(5)

OBS & GYN PG-NOTES

2017

JAIN STATIONERY

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Revelant Anatomy

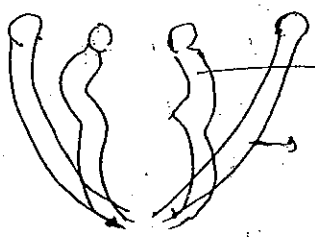
Ovaries → Gonads

Uterus & cervix, Fallopian tube → Internal genitalia

Vagina → ext. genitalia

→ Uterus: Embryologically derived from
↓
Mullerian ducts / Paramesonephric ducts
↓
they form female int genitalia

→ Mesonephric duct / Wolffian ducts → both are in fetus that appear in 6 weeks
↓
they form male internal genitalia
↓
and disappear in 9 week acc to sex (only one duct is disappear)



Mesonephric duct

Paramesonephric duct

→ Male baby produce MIS (mullerein Inhibiting substance)
↓
Sertoli cells
↓
from testis
↓
they act on mullerian duct of ipsilateral
↓
at 7th week have sertoli cells & release MIS

→ Wolffian ducts are acts upon by testosteron
↓
from leydig cells

→ Wolffian ducts
↓
Internal genitalia : S Seminal vesicle
E Ejectulatory ducts
E Epididymis
D Ductus deference

→ Mullerian persists due to absence of MIS

→ oestrogen is not required for fetus to become female

→ but testosterone is needed in fetus to be a Male

→ Wolffian duct need testosterone hormon in fetus to persistance of Wolffian ducts

→ Remnents of Wolffian ducts are
⑤ Appendix of Epididymis
contents of the broad Ligament.
① Epopheron → Cranial remnant → Mesonephric tubule
② Paropheron → Caudal remnant → Mesonephric tubule
③ Gartner's duct → Mesonephric duct
④ Hydratid of morgagni (paratubal cyst)

Remnents of Mullerian ducts →
Appendix of testis (Hydratid of morgagni)

→ 2 mullerian ducts cross from lateral to medial aspect and fused to form int genitalia

fusion → Center then
- Cranial (up)
- caudal (down)

MCQ : Direction of fusion : Caudal to cranial

when u the fusion completes: by 10 weeks

→ Internal genitalia into Male & female can be differentiated by 10 weeks

→ time of cavity formation of uterus & cervix by 18-20 weeks

↓

dissolution of Midline septum

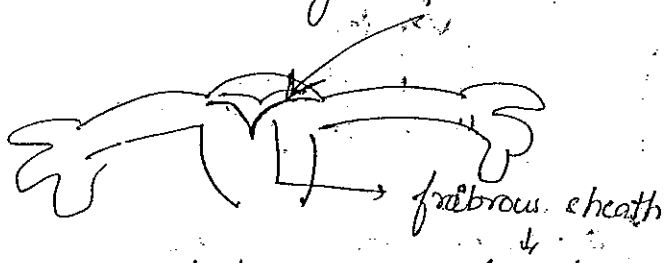
↓

Direction of dissolution

↓

Caudal to cranial

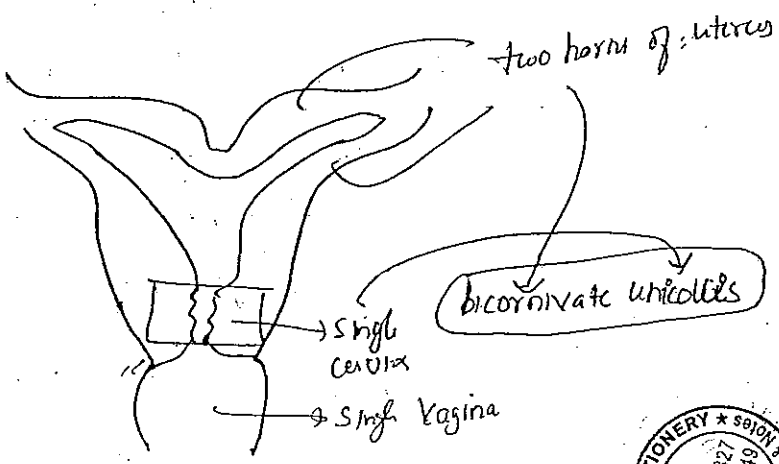
→ (1) M/c congenital Mullerian anomaly: septate uterus



(2) It presents as abortion

(3) worst reproductive outcome

(4) Rx with TOC is Hysteroscopic Resection of septum



→ Bicornuate unicollis

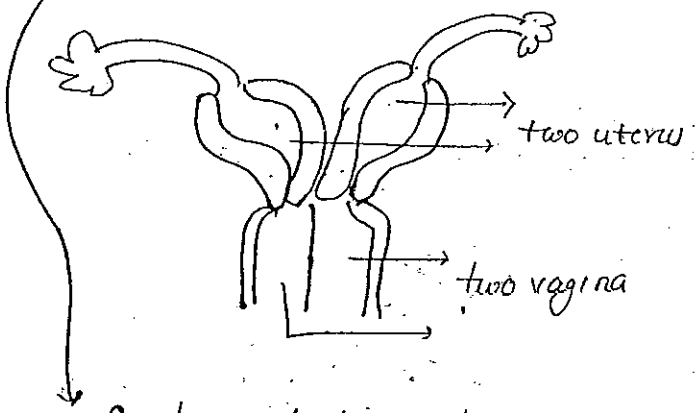
a) it has good reproductive outcome

b) Cause is incomplete fusion

→ if it is complete unfused

completely lack of fusion

uterine didelphius



Good reproductive outcome

→ UTERUS : a) Pear shaped

b) shape of the cavity : triangle

on coronal section

but in saggital section: Cleft like shape

c) weight in nulliparous : 50 - 70 gms

Multiparous : 80 gms

pregnancy : 1000 gms / 1.1 kg

d) Length of uterus : 6 - 8 cm

e) Volume " " in Non pregnant → 10 mL

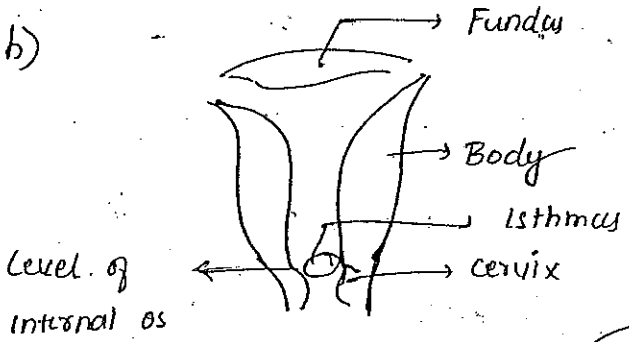
pregnant → 5 litres

f) pregnant uterus undergoes both, Hypertrophy and Hyperplasia

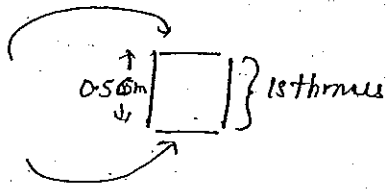
Hypertropy is predominant

a) wt of uterus in 6th month postpartum women
↓
80 gm (Multiparous women)

b)



Internal os
- Anatomical
- Histological



Isthmus : gap b/w Anatomical & Histological Int os

g) Lower Uterus segment
↓
a) only seen in pregnancy
b) Starts forming after the 1st trimester
iii) length of LUS at term = 5 cm
iv) LUS formation at term Isthmus + cervix
70% 30%

v) Effacement : moving up of cervix to form LUS and it become shortening
↓
length of cervix short

→ Complete effacement is in Labour
→ length of LUS in labour is 10 cm

→ LUS is used in Incision

↓

KERR'S incision → transverse incision (or) Pfannenstiel incision } shape is transverse

↓ → on uterus

M/c ~~lac~~ uterine incision

low vertical incision: kronig's incision

upper segment vertical incision: classical incision

KERR'S incision: strongest incision

↓

risk of rupture is min

Previous 1 kerr's 0.5 - 0.9 %

2 kerr's 0.9 - 1.5 %

vs vertical / classical incision: 4-9%

↓

Risk of rupture is Max

↓

Absolute Indication for: Repeat caesarean section

but not Absolute indication for Repeat classical incision

↓

You have to given lower segment

Indications for classical incision Cesarean

a) Ca cervix

b) Dense adhesion b/w lower segment and bladder

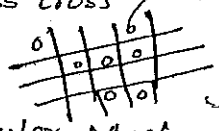
c) postmortum

d) repaired VVF (vasicovaginal Fistula)

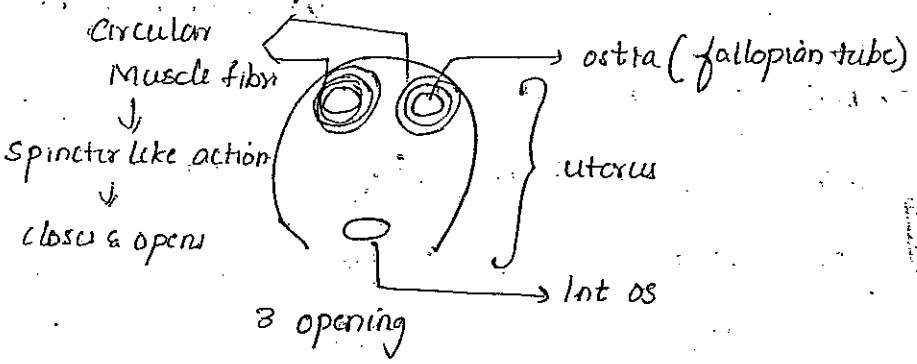
→ BODY OF UTERUS

Body is made up of smooth Muscle
↓ myometrium → 2.5cm thick
also made of corpus

Outer layer → longitudinal

Middle layer → cross cross → blood vessel
↳  } Living ligature
↓
Natural defence mechanism

Inner layer → circular Muscle



→ In the inside of myometrium there is Endometrium

Endometrium → Glands + stroma = equal amount
↳ single layer columnar epithelial lining
ciliated columnar is in near gland openings

Simple tubular

Endometrium

functional
↓
2 layers { stratum compactum
" spongiosum
↓
shed off
↓
Tk thickness

Basal
↓
stratum basal
↓
does not shed off
↓
takes part in endometrial regeneration

→ basal layer is distrubed in curatage
↓
vigorous curatage
↓
basal layer shedded off
and raw area is exposed
↓
Healing occurs
↓
fibrosis is formed
↓
intrauterine adhesions occur

thickness of endometrium

→ Immediatly after menses / Day 5 0·5 mm

→ periovulatory 5-6 mm

→ secretory

→ Implantational 10-12 mm

FUNDUS OF UTERUS

Ant-Post relation: Utero ovarian pedicle ← { Round ligament, Falloplan tube, ovarian ligament }

Most superior structure: Falloplan tube

RL OL → are attached at same level

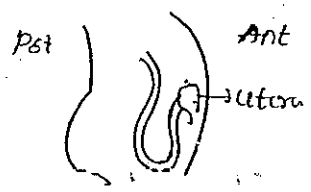
→ Fundus is part of uterus that lies above the FT.

Blood supply: uterine Artery
↓
branch of Int iliac Artery
↓
Ant devision of Int iliac Artery

→ M/c site of injury in gynaec surgery is
ureter
↓
water under bridge
↓ ↓
urine Ant devi of int iliac artery

crossing over takes place 2cm lat to Cx

→ 2nd m/c site of injury pelvic brim



(d) → Ureter is ant to uterine artery

→ Uterine artery ligation: bilaterally done
and at the level of int os

Lymphatic drainage: Int & Ext Iliac lymph Nodes

lymphatic drainage at fundus is paraortic
ostia → superficial inguinal

Nerve supply: T_{10} to L_1 (Pain in labour)

Franken hauser ganglion (inf hypograstic plexus)

Epidural analgisia (to relief from pain)

level of block for Labour analgisia: T10

" " " " caesserian: T4

↓

to knock out the peritoneal layer pain

m/c anaestheria in C.S is spinal anaestheria

Drug used in labour analgisia: Bupiracaine 0.125 - 0.25 %

↓

above 0.25 we put on Motor analgisia that is not required

Prolong labour:

= active phase 1 hr

2nd stage : 20 - 25 min

→ the block that is given in forceps (or) instrumental delivery is pudendal nerve block.

↓ S_2 S_3 S_4

★ Landmark : Ischial spine

↓

canal of pudendle

★ Ligament pierced: Sacrospinal ligament

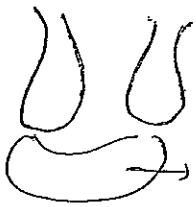
★ Direct of needle: Posteromedial

CERVIX (Cx) : Length 3-4 cm

Conical shape

Mullerian ducts

Shape of the canal : fusiform (or) spindle shape



endocervix → glands → Compound Racemosed

↳ Tall columnar

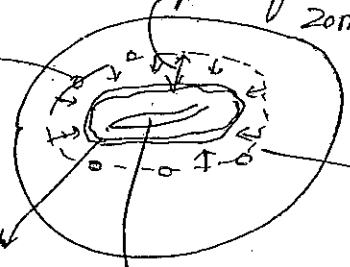
→ Ectocervix

↳ stratified squamous non keratinized

Arbor Vitae

↓

Numerous branching glands



Transformation Zone (M/c site of premalignant & malignant lesion of Cx)

Most distal cervical crypt opening

↓

Nabothean cysts

↓

Neo SCJ

Ext os

original squamocolumnar Junc

↓

has age goes on it moves inward towards ext os

↓

Neo S.C.J

WALL of Cx : Connective tissue collagen + Little smooth muscle (10-15%)

↓

effacement can occur due to this

↓

(softening + shortening)

→ due to (↓) collagen

↓

(↑) Hyaluronic acid

(↑) water

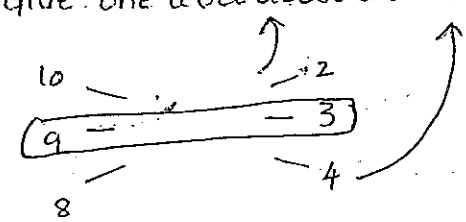
(↓) Decorin (dermatan SO4)

(↓) Chondrotin SO4

	Cervix : corpus (body)	Corpus : cervix
at Birth	1 : 1	1 : 1
befor pubertj	2 : 1	1 : 2
after pubertj	1 : 2	2 : 1
throughout reprodtj	1 : 3	3 : 1
menopause	1 : 1	1 : 1

Blood supply : descending cervical artery
↓
Branch of uterine artery

paracervical block: should not give at 9 & 3 o'clock position (there lies the blood vessel des cervical artery)
↓
Give one level above (or) below



Branches of Des cervical artery

U uterine A
↓
A Arcuate → outter 1/2 of myometrium
↓
R Radial → inner 2/3rd of myometrium
B ✓ Based → basal endometrium
S ✓ spiral → functional endometrium

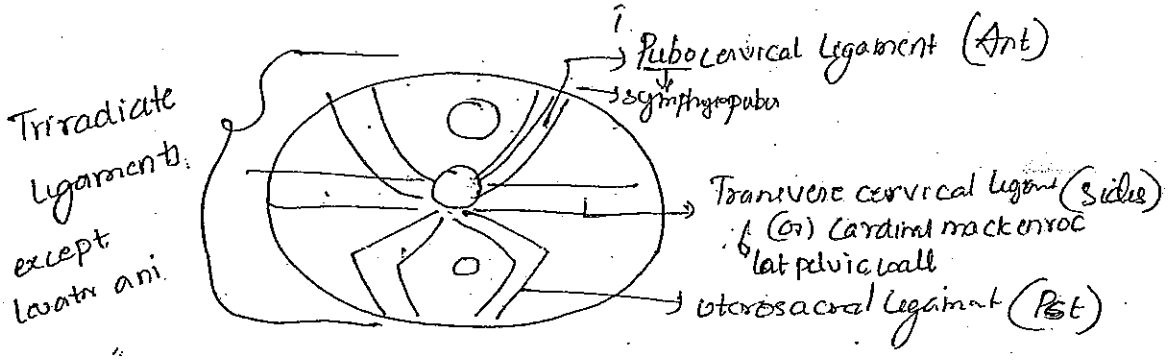
→ Lymphatic drainage of Cervix

Cx → ex iliac L.N → common iliac → Para aortic
Cx → Int iliac LN → Para aortic
Cx → Presacral

