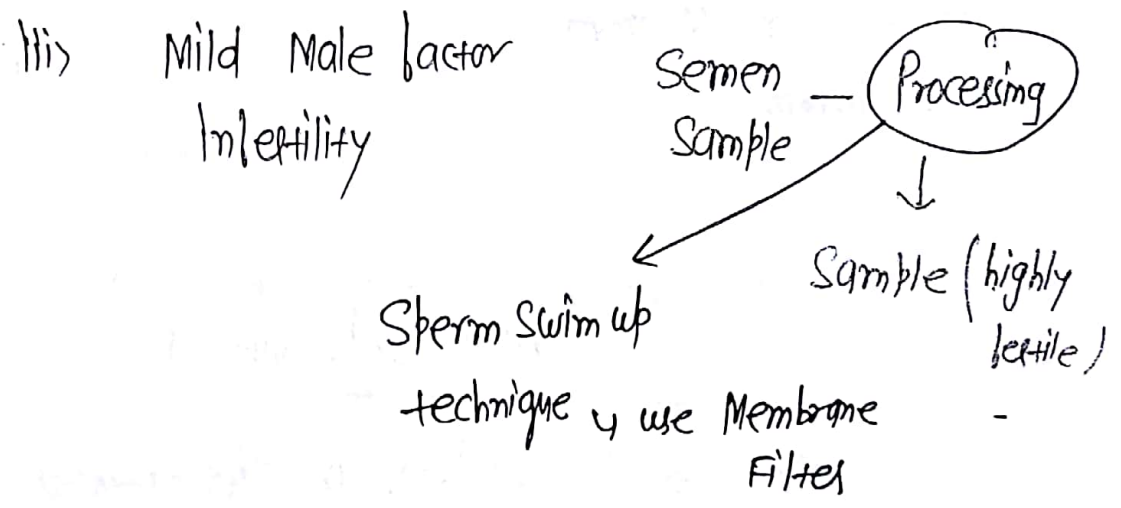
ASA \oplus — $\textcircled{\text{AbN}}$ Motility
 circulatory }
 Shaky }

done \textcircled{a} 12-14th day of cycle

* Newer test ⇒ Immunobead Assay } Not doing in
Sperm Agglutination } Current clinical
Sperm Immobilization } Practice.

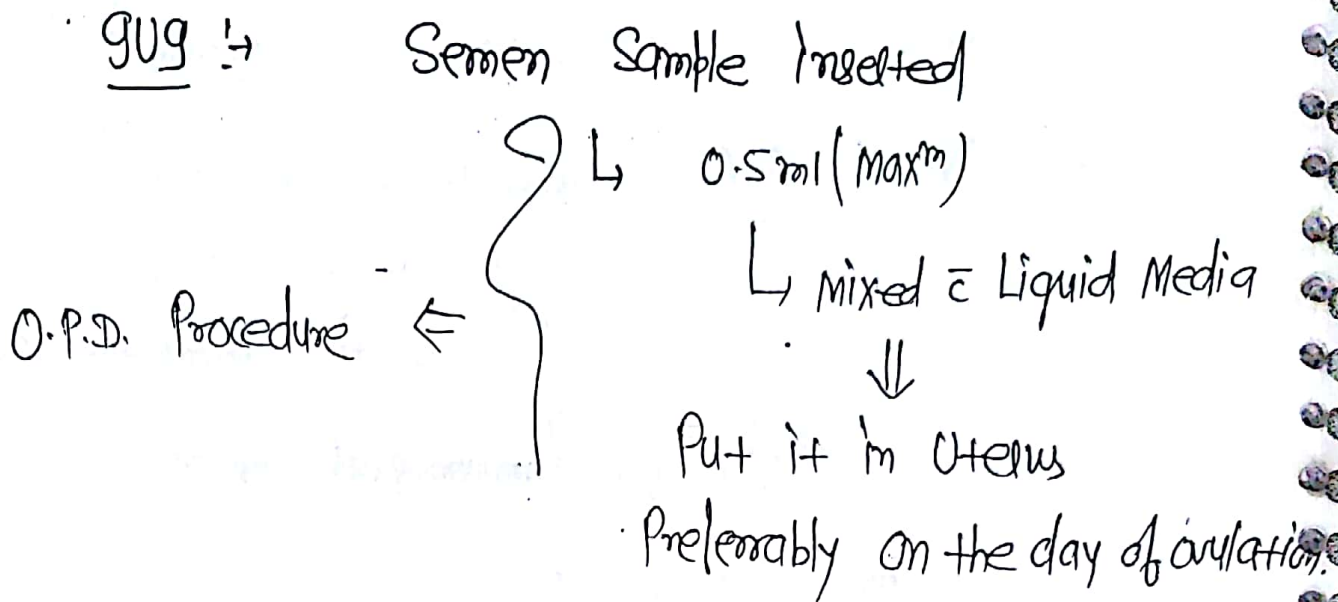
* Tx of ASA ⇒ i) Intrauterine Insemination (IUI)
↳ Put sperm directly into Uterus; bypass the Cervical Mucus
IOC for Immunological Infertility.

ii) CC + IUI
↳ 6 Month — 3 month CC alone
↳ 3 month CC + IUI



iv) Disorders of Sexual Intercourse
↳ Vaginismus } Sexual dysfunction
Failure of erection }

v) Absence of Male partner (Popular in developed world).



Pell's patient } - to reach the site of fertilization in
to be injected } 10 minute
Position in Minimum
of 10 min.

Male Infertility Factors

MIC ⇒ defect in spermatogenesis

Semen Analysis ⇒ Best ⇒ Masturbation

Abstinence ⇒ 9d - 7day

Reach the Lab in 60 min

done after liquefaction

avg. time ⇒ 2-30 min

* Wetter Parameter for Semen analysis 4
Lowermost value → for fertile

Volume > 1.5ml

pH > 7.2

Total sperm count → > 39 million / ejaculate

(N) > 100 million / ejaculate

Sperm concⁿ → > 15 million / mL

Total Motility → > 40%

Progressive (Forward) → > 32%

Morphology → > 4%

Vitality → > 58%

WBC count → < 1 million / mL

* Aspermia ⇒ Absence of ejaculate

Azoospermia ⇒ Absence of sperm in ejaculate

Oligospermia ⇒ < 15 million / mL

Asthenospermia ⇒ (Abn) Motility

Telatospermia ⇒ (Abn) Morphology

Necrostermia ⇒ Dead Sperms

Globospermia - Sperm - Rounded head

↓
Lack Acrosomal cap

*

AZOOSPERMIA

↓ then 1stly do

- Confirmation on a 2nd sample

(1-4 week interval)

↓
if azoospermia confirm

Do LH | FSH | Testosterone

if comes (N)

↓

Klas "obstructive" } ⇒ obstruction in
Azoospermia" } Semen Pathway

→ if site of ejaculation block
in vas deferens

⇒ Scrotal USG

→ if site of ejaculation block
on ED
(Volume ↓)

⇒ Trans-Rectal
USG

↓
dilated seminal vesicle

→ absent seminal vesicle

↓
Cystic fibrosis (CFTR-gene)
Analysis

if Non-obstructive case
defective ⇒ (Testes) - M/c
Spermatogenesis

↓

↑ LH | ↑ FSH | ↓ testosterone

• ↑ FSH | LH (N) | Testes (N)

↓

- Sertoli cells affected
(Leydig cells (N))

• LH ↑ | Testosterone (N) / FSH (N)

↓

Partial AIS

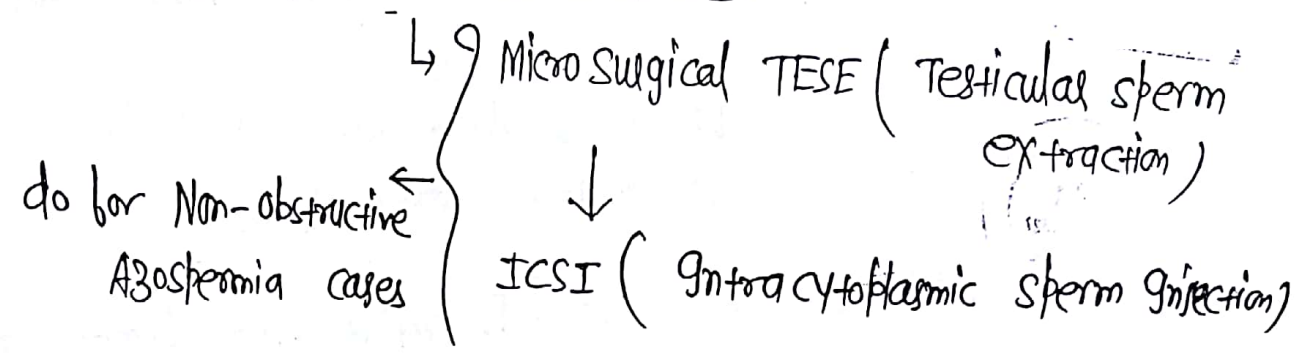
↳ Testosterone (N)
↳ LH ↑

FSH (Sertoli cell inhibition)

* ib Testes Involve \Rightarrow 1^o Hypogonadism (Hypergonadotropic) (152)

~~Testosterone~~ \downarrow Testosterone

* Non-obstructive Azoospermia



- * ib Sperm count (15 million/mL) $\xrightarrow{\text{do}}$ gug
- (5-15 million/mL) $\xrightarrow{\text{do}}$ gvf
- (<5 million/mL) $\xrightarrow{\text{do}}$ gcsi

* Sperm concⁿ + Motility > Morphology (Most imp. parameter in (N) sperm)

* ART \Rightarrow Simplest form \Rightarrow gug

- All are ART except \Rightarrow ~~gug~~
- ZIFT
 - GIFT
 - gcsi
 - gvf

GVF

- do for -
 - i) Tubal factor infertility
 - ii) Male factor infertility
 - iii) Unexplained infertility



give 1st clomiphene citrate + gug x 3 cycles

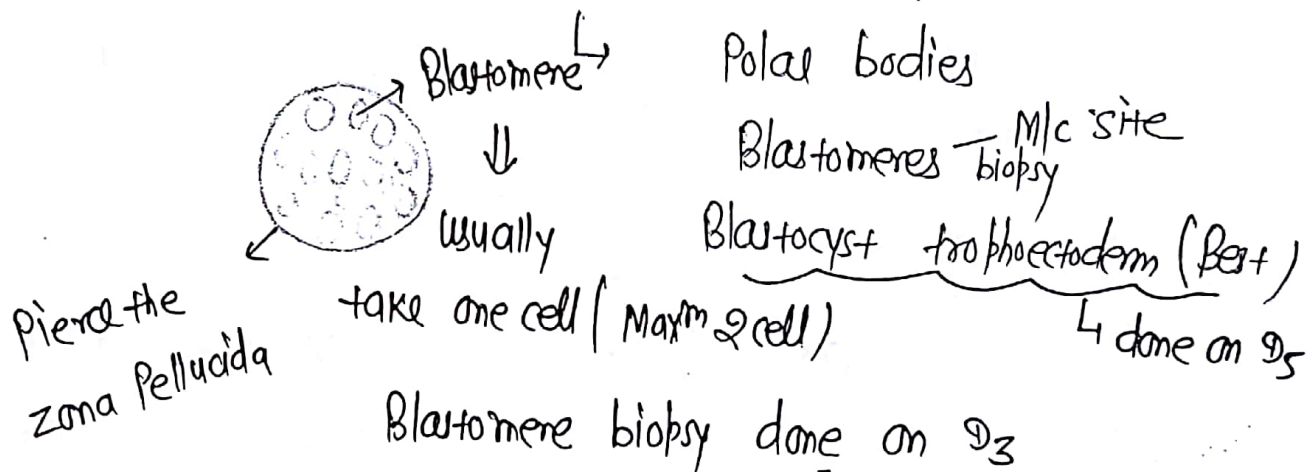
for superovulation; Not
for ovulation induction do gvf

if don't conceive

iv) PIGD (Pre-implantation Genetic diagnosis)



Site of Genetic Material taken



v) Premature ovarian failure

- Parts of GVF :- 1st ovulation induction

↳ by Gn1. Gonadotropins

↓
Follicular grow (Monitor growth)
↓
by Number size; F₂

(153)

↓
Ovulation triggered by Gn. HCG

↓ 34-36 hr after

ovum pick-up (under USG guidance)

↓ on same day

GVF (test-tube Embryo formation)

↓

do Embryo transfer (M/c on D₃).

↳ Never put one embryo; Min^m - 2
embryo puts at 2cm below fundus
under USG guidance

ENDOMETRIOSIS

- Presence of Endometrial glands & stroma outside the Uterus.
- M/c site ⇒ Ovary > Pouch of Douglas > Posterior^{leaf} of broad Ligament > Uterosacral Ligament > Fallopian tube
- Occur at all sites except ⇒ Spleen.
- dependent on Estrogen for growth (ovarian steroids)
 - ↳ so; disease of Reproductive Age g/f (25-35yr)

- It is Rare in adolescent & Perimenopausal / Post Menopausal women

- In Pregnancy — Max^m time endometrium Regress

↳ b/c of continuous - Progesterone Rich condⁿ

↓
decidualization

PGI Theory of endometriosis

↳ Most accepted theory

↳ Retrograde Menstruation
(Sampson's theory)

2nd Most accepted theory

↳ Coelomic Metaplasia

3rd ⇒ Immune Mediated theory

↓

women w/ endometriosis have deficient cell Mediated / humoral immunity

4th ⇒ Genetic theory ⇒ K-ras

if one first degree Relative affected

↳ chance of endometriosis is 7 times more

5th ⇒ Lymphatic/ hematogenous

(154)

↓
Umbilical Endometriosis

↳ Lymphatic explains it very well

IOC ⇒ Diagnostic Laparoscopy

confirm the diagnosis → Stage

↓
Biopsy (HPE)

↳ if altered coloured blood ⊕

↳ Chocolate cyst ⇒ fr. in ovary

↳ Red flame lesion (New lesion) of endometriosis

↳ Powder burnt lesion (chronic lesion of endometriosis)

Minimal → Superficial isolated implants

Mild → Superficial + Multiple aggregates diameter < 5cm

Moderate → Superficial + deep lesion (7.5mm deep)

Severe → if endometriosis distorts Pelvic Anatomy
↳ dense adhesion
↳ chocolate cyst

On USG \Rightarrow Chocolate cyst can be pickup

\hookrightarrow Homogenous ground glass appearance

On MRI \Rightarrow Only pickup the chocolate cyst

\hookrightarrow do in Adolescent girl comes \bar{c} Endometriosis.
 \hookrightarrow "Mushroom gap sign" seen.

* CA-125 \Rightarrow Raised in Endometriosis

\Downarrow
Normal < 35

\Downarrow
In Endometrioma \Rightarrow CA125 > 100
(Rupture)

24/5/18

Presentation \Rightarrow Pain + Adnexal Mass + Infertility.
(M/c)

\hookrightarrow Menstrual complain — Menorrhagia
 \hookrightarrow Bowel/Bladder symptoms \oplus
 \hookrightarrow Catamenial Hemo-thorax / Pneumo-thorax

\hookrightarrow @ the time of Menses (May be endometriosis goes to Lung)

PAIN \Rightarrow M/c \Rightarrow Dysmenorrhoea $>$ Chronic Pelvic Pain $>$ Dyspareunia $>$ Low Back Ache

\hookrightarrow 20 dysmenorrhoea

[10 dysmenorrhoea

d/t Progesterone withdrawal

20 dysmenorrhoea

d/t underlying disease process
(Endometriosis)
P12

1° dysmenorrhoea
Spasmodic

center (suprapubic)

beginning Mostly on the 1st
day of Menses

Improves \bar{c} blood flow
 \bar{c} in 72 hrs - Relief

Rx \Rightarrow Very Responsive to
NSAIDs

2° dysmenorrhoea 307
Congestive (SS)

Localised (on one side)

PreMenstrual
(week before Menses)

doesn't improve \bar{c} flow;
persist even Post Menstrual

Less Responsive to NSAIDs
* also has Dyspareunia;

\downarrow semin
} Rectovaginal septum;
} deep endometriosis

Why Pain in endometriosis \Rightarrow

Implants \rightarrow [Bleed] \rightarrow Inflammatory cells

\downarrow Release

Inflammatory Mediators

Sign of Chronic Inflammation

cause of Pain

\hookrightarrow healed by Fibrosis

\hookrightarrow cause Adhesion

\hookrightarrow also cause Pain

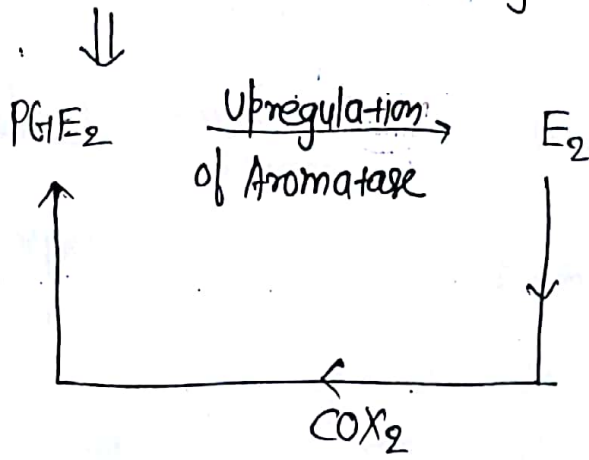
* Pain in - Implants

\downarrow

Neuromodulation (\uparrow Nerve Endings - Pain sensation)