→ Inguinal L.N are not part of Cx ca → No Radiotherapy

Para aortic LN in abd [all other are in pelvis L.N's]

MAIN SUPPORTS OF UTERUS



4th (below the cervix) Levator ani
puborectococcygeus

→ Broad ligament is not a support for uterus
→ Broad ligament is not a ligament at all

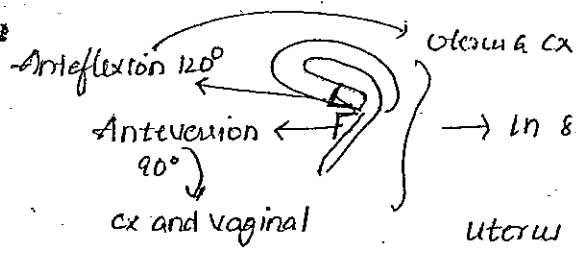
→ upper end of uterus
↓
deep inguinal ring
↓
superficial ring
↓
labia majora

→ R.L, O.L } comes from gubernaculum
gubernaculum → distal part → R.L
gubernaculum → Proximal → O.L

Canal of Nuck: a fold of peritoneum that carries round ligament into the inguinal canal in fetus and then in adult and presents as herniation

→ Uterus is bends in two angles

Angles



→ In 80% of female Antiversion, Antiflexion

uterus bending on bladder

uterus bending on Rectum → Retroverted, Retroflexed

found by bimanual examination

→ while doing PV → Ant lip of cervix : Anteverted

Pst " " " : Retroverted

ⓓ Long axis of uterus ⊼ Cx

ⓓ Anteverted Antei

FALLOPIAN TUBES:

Length : 10cm

Most medial → Intramural / Interstitial

↓

Isthmus (3cm ; 1mm wide)

↓

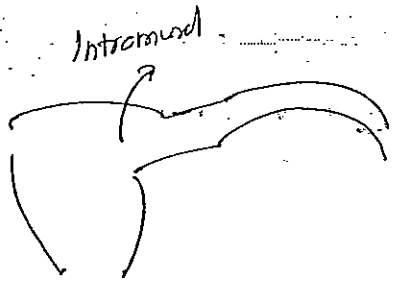
Ampula

↓

Infundibulum

Widest part : Ampulla (5cm long, 6mm wide)

Narrowest part : intramural / Interstitial (2cm long, 7mm wide)



Site of fertilization : Ampulla

m/c ectopic preg : Ampulla (bcoz it is site of fertilization)
↓
pt More no of plicae / folds

How long conceptus remains in Ampulla : 3 days

Narrow 1mm wide ← Isthmus of FT is physiology sphincter

Intramural part is Anatomical sphincter
↓
circular fibre

Lining : single layer ciliated columnar epithelium

3 types of cells : ciliated cell
Secretory cell
★ PEG cells → resting cells of FT

movement of cilia is toward uterine cavity

→ The main reason for transport of conceptus towards uterus is Tubal peristalsis (but not cilia)

Risk factors of ectopic preg

① ~~pro~~ PID (adhesion)

② progesteron only pills

③ sterilization failure

Blood supply : dual

Medial 2/3	lat 1/3
Uterine A	cervical A

↓

3 time dilate

→ Uterine Artery ligation : ~~at~~ level of Int os

at anastomosis c̄ ovarian artery

→ Unruptured Ectopic : pain occur due to tubal stretching

↓

Pain travels in, which Nerve route

T11 T12 & L1 (thes is the NS of FT)

→ Lymphatic Drainage : Para aortic LN

Intramural part : supficial inguinal LN

ostia : " " LN

→ VAGINA ←

① 7-10 cm longs

② 4 wall < Ant, Pst, 2 Lat.

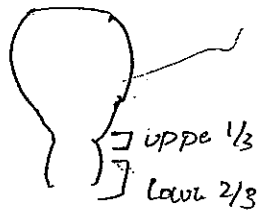
* post wall longer than Ant wall by 2 cm

Ant & post walls are opposed each other

↓

they in contact w each other

(d)

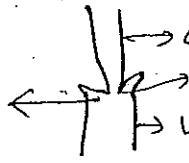


Mullerian duct

upper 1/3 → mesoderm

lower 2/3 → endoderm

Spaces (fornix)



→ Cx

→ Cx inward into vagina (formin fornix)

→ vagina

↓

4 formixes

Ant Pst 2 lat

↓

long fornix

↓

does long Pst wall

Vesicouterine flod of peritoneum

→ Rectouterine pouch / Pouch of douglas

site where LUS present

↓

Most dependent part

↓

So small amounts of any fluid peaches here,

fluid / Blood / Pus

↓

if Pus is collected we have to do

Colpotomy

↓

open POD to drain Pelvic abscess

Culdocentesis

↓

blood collected in ectopic rupture

↓

Int bleed

→ Vagina makes angle 90° c̄ cx
45° c̄ Horizontal

→ Vagina has **No glands**
↓
but for vaginal secretion
↓
comes from cervix
↓
Cervical secretion : alkaline nature
↓
Vagina secretion : acidic pH
(N) pH of vagina **7.5 – 4.5**
↓
Natural defense mechanism

Glycogen ⟶ lactobacillus (Doderlein bacilli)
↓
(oestrogen presence)
⟶ Acidic nature

☆ { if any one is missing the secretion becomes alkaline
↓
Bacterial vaginosis – gardenella replace doderlein bacillus

post menopausal women
pre pubertal girl
at the time of menses
} pH is higher > 4.5

→ pH in preg: 4

→ New born girl: 5.5 – 6 (Acidic)
↓
bcoz no vaginal flora produced

→ Candida grows in acidic medium

Candida is commensal, Not a STD
↓
Sometimes it grows more in HIV & DM

- women who consume oral pills & pregnancy candida is grown more

Nerve supply: S_2 S_3 S_4

	Blood supply	Lymphatic drainage
upper 1/3	Br of uterine A	Ext Iliac
Middle 1/3	Inferior Vesicle	Int Iliac
Lower 1/3	Middle Rectal	Sup f inguinal grop LN

→ EXTERNAL GENITALIA ←

	Female	Male
Genital tubercle (6 weeks) =	Clitoris	Penis
Genital fold =	L Minora	Penile urethra
Genital swelling =	L Majora	Scrotum

glans of skene Homologous to Prostate in male
↓
present in periurethral area

Bartholin gland = Bulbo-
(Cooper's)

Ext Genitalia
- Male (Testosterone)
  - ↓ ← 5α Reductase
  - active form dihydrotestosteron (DHT)

5α Reductase Deficiency is an ex for (Male pseudo hermophrodite)

→ by 12 weeks ext genitalia form into Male (or) Female

ext genitalia
sex differentiation } 12 weeks
Male & Fema

→ CLITORIS : L. Majora
↳ supt inguinal (drains)

Glans Clitoris : Drains into deep inguinal Lymph Node (or) called Lymph node of Cloquet

L. Minora drains

→ BARTHOLIN'S GLANDS : Pea sized

→ it is present in groove b/w L. Majora and Minora at the Junction of ant 2/3 & post 1/3

→ Duct will go forward and medial opening inside the vagina b/w L. Minora and Hymen

→ they are contents of supf perineal ~~glan~~ pouch

Gland lining epithelium : Short columnar

duct " " : Transitional epithelium

PH is alkaline

This gland secreates only in coitus

→ Bartholin's cyst → post lat wall of vagina

D/D

Gartner's duct cyst → ant lat wall of vagina

★ M/c cyst of vagina/vulva is INCLUSION CYST

Asymptomatic → No treatment

Symptomatic → Treatment is done

TOC : Marsupilization

↓

permanent exterrosation

M/c Abscess → E coli > gonorrhoea

↳ I/D c̄ word catheter

treatment : ≥ 40 yrs → symp / Assymptomatic

↓

TOC : Excision

↓

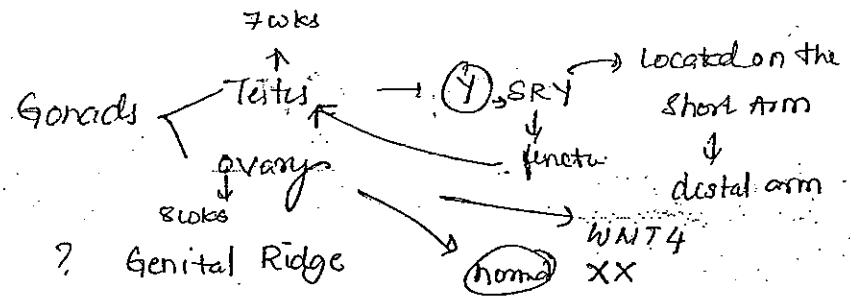
bcoz risk of cancer

Honan criteria

↓

location : deep

skin : Normal skin



ovary in ovarian fossa
↓
(lat pelvic wall)

→ boundaries of ovarian fossa

Superiorly ext iliac vessel
Inferiorly Levator Ani
Medially ovarian ligaments
↓
Connects ovary to uterus
Laterally obturator N & vessels

MCQ Posterior ureter / int iliac vessels

Anteriorly obliterated umbilical Artery
Mesovarium
post leaf of broad ligament

→ Ovarian ligament extends from uterus to inferior pole of ovary

→ Infundibulo pelvic ligaments
⇒ also called suspensory ligament

⇒ attach upper pole to lat pelvic wall

⇒ primarily carry ovarian vessels

⇒ oophorectomy

Mesovarium: fold of peritoneum
↓
attach. to post ligment of B.L

	Reproductive women	Post menopausal
Normal →	upto 20cc	upto 10cc
~~Average~~		
Average →	7-8 cc	3-4cc

Blood supply to ovary

ovarian Artery
↓
From abd aorta
↓
at level L2

ovarian vein
↓
on (Rt) side
↓
drain to
↓
IVC

(Lt) side
↓
Renal vein
↓
Same in (Lt) testis
↓
Varicocele is more common on (Lt) side
↓
bcoz Lt testicular vein connect (Lt) Renal vein at 90° angle
↓
More reversible male infertility

Epithelium: Single layer cuboidal epithelium
Germinal epithelium

Cortex (follicle are present only in cortex)

Medulla (vascular part of ovary)

Genetic Material : PGC

primodial germ cell

↓

dervied from EPIBLAST

↓

from Epiblast

↓

reach Yolksac (3wk Gst age)

↓

genital ridge (6 weeks)

↓

OOGONIA (9 weeks)

↓

1° OOCYTE (12 weeks)

↓

Follicle (14 weeks)

formation starts

↓

completed at (24 weeks)

MCQ

Max no. of follicles

8th month IU life

20 weeks IUG

↓

2 million - Birth

↓

400,000 Puberty

↓

400 ovulate throughout the reproductive life

↓

10000 atresia / month

Folliculogenesis / oogenesis

↓ ↓

Genetic material when genitic material changes
called

1° oocyte

↓

when cells supporting
or present around the

Primordial cell → flat cells

↓

granulosa cells of ovar

primary cells → cuboidal cells

secondary follicle → theca cells

tertiary follicle → can CAVITY develops

seize of Mature follicle → Graffian follicle
18-20 mm

↓

(Mature follicle ruptures)

→ Oogenesis

- begins in IUL
- very long
- OOGONIA (Diploid) (2n)

↓ (MITOSIS)

1° oocyte (2n)

↓

meiosis I

↓

Prophase I (arrested ?)

↓

Diplotene

↓

DICTYOTENE

↓

name of arrested phase which is b/w prophase & metaphase

↓ when is meiosis I completed

** At ovulation (3-4 hrs before ovulation)

2° oocyte → (ovum contains 2° oocyte which is haploid)

1st polar body → very little cytoplasm

Mature Ovum (120 μm)

Fertile period of ovum: 24 hrs

↓ 2° oocyte enter into meiosis 2

arrested at Metaphase

Meiosis 2 completed → After fertilization

↓

female pronucleus

↓

Premale nuclei, Prefemale nuclei → both combine & form zygote

single cell

Spermatogenesis

↓

→ . Begin in Puberty

- duration : 72 days
- spermatogonia (2n)

↓ MITOSIS

1° spermatocyte (2n)

↓ meiosis I

Absent stage : Dictyotene

2° spermatocyte (n) — 2° spermatocyte (n)

↓ meiosis 2

spermatids spermatids spermatids spermatids..

1 spermatocyte = 4 spermatids

1 spermatogonia = 64 spermatids

16 1° cyte → 4 spermatids

avg length → 55 um

Life span –

Spermatids should through the epididymis

↓

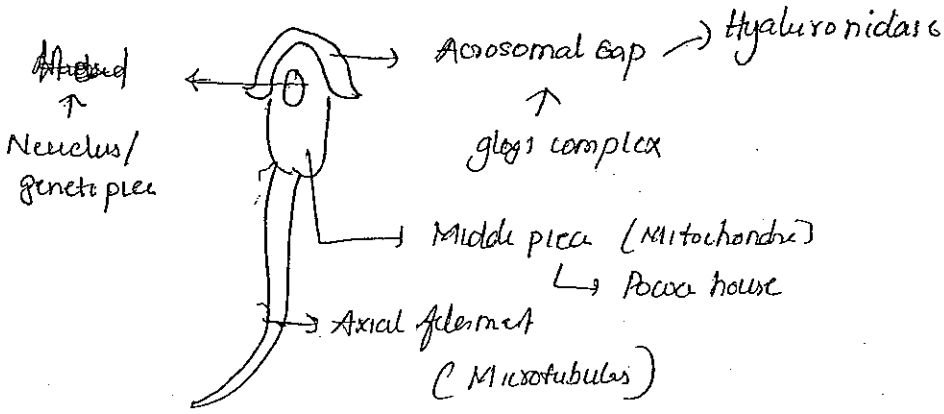
achieves maturity/motility

↓

It takes 14 days to maturition

tail (caudate)

duration of spermiogenesis = 14 days



→ Motility by Calcium ions

→ Gene required for Motility : CATSPER Gene

→ How long motile sperm in female genital tract is 12 hrs

→ After release the sperm reach site of fertilization in 30 min

→ capacitation : begin in cervix

main site Fallopian tube

duration 7 hrs

* { ① they get ability to undergo acrosomal reaction
② Hypermotility → Imp to bind zona pellucida

protective layer of ~~ovum~~

↓

protect against polyspermy

↓

cortical formation

Final maturity in female

→ Maturity at Epididymis

↓

Cortical reaction

↓

Release granules

↓

c help of Ca^{2+}

→ Receptor ~~sperm~~ of sperm to zona pellucida

ZPGP-3

→ Zona pellucida is lost Just before implantation

(a) on Day 5 post fertilization

↓

fertilization

↓

Zygote formation (conception)

↓

Embryonic period

↓

fetal life

Weeks = POG = from LMP (1st day)

day = from fertilization

1) every female 28 days cycle

2) 14th day = ovulation

3) day of ovulation = day of fertilization

Site of fertilization : Ampula

↓

3 day remain here (conceptus)

conceptus enter the uterine cavity 4th day

↓

enter in form of morula

↓

cell stage : 8 cell stage (enter the uterine cavity)

Morula → what cell? → An) 16 ~~cell~~ staged cell

when does First cell division : approx 20 hrs after fertilization

↓

2 cell stage

↓

4 cell stage

↓

8 cell stage (enter uterus)

→ Embryonic stage : (3 – 8 weeks) from fertilization

2 zygote befor 3, 3–8 ↓ embryon, after 8 – fetus

When does fetal stage start : 8 weeks

from fertilizat " " " : 10 weeks

implantation begin : Day 6 – 7

from : BLASTOCYST

↓

what day blastocyst formed Day 5

↓

→ Site of implantation : upper post wall : dent crate fundus

Implantation in 3 phases

	Molecule
1) Apposition →	Selectin
2) Adhesion →	Integrin
3) Invasion →	Matrix metallo proteinase

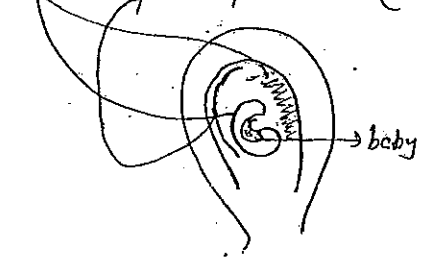
Implantation = Eccentric

Implantation window = D_{20} to D_{24} menstrual cycle

Hartman's sign = little bleeding at the time Implantation

→ Decidua (Endometrium of preg)

Decidua basalis (beneath endometrium)
Decidua capsularis (cover the baby)
Decidua parietalis (remaining decidua)



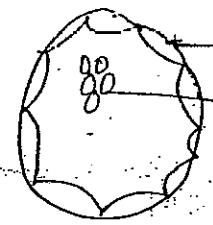
fusion of capsular & parietalis : 14-16 weeks
↓
Called as
decidua vera

In Ectopic : decidua ~~vera~~ sheded off this is also called decidua vera

Superfetation: 1 fetus + Implantation of 2nd fetus
↳ does not occurs in humans

Superfecundation: menstrual cycle → 2 ova get fertilized by 2 sperm in One menstrual cycle

two diff cycle
↳ superfetation



→ Trophoblast — placenta ? fetal membranes

→ Inner cell mass
↓
fetus proper

cytotrophoblast — syncytiotrophoblast

differentiation by
8th day post fertilization
(D6 - D8 range)

→ all 3 germ layers found : D21

Endoderm — Meso — Ecto (sequence)

Anterior Neuropore Close : Day 25
post " " : Day 27

☆ Cytotrophoblast ☆

Villous cytotrophoblast
↓
from placental villi

Extravillous cytotrophoblast
↓
go into decidua
↓
into the spiral artery in decidua
↓
so called endovascular
↓
converts the spiral artery to permanent vasodilated
↓
this vasodilated artery provides uteroplacental ~~fetoplacental~~ circulation
nutrient & blood exchange

} vascular remodeling

Syncytiotrophoblast
↓
— all hormones produced by it (protein / steroid)
— exchange (nutrients gas)
— source: cytotrophoblast

Vascular remodeling: ends in two phase

2 phases < 12 weeks
16 weeks

→ Controller of vascular remodeling: NK cells in decidua

→ Preeclampsia: Vascular remodeling is absent

→ Fetal membranes: ① Amion
② Chorion
③ Yolk sac
④ allantois

Amnion: a) inner most of placent
b) Avascular
c) Max tensile strength
d) fetal ectoderm
e) Aminon fluid rich in Prostaglandins

Chorion: a) two types < trendosum → forms the Placenta ↓ bulk
Laeve ↓ fuse c̄ Amnion

bed of placenta: basalis

Allantois: a) diverticulum from hind gut
b) grows into connecting stalk
c) forms umbilical cord

Yolksac: Hematopoiesis occurs in this.

a) site of Hematopoiesis

b) 3-6 weeks

c) Gower 1, Gower 2 & Portland

d) after 6 weeks → Extramedullary
↓
Liver
↙
HbF
↓
→ $\alpha_2\gamma_2$
→ High affinity for O_2
→ fetal curve to the right of maternal curve
→ It is resistant to alkali and acid denaturation
↓

Alkali denaturation test { Apt test (or) Singers test
↓
Qualitative test
↙ bedside test
↓
(fetal / maternal blood)
→ 1% NaOH

acid denaturation
KB
Kleihauer Betke test
↓
Quantitative test
↓
Laboratory
↓
- citric acid PO_4 buffer is used to test
- Rh -ve preg → to calculate the dose of Anti D
↓
(to differentiate RBC's)

fetal RBC : a) Large in size

b) life span 90 Day

c) Hb% at term 18 gm%

d) HbF% at term 75-80%

e) transition from HbF → HbA ↓

It begin at 36 weeks

it is completed at 6 month of age after birth

↓

at this age <1% is HbF

→ PLACENTA

→ Def: Hemochorial

Discoid

Decidiate

2 sites of placenta: fetal site

- Maternal site → lobes → lobules

↓

functional unit

↓

"cotiledon"

fetal site: smooth & shiny

umbilical cord is attached

a) wt of placenta: 500 gm

Vol: 500 ml

diameter: 20 cm

thickness: 2.5 cm (upto 4)

PL: fetal wt at term 1:6

PL: fetal wt = 1/7 wt

Uteroplacental circulation = 450 - 650 ml/min

Uterine blood flow at term = 750 ml/min

When is uteroplacental circulation established = 12 day of post fertilization

When is fetal circulation establish = day 21

Anatomical formation of fetus = 16 weeks

As POG (↑)

① cytotrophoblast layer is thinned out and at term it is absent

② At term only syncitiotrophoblasts are present

③ stroma (↓)

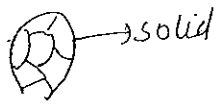
④ fetal blood vessels (↑)
↓
They also moves to periphery
↓
to have good exchange

⑤ Appearance of some cells called Hofbauer cells
↓
fetal macrophages

Intervillous space → maternal blood

In villous space → fetal blood

Villous can be divided into: 1° stem villous



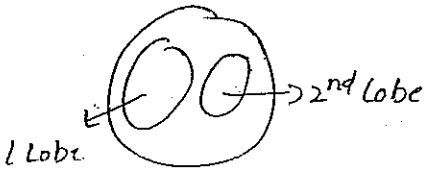
2° stem →  → mesodermal core

3° stem →  → fetal blood vessels

[Day 21]
↓
fetal circulation is started

Intervillous space = Maternal blood
↓
a) 140 ml in each space
b) 120 spiral arterioles
c) O_2 saturation 65-75%
d) PO_2 = 35-40 mmHg
e) 2-3 time/min is replaced

Gross structure of placenta



Succenturiata
↓
smaller lobe is retained after delivery and the women as PPH

→ Cord should be attached to the central of the placenta

→ Battle door placenta: Cord attached to periphery
↓
this cause PPH

→ When the blood vessels of fetus attaches to membrane of placenta it is called Velamentus placenta
↓
type of it is ~~placenta~~ Vasa previa
↓
blood vessels of fetus outside cord travel & attach to membrane of placenta
↓
travelling over int OS

↓

Rupture of membrane

↓

APH ← fetal blood

↓

fetal distress

↓

Quickly take women for LSCS

(An Emergency)

Chronic plate smaller

→ Circumvallate: ~~a~~ differentiated

→ Circummarginate:

Umbilical cord: a) center

b) fetal side

c) avg length 55cm

d) 30–100 cm

e) Short cord < 35cm

f) 3 vessels 2 UA + 1 UV

↓ Carry deoxygenated blood

↓ Left umbilical vein

↓ Carries oxygenated blood

the 1st BV to show you change in umbilical artery

the last " " " " " " " vein (co)

Ductus venosus

g) Respiratory function

Excretory "

nutritional "

Endocrine "

Endocrine: Progesterone / oestrogen / HPL / HCG

Progesteron: early source is corpus luteum
↓
Rescues the CL from Lutolysis a HCG

Placenta takes over function of CL = 8-10 weeks

Placental source of progesteron: maternal LDL cholesterol
↓
Imp for preg = ① decidualisation
and
② smooth muscle Relaxant

Luteal phase Defect (LPD): Deficiency of Progesteron
↓
they present c̄ recurrent preg loss

Areas stella Reaction: ← Progesteron.

HPE → Hypersecretory change

→ Oestrogen:

a) ~~total~~ specific to preg: E3 (Estriol)

b) Placenta cannot syn on its own
↓
beoz: 17 Hydroxylase
↓
it takes help from fetus
↓
fetal DHEAS source
↓
from fetal adrenal gland
↓
oestrogen syn done

⇒ Hormone that can say fetal well being is E3 (Estriol)

HPL (Human placental lactogen)

a) produced by placenta have endocrinal function
↓
⇒ Insulin resistance in Pregnancy

b) Placental functioning

- As the PL mature more peak levels of HPL is produced
  ⇒ peak level = 36 weeks

HCS (Human chorionic Somatomammotrophin)
↓
Old name of HPL

⇒ HPL Hormone is max produced amount by PL at term

- Source is syncitiotrophoblast
- early product^n : 3 weeks POG
- $t\,1/2$ = 3 min

HCG

- Source is Syncitio
  - α — Non specific (Same LH/FSH/TSH HCG)
  - β — specific ↓ similar to β of LH (84A)

→ secretion : 8 days post ovulation

the day ovulation = the day of fertilization

Range 8-9 days

→ doubling time of Hcg = 48 hrs (1.4 to 2 days)

↓

- doubling time for Intra uterine ~~and~~ viable pregnancy

↓

D1 to D3 (55% to 66%)

- if Day 3 (↑) only 20% this is seen in Ectopic preg
- if value is (↓) Abortion

- t1/2 = 24-36 hrs
  ↓
  more imp

- critical titer : for transvaginal u/s is 1500-2000 IU/L (TVS)
  (TAS) transabd scan is 6500 International unit (IU)

Clinical Q → 5 weeks Amenorrhea and little

→ m/c test for preg : UPT
↓
a) Sandwich ELISA
b) Sensitivity 20 IU/L
(10-20 IU/L)

→ Serum test is more sensitive than urine test

Serum → 1 IU/L
→ early detected D12-14 after fertilization

Fluroscence Immune-Assay

V

RIA

V

ELISA = RRA

V

IRMA

FIA > RIA > ELISA = RRA > IRMA

* HCG throughout preg

peak 10 wks. (100,000 IU)

↓

after 10 wks (↓)

minimum at 16 wks

after 16 weeks → Plateus (only 1st trimester)

(HCG only in 1st trimester)

→ HCG (↑) than expected

a) Gestational trophoblastic disease (GTD)

b) Multifeatal preg

c) Hyperemisis grandarum

d) Down's syndrome

e) underestimated Gestational Age

→ HCG (↓) ~~expect~~ than excepted

a) Ectopic preg

b) abortion (inviable preg)

c) overestimated GA

→ Return to Normal ~~Full term Preg~~

a) Full term Preg – 4 wks

b) Abortion – 6 weeks

c) Molar – 9 weeks

Imp function of HCG

1) maintainance of Corpus luteum

2) 1st stimulus for Release of testosterone from fetal testes

3) suppresses the myometrial constractions

4) growth of umbilical cord

5) Play role in immunosuppression

a) Villous trophoblast – lack HLA (MHC)

b) Extravillous " lack HLA G (only in human)

↓

Immunosuppressive

c) decidual NK cell have deficient cytotoxicity

fetus as a foreign body, but no Antibodies formation in the Mother becoz of these reasons.

DOWN'S SYNDROME

→ Tri 21

→ m/c Nondysfunction 95% (not inheretory)

→ recurrent risk of down's synd is 1%

→ 4% Translocation } Inhereditory
1% Mosaicism }

Clinical Q) 1st child down' baby
T 14, 21

Preg: Advise
↓
Prenatal testing

2nd clinical Q) 1 child – down
T (21, 21) → 100% risk of down's → translocation
Preg: 10 wks
↓
Advise Abortion

⟹ Screening for down's:

Age: Age is no longer a criteria

OPTOUT screening: to all preg women

if 1st child Down → diagnostic testin > screening
↓
Invasive test

Screening can be done in 1st or 2nd trimester
(1) done by U/S (or) by maternal serum markers
(2)

In U/S → NT nuchal translucency is seen
↓
Ideal when 11 to 13+6 wks.
↓
≥ 3mm (+ve screening test) for Down's

fluid collection can be seen

- (↑) NT is also seen in
↓
Down > monosomy X

- (↑) NT but (n) karyotype then look for CVS anomalies

→ D/D of Nuchal translucency is cystic Hygroma

- If it is large collection
- " " septic "
- extends along length of spine

Cystic Hygroma is assoc c̄ aneuploid Rate High i.e more than Down's

- Duel test: (↑) HCG + PAPPA

  1st trimester screen test done in 11 to 13 wks

  Preg ass plasma protein A

- Combined test: 1st tri

  USG (NT) + Serum Markers

2nd trimester screening test

a) U/S → soft markers (these markers can also seen in (N) babies)

- 15-20 wks
- ≥ 2 (↑) High risks
- ① absent Nasal bone (or) Hypoplastic Nasal bone
- ② Nuchal fold thickness
  - ≥ 6 mm (+ve test)
  - Skin fold thickness
  - (Not) NT Nuchal translucency
- ③ Short femur
- ④ " Hemur
- ⑤ " ear length
- ⑥ " frontal lobe
- ☆ ⑦ Sandle gap → 

(8) choroid plexus cyst

(9) Mild pyelectasis (dilatation of renal pelvis)

(10) Ecogenic foci → Intracardiac

(11) Ecogenic foci → Bowel

→ Endocardial cushing Defects in Downs synd

→ Gastrochisis is least assoc Downs [one of this contain memb]

omphalocele " " " " "

Triple tests: Hcg + AFP + UE3

↓ ↓ ↓ unconjugated ↓

(↑) (↓) (↓)

Quadriple tests " + Inhibin A (↑)

Diagnostic test: Karyotyping

1st cus — chronic villus sampling ≥ 10 wks, m/c 11-13

→ should not do ≥ 9 wks → cause limb defects

using: Trophoblasts → fetal loss 1%

2nd T — Amniocentesis ≥ 15 wks, m/c 16-18 wks

should not be done 11-14 wks → early amniocentesis → cause fetal loss

using ① Amniocyte ② dermal fibroblasts → fetal loss <0.5% (safer)

Amniocentesis →

c̄ help of USG probe

↓

fluid collected is not more the 20 ml

Cordocentesis : ≥ 20 wks (not less than 20 wks)

- sample from umbilical vein
- take from placental end of cord bcoz it is most static part of cord

CVS :

Catheter inside placental tissue: Trans vaginal, Tram abd } USG guided

→ fetal skin biopsy : Not used in karyotyping

→ ** PHYSIOLOGICAL CHANGES ←

Presumptive signs

Probable signs

Positive signs

Signs

1) Hegar's sign: softening of the Isthmus } by 6 weeks
2) Goodle's sign: " " " cervix }

3) Piskacek's sign: unequal growth of uterus
due to lat implantation

4) Chadwick's sign (Jaquemier) : Bluish discoloration of Vagina & vulva

5) Osiander's : Lat vaginal fornix pulsations
↓
(↑) vascularity of uterine vessels

6) Palmer's : Regular rhythmic ut contractions

⇒ these ③④⑤⑥ 8 wks of preg

7) Melasma (Chloasma) Hyperpigmentation of malar bones
↓
Due to of oestrogen (oestrogen cause proliferatn of melanocyte)

8) Pyogenic granuloma : preg tumour
a) Not a tumour, simple a vascular lesion
b) Vascular lesion seen on Nasal septum
oral mucosa (gingiva)

⇒ Most of the changes in preg = brought by Progesteron

9) Aréa's stella

→ Calori Requirment: 350 kcal / Day (Additional Caloric)

→ 1st trimester → 150 kcal
2nd " → } 350 kcal
3rd " →

→ Avg wt. gain → 12 kg
(10 - 14 kg) Range

→ BMR ↑ by 20%

→ Salt & water: Retention occurs (brought by oestrogen)

Water retension : 6.5 L

— Plasma osmolality falls bcoz of salt retension

Water retension > Salt retension

↓

Plasma osmolality (↓)

→ Insulin Resistance : (HPL, E, P, cortisol)

fasting Hypoglycemia

PP Hyperglycaemia

→ Plasma Volume : (↑) by 40%

starts (10 wks)

Red cell Mass : (↑) by 20% (by 8 wks)

Haemodilution : upto 11 gm% loss is called Haemodilution

Anemia : → Hb < 11 gm% (if)

→ Hct < 33 %

Severe Anemia : < 7 gm%

Very S. Anemia : < 4 gm%

M/c Anemia in Preg : Iron Def Anaemia

⇒ Best test for IDA is : S. Ferritin

IFA drug → 100 mg of element Iron (Fe sulphate)

100 mg Fe sulphate + 500 mg (FA)

1 tab for 6 months (Preg)

1 tab for 6 months (after Preg)

→ for Rx of IDA : 2 tabs

→ Total Fe requirment is 1000 mg
↓
In the Fetal requirment is = 300 mg

Fe requirment in 1st half of Preg → 3-4 mg/dl
2nd " " " → 6-7 mg/dl

→ Switch from oral to Injectable ←

conditions

① Non compliance to oral drugs

② Malabsorption Sx

③ Worse c̄ Intorable S/E

→ Indication for Blood transfusions ←

① Impending CHF

② already c̄ "

③ gest age > 36 wks c̄ S. Anemia

Total leucocytic count : ↑3 15000 (trimester)
25,000 (post partum)
↓
A physiological change don't try to Rx

DLC :

→ Avg Platelet count (↓s)

↓

but it will remain in the normal range

does not goes into thrombocytopenia.