\hookrightarrow cause by Estrogen,

* Implants → Estrogen is in Permanent Excess state



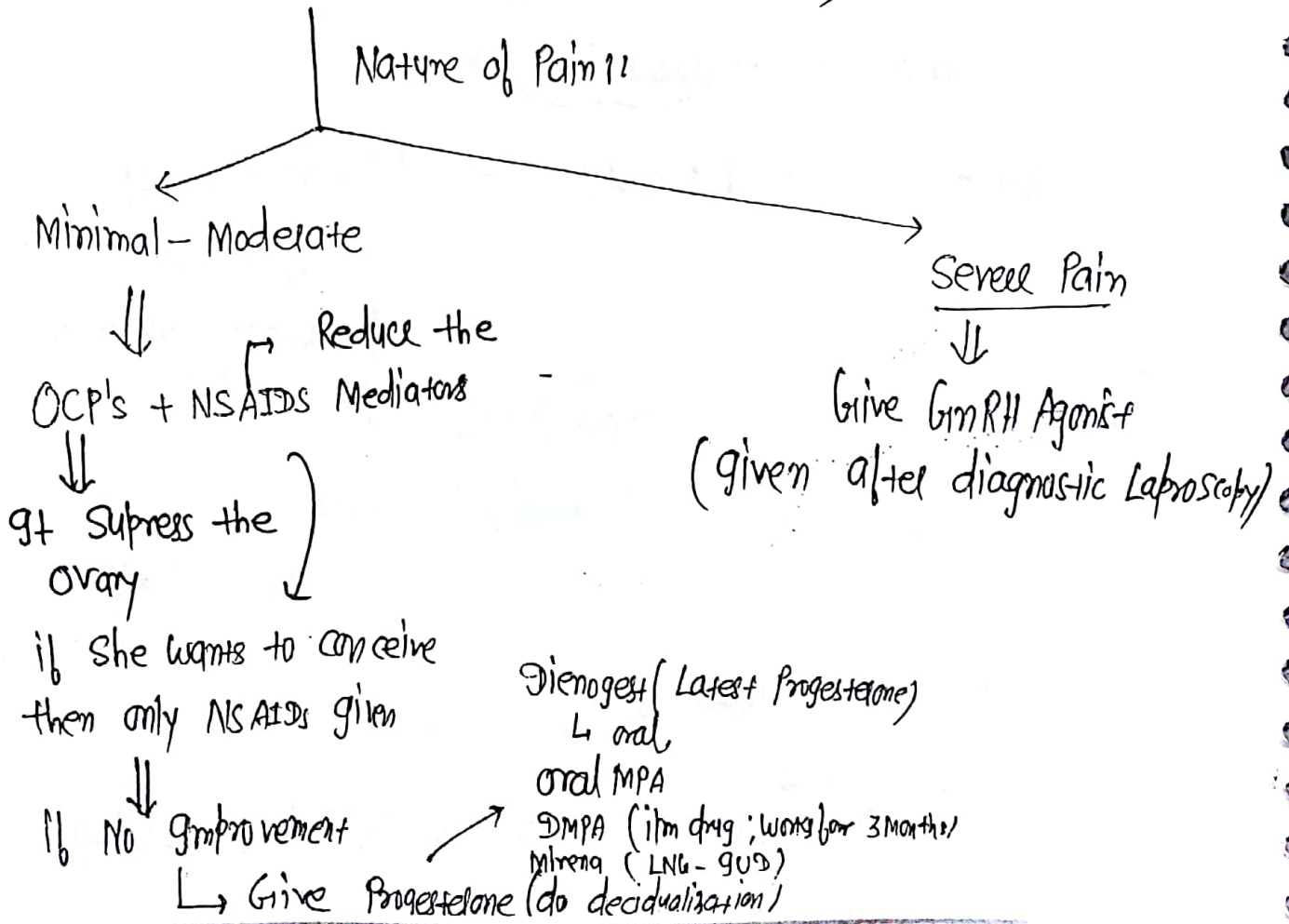
(N) Endometrium doesn't have excess Aromatase for Upregulation of Aromatase

↓
Eutopic

(N) Endometrium has 17-βOH dehydrogenase; which Metabolizes E₂.

* What we do for Pain in Endometriosis ⇒

Pain (Suggestive of endometriosis)



(156)

If After Progesterone female
is Not Responsible

↓
Give GnRH Agonist. (given after confirmation of endometriosis)

↓
by Laparoscopy

↓
given in continuous manner

can also Give GnRH Antagonist.

↓ if Not Responsive

Give Danazol

→ highest Androgen S/E

Aromatase Inhibitor → it can produce hypoenstrogenic state

if given for more than six months ⇒ Result in Bone Loss;

So; given "ADD-BACK THERAPY" ⇒ Given NORETHINDRONE

↓
to protect bone loss.

* Surgical Management of Pain ⇒ i) Adhesiolysis;

ii) Fulguration of Gmplant;

iii) LUNA (Laparoscopic Uterosacral Nerve Ablation);

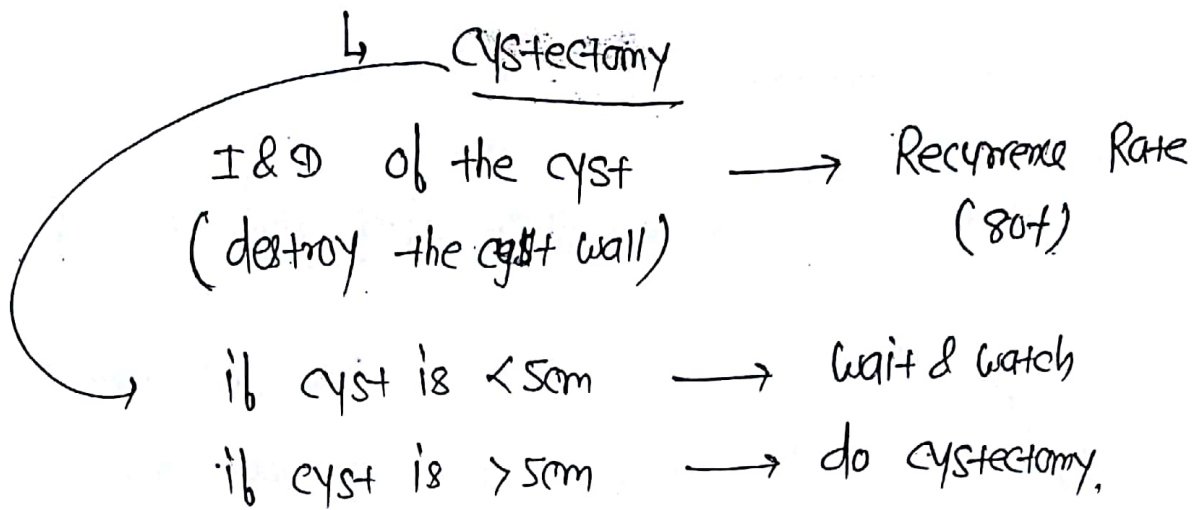
iv) Presacral Neurectomy

↳ Not effective;
Not done Routinely.

v) Adrenal Mass

↳ Endometrioma ⇒ Not Responsive to Medical Mx + or

* TxOC for Endometrioma



WIS Hysterectomy (Last Resort)

↳ only in case of family is complete

ADNEXAL MASS

PV examination ⇒ findings who tells about endometriosis ⇒

- i) Tenderness in Pouch of Douglas;
- ii) tender Nodules on Uterosacral Ligament;
- iii) Fixed Retroverted uterus
- iv) Adnexal tenderness.
- v) Adnexal Mass

⇓
Ground glass appearance on USG

Inferility in Endometriosis

⇒ Main Reason ⇒ Ovarian (Not Anovulatory) (157)

- folliculogenesis ⇒ defective
- Genetic Material ⇒ Not good of ovum

In Moderate - Severe endometriosis

⇒ Main Reason ⇒ Ovarian + Tubal

In Minimal - Mild Endometriosis

⇒ Rx Clomiphene Citrate + Intrauterine insemination X 3 cycle (3 Months)

↓
Superovulation

↓
don't conceive

↳ do IVF

In Moderate - Severe Endometriosis

⇒ Rx IVF + Sx

↳ do Mainly; Surgery is Not Necessary to do.

* GnRH Agonists has No Role in improvement of Inferility in endometriosis

* Endometrioma ⇒ If during the IVF; we find the chocolate cyst

⇓

don't operate; b/c if operate, b/c of follicle damage; Inferility may seen.

wait & watch

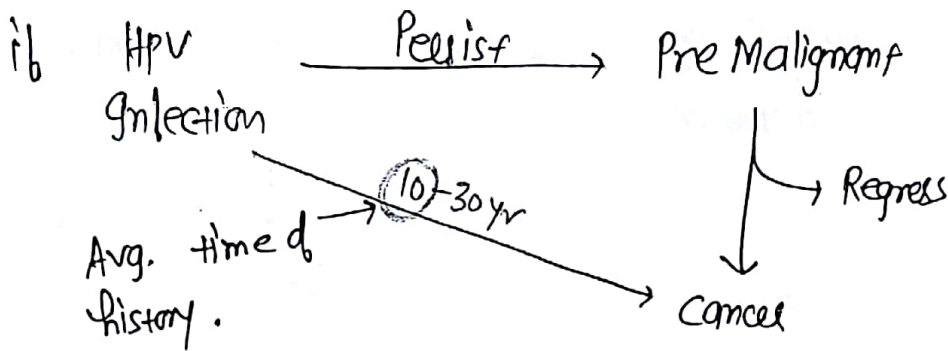
* MALIGNANCIES

* M/c Cancer among Indian women \Rightarrow Breast Cancer
 2nd " " " " \Rightarrow cervical cancer

M/c cause of Mortality d/t Cancer in Indian women \Rightarrow "

CERVICAL CANCER

Very commonly a/w HPV Infection (Most women clear this Infection)



if all history is given do " Universal Screening "

- Acc- to ACS/FIGO

Starts screening \Rightarrow 21 yrs (irrespective of sexual activity)
 In

How? \Rightarrow PAPS \rightarrow 3 yearly in 21-25yr; everyone will turn out +ve; so: Nt do.

Higher specificity \leftarrow PAPS + HPV (Co-test) \rightarrow 5 yearly \rightarrow Higher sensitivity (ideal \geq 30yr can do in \geq 25yr)

Stop \Rightarrow @ 65yr (provided the PAPS in last decade \textcircled{N})
 if Not Normal stop @ 75yr

WHO Programme

Start of Screening ⇒

begins @ ≥ 30yr

3rd-4th decade } More targeted
(30-49yr) } Population Age Group



WHO says "SEE & TREAT"

How?? ⇒

do single test

Via HPV > VIA > PAPS
≡ (visual inspection & Acetic acid) ↳ ideal ↳ for confirmation

Best Method ⇒ HPV + VIA

if HPV alone done ⇒ 5 yearly do

if VIA & PAPS done ⇒ 3-5 yearly do

Treatment Protocol also to WHO ⇒

do cryotherapy (LEEP) & Conization

if Not eligible goes for LEEP

* Screening Methods ⇒

HPV testing (DNA testing)

↓
• For high-Risk viruses

↳ do via Hybrid capture Technique

↳ expensive

• if HPV + PAPS → cotest

• if do PAPS 1st then follow up by HPV ⇒ Reflex testing

* High Risk viruses

↳ HPV 16, 18, 31, 33, 35, 45, 52, 56, 58
 ↳ 70% of cervical cancer
 ↳ 30% of cervical cancer

* Routinely in India we do ⇒ VIA (visual inspection w/ Acetic acid 5%)

↳ Abnormal → White area (dysplastic cells ⊕)
 ↳ Aceto white area (stained test)
 ↳ Unstained Area ⇒ Normal

VILI test — Lugol's Iodine — 4-5%
 ↳ Klas "Schiller's test" → Brown ⊕ test — Normal cells w/ Glycogen
 ↳ Unstained (Abnormal test) — ↳ ⊕ in mature cells
 ↳ b/c of ⊕nce of dysplastic cells (immature cells)

• PAPs Smear — Screening test (highly specificity)

↳ Taken by "Ayer's spatula & Cytobrush"

↳ taken secretion from endocervix secretion
 ↳ Pap smear is always taken from Blind end of it & turn it by 360°.



Taken from Transformation Zone > Squamocolumnar Junction > Ectocervix of cervix.

* Smear taken ON ONE SLIDE

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No Air drying do



Fixative - ? ^{AIIMS Nov/17} 95% Ethyl alcohol +
5% ether

* No Absolute Contra Indication for PAP Smear; but if actively bleeding patient comes then tell her to come after bleeding stops.

* PAP Smear is also klas " Secretion Cytology":

* BETHESDA CLASSIFICATION ⇒

① Normal Report ^{↳ tells about Report comes on PAP Smear.}

② Reactive & Reparative changes (Healing Inflammatory changes);

③ Infection — Specify the organism

* Organism lies inside endocyt; tough to

catch in PAP Smear ⇒ Chlamydia
Gonorrhoea

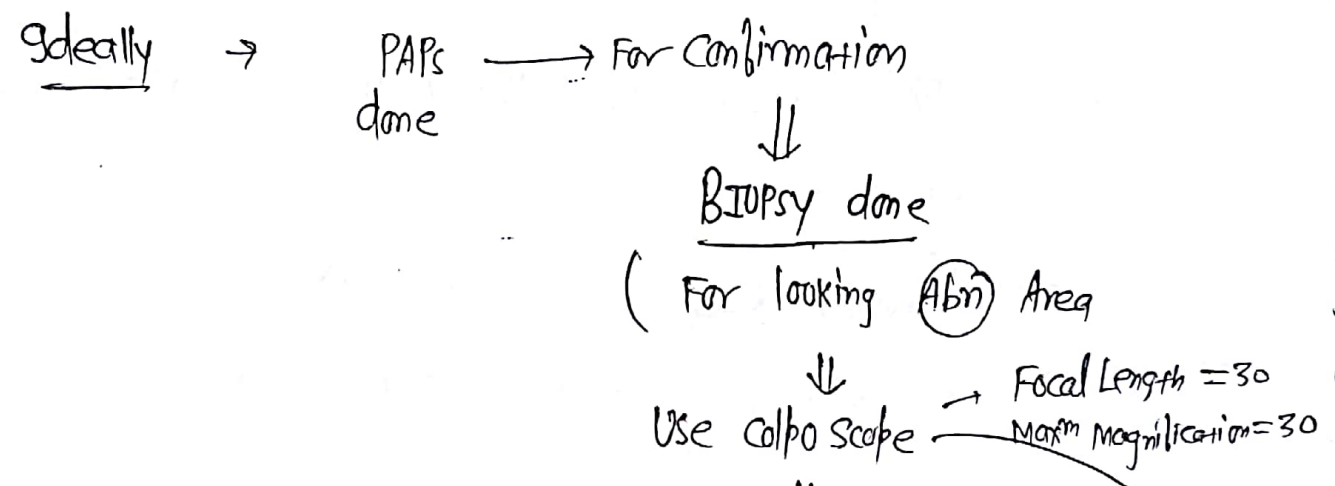
④ ASCUS — Atypical Squamous cells of Undetermined significance
Asc^{us} (Modified Bethesda classification)

⑤ LSIL — ^H Low grade Squamous Intraepithelial Lesion
_{High Risk}

⑥ HSIL — High " "

⑦ Cancer

*	<u>LSIL</u>	<u>HSIL</u>
<u>Nucleus</u>	↑	↑↑
<u>Cytoplasm</u>		
<u>No. of cells</u>	Less	More
<u>Granules</u>	evenly distributed (Granular chromatin)	clumps
<u>Membrane</u>	Shrivelled Membrane <u>Doubt</u>	



We can see Ectocx
vaginal wall
vulva;
but we can't see Endocx

* Abnormal Area from where Biopsy should be taken =>

i) Irregular Surface contour

ii) Mucosa - Pale in colour

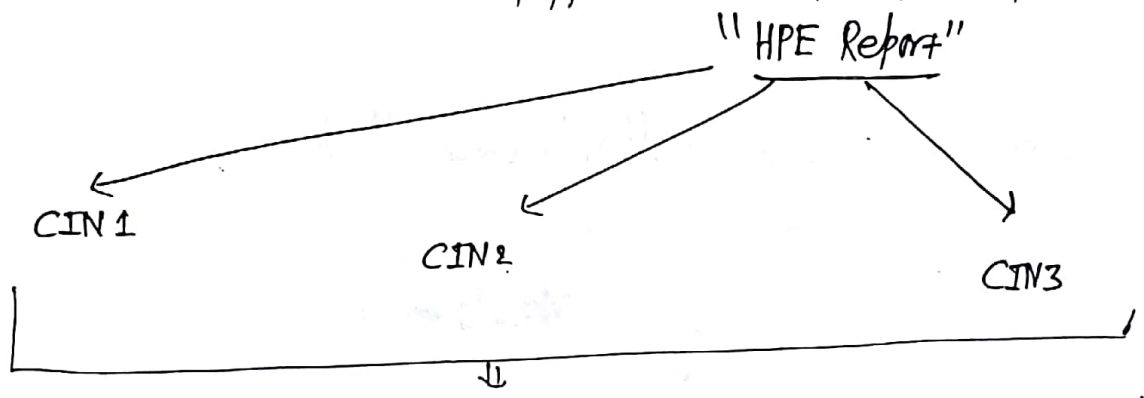
iii) In Acetic acid -> white area seen

iv) In vascular pattern of

- Reticular
- Mosaic
- Punctate

to visualise vascular pattern clearly; we add Green filter to colposcope

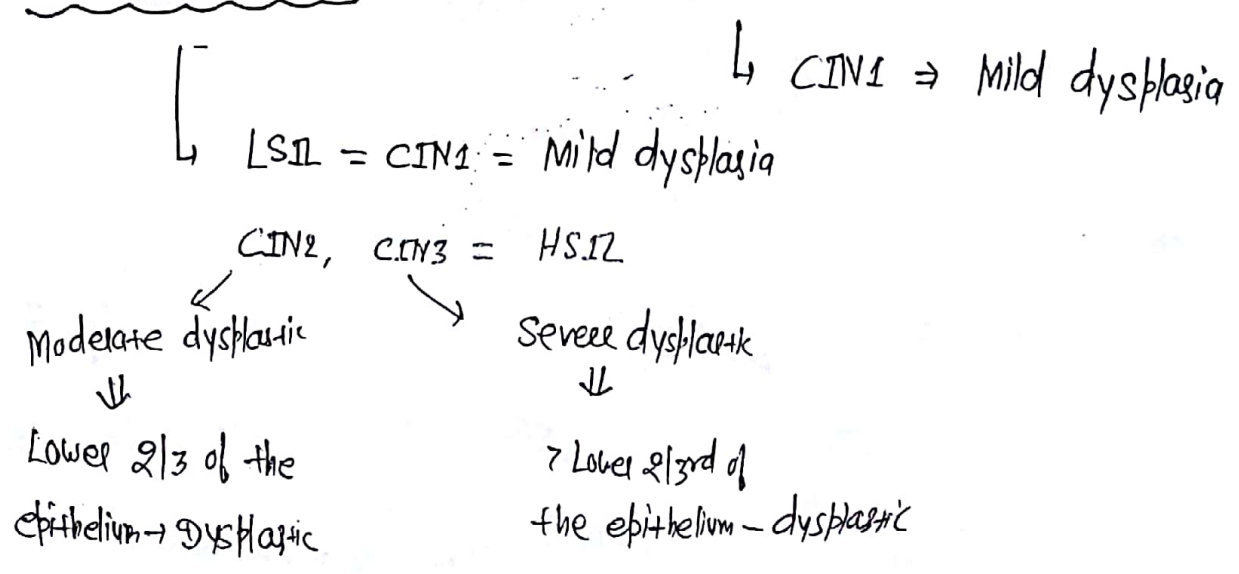
* Since it is Biopsy; so; Colposcope Report called as



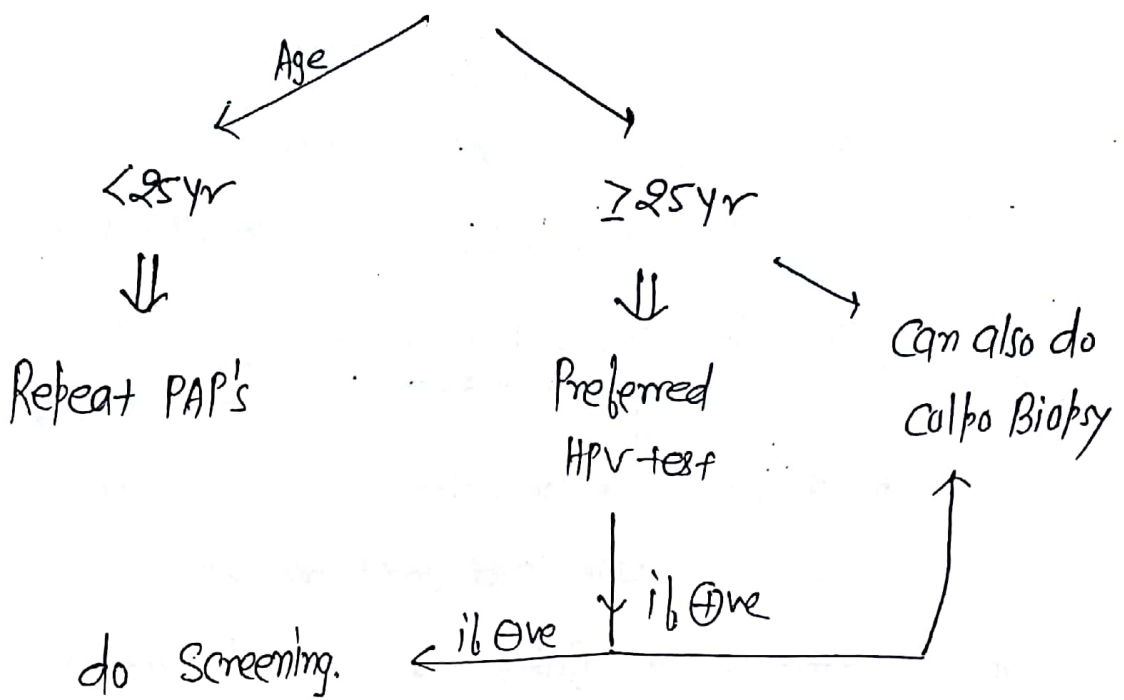
all are Pre Malignant condn i.e all have basement Membrane Intact.

* HPV cells affects basal & parabasal cells; so; Cancer starts from Lower part of epithelium

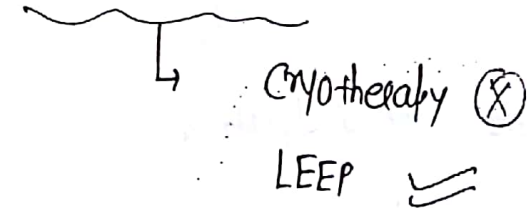
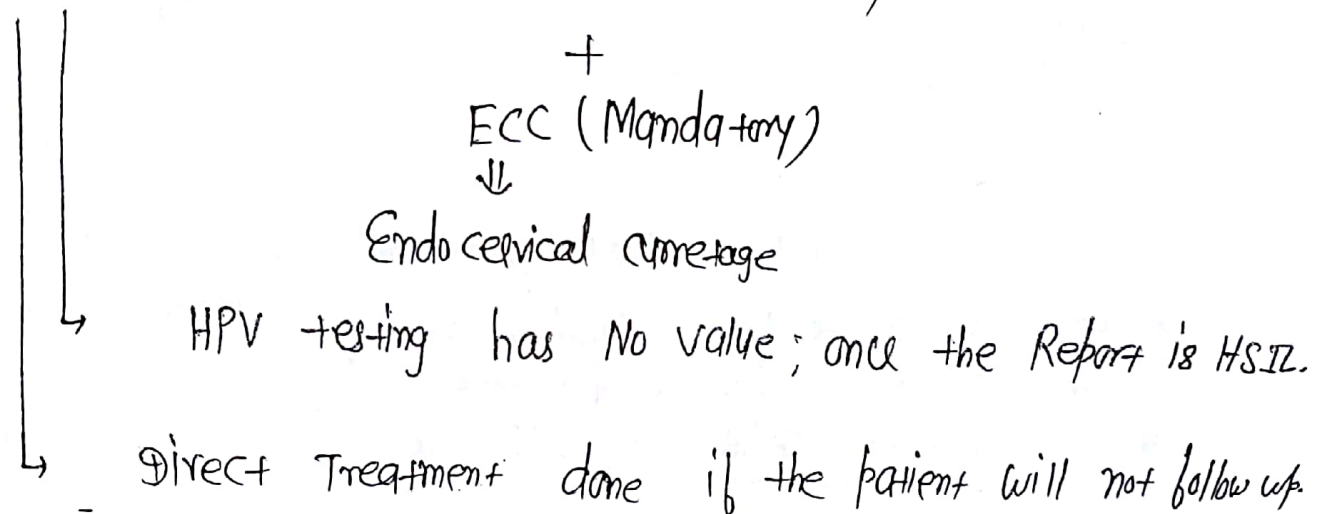
* Dysplastic cells - all seen in Lower 1/3 of the epithelium



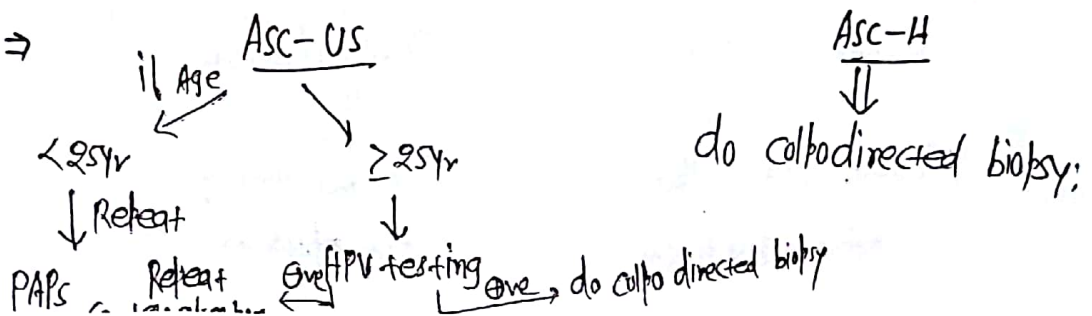
LSIL ⇒ if the case is of LSIL



HSIL ⇒ do colpodirected Biopsy



ASCUS ⇒



* if colpo Reports — Confirming the Lesion

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CIN-1



Ca 1-1.



Follow up



Co-test Yearly for 2 years

↳ Usual time of CIN1 to Regress



if Persistent for 2 years



Treat

CIN-2 → 5+ (cancer) } Treat.
CIN-3 → 12-40+ }

* How to Treat ⇒

Cryotherapy > LEEP > Conization

Who is eligible — 21 criteria

- i) Entire Squamo columnal Junction visible
 - ii) Entire lesion should be on Ectocervix
 - iii) if lesion should occupy < 75% of Ectocervix
- all are known by visual inspection by acetic acid

do CRYO ABLATION →
↳ OPD procedure

Apply cold gases (CO₂ / Nitrous oxide)
↳ destroy the cells in tissue

Mechⁿ of cryoablation

↳ (Freeze - Thaw → Freeze)

do crystallization of Intracellular water



Causes Desiccation

Pain Managed by Analgesics

Long term complication ⇒ Persistent watery discharge

↳ bleeding is Not a complication of cryoablation.

* Laser Ablation do → i) Depth of Lesion is More;
→ ii) Lesion extends on the vaginal wall

* LEEP = LLETZ →
↓ ↓

Loop electro-surgical excisional procedure Large loop excision of Transformation zone

→ electric current
do cutting + Coagulation

* Bleeding is Not the S/E of LEEP.

*** } No training Required;
OPD Procedure!
takes < 4min in completion of Procedure

* Cryo-therapy doesn't give us any specimen; while LEEP give us Specimen after procedure. (Specimen of Transformation zone)

* CONIZATION \Rightarrow Invasive ;
O.T. procedure ;

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Remove Tissue ;

* Indication of cone / C/I for LEEP \Rightarrow

⊙ \rightarrow Apex towards guide

\rightarrow Cut from Transformation zone

Shallow
coneDeep
cone

- ectocx
endocx
Junctional zone
(Mucosa & Stroma

} given by cone

i) Unsatisfactory colposcopy

↳ entire Transformation zone is Not visible ;

ii) When there is discrepancy b/w cytology & HPE

iii) if PAP's smear is HSIL \rightarrow do Colpo. + ECC

↳ \oplus ve

iv) if there is suspicion of Microinvasion

v) if Biopsy Report says \Rightarrow AdenoCa (Suspicion of endocx involvement).

Q.

38yr old Lady P₃L₃ CIN3 ??

a) cryo

b) LEEP

c) conization

d) Hysterectomy.

we need for follow up

↳ only indication to pre-invasive lesion

↳ In Recurrent CIN :

• if Patient will not follow up;

• if suspicion of Adeno histology & family complete

↓
May be case of endometrial ca;

so, do hysterectomy

• Other pelvic pathologies which justify a hysterectomy.

CANCER

RIF
↳ For Cx
Cancer

i) early Age of 1st intercourse

ii) " " " " 1st child birth;

iii) Multiparity

iv) Multiple sexual partner

v) Low Socioeconomic status

vi) STD

vii)

Smoking

↑ Sq cell ca

⊗ Adeno ca.

viii) Pre Invasive Lesion ⊕

ix) OCP → Tes Risk if use beyond or equal to 5yr

- ↳ Nullify Risk if leave for ≥ 10yr
- ↳ ↑ Related to Adenoca

x) Family history

Early Menarche

Late Menopause

⊗ Not a R/F for Cx Cancer

Etiology of Cx cancer ⇒

HPV

- ↳ also also cervix; vagina; vulval cancer;
- Penile; Anal; oral

High Risk HPV ⇒ Cause cancer

Low Risk HPV ⇒ 6, 11 → Cause genital warts

↓

condylomata Acuminata

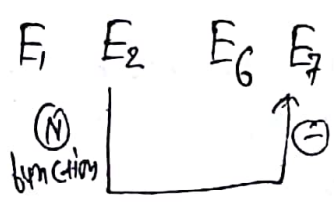
↳

Laryngeal Papillomatosis

* On HPE of HPV Infected cells

↓

Cancer ⇒



↳ Koilocytes

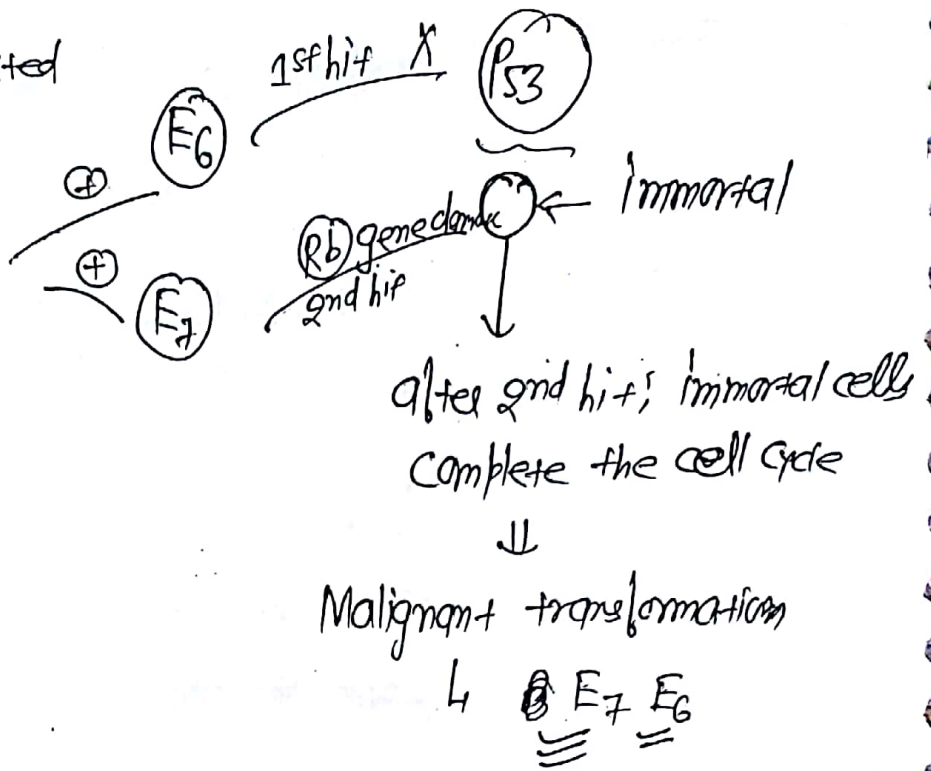
↳ Perinuclear halo-

* When virus integrated

in host genome



E₁, E₂ altered



* HPV vaccines

Gardasil

CERVARIX

Quadrivalent

Bivalent

- Made from

inactivated capsid protein

16, 18, 6, 11

16, 18

- Latest FDA approved Gardasil-9 (Nonvalent)

16, 18, 6, 11, 31, 33, 45, 52, 58

- given 0.5ml i/m

given 0.5 ml i/m

- 0, 2, 6 Months

0, 1, 6 Months

- Ideal age to give the vaccine \Rightarrow 11-12yr

can be given \Rightarrow 9yr - 26yr

- Girls/Boys both taken

Girls taken

S/E of vaccine \Rightarrow Syncope attack

\Downarrow

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So; there has to be observation time of 15min; then send her to home

* Protection Rate - Quadrivalent Gardasil \Rightarrow 70%
Gardasil 9 \Rightarrow 95%

* M/c virus a/w cancer \Rightarrow HPV16 = Squamous
Most specific virus a/w cancer \Rightarrow HPV18 = Adeno

* For cervical cancer \Rightarrow

M/c Age \Rightarrow 3rd-4th decade

(Shows Bimodal Peak

\nearrow 1st 3rd-4th decade

\searrow 2nd 5-6th decade

M/c histology \Rightarrow Squamous cell ca = 69%

Adenoca = 25%

Large cell Non-Keratinizing Squamous cell ca

M/c Route \Rightarrow Direct; Lymphatic

M/c Presentation \Rightarrow Irregular vaginal bleeding

1st Presentation \Rightarrow

Most specific presentation \Rightarrow Post coital bleeding

\Downarrow Next

Clinical examination

clinical examination

↓ ⊕

do PAPs Smear*

* Persistent Postcoital bleeding ≥ 6 Months

↓

even with (N) PAPs

↓

do colpo Biopsy

35yr P₂L₂ \bar{c} Postcoital bleeding; o/E 2x2 cm growth on the Anterior lip of cervix

do PUNCH Biopsy.

M/c site of distant Metastasis \Rightarrow Lungs
In Cx cancer

M/c cause of death \Rightarrow Uremia (Renal failure)
In Cx cancer

Risk of ovarian involvement \Rightarrow < 1%
In Cx cancer (Ovaries - Spared)

Most imp. Prognostic Marker \Rightarrow Stage > Lymph Node status
In Cx cancer

* Staging for cervical cancer ⇒

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FIGO - clinical staging

Investigation should not be used to change stage of cancer ⇒

USG
CT
MRI
PET } (X) Not used.

• Cystoscopy (to look bladder cavity);
↳ Part of Staging

• EUA (Examination Under Anesthesia);
↳ Part of Staging

Stage 1

A ₁	} - Micro Invasion	- depth < 3mm	Horizontal Limit - < 7mm
A ₂		- depth 3-5mm	" - < 7mm
B ₁	} → Macro Invasion	- depth ≤ 4cm	} & all Microscopic cond ⁿ above than A ₂ .
B ₂		- depth > 4cm	

Stage 2

Upper 2/3rd of vagina

A - Without Invasion of Parametrium

B - With Invasion of Parametrium

do MRI; but we can't
↓ change the stage
connective tissue

Clinically seeing
obliteration of Fornices

Stage 3

A → Lower 1/3rd of vagina is Involved

B → Spread to Lateral Pelvic wall

(Ureter → hydronephrosis)

↳ seen by gyp/CT → approved to change stage to look for hydronephrosis.

Stage 4 ⇨ A → Spread to bladder and/or Rectum
 B → distant spread
 Inguinal L.N.

* Early Stage Cancer ⇒ WHO Stage IB₁
 ↳ Primary Tt ⇒ Surgery

Locally Advanced Cancer ⇒ ≥ IB₂
 ↳ Primary Tt ⇒ Chemo Radiation

Tt of Cancer Cervix ⇒

IA₁ → if Family is complete

↳ Hysterectomy

↓

if Family is Not complete

(Type-I Hysterectomy / simple Extrafascial hysterectomy)

⇓

Fertility preserving amputation.

(Therapeutic amputation)



→ Cut Uterus; Cx & Overlying fasciae and Remove

IA₂ → if Family is complete

↳ Type 2 Hysterectomy + Pelvic LN (Wertheim / Modified Radical) dissection

Cut Midway b/w Utero sacral Ligament and/or cardinal Ligament

Remove Uterus; Cx & half of Parametrium



↳ Lateral Pelvic wall

↳ Parametrium

IA₂ - if Family is Not complete

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do Radical Trachelectomy



Remove Gx + Parametrium + Pelvic LN

Saves Uterus

IB₁ - if Family is complete



Type-3 Hysterectomy (Meigs/ Radical hysterectomy).

+

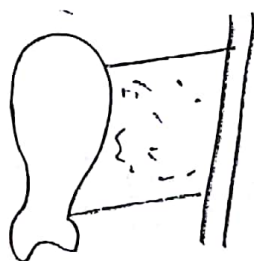
Pelvic L.N. dissection

+

Para-aortic LN Sampling.