→ Clotting factors : (↑) (except XI & XIII)

fibrinogen (450 mg) dl

→ Immunity : TH1 ⟶ TH2

↓ ↓

activity is suppressed ← (↓) IL 2 IL 4, 6 (↑) → activity is increase

↓ ↓

RA / Hashimitus SLE (flare up)

women ~ Preeclampsia does not show these changes

→ Cardiovascular Changes

① ↑ plasma Vol 40%.

② ↑ Red cell Mass by 20% (Not) Hb ↓

③ CO ↑ by 40% (starts - 5 wks) ↓ O_2 carrying capacity

④ O_2 consumption (↑) by 20%

→ Aterovenous oxygen gradient = decrease

A-V O_2 gradient = ↓

→ O_2 carring capacity is = ↓

↓

but no Hypoxia to tissue

↓

bcoz other mechanism play role in O_2 carrying

→ Max CO_2

→ Max Cardiac output ↓

a) (Only ~~to~~ (↑) in 2 stage of labour)

→ Heart Rate a) 20% (↑)

b) 18 bpm above baseline at term.

c) all Heart sounds are loud

d) S_3 can be Heard (gallop Rhythm)

e) ~~Sty~~ systolic Murmour (ESM) upto grade 2
↓
normal physiological
↓
above 3 is pathological

f) Diastolic murmour : pathological

g) S_1 → splitting

→ Chest X ray (CXR)
↓
Image of Heart
(Silhoulte) bigger
↓
No cardiomegaly

① bigger due to rotation in preg
↓
Rotates Anteriorly

② pericardial effusion (Physiological)

→ ECG: LAD

Left axis division (Physiological)

→ Blood pressure **

Fall in BP → both systolic & diastolic
↓
More Fall then SBP
↓
(10 mmHg) more

Estrogen : Vasodilation (Relaxation)
↓
due to release of ↑NO
↑Prostacyclin

Progesteron :
↓
Relax smooth muscle

[Starts falling by 6 weeks]

[Max fall in 2nd Trimester
↓
24-26 weeks]

In 3rd Trimester (↑) (to pre preg value)

⇒ M/c Pre eclampsia present in 3rd T

→ Preload (↑)

→ After load (↓)

→ EF (No change)

→ CVP (No change)

→ Respiratory changes in Pregnancy ←

① ↑ Tidal Vol ② ↑ minute ventilation

⇒ *** In Preg the depth of respiration is (↑)

No (↑) in Resp rate

bcos of Hormone Progesterone

↓

(↑) sensitivity of Resp center to CO_2

↓

more CO_2 is washed out

↓

PCO_2 (↓) and PO_2 (↑)

↓

Preg is a state of MILD RESP ALKALOSIS

↓

No ph of blood ~~changes~~

↓

does not change

↓

compensated by kidney

↓

kidney will ↑ HCO_3^- excretion

⇒ diaphragmatic excursions ↑ in pregnancy

⇒ Diaphragm Rise by 4cm

↑ transverse Thorasic diameter by 2cm

Total lung capacity (↓) slightly

VC → No change

ERV (↓)

IRV shows No change

⇒ No change in Pulmonary capillary wedge

⇒

→ GIT

⇒ GERD is seen

↓

due to progesteron (it will ↓ lower oesophagal spintor tone)

↓

So gastric pressure (↑)

Gastric emptying time (No change in all trimester)

↑

(↑) during labour

⇒ Nausea & Vomiting → I line → pyridoxine + doxylamine

Last line → ondansetron

"Hyperemesis Gravidarum"

↓

this cause lot lose by excess Nausea and Vomiting

a) HCG Hormone

b) H pylori infection

c) Vernikes encephalopathy cause it

→ LIVER

AST (↓) ALP (↑)

ALT (↓)

↓

physiological

↓

due to placental release ~~placent~~

↓

placental alkaline phosphotase isoform

which is heat stable

Total albumin } ↑ absolutely
Total globulin }

(↓) levels in serum

→ SPLEEN (↑) 50 % in surface area

size
Increases

→ NERVOUS SYSTEM

Pituitary : Massive (↑) by 135 %

↓

It cause

Severe PPH → Sheehan's synd

↓

→ In the ant pituitary is effected

but pst " is spared

↓

a) M/c symp is failure to lactate

b) 2nd M/c symp : amenorrhoea

c) Women present in adrenal crisis

Prolactin is : (↑) in preg

Max (↑) seen in preg not in lactating mother

In lactating mother fall by 50 %

It is not required for preg maintenance

→ Milk let down Hormone
Milk ejecting Hormone } OXYTOCIN

→ Milk synthesizing → Prolactine

→ 1st stimulus for initiation of lactation is
fall in Progesteron and Estrogen

→ THYROID ←

~~Hypertrophy~~

a) Gland undergoes Hyperplasia

b) goiter: pathological

c) TBG: Thyroid binding globulin (↑) in preg

d) Serum T3 & T4 (↑) in preg

Hcg α → Thyroid to stimulate
↓
(↑) T3 T4

e) TSH (↓) slightly (N Range)

f) Preg & Euthyroid state

I2 Requirment (↑) → RDA - 250 mg/dl

I2 excreation in preg (↑)

M/c cause of Hypothyroidism in preg: developing country
I2 deficiency
developed countries
Hoshimotos thyroiditis

→ K/c/o Hypothyroidism
↓
women preg
↓
↑ the dose by 50% in Rx of Hypothyroidism

→ Insulin Levels ← (↑)

→ Growth Hormone (↑)

→ cortisol (↑)

→ PTH ↓ in 1st T
↑ } 2nd/3rd T
↑

→ Maternal Hormones that do not cross placenta
① Insulin
② TSH
③ PTH
④ Calatonin
⑤ Erythropectin

⇒ M/c Nerve injury Lat cutaneous N of thigh > Femoral Nerves

→ RENAL CHANGES ←

kidneys enlarge (length 1-1.5 cm)

RBF = ↑ 80% (2nd T)
= ↑ 50% (3rd T)

** GFR = ↑ by 50%

⟹

		Pre eclampsia
S. creatinin	↓	↑
BUN	↓	
Uric acid	No change	↑

→ Hydroureter (Hydronephosis)

Rt kidney > Lt kidney

↓

bcoz the uterus is Dextro rotated

↓

it compresses the (Rt) ureter on pelvic brim

→ (↑) Bladder Pressure

↓ 8 cm H_2O → 20 cm H_2O

it leads to stress urinary incontinence

→ Asymptomatic Bacteriuria Incidence is not (↑) in preg

all preg women are Rx for this

↓

if this is present

↓

No treatment

↓

pyelonephritis (25%)

treated with NFT
_ Nitrofurentoin

→ Gross body Movements
seen from 7 weeks

Fetal swallowing : seen 10 wks
↓
Fetal urine production : 12 wks

Fetal breathing movement : 11 wks

Meconium : 16 wks

IgM (only antibody) : starts 2 wks

IgG (from mother) : transport 16 wks

surfactant synthesising : 20 wks
↑ AF : 28 wks

ACTH : 7 wks

glucagon : 8 wks

Insulin : 12 wks

Ant pituitary : 12 wks

TSH : 12 wks

Gonads : Male 7 wks
Female

Int genitalia : 10 wks

→ USG and Early Pregnancy ←

In USG : G sac on TVS → 4^{1-3} weeks (4.5 week)
4 wks 1 to 3 days
↓
a) empty bladder
b) Higher frequency (less depth)

TAS : 5 weeks (Full bladder)
(Low frequency → More depth)

→ G sac + white color around sac
↓
Intradecidual sign

→ Double decidual sign ⇒ 2 sacs
1 sac : decidua capsularis (inner)
2 sac : decidua parietalis (outer)

→ Double bleb sign : Yolk sac and Aminotic sac
bleb

→ Earliest time G sac on USG : 5 weeks
↓
(TAS) = 5 weeks
(TVS) = 4 weeks

→ Earliest time G sac on TVS from LMP
4 week 1 days = 29 days

→ " " " " " " fertilization
↓
(−) 14 days

→ Yolk sac : Cardiac Activity
TVS 5-6 weeks
TAS 6-7 weeks
CA on USG → 6 weeks (if type of scan is not mentioned)

→ Amniotic Fluid ←

a) Source : Main source : Fetal urine

Source in 1st 12 weeks : Maternal plasma

Ultrafeltrate of " "

Source in 12 - 20 weeks : Fetal skin

> 20 weeks : Fetal Urine

b) Colour : Colourless (or)

Strach colour.

c) pH : 7.0 to 7.5

↓

More Imp

d) Made up of Water 99%

↓

(No Nutrition function)

Osmolality of AF : 260 mosm

water gets replaced every 3 hrs

e) Colour changes with certain pathologies

① Green - Meconium stained

② dark - abruption

③ dark brown - IUD

(tobacco juice)

④ greenish yellow (saffron) → post dated preg

⑤ Golden - Rh incompatability

f) Max Amount of AF : 32 wks Max

(32 - 34 wks)

12 wks 50 ml

16 wks 250 ml

[illegible] 400 ml

32 1Lit

36 900 ml

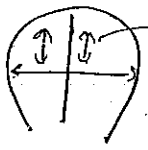
40 (term) 800 ml

42 weeks 200 ml

AFI — DVP (deep vertical Pocket)

m/c better

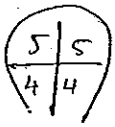
(n) 5-2~~4~~ ~~2-8~~



→ vertical value
(value in cm bcoz vertical)

2—8

↓



18 cm

Oligohydrominous	≤5	<2	<200mL
Poly hydrominous	≥25	≥8	>2 lit

→ m/c/cause Poly/oligo

↓

If Mild → Idiopathic

Moderate to severe → gCA (gross congenital anomalies)

↓

Poly → Fetal cannot swallow

Oligo → No urine passed by Fetus

Poly	Oligo
① GIT anomalies and NTD ex: cleft palate	① Renal anomaly ex B/L renal agenesis PUV (post urethral valve)

Polyhydrominous problem to Mother

a) Cause Resp distress

b) Preterm labour

c) Malpresentation

d) Abruption → controlled ARM (sterile needle & small pin holes made)

e) PROM (Pre rupture of Membr)

f) Cord prolapse

↓

when falls outside

↓

Sudden temp changes

↓

vasospasm of cord

↓

fetal distress

↓

Obstetric emergency

↓

Caesarean Section

↓

if only (cord pulsations) are present

↓

if absent the baby is dead

⇒ cot is not a creteria

⇒ Gestational age is Imp in cord prolapse

g) AFE amniotic fluid embolism

h)

→ Oligo Complication

1) Early onset → Pulmonary hypoplasia
2) Cord compression
3) PE (Pre eclampsia)
4) IUGR
5) IU infection
6) Amniotic band sequence — tears in amniotic membrane ↓ tears forms bands ↓ bands wraps around the fetus ↓ fetus does not receive vascular supply ↓ M/c they cause limb amputation ↓ 2nd M/c craniofacial abnormality
7) Amnion nodosum
8 Oligo

→ Indication for diagnostic Amnio ←

① Aneuploidy (Chromosomal Anamolie)
karyotyping

② Neural tube Defect AF - AFP
↳ Ach E

③ Haemolytic Anemia

④ Chorioaminonitis

⑤ Acute viral infections in fetus
↓
AP PCR analysis

6) FLM

Fetal lung Maturity.

M/c test L/S ratio → >2

Best test ~~Phosphatidyl~~

Phosphatidylglycerol

7) X Linked disorders

Inborn errors metabolism

29/JUNE/2017

PATHOLOGIES:

POST PORTUM HAEMORHAGE

3rd stage of labour: Placental expulsion

↓

Imp property is uit. contractions

↓

Line of separation: (Spongiosa) lyer separation

decidua spongiosa

duration: 20 mins

Prolonged: >30 mins

4th stage of labour: Stage of observation

↓ → 1 hr

↓ blood loss

ot resolution

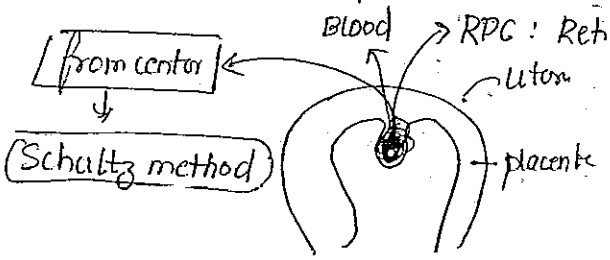
shivering (↑) ⇒ 4 stage

Signs & symptoms of placental seperation

1. Suprapubic bulge
2. gush of blood
3. lengthening of the U-cord → (Apparent) lengthing / (Permanent) lengthening
4. * Fundal Height (↑)
   ↓
   placenta come down and push up uterus

⇒ Best sign: Placenta lying in the (vagina)
(or)
Permanent lengthening of the cord

Placental seperation: 2 Methods



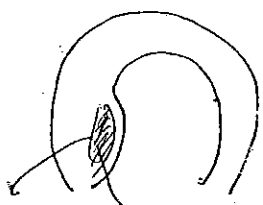
from center → (Schultz method)

80%

ext bleeding: only after complete seperation

→ Less blood loss

→ Presents at vulva fetal side
↓
Schultz method - Smooth shiny



from periphery → comes out from vagina → (Duncan's Method)

20%

Begin c̄ the (onset) of seperation
ext bleeding
→ More blood loss

→ D = Dirty
maternal side

→ If the placenta is not seperated
↓
1st give oxytocin (promotes ut contraction)

⇒ Normal blood losse :

Vaginal Deliver	: 500 ml	If this volume exceeds cause PPH
C.S.D	= 1000 ml	
twin VD	= 1000 ml	
C.S Hysterectomy	= 1500 ml	

⇒ PPH is blood loss such that causes fall in Haematocrit ≥10%

1° Haemorhage : < in 24 hrs
2° Haemorhage : ≥ 24 hrs

2° Haemorhage : ≥24 hrs upto 12 weeks

M/c/c of 1° Haemorhage : 1° PPH
M/c/c of 2° " : Retained Placental tissues

Cause : 4 Ts : ① Tone : Abnormal tone
1st leading cause ② Trauma : to ut tissue
2nd leading cause ③ Tissue : Retained tissue
④ Thrombosis : defects in thrombosis

Risk factor of ut atony :

Overstreching — Over contraction and relaxation

1. Multifetal preg
2. Macrosomia
3. Polyhydromion
4. Induction of labour
5. Augmentation " "
6. Precipitate labour
   ↓
   (labour ≤ in 3 hours)
7. fibroid in uterus
8. Pre eclampsia
9. DM →
10. Any kind of infection in ut :g: Chorioamnionitis
11. Multiparity

Z ~~Methods~~ Matural mortality in India
↓
(Hemorrhage > Anemia > PPH > APH)

* * * Prophylaxis for PPH (AMTSL)

Active management of Third stage of Labour

(WHO guidelines)

1. give uterotonic agent Immediately after delivery of the baby.