↳ When we Remove all the Lymph Node

↳ Few L.N. Remove



Insertion on the
Lateral Pelvic wall we
cut the cardinal/uterosacral
Ligaments

Type 1, 2, 3 Hysterectomy

↳ Only for Malignancy

(Piver Rutledge classification)

Q. Q. Cancer 1cm ; 4mm deep and 9mm $\frac{1}{2}$ Limit II Stage \rightarrow

In case if tumor is ≤ 2 cm \Rightarrow type 2 Hysterectomy
(Wertheim's)

Mic Surgery in the Cx Cancer \leftarrow

* I-B₁ \rightarrow Family is Not Complete

\Downarrow

if ≤ 2 cm \Rightarrow do Radical Trachelectomy.

CHEMORADIATION

Stage \geq IB₂

\Downarrow

Chemo-therapy + Radio-therapy

(give concurrently)

Radiation sensitizers

Chemo Agent \Rightarrow cis-platin > SFU

In 2A1 (< 4cm)

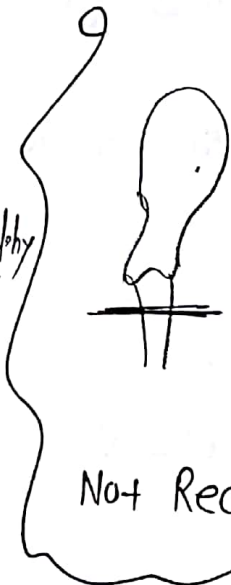
\rightarrow Type 3 hysterectomy

\Downarrow

Upper 2/3rd of vagina

Removed (or half of vagina removed)

Not Recommendation; but we do.



* Radio-therapy $\xrightarrow{\text{Distant}}$ External beam RT

\downarrow Close

(EBRT) - klas "Teletherapy"

Brachy-therapy (Intra cavity)

Mic used = Iridium

\rightarrow 121

* 1stly EBRT gives then follow up by Brachy-therapy.

\Downarrow
Mic used = cesium

* EBRT gives to Pelvis

(167)

↳ Dose \Rightarrow 50 Gy in 5 weeks
(5 fraction every weeks)

* EBRT gives to Pelvis + Abdomen (Extended field RT)

↳ Dose \Rightarrow 30 Gy

* Brachytherapy gives to Pelvis

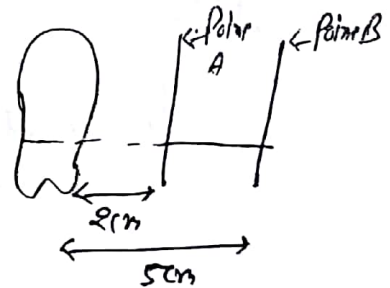
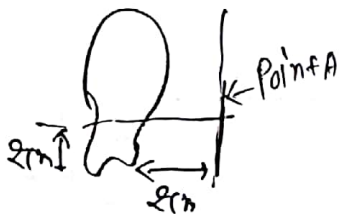
Point A

Point B

• 2cm above external OS

• 3cm Lateral to Point A at the same Level

- 2cm Lateral to cervix



• Corresponds to Paracervical Lymph Nodes; Ureters

• corresponds to ureter

Dose \Rightarrow 8000 cGy

6000 cGy

Radiosensitive order \Rightarrow Ovary > Rectum > Bladder > Vagina
 ↳ can tolerate 7000 cGy
 ↳ Most Radiosensitive (can't tolerate Radiotherapy)
 ↳ can tolerate 7500 cGy
 ↳ for tolerant Mobilize it.

* Adjuvant Chemoradiation

↳ give Post-operatively

- Reports says \Rightarrow
- i) Lymph Node \oplus
 - ii) Parametrium Invasion \oplus
 - iii) Tumor Margins all \oplus ve

Qq. What to do in Recurrence of Cx cancer \Rightarrow

Primary Rt if Sx $\xrightarrow{\text{do}}$ chemoradiation

if Primary Rt chemoradiation

Inoperable

operable

Seen by Looks for
 hydronephrosis;
 ULV ^{Loos} Limb edema;
 Pain Radiating on one Leg.

$\left\{ \begin{array}{l} \text{Reached Lateral} \\ \text{Pelvic wall} \end{array} \right. \Rightarrow$
 \Downarrow
 Palliative Rt.
 \Downarrow
 Chemotherapy

Central Recurrence
 \Downarrow
 do Pelvic Exenteration
 Surgery

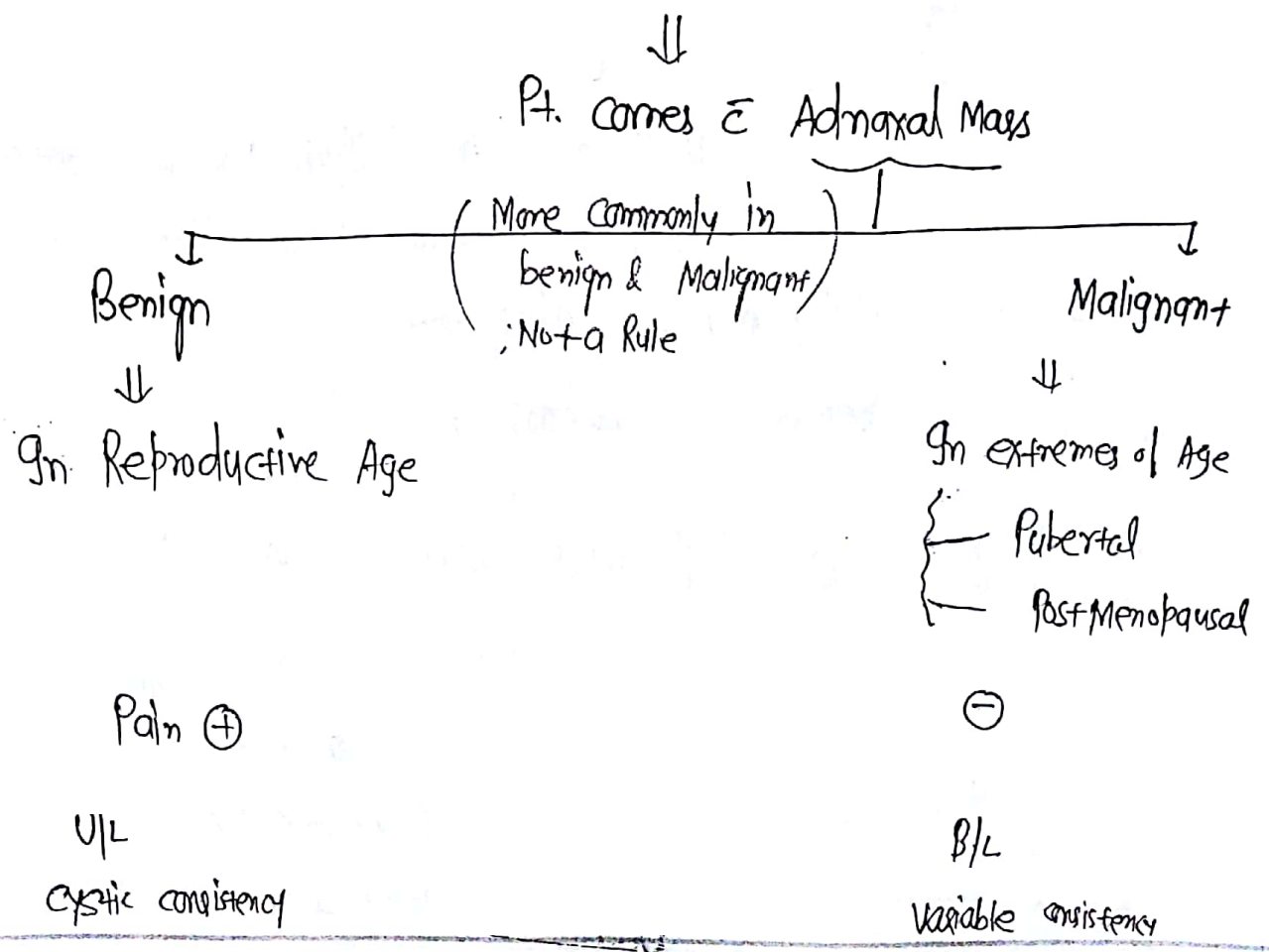
90. Known case of Cancer cervix 3cm growth upper vagina
 is involved \bar{c} Parametrium; CT - hydronephrosis; cystoscopy
 bulbosa edema of bladder Mucosa (168)

Stage 3B.

doesn't Mean Invasion of
 bladder; i.e Lymphatics
 obstruction; so, Not change
 the stage

OVARIAN CANCER

- Not very common like of cervical ca
- No specific sign & symptom
- Routinely Not screening for ovarian cancer



Benign
tender

Malignant
Non-tender

IOC for Adnexal Mass \Rightarrow TVS

{ U/L; Unilocular; Anechoic } \Rightarrow Benign cyst
Most commonly

* M/C cyst of the ovary \Rightarrow Follicular cyst^{*}

B/L; Solid component; Septae; \uparrow vascularity; Ascites; Enlarged LN/
(thick 2-3mm) / \ Matted Bowel Loops
Ground Mass Septae



Malignant cyst. (Highly Risk of Malignancy)

Sx \Rightarrow Malignancy - Laparotomy

Benign - Laparoscopy

Indication

i) High Risk condition on USG

ii) Ovarian Mass > 7cm

Adnexal Mass > 10cm

iii) Raised CA125 (\odot value < 35)

↳ only in Post Menopausal age group.

iv Mass present as acute Abdomen ^{d/t Rupture} _{d/t Torsion}

* Benign looking Mass
(UL; Unilocular; Anechoic Mass)

in Reproductive Age
Group patient
↓ check size

if 3-5cm ⇒ wait & watch (Maybe follicular cyst)

if 5-7cm ⇒ Follow up 6wks; 12 weeks

* Give OCPs → Pain
→ Menstrual Irregularity
↓
contraception

OCP prevents occurrence of New adnexal cyst;
but Nothing do if currently exist cyst

in extremes of Age
patient
↓

Tumor Marker

• Pubertal (Adolescent)

↓
AFP + HCG

• Post Menopausal

↓
CA-125

* Adnexal Mass in a pregnant women ⇒

in 1st Trimester ⇒ wait & watch;

in 2nd Trimester ⇒ i) High Risk features

↓
Safest time for
elective Surgery.

ii) >10cm (size)

iii) Acute Abdomen

cyst seen common in ♀ ⇒ i) Theca Lutein cyst → HCG (Pregnancy Marker)
(Adnexal Mass) ↳ also in clomiphene citrate therapy

iii) Luteoma → In Pregnancy

↓
virilizing ovarian tumor

↳ In Mother

Less common - In fetus

- Spontaneously Regress after ♀.

More the ovarian cycle
↓
More the Risk of ovarian tumor
More the estrogen
↓
More the Risk of ovarian tumor

* RIF for ovarian tumor

i) Early Menarche;

ii) Late Menopause;

iii) Infertility;

iv) obesity;

v) BRCA 1
2

HNPCC

vi) Smoking (only in Mucinous variety of ovarian tumor)

vii) PCOS

Protective for ovarian tumor

i) O-CP

ii) Breast feeding;

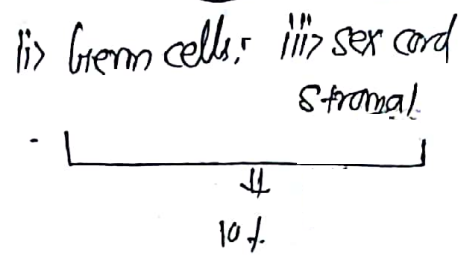
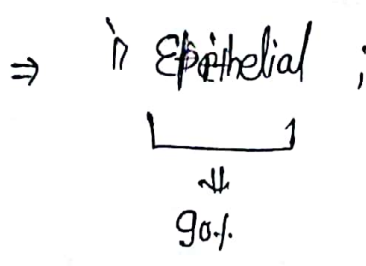
iii) Only Anovulation (Not also Any disease)

iv) Salpingectomy
Tubal Ligation
Hysterectomy } Ascending Mitogens don't reach ovary

* Fallopian tube ca ; ovarian ca & peritoneal carcinoma

↳ all have common origin ⇒ Fallopian tube

* Types of ovarian tumor



(A) Epithelial ovarian TUMOR (Features) -

- Serous - 75%
- Mucinous } 10% each
- Endometrioid }
- Brenners
- clear cells

Presentation Peak age - 60 yr. (6th-7th decade)

- B/L
- Non-specific sign & symptoms
 - ↳ Irritable bowel Sx
- Late stage
- High Mortality Rate
- Sporadic - 90%

So, only 10% tumor are familial.

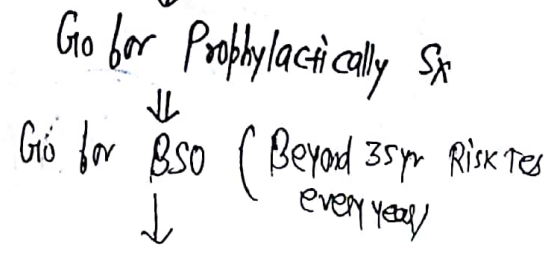
Can tend to occur a decade earlier (50yr)

Gene involved ⇒

- BRCA1 - 45%
- BRCA 2 - 25%
- HNPCC - 15%

* if First degree Relatives affected → Risk Tes by 3 times.

aa if patient is k/clo Genetic Mutation



BSO (Bilateral Salpingo-oophorectomy)

↓
done @ 35yr or as soon as family complete

↓
This surgery protects against ovarian cancer
also protects from breast cancer by 50%.

2nd Line approach

⇒ if Patient doesn't want to go for BSO

↓
Give OCP's + Screening - High Risk

High Risk {
i) Strong family Breast/Ovarian ca
ii) K/clo Genetic Mutation

Screening ⇒ TVS + CA125

Start @ 35yr 6 Monthly / 12 Monthly

Ovarian Tumor

⇒ M/c ovarian tumor = Serous cyst Adenoma

M/c ovarian ca = Serous cyst Adenocarcinoma

* Mucinous ovarian tumor ⇒ i) Decade earlier

ii) Grow to Large size (20cm)

iii) Diagnosed early

iv) Better prognosis

v) UEL (BIL in 10% cases)

vi) CEA (Tumor Marker)

CA125
↓ Tumor Marker
for Serous tumor

vii) Pseudomyxoma Peritonei

(171)

(Loculated mucinous collections in Abdomen)

M/C Seen in tumor of Appendix; Not ovary.

* Endometrioid ovarian ca - high Risk for co-existent "Endometrial ca".

↳ lot of total Epithelial
↳ UL in Nature

* Clear Cell ovarian ca - highest association with Endometriosis

↓
also w/ Endometrioid ovarian ca.

↳ HobNail cells ⊕ (O)
↳ HPE findings.

* Brenner's - UL; Benign; solid lesion

↳ on HPE - Bladder Like epithelium - Transitional epithelium.

(B) GERM CELL TUMOR → 5-8% of all ovarian tumor

• Teratoma → Mature cystic (Dermoid)
Teratoma

↳ Immature

• Dysgerminoma

- Embryonal cell ca
- EST (Endodermal sinus tumor) | Yolk sac tumor
- Choriocarcinoma
- Mixed tumor

M/c Germ cell tumor of ovary \Rightarrow Dermoid

M/c Germ cell cancer of ovary \Rightarrow Immature $\left\{ \begin{array}{l} \text{Dysgerminoma} \\ \text{Teratoma} \end{array} \right.$

* Feature \Rightarrow i) U/L (Unilateral);
 of GCT which GCT has highest Risk of Bilaterality \Rightarrow

Dysgerminoma $>$ Dermoid
 (15%) (10%)

ii) Seen in younger girls (10-30yr)

What % of ovarian tumor in this age grp. are Germ cell tumor \Rightarrow 70%
 \Downarrow

iii) Non-specific sign & symptom + (A) Precocious Puberty
 (B) acute abdomen

iv) Pick up in early stage;

v) Good Prognosis

vi) Conservative.

b/c GCT Release hCG
 \Downarrow
 & Submit similar to LH/FSH
 EST / Yolk sac tumor
 \hookrightarrow Most Rapid growth of tumor

(172)

* Dysgerminoma ^{TUMOR} ⇒ LH (HCG + PLAP) ^{TUMOR MARKER}
 (Tumor Marker)
 ↓
 Fleshy; Lobulated & Tan in colour. Not secrete AFP.

* Endodermal Sinus tumor ⇒ AFP (LH)
 (Tumor Marker)
 ↓
 Not secrete HCG.

* Choriocarcinoma ⇒ HCG

* Embryonal ⇒ HCG + AFP

* Dermoid ⇒ No Tumor Marker

↳ Rarely HCG secrete
 ↳ Rokitansky Protuberance / Tip of Geelberg sign
 ↓
 White Area Inside cyst (black) in USG.

* M/c ovarian tumor in Reproductive Age ⇒ Dermoid.
 " " " In ♀ ⇒ Dermoid
 " Cancer ⇒ dysgerminoma

* Dermoid ⇒ Benign
 Risk of Malignancy (0.2-2%)
 type / site
 Sq cell Ca / Rokitansky protuberance
 ↳ M/c ovarian tumor to goes in torsion.

* Germ cell cancer = Best prognosis \Rightarrow Dysgerminoma
" " " worst prognosis \Rightarrow EST / Yolk sac tumor

* Only ovarian tumor = is Radiosensitive

\Downarrow
Dysgerminoma (Moderately Radiosensitive)

© Sex cord Stromal tumor (3+)

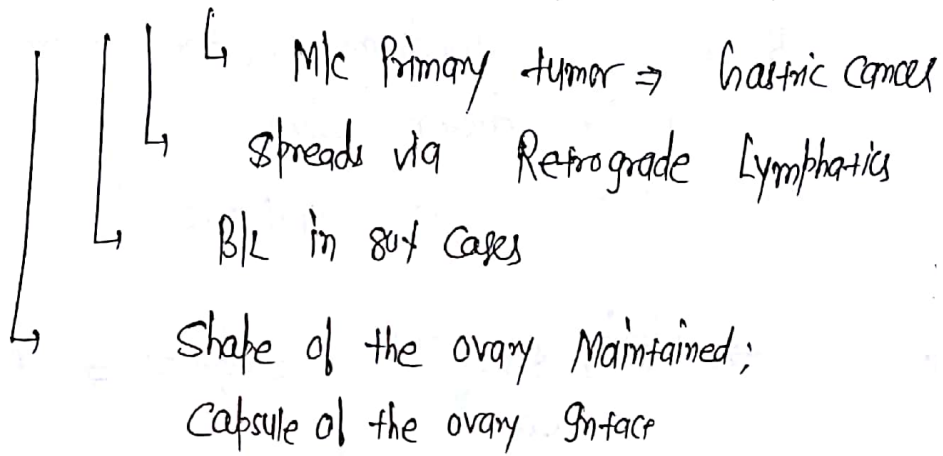
- Granulosa cell tumor
- Sertoli-Leydig cell tumor
- Leydig cell tumor
- Thecoma
- Fibroma (stromal tumor of ovary)

Features \Rightarrow , U/L; occur in all age gp / Peak incidence in Peri Menopausal women;

- Non specific sign/symptom,
- AUB \longrightarrow Estrogen
or
virilization \longrightarrow Testosterone
- early stage pick up
- don't show any Lymph Node Metastasis.
- Best prognosis:
- Granulosa cell tumor \longrightarrow Inhibin
 \hookrightarrow secrete estrogen \rightarrow Risk of endometrial ca.

- On HPE \Rightarrow
 - Call exner bodies (follicle) (173)
 - Rokitansky protuberance (also in dermoid)
 - Wall-thard cell Nest
 - coke-bean cells (also seen in Brenner tumor)
 - Reinke's crystal \rightarrow Leydig cell tumor
 - Schiller-dural bodies \rightarrow EST tumor
 - Psammoma bodies \rightarrow Serous cystadenoma of ovary

Krukenberg's tumor \Rightarrow Not a Primary tumor; Secondary tumor.



On HPE \Rightarrow Signet Ring cells*

* STAGING OF OVARIAN CANCER \rightarrow by FIGO; Surgical Staging.

- | | | | |
|---------|----------------|---------------|---------------------------------|
| Stage 1 | A | \rightarrow | U/L ovary |
| | B | \rightarrow | B/L ovary |
| | C ₁ | \rightarrow | Intra operative capsule Rupture |
| | C ₂ | \rightarrow | Pre operative capsule Rupture |
| | C ₃ | \rightarrow | Malignant Ascites |

- | | | | |
|---------|---|---------------|--|
| Stage 2 | A | \rightarrow | Cancer Spread to uterus / Fallopian tube |
| | B | \rightarrow | " " pelvic structures |

Stage 3

A $\begin{cases} 1 \\ 2 \end{cases} \Rightarrow \oplus$ Retroperitoneal Lymph Node
 \Rightarrow Microscopic extra pelvic peritoneal spread


B - Macroscopic ≤ 2 cm extra pelvic peritoneal spread

C - Macroscopic > 2 cm extra pelvic peritoneal spread

\hookrightarrow if there is involvement of capsule of liver/spleen

Stage 4

A \rightarrow Malignant pleural effusion

B \rightarrow  Parenchyma of the abdominal organ
distant spread
Inguinal Lymph Node

Stage 1/2 \Rightarrow early Stage Ca

Stage 3/4 \Rightarrow Advanced stage carcinoma

* For early diagnosis of ovarian Ca \Rightarrow TVS
or

K/C/O ovarian Ca \Rightarrow Best \Rightarrow CT-Scan

\hookrightarrow can't do staging \bar{c}
CT-Scan

* on Follow up; if CA 125 \uparrow

\hookrightarrow it Means Recurrence happen

do PET scan

Staging Laparotomy ⇒ Steps

(174)

i) Midline vertical incision

Never give Pfannenstiel incision or transverse incision; Rarely Paramedian incision given



ii) Ascites → Sample taken → Cytology for malignant cells
OR

Saline wash → 50-100ml → Cytology for malignant cells

iii) Inspection & Palpation of all abdominal organs

iv) Random Peritoneal biopsies



Paracolic gutters
Pouch of Douglas
Surface of diaphragm → Scrapping (acceptable)

v) TAH ± BSO (Pan hysterectomy)

↓
type 4 hysterectomy

vi) infracolic omentectomy.

vii) Pelvic & Paraaortic LN Sampling

viii) closure

* In Stage 3/4 \Rightarrow Primary T/t \Rightarrow Surgery
 \Downarrow
Debulking &

* Post-operative \Rightarrow Chemotherapy.

* conservative sx for ovarian cancer \Rightarrow U/L Salpingo-oophorectomy

\hookrightarrow eg \Rightarrow Germ cell tumor

(U/L; Younger age patient)

• Stage IA (if fertility is desired).

• Borderline epithelial ovarian tumor

\hookrightarrow epithelial ovarian potential \bar{c} Low

Malignant potential

(Stromal Invasion - Absent)

Very good prognosis

U/L

decade earlier

Post-operative chemotherapy

(175)

↳ Epithelial ⇒ all stages except ⇒ 1A & 1B graded cell ca
Need Post-op. chemotherapy,

Chemotherapeutic Agent ⇒

Carboplatin + Paclitaxel } 6 cyc.
↓
Cisplatin + Paclitaxel }
Given i/v + Peritoneal.

Germ cell ca } ⇒ All Stages Need chemo. except ⇒
sex cord ca } }
↓ } }
Dysgerminoma stages
II
No Need of chemotherapy

Only Advanced stages Need chemotherapy.

Chemo. Agent ⇒ BEP (Bleomycin, Etoposide, cisplatin).

ENDOMETRIAL CANCER

Etiopathogenesis ⇒ ↑ Estrogen (unopposed)
↑ Menstrual cycles

• RIF ⇒ Early Menarche;
Late Menopause;
PCOS;

- iv Infertility
- v obesity
- vi HTN
- vii Diabetes
- viii Tamoxifen → highest - 70%
- ix BRCA1, 2, HNPCC → Lynch Syndrome
 - ↳ also ovarian ca; Not of Breast Cancer
- x HRT (only Estrogen)

- Protective of Endometrial ca
- Smoking
 - OCP (↓ Risk by 60%)
(↓ Risk of ovarian ca by 50%)
 - Exercise
 - Green tea

<u>Histology</u>	⇒	<u>Type 1</u>	<u>Type 2</u>
		Endometrioid	Papillary; serous; clear cells
		Most common in 80% cases	In 20% cases
		Estrogen Responsive	Non Responsive
		Pregnant	⊗
		Good Prognosis	Poor prognosis (Worst prognosis clear cells)

Type I
Gene association => PTEN ← Gatekeeper Gene
KRAS

Type II
p53

(176)

Microsatellite deletions

High grade serous show p53 Mutation

Low grade serous show KRAS Mutation

Peri Menopause
early Menopause

Late More Age More type II

Obese Lady

thin Lady,

* Pre Invasive Lesion =>
which changes into the
endometrial Ca

Endometrial hyperplasia

↓
on HPE by taking endometrial Biopsy.

	Klau "cystic glandular hyperplasia"	Glands & stroma both proliferate
Simple hyperplasia	̄ Atypia	→ 1+
Complex II	"	→ 3+
Simple	̄ Atypia	→ 8+
Complex	̄ Atypia	→ 29+

Glandular Proliferation is Much more than ~~stromal~~ stromal prolifer

↓
Back to Back arrangement of gland.

Hyperplasia c/out Atypia

↳ given Progesterone 1stly

M/c ⇒ MPA X 6 Months
(daily)

⇓
Repeat Biopsy

also we DMPA
Mirena

Hyperplasia c Atypia

Reconfirm ← ↳ do Hysterectomy (TOC)
(Frozen section)

↳ Intraoperatively send & get a
Immediate Report & Reconfirm

⇓ if Not possible

do Endometrial sampling (Optional)
(FC + Hysteroscopy)
↳ Functional curettage

2nd Line ⇒ if wants Preserving the Uterus

Reconfirm on Repeat Endometrial sampling
(FC + hysteroscopy)

2ndly Progesterone (Prefer - Megestrol Acetate X 6 Months)
Other MPA
Mirena

* Simple hyperplasia
 Cystic glandular hyperplasia → Metrorrhica
 Hemorrhagica (177)

Age ⇒ 40-45yr

8 weeks of Amenorrhoea ⇒ History of bleeding
 (Anovulatory cycles
 b/c Unopposed Estrogen) ↓ Painless

Endometrium ⇒ Absence of secretory pattern
 on HPE

Ovary ⇒ Swiss cheese appearance
 cyst

Ca ⇒ 1%

TOC ⇒ Progesterone

* Peak age for endometrial cancer ⇒ 60yr (5-7th decade)

M/c presentation / 1st presentation = Irregular vaginal bleeding

Most specific presentation = Post Menopausal bleeding (PMB)

f. of PMB have endometrial Ca = 10%

M/c cause ⇒ Senile endometrial Atrophy
 (Atrophic endometritis)

M/c cause of PMB in India ⇒ Ca cervix

* M/c Cause of Pyometra \Rightarrow Endometrial ca
in India \Rightarrow cervical ca

* M/c Route for endometrial ca \Rightarrow Direct spread
" " ovarian ca \Rightarrow Tumor Exfoliation

* No Routine screening done for endometrial ca

\downarrow
if pt. is K/c/o "HNPC" - do Routine screening

\hookrightarrow age ≥ 35 yr

do b/nctional curettage (FC)

6 monthly / 12 monthly.

* Any women comes \bar{c} Post Menopausal bleeding

\hookrightarrow Rule out Endometrial ca

* if women ≥ 45 yr \subseteq AUB

\hookrightarrow Rule out Endometrial ca

Next Step

do TVS (see endometrial thickness)

\Downarrow

QC. to ACOG if thickness ≥ 4 mm \rightarrow high Risk for cancer

AIIMS May 18

" American
Radiological

"

≥ 5 mm

cancer

ib endometrial thickness < 4mm / < 5mm
(ACOH) / (American Rodology) (178)
↳ No Tes In Risk of Cancer

* IOC =>

Endometrial Biopsy

* TVs do Polyp

to Rule out other pathologies like fibroids

O.P.D Procedure; Aspiration do
(Endometrial Aspiration cytology)

↓
we use "KARMAN'S CANNULA"

Other device "PIPELLE DEVICE"

"VATIRA ASPIRATOR"

* Gold Standard
technique to Rule out
endometrial ca

↓
Most Invasive

⇒ Functional curettage + Hysteroscopy
May (D&C) use synonym = Functional curettage
O.T. + Anesthesia Needed

→ 1st ly do cervical curettage - (Endocervical curettage)

↓ then
dilate Internals

↓ then
Endometrial curettage

ib 1st ly do endometrial cells comes in cervix.

Condition where Functional curettage done

- i) Endometrial biopsy — Benign ; while Symptom Persistent
- ii) Endometrial biopsy — No Endometrial cells seen
- iii) Hyperplasia \bar{c} Atypia
(preserve the uterus)
- iv) Cervix Stenosed

* Should we do PAP Smears in Post Menopausal bleeding \Rightarrow Yes.

Staging \rightarrow FIGO \rightarrow Surgical Staging

Stage 1 $\left\{ \begin{array}{l} A - \text{Only Endometrium or } < 50\% \text{ of Myometrium} \\ B - > 50\% \text{ of Myometrium} \end{array} \right.$
Intraoperatively cut open the uterus

Stage 2 — cervical spread

\Downarrow
only if cervical stroma is involved

Stage 3 $\left\{ \begin{array}{l} A \rightarrow \text{Cancer spread to Serosa/Adnexa} \\ B \rightarrow \text{'' Vagina/Parametrium} \\ C_1 \rightarrow \text{+ve Pelvic LN} \\ C_2 \rightarrow \text{+ve Para-aortic LN} \end{array} \right.$

Stagey A \Rightarrow Spread to Bladder and/or Rectum (179)
 B \Rightarrow Distant spread / Inguinal L.N.

Staging Laparotomy

\hookrightarrow Omentectomy is Not done Routinely

\hookrightarrow for type 2 Endometrial ca.

do TAH \bar{c} BSO

\hookrightarrow type 1 — Stage 2 — Type 3 hysterectomy
 \Downarrow
 Cervical spread

- Pelvic & Paraaortic LN sampling \Rightarrow Type 2 cancers L.N. dissection

- Stage 3/4 \Rightarrow Advance stage Ca

\hookrightarrow Primary Tx \Rightarrow Debulking surgery

Post-operatively

Low Risk

Intermediate Risk

High Risk

- all three
- a) Endometrioid
- b) Grade 1
- c) only endometrium

In b/w Low & high Risk

- a) Stage 3/4 disease OR
- b) type 2 cancer

Tt of Low Risk → Need No Post-operative Tt;
do Follow up

Tt of Intermediate Risk → Pelvic Radiotherapy

Tt of high Risk → Chemotherapy + Radiotherapy
↓
(cisplatin + Paclitaxel)

PROLAPSE → Recent classification
↳ POP-Q classification
↳ Pelvic organ prolapse
quantification
↓
Reference → Hymen.

* Delancey Level of Support

Level I — Uterosacral Ligament + Cardinal Ligament
↓ ↓
Weak ^{cases} → Uterocervical descent

[earlier classified as

1st degree → descent above vaginal opening

2nd degree → Uterus descent @ the level of vaginal opening

3rd degree → Uterus descent outside the vagina

Procidentia → Uterus completely outside the vagina

↳ Fundus is also outside

Apical Prolapse

Enterocoele → Prolapse of GUT into Pouch of Douglas.

Vault Prolapse (apex of vagina)

(Post hysterectomy*)

Uterocervical descent

Level 2 ⇒ Paravaginal tissue & their attachment
basia covering Levator Ani (180)

↳ weak $\xrightarrow{\text{causes}}$ Cystocele (upper 2/3rd Anterior wall)
↳ M/c type of Prolapse

Level 3 ⇒ Perineal body & Muscle attach to it

↳ weakness $\xrightarrow{\text{causes}}$ Rectocele (Post. vaginal wall)
↳ LEVATOR ANI MUSCLE;
E ⇒ EXTERNAL ANA SPHINCTER
S ⇒ SUPERFICIAL TRANSVERSE PERINEAL MUSCLE
S ⇒ SPHINCTER URETHRAE
D ⇒ DEEP TRANSVERSE PERINEAL MUSCLE

* Prolapse is disease of older Age.

RIF of Prolapse ⇒ i) No. of deliveries & process deliveries
(Prolonged Labour)
Instrument delivery
Episiotomy "
Perineal tear
obstructed Labour

ii) Old Age (d/t loss of estrogen)

* Younger age women also have Prolapse of uterus by \rightarrow
May

- i) Connective tissue d/o.
- ii) Congenital elongation of cx
- iii) Spinal cord injury

* Smoking ↑ Risk of Prolapse

* Intra abdominal Pressure ↑ ⇒ ↑ Risk of Prolapse

↳ Chronic cough; Ascites.

Case I ⇒ Old woman P₃L₃; 3rd degree Uterocervical descent
+ Cystocele + Rectocele

Uterocervical descent → do Vaginal hysterectomy

Sequence of cutting & clamping the structure in TAH

↓

Reverse the sequence of vaginal hysterectomy

Sequence of cutting & clamping of sequence

Firstly Uterosacral Ligament

↓

Cardinal Ligament

↓

Uterine Artery

↓

Utero ovarian Pedicle

* Sequence of cutting & clamping the structure in TAH & BSO ⇒

Uterosacral Ligament

→

cardinal Ligament

→

Uterine Artery

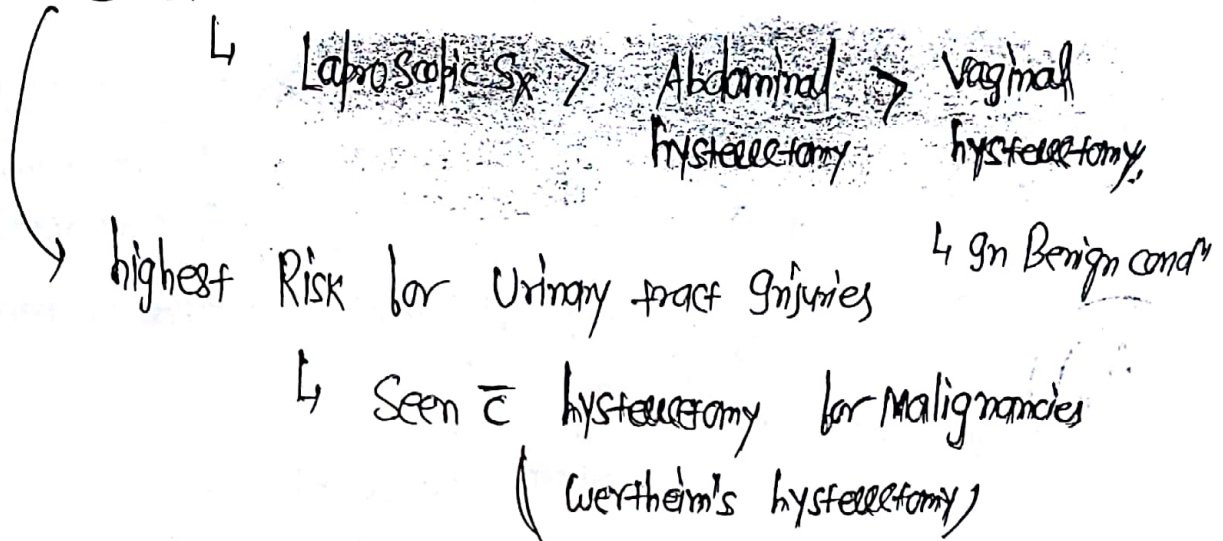
Infundibular Ligament

↓

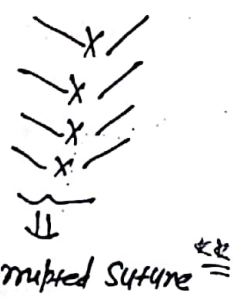
Utero ovarian Pedicle

* Usually we prefer vaginal hysterectomy; but
 it is difficult in — Uterine size > 12wk
 obese
 Pelvic Adhesions.

* Urinary Tract Injuries

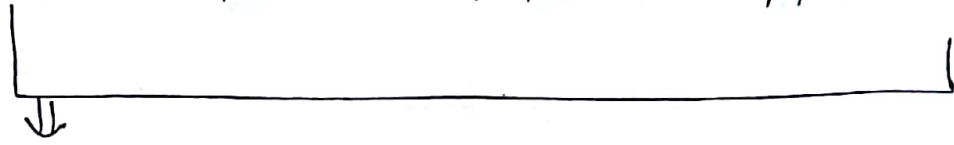


* Rx of cystocele → Anterior colporrhaphy
 (Vagina)



Rx of Rectocele → Posterior colporrhaphy
 ↓
 Strengthen the Levator Ani Muscle

VH + Ant. colpo + Posterior colpo/epineorrhaphy



VH + Pelvic Floor Repair

+ Enterocoele Sx



ward Mayo Sx**

do prophylaxis; also do

for vault + Prolapse



McCalls culdoplasty

↳ vaginal Repair

Other surgeries for Enterocoele ⇒ Moschowitz Repair

Both are Abdominal Repair; Not used Now a day

Halban's Repair

Support the vaginal vault =

Uterosacral Ligament in McCalls culdoplasty.