Uterotonic agent : DOC : oxytocin
↓
~~C~~ in 1 min

Oxytocin : ① 10 IU given

② I/M Bolus

I/V Infusion (Not Bolus)

③ Recomended IM Bolus (onset take 3 min, action lasts for 3 hrs)

④ IV infusion (onset of action: immediate)
(Last for 1 hr)

⑤ IV Bolus : Causes Hypotension
↓
arrythmias / MI / Cardiac arrest

⑥ Syn of oxytocin : Hypothalamus
↓
~~Paranuclear~~
Paraventricular nucleus

octapeptide (synthetic)

Nonapeptide (Naturally)

⑦ t1/2 : 3 min (3-5 range)

⑧ Ideal storage : Cool storage [2-8°C]

⑨ Mechanism of Action : Oxytocin
↓
① Ca into uterus
(Ca)
② Cause release PG_2 & $F_{2\alpha}$ from decidua
↓
Contraction

(10) type of contraction: Physiological ut contraction
- Contract
- Retract

(11) OTHER DRUGS:

(1) Methergin (1) 0.2 mg
(2) IM Injection
(3) comes in ampule Brown in colour Bottle (Photosensitive)
(4) S/c transient Hypotension
a) PE
b) Eclampsia
c) HD
d) PVD
e) after delivery of 1st baby in twin
↓
bcoz: ↓ contraction in (Lower segment) is atonic contraction
f) Rh-ve preg
bcoz: i) ii)

(2) Syntometrine
5 U oxytocine + 0.5 mg methergin
(Very potent)

(3) CARBETOCIN:
(1) synthetic oxytocine analogue
(2) octapeptide
(3) Longer t½

★ ④ 100 mcg IV over 1 min

★★ ④ MISOPROSTOL

★

ⓐ PGE1 analouge

ⓑ Dose: 600 mcg per oral

(only drug given [oral])

(whenever injection cannot given tab can begiv)

★ ⓒ can be given to Asthmatic

(it is bronchodilator)

ⓓ S/E: Hyperthermia

Chills

it does cause Nausea, Vomit, abd pain

most imp S/E are. Fever & chills which are dose dependent

2nd METHOD

Delivery of placenta by controlled cord traction

[Modified Brandt Andrews methods]

controlled traction: forced traction cause uterine inversion

3RD METHOD

CORD CLAMPING [LATE]

Delayed

late (or) delayed cord clamping: ≥ 1min

↓

bcos it provides blood to baby

80 ml blood
↓
50 mg of Iron in it
No difference of 2 gm/.

Early cord clamping : ① Baby Needs Resuscitation
↓
(only in term baby)

② Also in : Rh -ve pregnancy

⇒ ~~Deta~~ in HIV tranmission also Delayed is recommended

③ Heart disease in Fetus

4th METHOD :

INTERMITTENT UTERINE TONE ASSESSMENT
↓
for every 15 min
(Uterine massage is not component)

Most Imp Component of AMTSL is 1st method
↓
OXYTOSINE
↓
Reduce blood loss by 50%

Despite AMTSL : Some steps into PPH
↓
then treatmented should be done
in logarythm form
↓
1 after another methods

TREATMENT

Symptomatic | Specific

1) Secure 2 large bore IV cannula (≥16 gauge)

2) IV fluids : Crystalloids

3) Arrange Blood & Blood products

4) Catheterise Bladder

5) Blood group / Rh / Hct / coagulation Profile

SPECIFIC (Stop PPH)

FIRST STEP: Bimanual uterine massage + Call for help

→ Rt hand into ut ; Lt hand suprapubic

→ then uterotonics

DOC: oxytocin (IV fusion)

↓

① 20 IU to 40 IU / 500 ml

② given in NS (or) RL

(in 5%D not given)

↓

large close

↓

long duration

↓

in a electrolyte deficient media

↓

Cause water-intoxication

⇒ If oxytocin is not Acting
↓
THEN Give

Inj TRANEXA (IV in 20min)

⇒ If these two not acting

THEN GIVE: CARBOPROST / methergin 0.2mg IM
↓
① Methyl analogue $PGF_{2\alpha}$
② 0.25mg IM
③ C/I, in ~~Bor~~ Asthmatic (Bronchoconstrictor)
④ M/c. S/E a) Diarrhoea
Can cau. Nausea, pain, Vomiting
b) Hypertension
⑤ 8 doses in 24 hrs at 15-30 min intervals
and given in 8 times

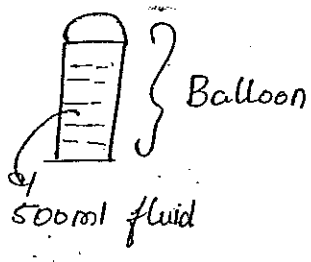
MISOPROST: → It is not effective than oxytocin
↓ → it is given only when Injectables cannot be given

Dose: 800 mcg : Sublingual: Immediate action

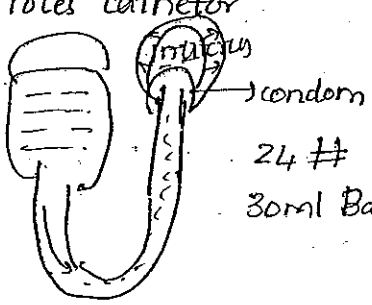
if all these done but still pt bleeding

Placenta completeness · genital tract trauma
↓ (Pl remains) ↓ (ut is atonically contraction)
↓
(ut is Intermittent Atonicity contraction)

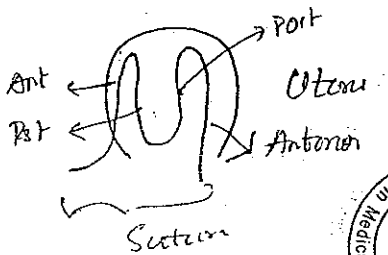
Uterus atonicity
↓
then Do: Balloon Tamponade
↓
we can use Sengstaken Blake move
(or)
use Bakri Balloon



Foles cathetor



if Balloon tamponade failure
↓ ← But in b/w these two there is
Surgical Managment
↓
⇒ Ut compression sutures
① B Lynch sutures (or)
Brench stures
② Cho square sutures
③ Hayman's sutures



Minimally invasive Method
↓
(i.e Ut artery Embolisation)
↓
Stops blood to bleeds from ut artery
↓
UAE: Pt must be Haemodynamically stable and facility of UAE must be there
↓
And Next comes Sx Mang

↓ Still Bleeding

B/L Ut Artery ligation

↓

B/L ligation of Ant Division of Ut A ligation

↓

Site: 5cm distal to the Bifurcation of common iliac

It reduces pulse pressure in pelvis by 80%

↓

It forms the thrombus

LAST METHOD: HYSTERECTOMY

↓

(Not the TOC)

If not we can do: Subtotal (Supracervical) Hysterectomy

↓ If Haemostasis is not maintain

then Do

↓

Total Hysterectomy

↓ still bleeding

2 Reasons (Post Hysterectomy)

① DIC

② Slopping of ligature

DIC: ① correct

② UMBRELLA (or) PARACHUTE PACK in the pelvis

↓

Only in Pst Hysterectomy pack

→ GENITAL TRACT TRAUMA ←

trauma to perineum (or) Vagina

⇒ Perineum

Perineal Tear

↓

preventional methods

① support to perineum

★ ② Apply warm compression on the perineum

NICE / RCOG guided

↓

New guideline (or) latest

③ Control delivery of Head

Head should be in flexion

Episiotomy

↓

① Surgically planned

② Epsiotomy not to be given as routine

③ Episiotomy only in special circumstances, like

⇒ Instructmental Delivery

④ Given in Mediolateral

↓

disadvantage: a) Blood loss more

b) Pain more

c) dyspareunia more

d) healing poor

e) cosmetically poor

only advant is no Anal sphinter extension

⑤ If given Medially

↓

extended into Anal sphinter

⑥ Angle

Grades of Perineal tear

Grade I : Perineal skin / Vaginal Mucosa
↓
II : + Muscles of perineum

III → A < 50% EAS torn
→ B > 50% EAS torn
→ C Both EAS & IAS torn

IV : Sphinctor : Rectal Mucosa

Managment of Perineal tear

Grade 1 and 2 : in Labour Room itself

3 and 4 : ① in OT (for analgesia + Muscle relaxation)
↓
local (or) General

② Regional anaesthesia

③ C̄ in 24 hrs (obs emergency)

⇒ Sequance of Structures that are repaired

Polyglactin
↓
(VICRYL)
for sutures

Rectal Mucosa } continuous sutures
↓
IAS
↓
EAS } End to end anastomosis
↓
Epsiotomy
↓
In epsiotomy : Vaginal mucosa → continous sutures
↓
Muscle → Interrupted
Bleeder - 8
↓
Skin → Mattress Sutures

Haemotoma
- Vulval → Branches of Int pudendal Artery
- Vaginal → Branches of Ut Artery

M/c/p of Haemotoma: Pain on the Local site
(or)
Inability to pass urine
(or)
they present in shock

Local examination; Bluish tender swellings

Management: Conservative
analgesics + Ice packs

⇒ surgical managment in

① Pt present in shock

② Expanding Haematoma

③ Excruciating pain
↓
Int expanding into the tissue
↓
into the Muscle layer (Carefull)

Sx: Incision and drainge I/D

⇒ if shock (ie int bleeding)
No I/D should be done
↓
Do emergency Laprotomy

⇒ CERVICAL TEARS:

① M/c site 3'o clock >> 9'o clock
↓
left side

② sponge Holder should be there to seen

Cx tear

↓

2 sponge Holder are used (Minimum requirment)

↓

12

9 3

6

⇒ UT TRAUMA

Inversion — Rupture (life threatening)

Inversion: Grades of Inversion

Inversion means Fundus travels through its own cavity

Grade I Fundus still in ut cavity

II Cross int OS

III outside the Vagina

IV : Vaginal wall are inverted

During inversion: there will be intense stretching on the ut ligaments

↓

Neurogenic shock

↓

atonic

↓

Haemorhagic shock

↓

Death (Haemorrhage is cause of death)

Managment of ut Inversion

① try for the manual reposition

↓

for (reposition) ~~but~~ should be (relaxed)

the part that comes last should put in reposition first

JHONSON'S METHOD (manual method)

↓

ut relaxation — NO / terbutaline

↓

HYDROSTASTIC METHOD (osullivan Method)

↓ (fails)

SURGICAL OT

→ HUNTINGTON AND HOULTAN'S

Huntington > Houltan's

⇒ spinelli method (sx done vaginally). Not used Now a days

⇒ lady develops shock after normal delivery most propable cause: PPH

⇒ Lady developed into shock immediately ~~after~~ (or) Just after delivery most propable cause: Inversion

⇒ lady goes into unexplained shock immediately after delivery: M/propable cause: Amniotic fluid embolism

AFE

⇒ AFE is also called Anaphylactoid synd of Pregnancy

AFE into pulmonary circulation

AFE : 2 phases

1st phase: (Do not survive in 1st phase) Very high mortality

① sudden onset of breathlessness

↓

Hypotension

↓

Cardiac arrest

↓

Coma

clinical Assessment:

→ Diag of Exclusion

→ neither confirmatory (Cos)

2nd phase: DIC

Haemorrhage

M/c/c of DIC in obstretic: ① ABRUPTION

② AFE

③ massive Hgc (APH/PPH)

④ IUD ⇒ risk of DIC (↑) only after 4 weeks

⑤ sepsis

⇒ In IUD the management is ~~watch & wait~~

Watch & watch

↓

Pcos in IUD the Female undergo spontaneous labour ∑ in 2 weeks

→ sign in IUD: Robert's sign (12 hrs)

other sign: Spalding's sign (7 day)

Hyperflexion of spine / Ball sign

→ UTERINE RUPTURE

~~① M/c/c : Previous C.S~~

② Nonscared ut rupture : a) obstructed Labour

b) Grand multiparae
↓
More 5 babies delivery

c) Ut anomalies

d) Drugs :⇒ misoprostol
↓
not used for augmentation
and C/I in Prev C.S

⇒ oxytocin (used for augmentation)

③ Managed by Emergency laprotomy
↓
Rupture Repair
↓
and then not No Haemostasis
↓
Hysterectomy

→ PRE- ECLAMPSIA ←
(Hypertension)

Gestational Hypertension:

① BP ≥ 140/90 mmHg on 2 occasions 4 hrs apart

② >20 weeks of gestational age

③ No proteinuria

④ return to ⓝ c̄ in 12 weeks postpartum
conformatory pnt

Preg women: 5th Korchoff sound (diastolic)
N women: 4th korchoff sound (diastolic)

Both systolic / diastolic
(or)
any one is high } PE

⇒ BP checked in sitting position

⇒ supine hypotension synd seen physiologically in all preg women

Pre eclampsia

① BP ≥ 140/90 mmHg on 2 occasion 4 hrs apart

② >20 weeks (beyond 20 weeks of GOA)

c̄ proteinuria
⇒ 300mg (or) 0.3gm in 24 hr urine sample

absence of proteinuria
↓
any of the following
↓

⇒ urinary protein / creatinine > 0.3

⇒ 1(+) in dipstick method (not ideal)

↓

① Platelet count < 1 lakh

② liver enzymes > twice the ① value

③ Serum creatinin > 1.1

④ pulm oedema

⑤ cerebral / visual symptoms

Once there is a Endorgan damage

↓

Severe Pre eclampsia

⇒ Severe Eclampsia:

BP ≥ 160/110 mmHg 2c 4 hrs apart

⇒ S.P.E removed criteria

① Oliguria

② Early IUGR

③ proteinuria > 5gm/24 hrs

→ ECLAMPSIA ←

Pre eclampsia + Seizure

→ Chronic HPN in Preg ←

↓

① k/c/o HPN before conception

② High BP in 1st 20 weeks

③ BP remains High > 12 wks after Postpartum

→ Chronic HYP T superimposed Pre eclampsia

① New onset proteinuria > 20 weeks

② End organ Injury > 20 weeks

③ Uncontrolled BP > 20 weeks

** Risk factors for Pre eclampsia

① Primigravida Women

* ② Prev H/o Preeclampsia in prev preg

③ known renal disease

④ known DM

⑤ Chronic Hypertensive

⑥ Extreme of Age
≤ 18 yrs to ≥ 40 yrs

⑦ APLA

⑧ Women who has molar preg

⑨ Multifetal preg

⑩ Obesity

Protective Factors for Pre eclampsia

① Smoking

⇒ there is +ve ass b/w Pre eclampsia and abruption
" " -ve " " " " Placenta Previa

Pathophysiology:

Extravillous endo cytotrophoblast
↓ Invasion
spiral arteriole in D. Basalis
(Rm)

↓

Vasocontriction

↓

Reduction in Uteroplacenta Flow

↓

Uteroplacental Insufficiency (UPI)

↓

Placental ischemia undergoing

↓

Ischaemia — Causitive organ is placenta

↓

it releases a few things into maternal blood

↓

(↑) sFlt-1

(↓) PlGF, VEGF

Inflammatory mediators

↓

enter into maternal blood

↓

Rise BP and damage capilary endothelium

↓

by inflamatory mediat

(Soluble fms like tyrosine kinase 1)

S. Eng (endoglin)

Vasopressor

↓

BP (rise)

leakage

(↓) IV Volume

3rd space elementes

leakage

→ odema

Hb Haemoconcentration

capilary of kidney (End organ damage)

(&)

capillary of brain (Cerebral symptoms are presented)

↓

(end organ damage)

M/c organ damaged 1st in PE: kidney

glomerular endotheliosis in H/p examination

Histopathological

⇒ 2nd ~~H/p~~ pathology ↑ TH_1 response ↓ not suppressed

[(N) in preg TH_1 ↓ suppressed and switch to TH_2]

⇒ Only specific Mx in Pre Eclampsia is TOP

↓

removal of placenta

→ PREDICTORS

① Ut Artery doppler

Notching → disappear by 22 wks

↓

due to vascular remodeling

⇒ if Notching present beyond 22 wks it will be a predictor

② New things of predictor

↓ VEGF ↑ s-Eng

↓ PLGF ↑ s-Flt 1

③ Findings (Not Predictors)

① Haemo ~~centric~~ concentration

② oedema

③ ↑ uric acid

⇒ Roll over test done at (28 - 32 wks)

(Considered to be a predictors but not now a days)

... Not a predictor

Roll the women from (Lt) Lat supine position

↓

Pre eclampsia

(↑) mean arterial BP

Prevention : → If only the women has ~~risk~~ high risk factor

① Low Dose Aspirin

80 mg OD X 12 wks ⟶ Continous throughout preg

② Ca supplementation

(in some condition) not in everybody

only in women ∵ ↓ Serum Ca befor conception

IMPENDING ECLAMPSIA (IE)

↓

Impending Seizures

a) So prevent her from seizures

b) Sign & symptoms : ① Epigastric pain, Nausea, Vomit

Subcapsular hematomas in liver

Strechin of capsule

② Headache / dizziness
↓
① cerebral Hypoxia
② at any location

③ Visual disturbances
(blurring of vision / diplopia / Scotomas (or) Blindness
↓
Due to HTN retinopathy
↓
central / occipital lobe Hypoxia
(or)
Retinal detachment

central blindness will return after delivery

⇒ Keith wagner's classification (Silver wiring / papilledema) } having IE

→ # ELLP SYND ←

ⓐ usually a feature of Severe Pre eclampsia
ⓑ diag of HELLP synd.
i° Haemolysis
↓
Peripheral smear : Schistocytes / Helmet cells
↓
If these cells are present
↓
Haemolysis

ii° T Bil ≥ 1.2

iii Elevated liver enzyme
AST ≥ 70

(v) low platelet count

LPC < 1 lakh

⇒ upto 15% - women may have (N) BP

↓

So BP is not a criteria in HELLP synd

⇒ 3rd trimester

M/c Pain: Epigastric pain (at (Rt) upper quandrant)

Other s/s: ~~Peripheral~~ Pre eclampsia

D/D: 1) HELLP

2) Acute fatty liver Preg (AFLP)

3) acute hepatitis

4) obstetric cholestasis

AFLP is more severe HELLP like synd

① Hypoglycemia

② Hepato renal synd

③ coagulation disorder

④ Development of 2° complication like pancreatitis

these features only in AFLP but not in HELLP

⇒ M/c/c of Acute Liver failure in preg: AFLP

⇒ High Mortality

⇒ conformation is PP liver biopsy

⇒ Liver span is reduced in AFLP.