2nd case

Younger - Reproductive Age group

+ Prolapse

(Uterocervical descent)

TOC ⇒

** Sling's Surgery

↳ can't do hysterectomy

↳ Modified Shirodkar's abdominal Surgeries

Anterior sling

(if we tie's at anterior aspect of Gstramus)

Posterior sling

(if we tied at posterior aspect of Gstramus)

* Pyramidal Sx

↳ Autologous (tendon fascia lata) and - Abdominis external oblique muscle

* Khamna's Sling.

Khamma's sling

(182)

↳ one end at posterior aspect of isthmus;
other end Anterior Superior iliac spine.

Shriodkar sling ⇒ Posterior sling

↳ Merselene tape used
↳ on the left hand side first pass through
Psoas Muscle (Form Psoas hook) then goes to Sacrum

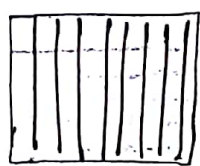


↳ to prevent compression & obstruction
of sigmoid colon / Rectum.

VikRud's sling ⇒ Composite sling
(Anterior + Posterior sling)

Abdomino cervicopexy ⇒ For putting Mesh we have to
clear Pre-Sacral Area; technically
difficult; but Results are bett.
↳ higher complication

Mesh

* Non Sling Surgery ⇒ Manchester Repair

(Klas" Fothergill's Surgery)
↳ * done in Reproductive age women who completed child
bearing.

- also done in congenital Cx Elongation
UCL (Uterocervical Length) Estimation

- Process \Rightarrow
of Manchester
Repair

\downarrow
D & C (to prevent the complication of
Cervical stenosis)

\downarrow
Cervical Amputation

\downarrow
Reattach Cardinal Ligament anteriorly

\downarrow
Cover the Cervix \rightarrow \bar{c} vaginal Mucosa

Cystocele done

Rectocele Repair done

if the UCL tes ; don't do sling operation ; do
Manchester Repair

UCL = Uterocervical Length

(N) UCL Length \Rightarrow 6 to 8 cm.

* Shriodkar Modification of Manchester

\downarrow
L Can be done even if women want
to conceive.

\downarrow
All steps are same in it except there is no cervical
Amputation in it

3rd case

old woman + Co-morbidities

(183)

PAC
(Pre-Anesthetic
Check-up)

↳ bit for short sx.*

Latzko colpocleisis



done for vesico-vaginal
Fistula.

Rx ⇒ Lefort's colpocleisis

Denude Mucosa - Anterior vaginal wall

Denude Mucosa - Posterior vaginal wall

Means scrape
Mucosa from Anterior
& Posterior vaginal
wall

1stly we Rule out Cx Cancer & Endometrial
Cancer

& Patient can't be sexually active

• Done Under Local Anesthesia,

colpocleisis ⇒ closure of vagina

4th case

Co-Morbidities + old woman

↳ PAC ⇒ Unfit for short sx.

Rx ⇒

Pessary


→ space occupying device
- Insert into vagina

Gellhorn
Pessary



Doughnut
Pessary

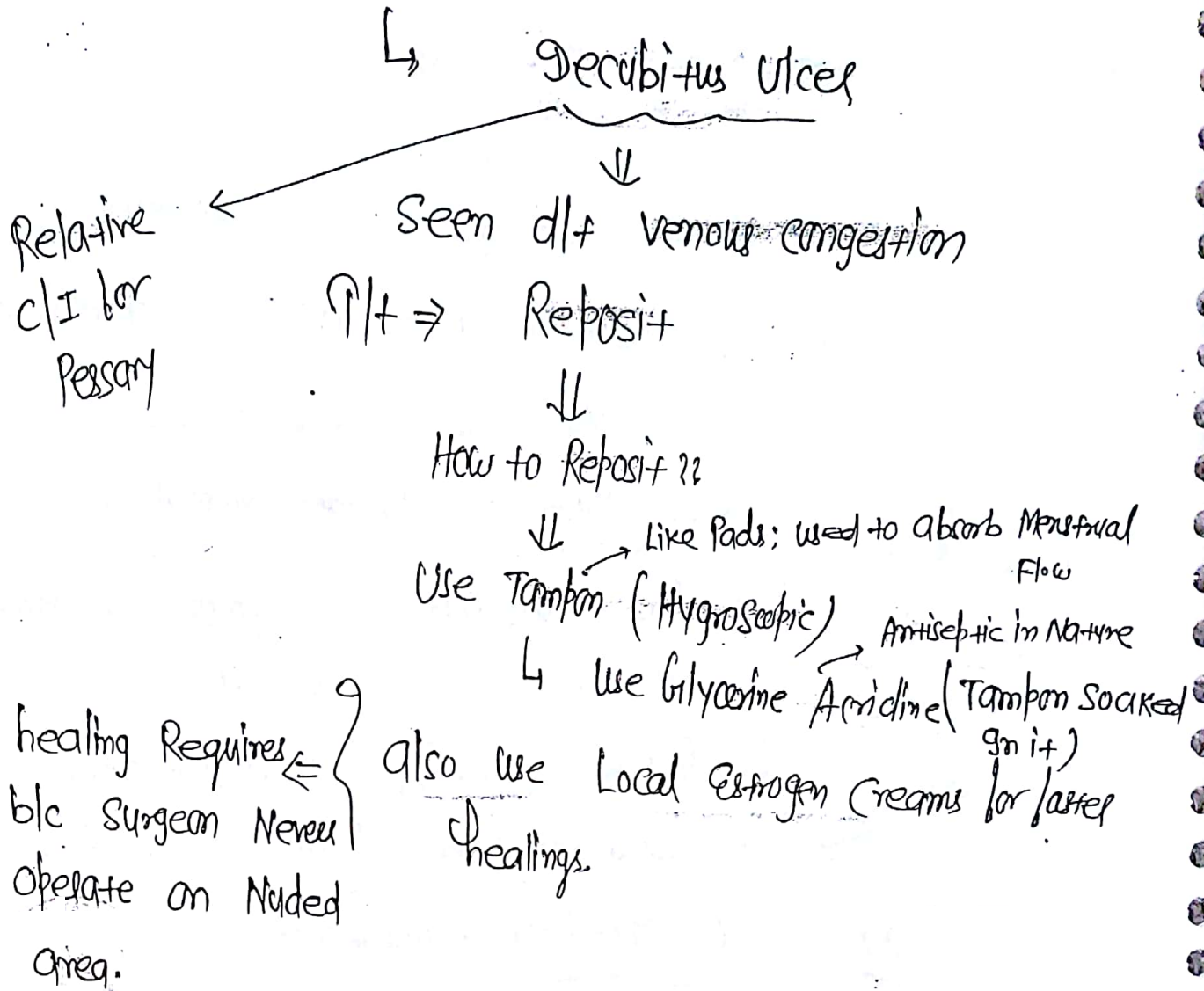


* Early  prolapse - also indication of Pessary

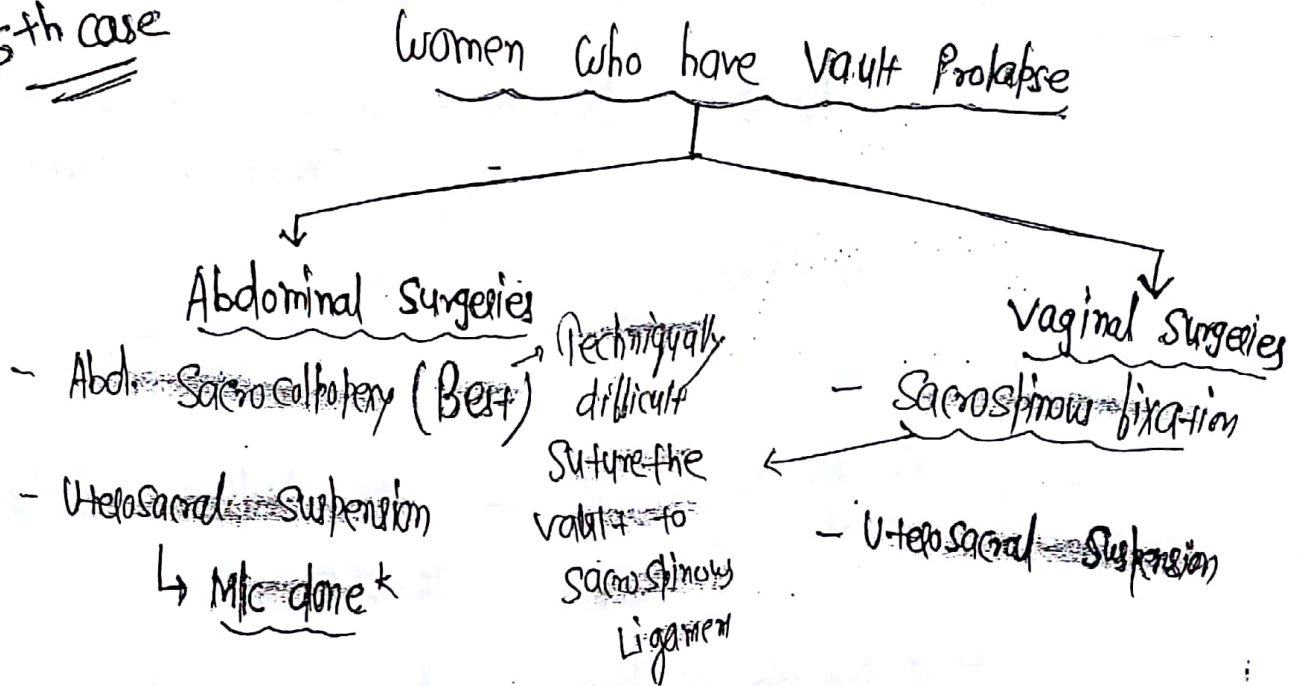
↳ Remove - 16-18 weeks.

* Other indication of Pessary - Puerperium

* Ulcer on Prolapse part

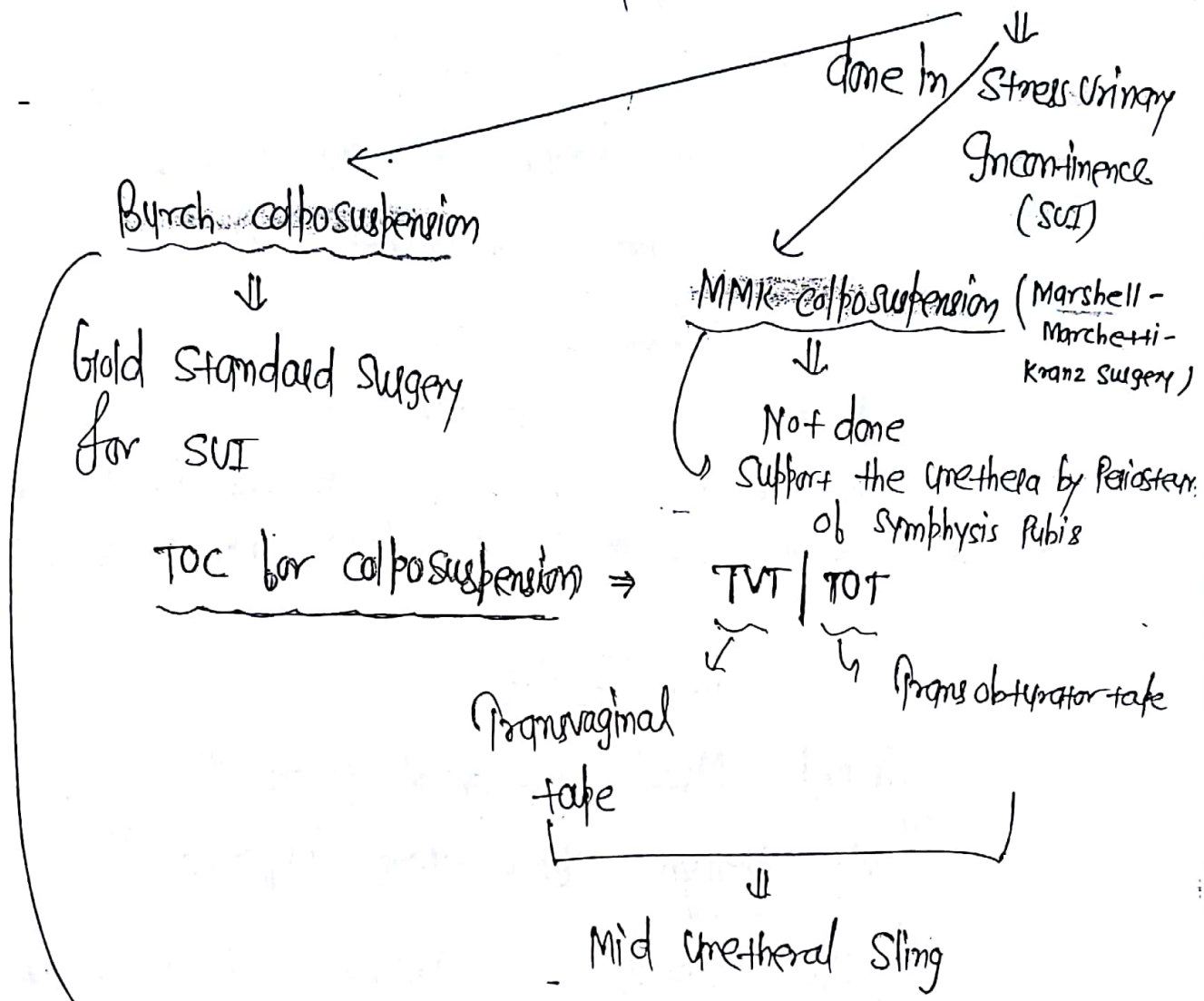


5th case



* Lebortte Colpocleisis can also done in vault prolapse (184)
↳ In short time sx.

* all all sx of vault prolapse except Colposuspension:



Kind of Abdominal Surgery
- Support the proximal urethra by Cooper's Ligament.

- ↳ They are as good as Burch colposuspension
- ↳ Day-care sx;
- ↳ Lesser complication;
- ↳ vaginal surgeries;

* Trans vaginal tape \Rightarrow Reproductive Surgery

Trans obturator tape \Rightarrow Not Reproductive Surgery

\hookrightarrow In it we enter the Space of Retzius

\Downarrow

So; More complication than TOT, 'so'
We prefer TOT Now a days.

* Pt \bar{c} Vault prolapse + SUI??

\Downarrow

Abdominal Sacrocolpopexy

Rx \Rightarrow Burch colposuspension.

* 1st Line Mx of SUI \Rightarrow Kegel's exercise i.e Pelvic floor exercise

* Drug Rx of SUI \Rightarrow DULOXITENE (only drug Rx of SUI).

FIBROID

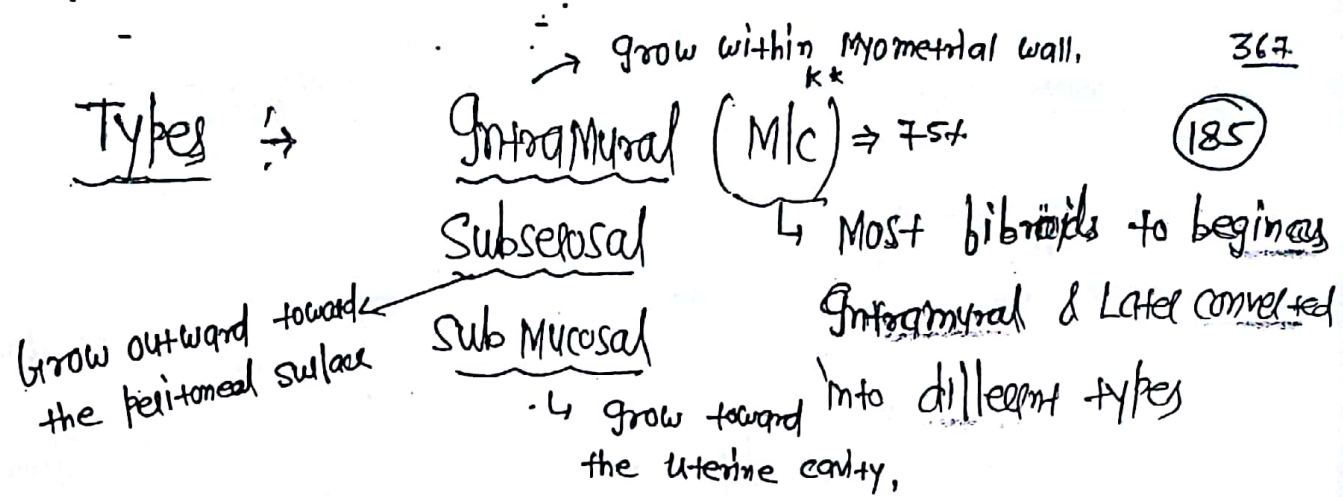
- Smooth Muscle tumor of Uterus ⁹⁹

- M/c benign Pelvic tumor in females. ⁹⁹

- a/w Estrogen; so; Seen in Reproductive Age
in > 35 yr women; It is More Common.

- In Postmenopausal women \rightarrow Regression seen.

- In Pregnancy — Most of the fibroid don't enlarge



Hysteroscopic appearance (Wansteckers classification).