→ ACUTE HEPATITIS ←

① → prodromal symptoms:
→ fever

② Liver enzyme (↑↑)

③ S. Bili (↑↑) (>10)

⇒ M/c Hepatitis (B)

M/c acute Hepatitis (E) → Very High Mortality rate

→ ***
OBSTETRIC CHOLESTASIS ←

① M/c/p : pruritis

② M/c/Pore : 3rd Trimester

③ Cause : Estrogens + Progestron
↓
single Best answer

** ④ Genetic involvement : ABC B-4
↓ ABC B-11
due to an enzyme Def called (LCHAD deficiency)
↓
In fetus the deficient
↓
it transforms in to mother

(↓) conjugation of Bile salts
(↓) excretions

△ Statusis Serum Bile salts

pruritis → more on Palms, Soles → More at night

↓

We have to give

↓

Ursodeoxycholic acid

(UDILIV)

↓

cause conjugation of Bile Salts and promote excreation

* Usually s. Bil [<5]

Fetus : (↑) Risk of still Birth

(5) women with obstetric cholestasis

do TOP (Not less than 37 weeks)

only above 37 weeks

⇒ Recurrance rate of OC 65-70%

Recurrance " " HELLP 4-7%

Manangment of Preeclampsia

Non specific (Symptomatic

Specific → TOP

stop & reversal the condition

BP- Anty hypertensives

Seizures - Anitconvulants

} They not essefur in PE but they reduce mortality and morbidity

Anti hypertensives

→ Indication to start Anti HTN

① BP ≥ 160/110 (severe PE)

Persistently → ≥ 150/100

a) DOC: HTN in preg is LABETALOL
↓
(α + β blocker)

b) DOC: chronic HTN in preg: METHYLDOPA
Safest Anti HTN in preg: ,,

c) Acute HTN in preg:

① Labetolol (I.V) > ② Hydralazine (I.V) > ③ Nifedipine (Sustained release oral tab) — 1st line drugs

↓

20mg I.V Inj

↓ wait 20 min / Neo guaid lenc 10 min

40 mg

↓

80 mg

possible
Max dose giving in 24 hrs : 300 mg

d) I.V Nitroglycerine NTG
for pulmonary odema

e) Sodium Nitroprusside → cause cyanide toxicity
↓
refractory HTN

Contraindicated Anti HTN in Preg

1. ACEI
2. diuretics ⇒ don't give it has routine Anti HTN
3. B Blockers
4. ARB's
5. Diazoxide (Acting on K^+ channels)

Targets : S.BP = 130-140 mmHg
BP = 80-90 mmHg

→ TERMINATION OF PREGNANCY ←

⇒ All pt's of gestational HTN & MILD PE = 37 wks
even BP well controlled

⇒ S. PE (well controlled) = 34 wks

⇒ V.D > C.S

⇒ TOP (irrespective of POG)

---

1) Impending Eclampsia
2) Eclampsia
3) Fetal distress / abruption
4) uncontrolled BP (or) worsening of lab parameters

Most imp S.creatanine
↓
↑S.C leads to renal failure

HELLP synd

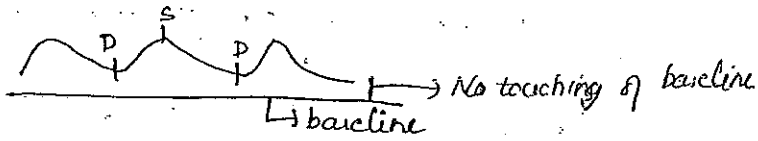
5) REDF (Reversal of end diastolic flow)

↓

Umbilical Artery Doppler

S/D ratio $\frac{\text{Systolic}}{\text{Diastolic}}$

⇒ S/D ratio (N) ↓ing as POG ↑ing

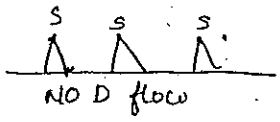


Uteroplacental Insufficiency:

Initially E/D (↑)

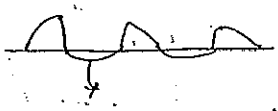
↓

TOP beyond 34 weeks ← AEDF → Absence of end diastolic flow



↓

TOP irrespective of Gest age ← REDF →



Reversal of end diastolic flow

↓

TOP (in REDF the baby dies in 48 hrs)

S/D ≥ 30 weeks → Tell about UPI

↳ ≥3

⇒ A placenta in Pt of PE the: it is small size (1)
pale colour (2)
and U/S show calcifications (3)

⇒ Eclampsia

PE + seizures

↓

GTCS (grand mal)

1) cerebral hypoxia
2) cerebral edema

Seizure that seen can be 3 phases

- Antepartum (M/c)
- Intrapartum
- Postpartum (L/c) → (only for 48 hrs after delivery)

⇒ Shorter seizure phase → better in prognosis

Symptomatic — Specific TOP

↓

tap (name)

↓

No response

↓

PR = 100/min → check Pulse & BP

BP = 160/120 mmHg

↓

stabilize the position

(she shouldn't get a fall)

↓

Protect airway → Mouth gag
→ O_2 by mask
→ aspirate secretions

↓

Inj: $MgSO_4$ (DOC in preg c̄ Eclampsia)

- It acts centrally i.e. brain → Not peripherally
- acts on NMDA receptor in brain
- It cause cerebral vasodilatation

↓

Anti HTN should be given (peripheral action)

⇒ $MgSO_4$ + CCBs Should Not be combined

$MgSO_4$ loading dose (IV + IM)
4gm 10gm

Maintainance Pritchard's Regimen

~~Loading~~

4gm → 20% solution → Slow IV

10gm → 50% solution → In buttock

Rate should not be more than 1gm / 1min

IV dose acts on Ca Channel blocks
blocks the Ca
↓
if given fastly the Ca is blocked and Ca omeat put in
↓
So check HR (or) PR

Maintainance Dose
↓
(every 4 hrsly)
5gm% (50% solution)
Deep IM → alternate site every time
↓
or else it forms the abscess

Befor maintainance dose given check for

⇒ 2gm I.V $MgSO_4$ even seizure came after loading dos

a) Deep tendon reflex (patellar reflex) should be present
B) Urine out ≥ 30ml/hr
c) Resp rate > 12/min

if absent; omit the dose of $MgSO_4$ and send S. Mg levels

⇒ In E pt they have oligouria
↓
if oligouria persists
↓
$MgSO_4$ not excreted
↓
$MgSO_4$ toxicity occurs

→ Therepuetic toxicity: (1st sign: seen at 10 meq)

9–12
- loss of petellar reflex
- slurring of speech
- diaphoresis (feelers of Vomit)
- Resp distress

levels
- > 15 meq : Resp arrest
- > 30 meq : Cardiac arrest

⇒ if ~~dose~~ 10 meq given anti dote
↓
10 ml 10% IV Ca gluconate

End point: 24 hrs after delivery
24 hrs after last seizure which ever is later

⇒ Prophylatic $MgSO_4$

Who have ① Impending E
② S.PE
③ HELLP synd

⇒ TOP: VD > CS

for anesthesia

blood loss

Deliver should be ≤ in 24 hrs from 1st seizure

Clinical Q) Primi 34 wks, c/o headache & 2 episodes of vomiting since morning → PR = 106 bpm

BP 170/120 mmHg, Urine acid 3+, FHR (+)

↓

Headache + Vomiting → Impending eclampsia

↓

Mx

a) MgSO4

b) Anti HTN given

c) TOP

→ DIABETES IN PREGNANCY ←

Pregestational
↓
also called as (OVERT DM)
↓
K/c/o type I/2 DM

Gestational
↓
The deranged Blood sugar are first diagnosed in Pregnancy
↓
IAPDSG [WHO] (International association of pregnancy diabetes study group) | DIPSI Criteria

common : 24 - 28 weeks (test should be done)

Insulin resistance get significant and blood glucose is raised

single step test
75gm oral glucose
both screening & diagnostic
2hr - OGTT (Oral glucose Tolerance Test)

IAPDSG

FBS < 92
→ 75gm of oral glucose
1 hr < 180
2 hr < 153

when even 1 value is deranged it is diagnosed as GDM

DIPSG

C (or) C out FBS
→ 75gm of Oral glucose
↓
after 2 hrs one blood sample
↓
2 Hr ≥ 140 → GDM
≥ 200 → Overt DM

⇒ to check GDM befor 24 weeks

1. significant family History
2. H/o DM in prev preg
3. H/o birth to macrosomic baby

④ H/o still birth

⑤ H/o gCA (gross congenital Anomalies)

⟹ Early
↓
1st ANC
↓
FBS (or) HbA1C

Glycosuria: It can be physiological
↓
Does not mean DM

(N) bcos in preg (Renal thersold ↓ed)

DM preg: High risk preg for both Mother & baby

→ Risk in baby

① gCA : Risk ↑ed 2-4 fold in a diabetic
5% fold

→ Hyperglycaemia: It is fetotoxic
↓
Mechanism is free radical injury

(gCA is only seen in overt DM)

M/c/anomaly: CVS > NTD

Most specific anomaly: Sacral Agenesis
Caudal regression Synd

M/c/ Cardiac anomaly: VSD

Most specific Cardiac Anomaly: TGA

M/c cvs finding: HOCM

⇒ finding is reversable: bcoz it a physiological adaptation
anomaly is not " : it is surgically corrected

Clinical Q) 8 week gest age, overt DM type 2
risk of ~~TGA~~ GCA gross congenital ~~Anomaly~~

Hb A1C test (for Hyperglycaemia)

<6.5 %	→ No risk
6.5 %	→ 3% "
7.5 %	→ 4% "
8.5 %	→ 8% "
9 %	→ 15% " (very high)

It only tells the risk but does not confirm whether the baby is affected c̄ GCA

For conformation do USG (If HbA1C do USG)

IOC = USG

→ level II USG (18-20 weeks) to best

→ anomaly scan

→ TIFFA → targetted imaging for fetal anomalies

When GCA can be picked up early on USG is Anencephaly

level II USG should be done in all preg women irrespective of DM

→ Anencephaly as early as 10 weeks
↓ ↓ Ideal = 14 weeks
in USG it look like: frog eye sign
Mickey mouse sign

⇒ But Screening test ⇒ USG

⇒ post term labour is more common than preterm labour
↓
Hypothalamus pituitary axis Not present
↓
CRH initiati

presentation: face presentation

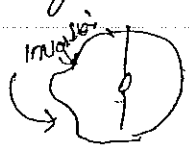
NTD: Screening for NTD
USG > S. AFP

→ diagnostic/best test for NTD: AF-Ache
Amniotic fluid Acetylcholenesterase

Recurrence Risk of NTD after 1st baby c̄ NTD : 2-4%
" " " " " 2nd baby " " : 10%

→ Holo Prosencephaly
Myelomeningocele
Hydrocephaly
encephalocele

→ Lemon sign and banana sign



Banana } Arnold chiari synd
Lemon } Chiari type 2 synd

→ Prevent GDM

1) tight control Blood glucose

2) ~~U~~FA (Folic acid)

↳ dose 4 mg

① diabetis
② antiepileptic
③ previous H/o NTD

Preg: Prophylaxis : 0.4 mg

~~FDA~~, IFA 0.5 mg in preg

3) High Risk women (ideal ~~dose~~)
↓
PReconceptionally given

Minimum requirement: 1 month before and period
3 month after conception

MACROSOMIA : the DM Mother develop it
↓
Hyperglycemia → Goss PL → Hyperglycemia features
↓ In mother
DED
↓
Pancreas → Hyper insulinemia → Inhibits lipolysis

Macrosomia Risk factors

1. diabetes
2. Male sex
3. multiparity
4. ↑ing maternal age
5. post dated
6. excessive wt gain in preg
7. obesity

⇒ 8. Smoking is Not a risk factor

⇒ Macrosomia (baby wt > 4 kg) done by USG

Abd circumferance ⇒ tells the growth of baby { IUGR / Macrosomia

Hockey stick sign (to measure the Abd circumferance)

In Rt plane : ~~Bowel sign~~ Bubble sign
↓
bubble of stomach

Double Bubble sign

stomach — Duodenum

⇒ Gestational Age Assesment

But is : CRL

1st trimester : CRL (upto 14 weeks)

2nd trimester : BPD (Biparietal diameter)

3rd " : FL

⇒ IUGR :

EBW < 10th percentile

Macrosomal baby undergo:

Shoulder Dystocia

↓

fat deposition is more in shoulder

↓

delay in the delivery (> 1min)

↓

Turtle sign

↓

☆☆☆ Sudden pulling back of the head ~~into~~ towards perineum

Mx of shoulder Dystocia

H → Help

E → Episiotomy & empty the bladder

L → 1st manuvre to legs → (Mc Robert's manuvre)

Sudden flexion and Abduction of the leg of mother to her abd

↓

S.P rotate

(Symphysis pubis rotate Cranially) ↑ available space

injury to lateral cutaneous nerve of thigh of the mother

↓

(Meralgia Paraesthetica)

Paresthesia of thigh

P – suprapubic pressure but not fundal pressure

↓

I/C in shoulder dystocia

E – Enter (wood's cork screw)

R – Removal of post Arm

↓

(Jaquemier's Manuvre)

R – Roll the mother on her 4 limbs

↓

(gaskin all 4's)

↓

but still it is not coming baby

↓

Last Manuivre done is

→ Zavanelli

↓

push back the head into uterus and take the Mother for C-S

⇒ Symphysiotomy } → symphysis pubis is cut off
Cleidotomy } → clavicle " "

M/c fetal complication : Branchial Plexus injury

M/c maternal : PPH

3rd complication of child in DM

⇒ IUGR : uncommon

Vasculopathy } UPI
Pre eclampsia }

4th) Still birth

a) 3rd trimester

b) Male sex

5th) Prematurity

↓

thu is iatrogenic (Mostly)

Spontaneous in DM Mother

6) lung maturity is delayed.