- ↳ Type 0 → Completely Intra cavity
- Type 1 → > 50% Intra cavity i.e < 50% in Myometrium
- Type 2 → < 50% Intra cavity i.e > 50% in Myometrium
- ↳ can't be removed hysteroscopically

Mlc presentation ⇒ Asymptomatic

Mlc Symptom ⇒ ① Bleeding → Menorrhagia

↓

Cycles — Regular

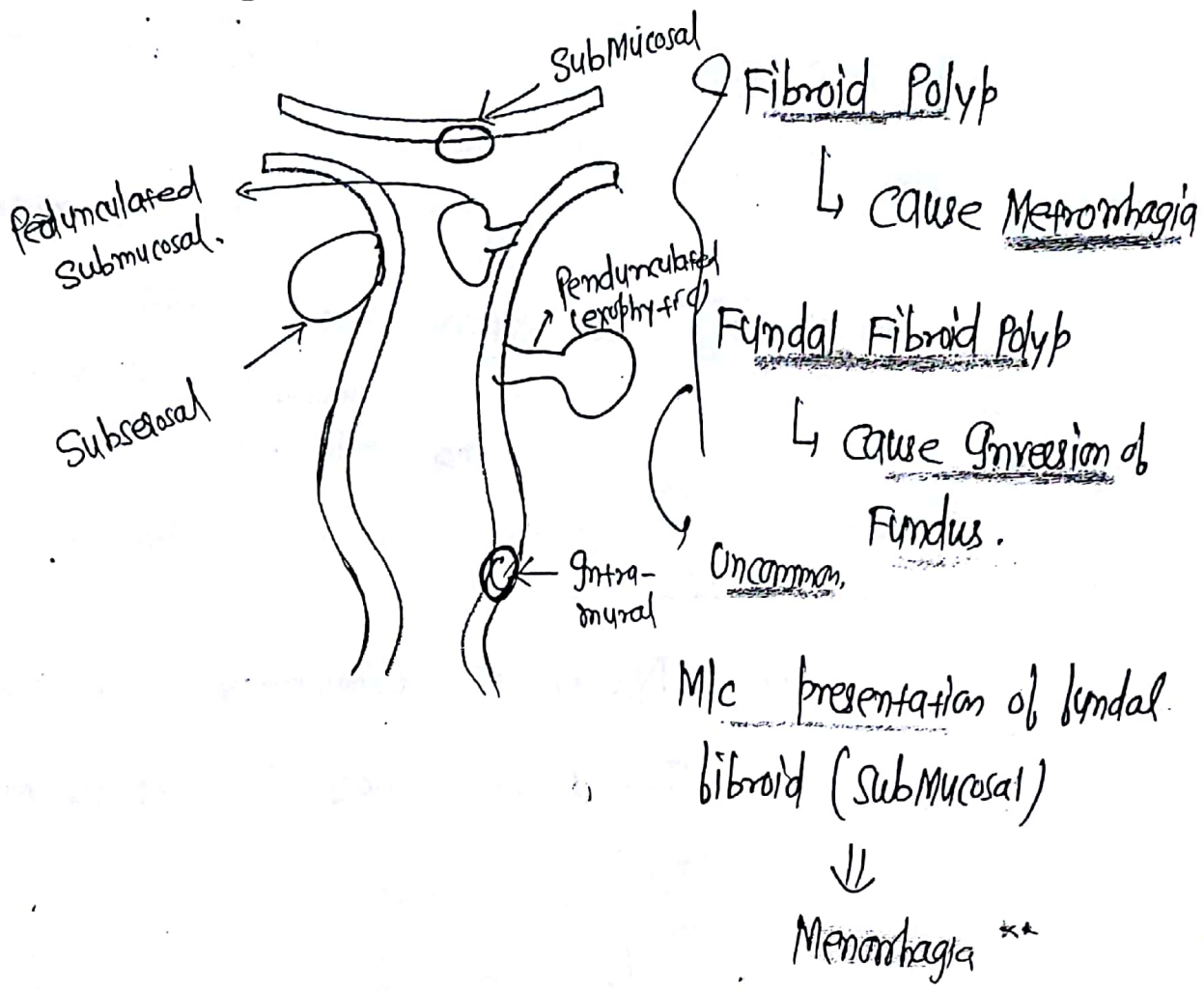
Flow — ↑↑↑

Mlc cause by which fibroid

↓

Submucosal

While Subserosal fibroid Not a/w bleeding



② Pain → if it undergoes - degeneration

Torsion - Pedunculated Subserosa

Mlc degeneration ⇒ Hyaline ***

Least Common " ⇒ Sarcomatous **

Rare Malignant change

↳ < 0.5%

↳ Postmenopausal fibroid enlarge & pain.

* Womb Stone - Subserosal fibroid Stone; (186)
 ↳ Old bladder stone ↳ calcification
 ↳ it is eccentric ↳ differential diagnosis

* Red degeneration ⇒ Seen in ♀

- Stained salmon pink^a or Red
 - Fishy odor^a
 - Histologically: evidence of thrombosis in some vessels^a
- M/C in 2nd Trimester.
 - presenting as acute Abdomen (Pain + Nausea; vomiting ± Fever)

- Mx - Conservative
 N.P.O + iv fluids + analgesic

aseptic condⁿ ⇒ No Role of Antibiotic **
 ↳ Fever is only Reactionary

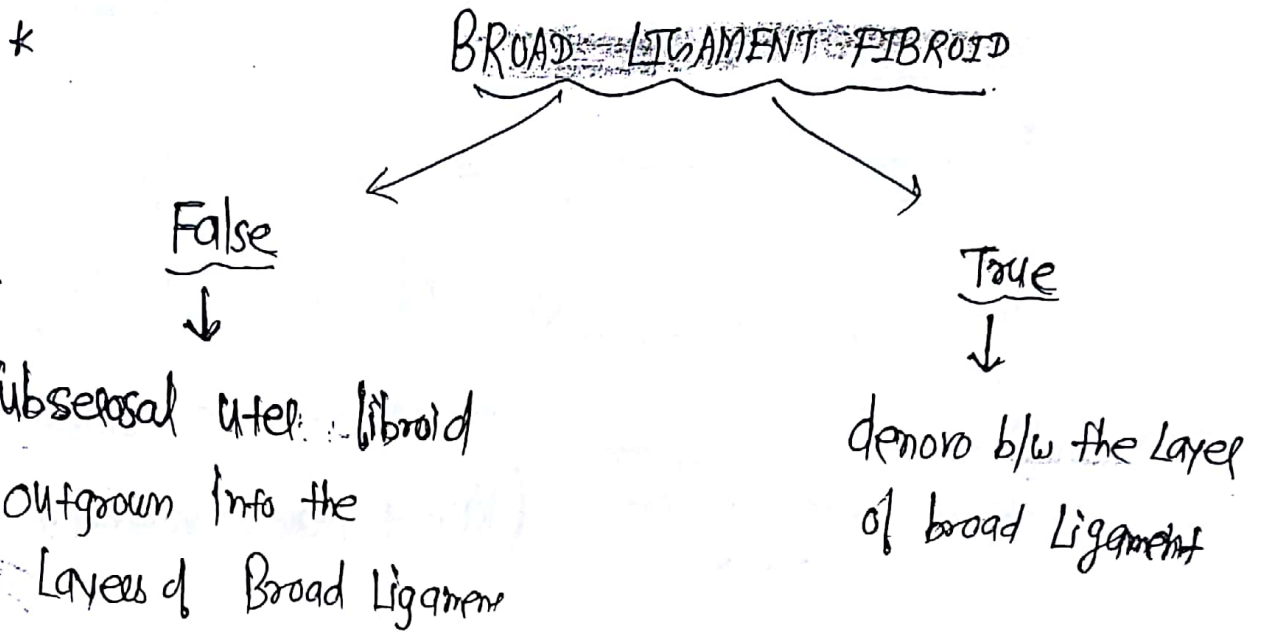
* In Fibroid ⇒ dysmenorrhoea ⊕; but Not a primary complaint.

* Fibroid doesn't cause dyspareunia

Fibroid May show pressure symptom

↳ Ant wall Fibroid ⇒ Urinary symptom
 ↳ Post wall Fibroid ⇒ Bowel symptom **

- * Ant. wall fibroid cause \Rightarrow \uparrow Frequency of Micturition
- Post. " " " \Rightarrow Urinary Retention



Lateral to the Fibroid \leftarrow Uterus \rightarrow Medial to the Fibroid.

- * Fibroid also prof. \bar{c} Infertility } Submucosal Fibroid
Recurrent @ Loss } only **

- * Pregnancy complication of Fibroid
 - abrupt loss RPL & Red degeneration \rightarrow
 - Abortion
 - Pre-term Labour
 - Malpresentation
 - dysfunctional Labor
 - PPH

IOC \Rightarrow USG (Hypoechoic)

↳ Well-circumscribed Masses;
Pseudocapsule

Small submucosal fibroid may miss in USG

↓

Best Ix \Rightarrow Hysteroscopy

2nd Best \Rightarrow SIS (Saline Infusion Sonography)

* Don't do MRI \Rightarrow Routinely

↓

do in pre-op. condⁿ to know

- No. size
- Location

* Diff Fibroid

- Smooth Muscle tumor
- > 35yr age
- Menorrhagia
- Non tender
- Irregular growth
(Lumpy Bumpy growth)*
- Grows upto uterus sized
20week

Adenomyosis

Endometrial glands & stroma
Inside Myometrium

40-45yr age

Menorrhagia + dysmenorrhoea

Tender (Half hen size)

Symmetrical Growth
(Globular)*

10-12 week

Fibroid

GOC ⇒

USG

alternate dark & light band

Adenomyosis

MRI

On USG we see ⇒

Salt + pepper appearance

Venation blind "

Myometrial cyst

Subendometrial cyst

Poorly defined Junctional zone

* Degenerations / Secondary changes in Fibroid

↳ Mnemonic

- 4 A void → Atrophy;
- Red → Red degeneration;
- Hot → Hyaline degeneration (Mlc)
- Fatty → Fatty degeneration or calcification
- Meat of → Myxomatous degeneration
- chicken → Cystic degeneration.

don't tell about where endometrium starts & Myometrium ends

On MRI ⇒ Junctional zone thickness

> 12mm ⇒ Likely adenomyosis

< 8mm ⇒ Unlikely "

For confirming ⇒ (HPE) - Postoperatively Adenomyosis

See depth of these endometrial glands in Myometrium

At least 1 HPF deep to the Junctional zone

TOC ⇒ Hysterectomy *

Tt for Fibroid ⇒

Medical

Surgical

Minimally Invasive (Not Responsive to Medical Rx; Not willing for surgical)

Bleeding

(don't Reduce the size)

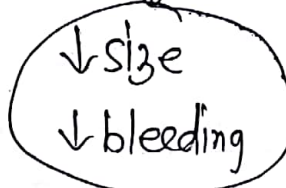
Doc →

OCP

Mirena

Tranexa

↓ less blood loss only.



↳ GnRH Analogue (continuous)

conceive

if stop

↳ Regrow fibroid

We don't use GnRH Analogue for long time

Used in pre-operatively to ↓ less Intraoperative blood loss

↓ before 6 month - GnRH starts

Fibroid → Regress

Intraoperative bleed ↓ less



(Sx Not prefer

to give 6 month

planes are lost

back to pre-op.)

GnRH against



Surgical planes are easily bind

Drugs that decrease size of Fibroid ⇒

U → Ulipristal

Are → Aromatase Inhibitor

Gynae → GnRH Agonist/Antagonist

M → Mifepristone

D → Danazol

ii) GnRH Antagonist

iii) Mifepristone } Progesterone (R)

iv) Ulipristal } Modulator

(Wants to conceive)
↳ don't give

↳ Aromatase Inhibitor - Anastrozole

(S/E - Severe Hypoestrogenic)

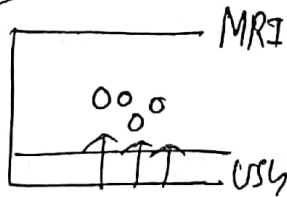
• Minimally Invasive

→ Uterine artery embolization
UAE → Embolization
↳ do if family is complete

MRg HIFU

• Rapid Symptom Improvement

• Fibroid Keep Shrinkage



Magnetic Resonance
Guided High Intensity
Focused USG

do if only family is complete;
expensive

* Surgical Pt

Radical

• Hysterectomy
Indication for TAH → Fibroid ***

Conservative

Myolysis
↓
Cryo Laser

Submucosal (Arboreo)
Myomectomy
↓
Hysteroscopic
Laparoscopic
others

* Gn Laproscopic hysterectomy

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↳ Slightly higher Recurrence Rate

↳ Not Statistically significant.

* Laparoscopy is better than Abdominal in all except \Rightarrow Recurrence

* Mlc Side effect of Hyster. scopy \Rightarrow Uterine Perforation
fluid overload

* Myomectomy \Rightarrow It is enucleation of Myomata from the Uterus leaving behind a potentially functioning organ capable of Future Reproduction

INFECTIONS

Bacterial vaginosis

Candida

Trichomonas

- all causing vaginal discharge (vaginits)

- Mlc - Bacterial vaginosis > Candida > Trichomonas

- Gardnerella vaginalis
(Mycoplasma Ureaplasma Mobilinac)
Doderline Replaced by Gardnerella
Candida albicans
T. vaginalis

after passing urine; Pain left
blc of excitation of skin

- Foul Smelling discharge
itching (X)
Dyspareunia (X)
Urinary symptoms (X)

- Pruritis
- Discharge may present
- Urinary symptoms (X) (SPLASH DYSURIA)

- Discharge
 \pm Dyspareunia
 \pm urinary symptoms
 \pm pruritis

Discharge \Rightarrow Foul Smelling
Off white / Greyish

Curdy
White

Green
(Yellow)

pH > 4.5

< 4.5

> 4.5

- IOC - Saline Microscopy

Clue cells \oplus

Pseudohyphae

organism

- Ratio of Poly Morpho-Nuclear cells
epithelial cells
 < 1

See itself
 \downarrow
dit + flagella

- Gold standard - Gram Staining

Nugent Score $\Rightarrow 7-10$

Culture has No Role in
Bacterial vaginosis

Culture in
SDA Media

Culture in
Stuart's medium

- Amsel's criteria

if 3 out of 4 - Bacterial vaginosis

Splash
dysuria

Strawberry
cervix

i) Foul Smelling (off/white)
discharge

\downarrow
Painful Urination
due to exfoliation of vulva

(Punctate Hemorrhage)

ii) pH > 4.5

Whiff test - \ominus

Whiff test - \oplus

iii) clue cells (at least 20% of
the epithelial cells)

iv) whiff test (addition 10% KOH to D/S \rightarrow Fishy Amine odour)

Not a STD

Usually

STD

Not

STD

Partner Not
treated

Usually Not

Yes

(done if partner
is symptomatic)

**
Doc \Rightarrow Metronidazole
 \downarrow
Clindamycin

Single dose
bicomazole

Metronidazole

Can cause Pre-term**
Labour in ♀

Recurrent
Vulvo vaginal candidiasis
 \Downarrow
 ≥ 4 episodes/year**

PID (Pelvic Inflammatory Disease)

Infection of upper genital tract
(uterus | Fallopian tube | ovaries)

M/c organism \Rightarrow Chlamydia
+ Gonorrhoea

age group \Rightarrow 15-25yr

highest R/F \Rightarrow Multiple sexual partners.

PID in virgin girl \Rightarrow d/f Tuberculosis

PID in GUD users \Rightarrow d/f Actinomyces

* Clinical diagnosis \Rightarrow

\hookrightarrow Pain Lower abdomen & Any of the following

a) Uterine tenderness

b) Cx Motion //

c) Adnexal //

Ix \Rightarrow * USG \Rightarrow Cog wheel sign; Beads on string sign; Waist sign

Endometrial Bx \rightarrow Plasma cell endometritis

\downarrow
Chronic endometritis

\downarrow
IIT of chlamydia

Laparoscopy \rightarrow Best (Gold standard)

\hookrightarrow FNA - High curis Sr.

FIB - Hugh Curtis Syndrome

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↳ Perihepatitis

Violin string Adhesions b/w Liver & Anterior
Abdominal wall

Rt. upper Quadrant Pain

Liver enzyme (N)

M/c caused by ⇒ Chlamydia & Gonorrhoea

* Long term complication of PID ⇒

↳ Infertility;

↳ Ectopic ♀;

↳ Chronic Pelvic Pain;

↳ Recurrent PID;

↳ Hydrosalpinx.

CONTRACEPTION

*

Methods of contraception

TEMPORARY METHODS

(Used to postpone pregnancy or space birth)

- BARRIER METHOD ;
- NATURAL CONTRACEPTION ;
- OCPs ;
- Injectables
- Implants
- IUCDs

PERMANANT METHODS

(Aim is to purposefully & Permanently destroy the Reproductive capacity of an individual).

Female

Tubectomy

Male

Vasectomy

* OCPs

* on the basis of Amount of estrogen; they can be classified

as → i) Low dose Pills : $< 50 \mu\text{cgm}$ (Avg: $35 \mu\text{cg}$) Ethinyl estradiol

ii) High dose Pills : $\geq 50 \mu\text{cg}$ (Ethinyl Estradiol)

iii) Very Low dose Pills : $\leq 20 \mu\text{cg}$ (Ethinyl Estradiol)

iv) Lowest Possible Pills : $10 \mu\text{cg}$ (Ethinyl Estradiol)

(Lo~~10~~ Estrogen)

* Mala D & Mala N

↳ both have $30 \mu\text{cg}$ ethinyl Estradiol + 0.15mg Levonorgestrel (LNG)

↳ Both have 21 hormonal tablets & 7 benzoin bumurate tablets;

Mala D available @ a cost of 2 Rupees ; while Mala N free of cost by govt of India

M.O.A of OCP → Mainly :

Inhibition of ovulation.

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* M/c side effect of OCP :

Breakthrough bleeding

* In Anovulatory DUB



Estrogen breakthrough bleeding



Progesterone breakthrough bleeding.

* If a Lady Misses 1 pill; take 2 pills the following doses; if she misses 2 pills → Backup Method of contraception

* In the event of Missing a pill →

Take the Most Recent Missed pill Immediately; use condoms for 7 days & continue the packet

Now if

≥ 7 pills are Remaining in packet

Finish the Remaining tablet & Start the New packet after 7 days gap.

< 7 pills are Remaining in packet

Finish the Remaining Pills & Start the New packet Next day without a 7 days gap.

Q: A women who is taking combined ocp misses 2 consecutive pills. There are 10 pills remaining in the packet. Next pill is

a) Take both pills immediately continue the packet & use condom for 7 days

b) Take the Most Recent Missed pill immediately; continue the packet & use condom for 7 days.

c) Take the Most Recent Missed pill immediately; continue the packet; use condom for 7 days & commence the next packet after a 7 days gap

* Fertility Return \Rightarrow \bar{c} (in 3 Months of withdrawal of the drugs in 90% cases)
 \downarrow
(Ovulation Return)

* Which contraception have least chance of Ectopic \Rightarrow OCP

* OCP also ↓ Risk of PID.

But \uparrow Risk of candida & chlamydia are seen by combined oral contraceptives

* Other M.O.A. of OCPs \Rightarrow Prevention of Fertilization;
Interference \bar{c} Implantation;

* OCP & Cancers \Rightarrow

• OCP & Cervical cancer \rightarrow Yes;

• Ovarian cancer \downarrow by 50%;

• Endometrial cancer \downarrow by 60%;

• Colon cancer \downarrow risk;

• Breast cancer : No Yes Risk

\hookrightarrow OCPs are protective against benign breast disease;
but Role of OCPs are controversial in ca of breast,

• Using HRT (Hormone Replacement Therapy) Yes chance of breast cancer.