↓

test for lung maturity : Phosphatidyl Glycerol

7) After birth complication

↓

Hypo-insulinaemia

↓

① Hypoglycemia

②

② Hyperbilirubinaemia

③ Polycythemia

④ Hypocalcaemia } bicoz of prematurity

⑤ Hypomagnesemia

⑥ RDS (Resp. distress synd)

⑦ HOCM (Hypertrophic obstructive cardiomyopathy)

↓

it is reversable

⑧ DM later in life if both parents are DM

the risk is ↑ 50%

→ Maternal ~~Risk~~ complication of Diabetes ←

① Abortions = bicoz of uncontrolled DM

② Polyhydrominos = more common

↓

Reasons → { ↑ glucose theory
Polyuria
GCA (gross congenital Anomal)

③ Oligohydron

↓

less common

a) diabetic vasculopathy

b) PE

4. PE in DM risk is 15%
5. Infections → UTI and candidiasis
6. Pre Term labour : due to Infections
7. Malpresentation & Surgical procedures
8. (↑) PPH
9. 50% GDM will develop type 2 DM later in life

→ Managment of GDM ←

Fetal monitoring

1) Start at 32 weeks
2) tests : fetal movement
   Non stress test (NST)
   BPP (Biophysical profile)
   USG

→ NST should be done once every 72 hrs (or) twice a week

Mother Monitoring

- OVERT DM → diabetic diet → Insulin (when gets Preg) → (tight glucose control)
- GDM → only diabetic diet
  1. 40% - Carbohydr
  2. 40% - fats → (<10% saturated)
  3. 20% - protein

Monitor → after 72 hrs send blood sample for sugar profile

→ Sugar profile (7 samples)
FBS / post Breakfast / prelunch / post lunch / Predinner / Postdinner / 2am sample

↓

if diabetic diet is not sufficient for control

↓

put on drugs

↓

when FBS is >95

1hr PP is >140

2hr PP is >120

Oral Hypoglycemic Agents (OHA's)

↓

(Not going to control in overt DM)

↓

only for GDM

↓

Glyburide / metformine

these cross placenta

↓

but do not cause any harm

Insulin

↓

M/c/ insulin in preg is (Regular Insulin)

other Insulin:

Lente/ Aspart/ NPH

↓

(Insulin resistance (↑) ⊖ (↑) PDG)

↓

So Insulin requirment also (↑)

1-12 wks 0.7 U/kg

12-28 wks 0.8 U/kg

28-34 wks 0.9 U/kg

≥35 wks 1 U/kg

Target insulin levels

F<95

1hr PP < 140

2hr PP <120

Avg Capillary Sugar = 100

HbA1c → < 6

Delivery at 39 weeks

Non medical indication for C.S is not before 39 weeks

Preterm
- < 37 weeks
- 37-38^{+6} Early term
- 39-40^{+6} Term
- 41-41^{+6} Late term
- ≥ 42 - Post term

→ Diabetes is not incidate for Caesarean Section

In diabetes it depends on the weight

EBW (expected baby wt)

EBW ≥ 4.5 kg in a DM Preg → CS

Indication of CS in non DM mother is 5 kg

4 kg: Macrosomia

4.5 kg: CS in DM Mother

5 kg: CS in Non DM Mother

→ VAGINAL DILEVERY

Insulin requirment in labour Reduces

① night dose is given before the day of labour

② Morning dose is omitted (on labour)

③ Intensive glucose monitoring

if required - insulin given by sliding scale

→ GDM

[6 weeks → Post Natal checkup → GTT should be done]

→ ECTOPIC PREGNANCY ←

Any preg which gets implanted outside the uterine cavity

cornual pregnancy : name for ectopic

Ectopic in interstitial / intramural part of fallopian tube

Angular preg : Intrauterine pregnancy

⇒ If the growth of the uterus lies medial to the attachment of Round ligament.

↓

(Angular preg)

⇒ lat to R.L is cornual

Heterotopic : 2 pregnancies simultaneously at different site of implant

M/c type of Heterotopic : 1 in intrauterine
1 in Fallopian tube

★ Risk factors of ectopic :

Highest RF : previous ectopic

1st ectopic : 15%

2nd ectopic : 30%

2nd Highest RF : H/o tubal surgery
↓
when sx done
↓
it is healed
↓
c̄ fibrosis
↓
ectopic can be formed here

3. M/c Risk factor : PID

4. cervicitis

5. multiple sexual partners

6. Infertility

7. ART (Assisted reproductive techniques)

8. Smoking

9. previous C.S

10. contraception
↓
all contraceptive agents reduce the absolute risk of ectopic pregnancy

Sterilization > Intrauterine Device > Progesteron only pill
↓
(↑) Relative risk

IUD's :

Progestasert > Mirena > ~~[illegible]~~ CuT
↓ High risk of ectopic
↓ Progesteron containing IUD

⇒ H/o ectopic is not a Absolute C/I for IUD.

⇒ Max reduction in Ectopic seen by OCP
oral contraceptive pills

M/c site for Ectopic: Fallopian tube

L/c site " " : Cesarean scar > cervical ectopic > Abd ectopic

Fallopian tube → M/c : Ampulla
↓
L/c : Intramural / Interstitial
↓
Muscle of uterus

→ tubal ectopics:

1st site — Ampullary ectopic outcome : ① tubal abortion
② tubal rupture
↓
8 weeks

2nd site — Isthmus ectopic outcome : ① Rupture : 6 week
↓
(M/c ectopic to Rupture)

3rd site : Interstitial ectopic outcome: Rupture : 12 weeks

⇒ ~~ec~~ in the FT L/c rupture : Fimbrial

⇒ Most life threatening rupture : Interstitial rupture
↓
bcoz there will be rupture of
Uterine Muscle
↓
Large Haemorrhage

Ectopic traid presentation (only in 50 % pts)

Pain
Bleeding — Amenorrhoea

(other 50% pts)

→ Most consistent / most common is Pain

⇒ The pt also comes c̄

① Syncopal attacks
② shoulder tip pain } Represent Rupture ectopic

↓

Haemoperitoneum

↓

pain in diaphragm

↓

referred pain to shoulder

→ Rupture ectopic does not mean Haemodynamically unstable

they are Stable if they present ĪĒ in time

③ tachycardia

④ Hypotension

⑤ tenderness in Lower abd
(She has rigidity and guarding)

⑥ P/V : ut is usually enlarged
but less than the period of Gestation

* ⑦ Cx motion tenderness
it means Salpingitis
↓
It is also present in PID
↓
but in PID ut is not enlarged

⑧ Adnexal tenderness
↓
present also in PID

⑨ Adnexal Mass
↓
Most ~~sig~~nant finding in ectopic in 50% of women

Next step is : UPT will be [+ve] (99%)
↓
but UPT does not say the site so it is
Not IOC in ectopic

IOC in Ectopic : TVS

8). What is the 1st finding in USG that raises suspecision of Ectopic preg :

Presence of empty uterus
↓ Then go for adnex

Not diagnostic ← ~~Complete~~ Complex adnexal Mass both solid & cystic complex
↓ Then keep the Doppler
Ring of fire sign
There will be (↑) vascularity around Mass

→ Pseudosac → Central in location
Pointed edges
Seen in 20% Ectopics

true sac
eccentric
round edges

Diagnostic:

⇒ extra ut G sac $\bar{c}$ cardiac activity.

① No IU G sac

② B/L adnexal (+)

minimum fluid in POD

2nd Investigation for ectopics

Serial Hcg : Day 1 sHcg

↓

< critical (1500-2000) → ① Live IU preg.

≥ critical titer ↓ likely ectopic → ② Ectopic (it may ectopic) ③ it may be Abortion

Day 1

to → after 48 hrs

Day 3

Hcg (↑) 55-65 % ↓ Live IU preg

Hcg (↑) (<55%) ↓ ? Ectopic

Hcg (↓) ↓ abortion

We do this still critical titer is crossed

Most imp diagnostic Investigation: USG + TVS (or) USG + β Hcg

rd Inv → Gold standard Ix for ectopic ⇒ Dstic laproscopy

th Inv Serum progesteron level (N) in live IU preg ≥ 25ng

≤ 5ng { abortion / ectopic

th Inv: Culdocentesis: Aspiration of fluid from pouch of Douglas

Significant fluid in pouch of Douglas ≥ 100 mL

ere x are no : in opic agnosis

⇒ ① colpotomy
② HSG
③ Hysteroscopy

} If UPT is +ve ↓ C/I in Ix of ectopic

⇒ FAST (~~pt into OT to operation~~)

c̄ localized signs present (guarding & rigidity) and Haemodynamically stable
↓
should go to OT & operate

If No localized sign & stable → FAST

Managment

- Medical
- Surgical
- Expectant

For medical Mx pt should be
① Stable
② Hcg value should be ≤ 5000 IU
③ sac size { < 3.5cm if Cardiac activity present / < 4cm if " " absent

4) preferably absent cardiac activity

Methotrexate Inj is given → due to the sac undergo Necrosis
IM ① 50 mg/m² / ② 1 mg/kg
↓
release more HCG
↓
HCG is raised upto D4 and then ↓ised by D7

D4 → S. HCG
D7 → S. HCG
↓
Succesful Medical Mx is when D7 value should fall by a 15% from D4
↓
failed Medical Mx is when if we give 8 inj and the fall is not more than 15% it is failed
↓
if D7 value is ↑ing then go for surgically Mx

Surgical Mx ~~Either~~

① Unstable
② failed Medical Mx
③ Criteria for Medical Mx not met
④ Ruptured ut
⑤ family is complete

Surgical Mx

1) Laproscopy > Laprotomy

2) unstable vitals → Do laprotomy

3) Salpingostomy → Salpingectomy

↓

Small incision on the Antemesentric end

Remove the products by Hydro dissection

keep open the incision & not suturing (ostomy)

↓ opening

It heals by 2° healings

Indication of Salpingectomy

(a) Ruptured ectopic

↓

means products came out so no role in ostomy

(b) family complete

(c) sac size >5 cm

(d) if Haemostasis cannot be achived

Clinical Q)

Expectant Mx

① Hcg <200 and falling trend

↓

It means it is aborting itself

↓

Just monitor HCG & vitals

Types of Ectopics

① Cervical Ectopic

Thu presents c̄ painless bleeding

Paalman's criteria (New criteria)

Rubin's criteria (old)

Medically stable

② Abdominal ectopic → 1%

Studdiford criteria

- only pain no bleeding
- Present later as abd pregnancy
- So do Laprotomy (Not c.s)

↓ bcoz not uterus

→ In laprotomy cut the cord as short as you can delivery the baby and leave away the placenta inside Autolysis

③ Ovarian ectopics Spiegelberg criteria

↳ Sx.

④ Heterotopic Ectopic

one in IU and one in tubal

Highest Risk = ART (IUF)

Incidence is 1 in 30,500 (olden days)

Nowadays 1 in 3000

→ Mx of Heterotopic

Heterotopic c/I for Medical Mx

TOC in Sx Managment

Treatment is : laproscopic salpingectomy
↓
usuall in Heterotopic
the USG have IU sac so
tubal ectopic is missed usually
and presented in Impending
ectopic (or) Ruptured ectopic so
we do salpingectomy

Can give Injection KCL → USG guidance into the
ectopic sac

****

→ ABORTIONS ←

"Any preg loss at less than 20 weeks"
(or)
"loss at less than 500 gm"

⇒ wt of fetus at 20 weeks : 300 gm
at 28 weeks (viability) : 1000 gm

⇒ Abortion should be confirmed by Abortion
USG cretrria : (1) g sac MSD (Mean sac diameter)
≥25mm no embryo (or) yolksac
(Non viable preg)
↓
BLIGHTED OVUM → characterised by Avascular villi

2nd criteria

Non viable pregna → Crown Rump length is equal to 7mm and no cardiac activity

*** CAUSES.

Spontaneous abortions

Recurrent abortions → ≥3 consecutive abortions

Induced abortions : If USG are present ≥2 abortions
↳ by the doctors (MTP)

Causes of spontaneous

① M/c is : Chromosomal anomalies

and common in 1st and 2nd Trimester

1st → 50%

2nd → 35%

Which chromosomal anomalies M/c for abortion is ANEUPLOIDY

↓

Trisomies Aneuploidy

↓

Single most common cause. 1st option monosomy X

↓

20% of all abortions

2nd option : Trisomy 16 – 16% abortion

↓

Most lethal

Most viable Trisomy : Trisomy 21

↓

Seen in live birth

Causes of Recurrent preg. Loss

M/c/c of RPL : in 1st T

2nd T

Leading cause: Idiopathic (>50%)

↓

Next leading cause: APLA (Antiphospholipid Ab)

↓

Immunological (16% of abortions)

↓

Next

Ut structural Anomalies

↓

Endocrinopathies

↓

Chromosomal Anomalies (Only 4% cases of RPL)

⇒ TORCH infection does not cause RPL

but cause spontaneous abortion

⇒ SYPHILIS is only infection causing RPL

↓

Kassowitz law: ∈ every preg the POG ~~after~~ of low

keeps increasing

syphilis is characterised by (still birth)

⇒ Chromosomal anomalies = 4% RPL

which anomalies cause RPL →

Balanced Translocations

→ karyotyping should be done in RPL

Endocrinopathies causing abortions

① Diabetes (uncontrolled DM)

↓

In this uncontrolled DM in case of RPL

No routine BS assesments should be done

② Thyroid (Hypothyroidism)

TSH routine Assesment → Yes

↓

Subclinical Hypothyroidism can cause abortion

③ Hyperprolactinemia

PrL → routine Assesment → Yes

↓

~~M/c/p → galactorrea~~

M/c presentation HyperPrL → Infertility

④ Poly cystic ovarian synd (PCOD)

⑤ Leutial phase defect (LPD)

↓

S. Prog <15 ng (Cause abortion)

But tat: Endometrial biopsy

Rx: Prog supplementation

↓

M/c prog type given is OH prog (Hydroxy) capronate IM. weekly

(Newer) micronised prog tab

→ Ut Anomalies

Congenital — Acquired

M/c: septate uterus < M/c: Cx insufficiency / Cx incompitence

which trimester losses most common: 2nd Trimester

septate can cause 1st Trimester Abortion

Δsu

History

1) painless Cx dilatation
2) 2nd Trimester loss only
3) Recurrent losses
4) c̄ every preg the POG loss is ↓ed
5) Preg: USG
   ↓
   a) we see Cx length → in 2nd T
      ↓
      if <2.5cm (Cx incompitance)
   b) serial USG show us shapes

      T – Y – V – U
      ↓
      Most imp for Cx incompitance

⇒ Cx incompitance in Non preg women
↓
by Hegar's dilator passed into int os
c̄ out resistance (no of Hegar dilator)
(Premenstrual stage)

NOTE foley catheter
↓
bulb 2cc NS
↓
pulled out easily

→ Rx of Cx incompitance
for m non pregn state: lash & lash

① Cervical circulage

M/c done / MC Donaldis procedure

purse string sutures

② Shirodkar Procedure

Mersclene tape

Ideal time → 12-14 weeks

Upto → 24 weeks

⇒ Absolute C/I for this circulage

① GCA gross congenital anomaly

② pelvic infection

③ Memb are ruptured

⇒ Strech is removed at 37 weeks

(or) SOS of

① ruptured memb

② Chorio amnionitis

③ Labour

APLA (Antiphospholid Antibody)

DIAG

- One clinical criteria
  - ① Arterial & Venous Thrombosis
  - ② ≥3 preg losses at <10 weeks
  - ③ ≥1 preg losses at >10 weeks
  - → But the baby morphologically Normal features
  - *** ④ atleast 1 preterm delivery at <34 weeks which is 2° to S.PE (or) Uteroplacental Insufficiency
  - → Characteristic is 2nd T Loss
- One Lab criteria
  - ① Lupus Anticoagulants (Russel viper venom test)
  - ② Anticardiolipin Ab (ACA)
  - ③ Anti β2 glycoprotein
  - IgG / IgM Ab
    - ↓
    - +ve in medium to High titers on 2 occasions ≥ 12 weeks apart

M/c : Lupus Anticoagulants

M/specific : Anti β2 glycoprotein
↓
Show disease activity

APLA IN PREG Rx

- Heparin
  - ① LMW Heparin
    can give unfractionated
- Aspirin
  - ① Low Dose Aspirin

② Start as ~~you~~ soon as you confirm IU preg

② Start as soon as UPT (+ve)

↓

Continued throughout preg

Stop Heparin at the onset of Labour

Stop 7 to 10 days before Labour

2nd Line Rx for APLA ① plasmapheresis ② IV Ig

↓ only given 1st line fails

→ m/c cause death in B.P.E / Eclampsia

↓

① Cerebral Haemorhage CVA ② pulmn odema

CVA > pulmn odema

→ overt diabetes

Investigation called Fetal Echo

↓

18-22 wks

→ Newer test available for Down's = Cell free fetal DNA

↓

Mother's Serum

↓

during preg woman has cell free DNA (circulating in blood)

→ done only beyond 10 wks

→ only a screening test = 2° screening

- If it become +ve then it is invasive
- Very expensive
- Using only in Trisomy 13, 18, 21 and Monosomy X
→ delay in report for 7-10 days

→ SPONTANEOUS ABORTION ←

Presentation

Missed Abortion	Threatened Abortion	Incurable
→ all ③ present ∵ pain + Bleeding		
• Os is closed	• os is closed	• os is opened ↓ expelled ↓ Incurable
• Size of ut < Pog	• Size of ut = pog	• Size of ut = pog H/o expelled
• USG CA is absent		✓ See the POC's in the Cx Canal

Incomplete abortion ↓ → Pain + Bleeding + H/o of passage of POC's (product of conception).