• Hepatic Adenoma : Yes Risk;

• HCC : No Yes Risk (Not decrease Risk also);

• Gallbladder cancer : No Yes Risk

* Absolute c/I of OCP (WHO category 4) ⇒

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Mnemonics

↳

Banks → K/clo Breast Cancer

Have → Severe Hypertriglyceridemia / Hypercholesterolemia

Various → Undiagnosed Vaginal bleeding

Scheme → Stroke ; Smoker over age of 35 years.

To → Thrombophlebitis | Thromboembolic disorder

Provide → Pregnancy

Home → Uncontrolled Hypertension ($\geq \frac{160}{110}$ mmHg)

Looms → Acute Liver disease (Hepatitis; Cirrhosis)

During → Dialysis & Vasculopathy
↳ NOT c/I of OCP.

May → Migraine & Aura (i.e Focal Neurological deficit).

also; Coronary Artery Disease is absolute c/I of OCP.

* Newly Married couple: choice of contraception: OCP

Living far apart; Meeting occasionally

↳ Barrier Method

* Safety: Which contraception is safest: "Barrier"

↓

S/E ⇒ very high failure Rate

∴ Pregnancy is S/E

PROGESTERIN ONLY PILLS (POP)

aka "Minipills" (75mcg Progesterone)

M.O.A. : Thickening cervical Mucus

* Should be taken on same-time Everyday

↓

(Safe Margin < 3hrs)

↓

If delay was for > 3hrs - Back Method should be used

* POPs are contraception of choice in Lactating Female

↓

↳ can be started immediately after delivery, POP > GUD

* contraception of choice in Lactating's Female : POP > GUD

↳ but Not Lactating Amenorrhoea

↳ b/c high failure rate

* Minipill available in India ⇒ "CERAZETTE"

↓

M.O.A. ⇒ Mainly Agaim

↳ Inhibition of ovulation

Safe Margin : 12hrs

(A delay of ~ 12hrs can be accepted)

* M/c side effect of POP : Irregular vaginal bleeding.

* Absolute C/I ⇒

i) Pregnancy;

ii) Undiagnosed vaginal bleeding;

iii) K/c/o Breast Cancer

} Same for all

PROGESTERONE INJECTION

DMPA (Depot Medroxyprogesterone Acetate)

385
194
Net En (Norethisterone enanthate)

Dose \Rightarrow 150 mg i/m

200 mg i/m

To be Repeated every 3 Months

Repeated every 2 Months

Pt. can wait upto 4 weeks late for Next Injection

Pt. can wait up to 2 weeks Late

* DMPA \Rightarrow 2 Good things \Rightarrow i) Useful in Female \bar{c} Epilepsy
 \uparrow Seizure threshold;
ii) Reducing Sickling crisis: Useful in Sickle cell Anemia;

2 Bad things \Rightarrow i) Significant Bone loss;
ii) Delay in Return of Fertility;
Avg. delay: 12 Months
Max^m. delay: 18 Months.

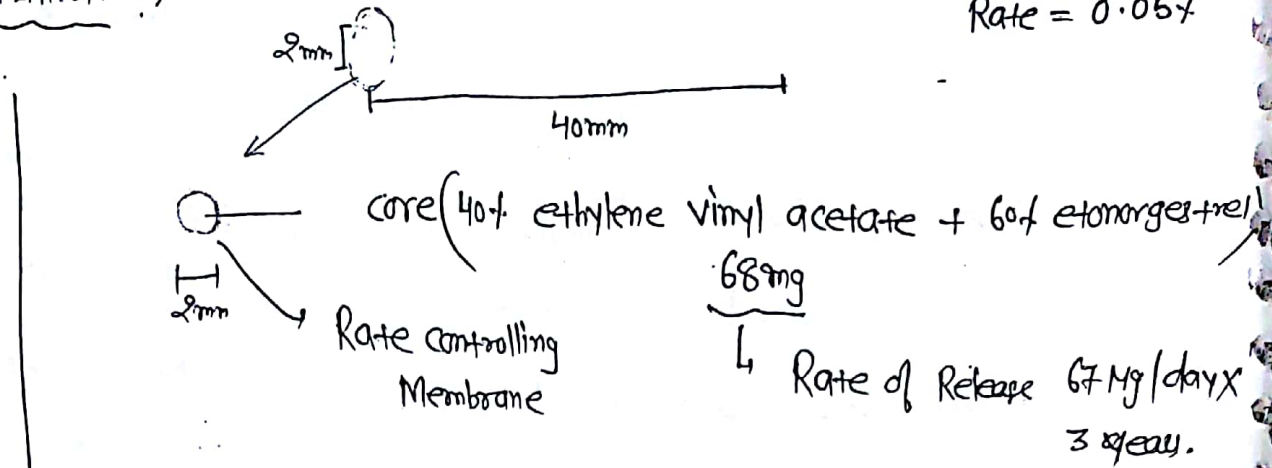
* M/c side effect of DMPA: Irregular Vaginal Bleeding.

* Absolute C/I: Same three S/E

↳ Undiagnosed vaginal bleeding
Pregnancy
K/c/o Breast cancer

PROGESTIN ONLY IMPLANTS ⇒ Among all contraceptives; Least failure Rate = 0.05%

IMPLANON ⇒



Single Rod = keto-desogestrel

OPD Procedures (Most Popular Implant Now a days);

M.O.A. ⇒ Inhibition of ovulation.

Site of Implantation ⇒ Non-dominant Arm (Medial aspect of Upper Arm).

It is Not Radioopaque.

∴ Next Generation of IMPLANON: NEXPLANON (Radioopaque)

Mic side effect: Irregular vaginal bleeding.

Absolute C/I:

Same 3

- i) Pregnancy
- ii) Undiagnosed vaginal bleeding
- iii) K/c/o Breast cancer

NUVA

It is Vaginal Ring (Blue or white);

It is E+P Compound (E = Ethinyl Estradiol : 15µg/day
P = Etonogestrel : 120µg/day)

M.O.A.: Inhibition of ovulation.

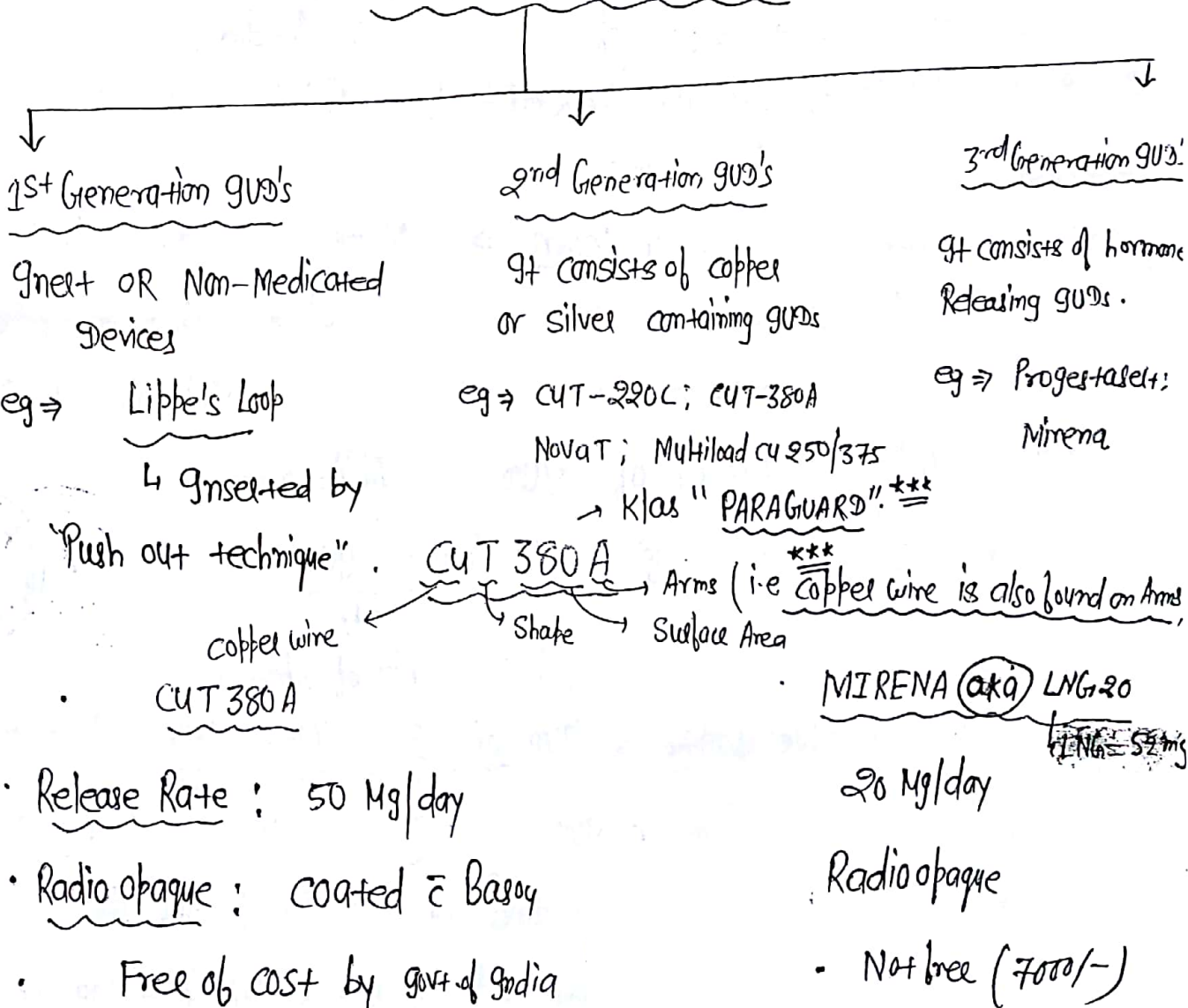
How to Use : ³⁸⁷ Insert in vagina on 1st day of her Menstrual cycle & keep it for 3 weeks; after 3 week; Last week is "Ring free" (195)

↓
then! ~~Insert~~ New Ring.

Safe period of Ring : 3 hours before sex.

It means if the expelled Nuva Ring has been out of vagina for more than 3 continuous hours; during weeks 1 & 2; you may not be protected from @.

INTRA UTERINE DEVICES



CUT 380A

- Effective for span of 10 years;
- presence of beak: to ↓ risk of Perforation & for identification and Removal.
- Can be used as Emergency contraceptives
- ↑ Menstrual blood loss

* M/c side effect of GUD \Rightarrow \uparrow Bleeding;
M/c cause for Removal \Rightarrow Pain
of GUD

* G.U.D.: Mech^m of Action \Rightarrow Mainly "Spermicidal"
 \therefore Ans \rightarrow Inhibition of fertilization
Inhibition of Implantation.

M/c Infection of GUD: Actinomyces;

* GUD insert \bar{c} \odot : Remove GUD
 \downarrow why
b/c ↑ Risk of Abortion

* Do USG b/c failure of GUD \odot \bar{c} G.U.D. Mostly: Ectopic*

* Max^m Infection \bar{c} GUD: \bar{c} in 20 days of insertion*

* Multifilamentous: Older days \rightarrow \uparrow Risk of Infection;

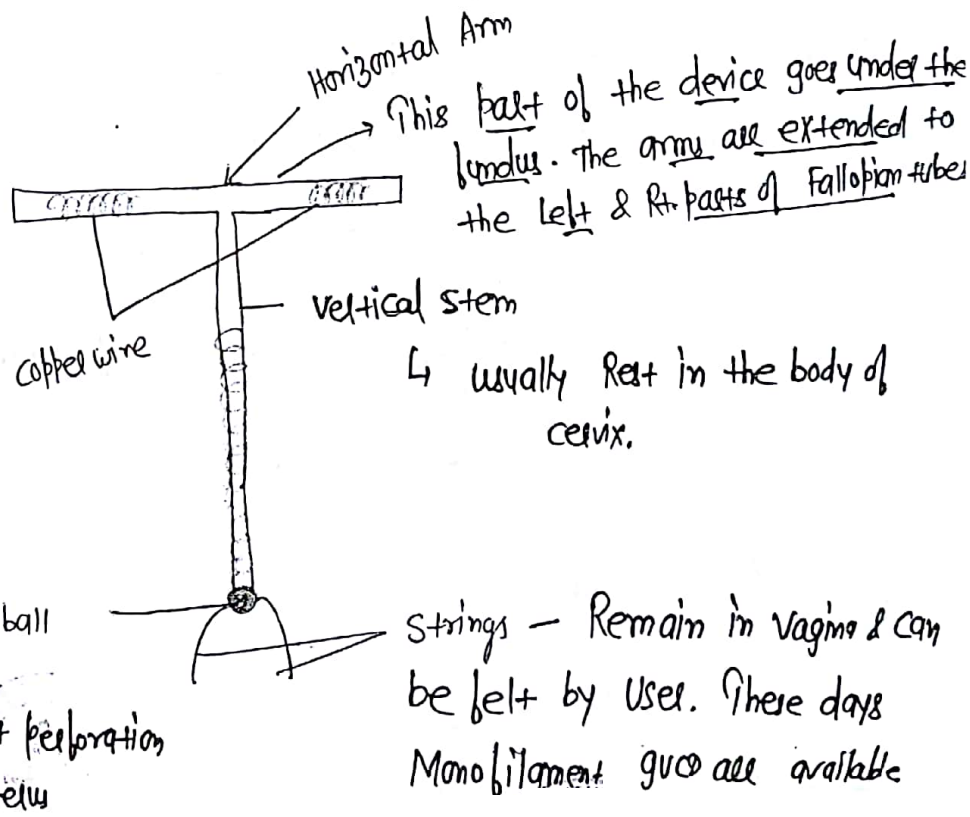
* Monofilamentous: Now a days \rightarrow \downarrow Risk of Infection \rightarrow that too by design

Mirena

- Effective for span of 5 years.
- Drug presents on Lower Limb only, (drug Release for a span of 7 years).
- Not an Emergency contraceptives
- ↓ Menstrual blood loss (amenorrhic for at least 6 Months).

Misplaced IUD (Missing thread) \Rightarrow G.O.C \Rightarrow USG ³⁸⁹
 \downarrow if Not found
 X-Ray ⁽¹⁹⁶⁾

- * IUD Missing \bar{c} Perforation \oplus in Abdomen: Mx: Laparoscopy**
- * if IUD Embedded in Myometrium: Mx: Hysteroscopy & Remove it.
- * Contraception in HIV \oplus Patient \Rightarrow IUD + Barrier
- D.M. \Rightarrow IUD
- Heart disease \Rightarrow IUD
- * M/c Mode of contraception used in India: Barrier Method*
- * Best contraception; if Family is complete: Vasectomy > Sterilization > IUD ^{ACP}*
- * Best Non-permanent contraception; if Family is complete: IUD > ACP.*
- * No Risk of Teratogenicity; if a \oplus Female continue \oplus \bar{c} IUD.



CU-T 380A

Silver Line CU 380 Ag \Rightarrow M/c CU \bar{c} used worldwide
 \hookrightarrow have flexible Arms: Silver core (A) & Rest same as CU-T 380A

* Absolute c/I of GUCD - category 4 of WHO ⇒

Mnemonic ⇒

- Please → Puerperal sepsis or Postabortal sepsis
- Don't → DUB / Unexplained vaginal bleeding
- Try to → Gestational Trophoblastic Disease
- Put → Current PID / STD or E in Past 3 Months
- Condom → Puerperal sepsis; know Pelvic TB
Ca Cervix
Ca endometrium

STERILIZATION (Permanent Method) ³⁹¹

* No. of children Required for sterilization?? (197)

Ans \Rightarrow At least 1 child of 1yr old.

* It is a legal procedure \rightarrow consent is Mandatory; but only of client.

* consent of spouse is Not Mandatory.

• 22yr - 45yr can undergo sterilization;

Q. Q. Unmarried women can undergo sterilization?? \Rightarrow NO
Married or ever married \Rightarrow Yes

• Most cost effective mode of contraception: Vasectomy

• Most effective contraception: Implant

• How many days after vasectomy should avoid coitus or use another contraception: > 3 Months till azoospermia

• Postpartum Sterilization \Rightarrow Within 7 days of delivery @
after 42 days of postpartum (can't do @ 8th or 9th days;

• This is klas "Interval Sterilization".

• M/C Method used for postpartum sterilization: Minilaparotomy (3cm Long)

\hookrightarrow by Pomeroy technique
 \downarrow
Babcock forceps

• We don't use Laparotomy / Laproscopic sterilization in post-partum sterilization.

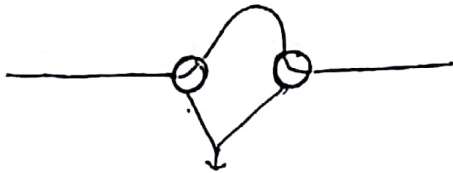
* Post-placental GUD \Rightarrow if we put GUD in 10 min of delivery;

* Post-partum GUD \Rightarrow if we put GUD in 48 hr of delivery.

- * M/c part of Fallopian tube that we ligate in sterilization
↳ Gsthmus.

Modified Pomeroy

AKA "Paekland Method"



double ligation of tube is done

Failure Rate = 0.2

Pomeroy



Middle part of tube (3-4cm from lundus) is formed into loop using babcock forceps, which is tied & excised. 0.4

- * Interval sterilization ↳ Non-pregnant state

Whenever we do sterilization in Non-♀ Female is k/as "Interval"

M/c Method used: Laparoscopy

- * M/c Method of Female Sterilisation ⇒ Laparoscopic Sterilisation

CO₂ gas used
↓
Intraabdominal Pressure b/w 8-12mm; Max^m 2 15mm of Hg

Not More than 15mm of Hg b/c it des ^{venous return}

- * M/c Method for Laparoscopic Ligation ⇒ Yarn balobe Ring / sigalistic band

- * Site of Ligation ⇒ Gsthmus

- * Among sterilization technique ⇒

Least failure Rate ! Unipolar cautery > Modified Pomeroy

(Never Used)

↳ b/c of Gntestinal burn

Highest Failure Rate ! Clips > Bipolar cautery

↓
HULKA CLIPS

Reversal Methods : Clips > Falope Ring > Modified Pomeroy ³⁹³ > Cautery

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Laparoscopic Ligation → P_t in Lithotomy position

↓
With help of Verres Needle (Introduced at
an Angle of 45°) Pneumoperitoneum is created

↓
↳ CO₂ gas used

Procedure is done under Sedation & Local
Anesthesia

* M/c used Method in Minilaparotomy : Modified Pomeroy

* Highest Risk of ectopic @ : Cautery > Madlenal > Modified Pomeroy.