Complete abortion ↓

- Os is open
- ✓ See the POC's in the Cx canal
- Present in shock ↓ bcoz the POCs are not completely expelled
- USG: Retained (RPOC's)

- os is closed
- Symptoms ↓ improve after expulsion
- USG: Empty ut cavity ↓ (No RPOC'S)

(Next step) → (Specific) → Mangement

IV canula + IVF + arrange blood

Evacuation The uterum

Suction and Evacution

MANAGMENT

Missed abortion → Induce abortion

Threatened abortion (only in 1st trimester)

→ marjority of abortion occurs in 1st trimester

→ pathology is mostly chromosomal

Empirical Rx

1) Bed Rest
2) Strat progesterone supplementation
3) avoid coitus
4) avoid lifting wt

Inevitable abortion → (naturally expelled

1) undergo spontaneous expulsion

→ INDUCED ABORTION ←

MTP act

- Abortions can be done upto 20 weeks as per law
- If continuing of the preg is risk to mother → this is called therapuetic

- Baby is anomely
  ↓
  It is called Eugenic
- Rape victim: called ~~social abortion~~ (Humanitarian)
- Contraceptive failure (social)

Conduct of Abortion (who conducts)

consents of < 12 weeks → 1 RMP

" " > 12 weeks → 2 RMP.

RMP

1) degrees / Diploma in obs & Gynae
2) 6 months of house surgency in obs & Gynae
3) 25 assisted procedure
   ↓
   out of 25, 5 should be done indipendently

Abortion can be done in 1st (or) 2nd trimester

Medical Methods :
↓
Outpt abortion

(mifepristone + Misoprostol)

Day 1 mifepristone 200 mg oral tab

Day 3 Misoprostol 400mcg SL / buccal / oral / vaginal
↓
under observation for 4 hrs

Day 15 → take the History and do the Accordingly

~~Method~~

→ Medical Method Abortion upto 7 weeks (Indian)

↓ upto 9 weeks (WHO)

but here 800 mcg misoprotol

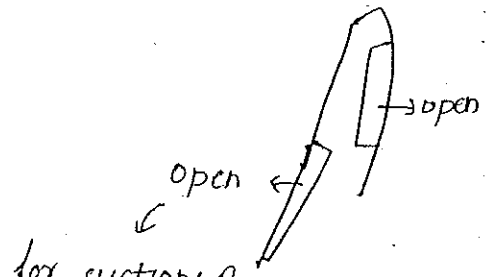
⇒ USG is not recommanded (Not mandatory)

↓

only in special circumstances like → Ectopic

→ Suction And Evacuation ←

- b/w 7 to 12 weeks
- best method in 1st trimester
-



for suctions of product

↓

Less than POG

(Only in even no)

12, 10, 8

↓

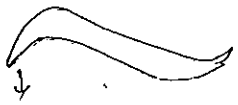
perforation can be made

↓

if perforation

↓

Astic laproscopy



① graduated ② for dilatation

③ holded in pen holder fashion

↓

to reduce the risk of perforation

④ if perforation, observe for bleed

End points in S/E

① Bleeding ↓

② Air bubbles in cannula

③ gripping sensation on cannula

↓

bcoz the ut will be involuted (closed)

Check curettage

⇒ It is a sharpe curettage

pressure during S/E is : 600 mmHg

Alternative S/E is : MVA (manual Vacuum Aspiration)

↓

Done upto 12 weeks

60ml syringe for it

600 – 650 mmHg pressure

⇒ Medical Methods : M/c method

↳ Drawback : May not complete abortion

cannot be done everytime

↓

options : prostaglandins : Misoprostol

→ Extraamniotic Ethacridine

→ Intraaminotic saline

→ oxytocine ⟨ large doses
long duration

Surgical Methods

Drawback: anesthisia

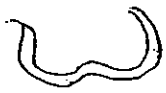
invasable

Ovum forceps:

Spoon (or) cup shaped ends

No lock (or) catch

Sims speculum (post vaginal wall retractor)



Cuscos speculum (self retaining)

↓

Specially used in Pap smear

AV retractor: used c̄ sims speculum

↓

(ant wall retractor)

Sponge holding forceps: A pregnant cervix is hold

~~due~~ due to cx is very vascular

A non pregn cx is hold by: ?

→ LABOUR ←

False labour pain		True labour pain
1) Not seen	←	1) ut contractions will ↑ in frequency & intensity and/or duration.
2) Not seen	←	2) Assc c̄ (show) ↓ Passage of blood mixed cx mucous

3) Not seen ← 3) Cx dilatation & effacement

10% (shortening & softening)

In primi: effacement preceed the ~~show~~ dilatation

multi: simultaneously

Gravida para

G T P A L

↓

~~No~~ No of pregnencies

T = term (any delivery beyond >37)

P = No of preterm deliveries 20-37 wks

A = Abortion (No of abortion <20 wks)

L = Live (no of live babies)

4) Rupture of membranes

5) It is suside the ut contraction

5) Effect of sedation

↓

Pain perception is ↓ed

ut contraction continuous

⇒ EDD (expected date of delivery) = 40 weeks

9 month + 7 days to LMP

~~LMP 1st day~~

AIIMS * { ± 1 week from EDD (50%) babies will delivered
± 2 weeks " " (80%) " " "

phase of labour / Stage of labour

True (or) False: Best ans

1) progressive cx dilatation & Effacement
2) Rupture of membrane

PROM: premature rupture of memb → <37 weeks

PPROM:

Phase of labour

Stages: 1st stage of true labour pain to full dilatation of cx

latent stage		Active stage	
0-3 cm →	WHO ←	≥ 4 cm	(WHO given partogram)
0-5 cm →	ACOG (New) ←	≥ 6 cm	

Duration

	Normal	prolonged
Primi	12 hr	20 hr
multi	8 hr	14 hr

Rate

① Rate of cx dilatation

Primi 1.2 cm/hr

Multi. 1.5 cm/hr

(min requirment the labour to progress is 1 cm/hr)

<1 cm/hr = slow

Dystocia (abn labour)

2) @ of descent of Head

Primi 1cm/hr

Multi 2cm/hr

Second stage: from full dilatation to expulsion of fetus

Primi = 1hr

Multi = 30 min

ARREST of Labour: then take for CS

Arrest of active phase (or) Arrest of 2n stage

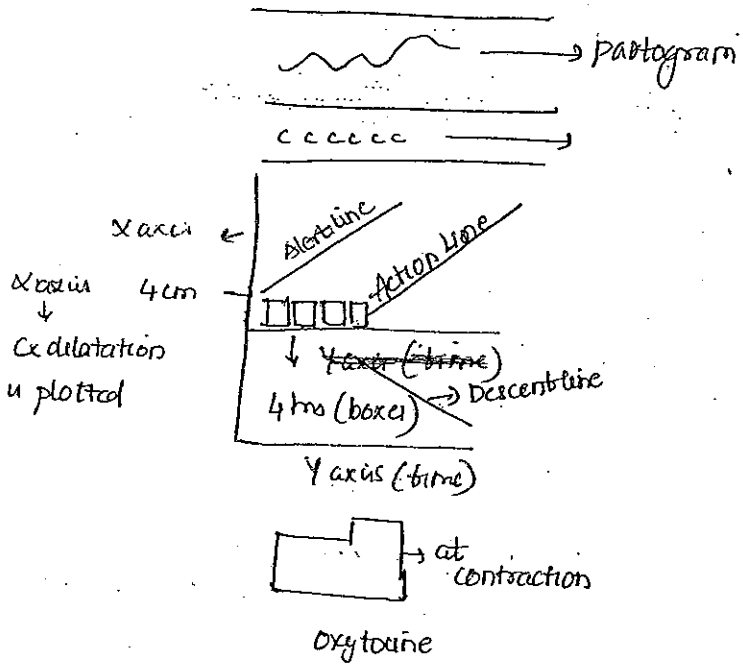
1st stage: Arrest: No change in Cx dilatation even after 4 hrs of adequate ut contraction

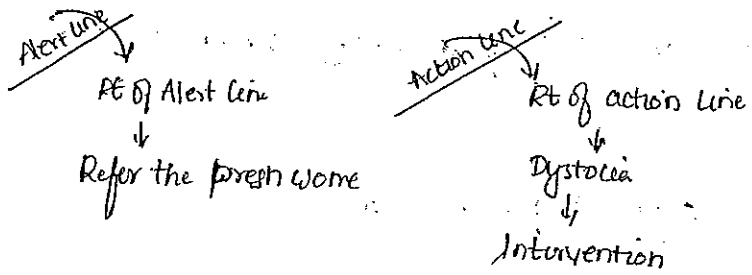
membrane have to be ruptured

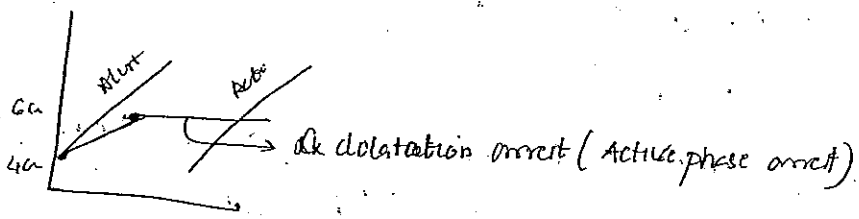
Arrest in 2nd stage: No descent (or) rotation with adequate constraction for 3 hrs in primi and 2 hrs in multigravida women.

If epidural analgesia Mean 4 hrs for primi 3 hrs for Multi

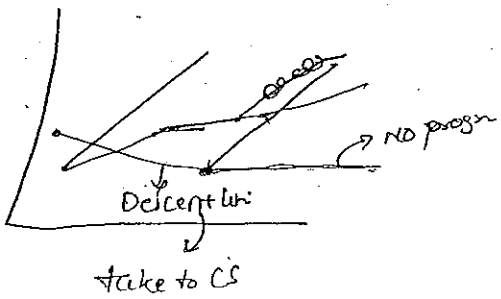
(ONLY when FHR is normal) this much waiting can be done)







→ Descent Arrest ←



→ LIE ←

Relationship of long axis of fetus to long axis of ut

→ Dextroversion

→ unstable lie: lie changes even beyond 37 weeks

m/c/c is Idiopathic

next c is Placenta previa

oligohydroamnios — not a cause of unstable lie

polyhydroamnios — cause of unstable lie, malpresentation

⇒ In Idiopathic condition the placenta is placed in normal site

Presentation: part of the baby which is foremost in birth canal

cephalic presentation

breech presentation

shoulder "

compound presentation: Hand ± head (m/c type)
↳ No need for CS

Cephalic: depends on degree of flexion & extension

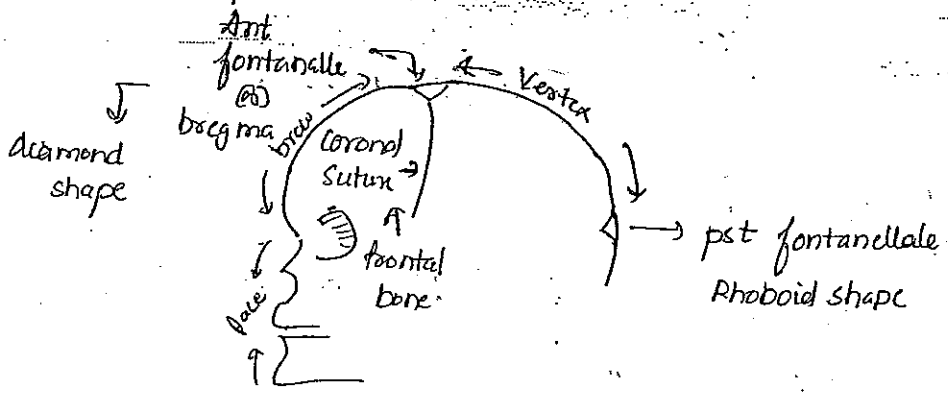
1) Head is well flexed: Vertex presentation

2) " deflexed: Sinciput "

3) partial extension: Brow "

4) Full extension : face presentation

Vertex presentation:



Denominator: Bony prominance on the presentation which is used to describe the poistion

Vertex : occiput

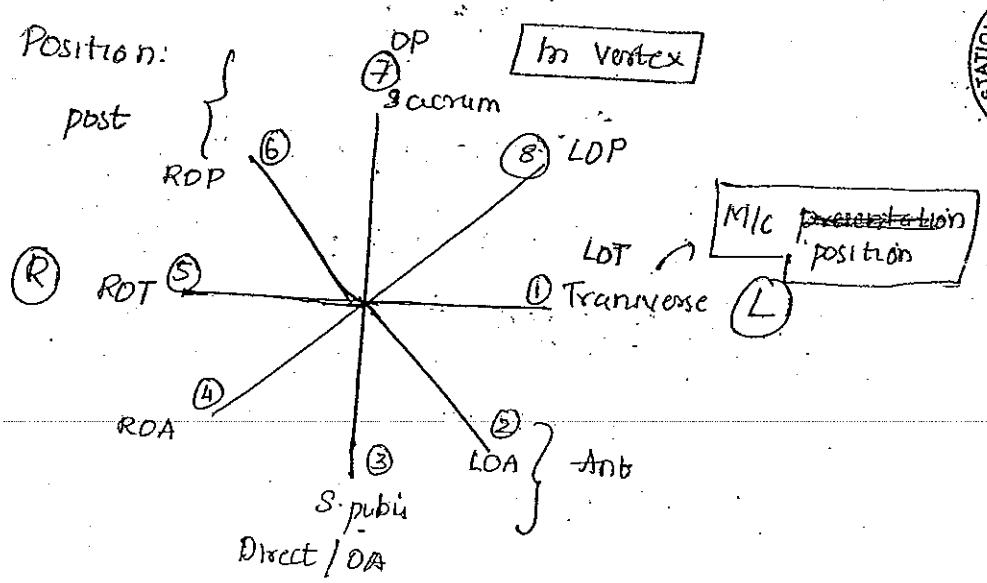
Brow : frontal bone

Face : mentum

Breech : sacrum

Shoulder : acromian process

Position:



2 oblique diameters are: 2 and 6
4 and 8

from sacrum: Left oblique : 8-4
Rt oblique : 2-6

→ Engagement ←

when largest fetal head dia crosses the pelvic inlet

Largest head dia : transverse diameter : BPD 9.5

Small head dia : Prismastoid : 7.5 cm
Pritemporal : 8 cm

MCQ ** Ant-post engagement dia

Vertex : Subocipito bregmatic : 9.5 cm
↳ flexion ↗

deflexion : → occipito frontal = 11.5 cm
(Highest point on sagittal suture)
→ Subocipito frontal = 10.5 cm

Brow : Mento vertical = 14 cm
↓
Largest fetal dia

Face : submento bregmatic = 9.5 cm

Breech : Bi Trochantric = 10 cm

⇒ engagement rules out CPD at inlet

Engagement < Primi = 37 wks
Multi = onset of labour

A lady comes to you at 37 wks head not engaged
↳ free floating

↓

Ans) Deflexed head

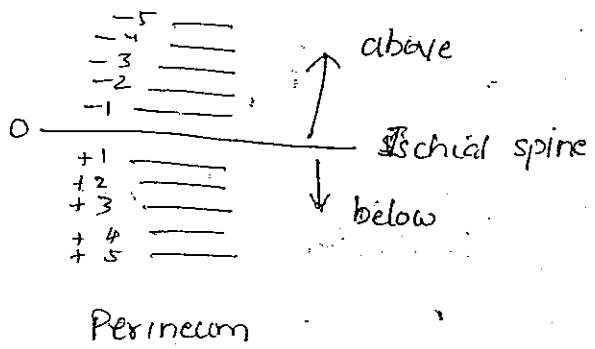
other ans: PP
CPD
Polyhydromnia
wrong date

During engagement:

P/A exam os done < Ut contraction
descent of head

↓ 2/5 (engagement into the pelvic inlet)

↓ represent as 5/5 5 parts
4/5 → 1 part into inlet ~~4~~ one palpable by P/A
3/5



Station: leading point of the fetal head

Loss of station:

↓

only a feature of Rupture ut

↓

Baby is expelled into the Abd

↓

from +1 to −3

Steps of the Labour

Vertex presentation

Cardinal movements (n) steps

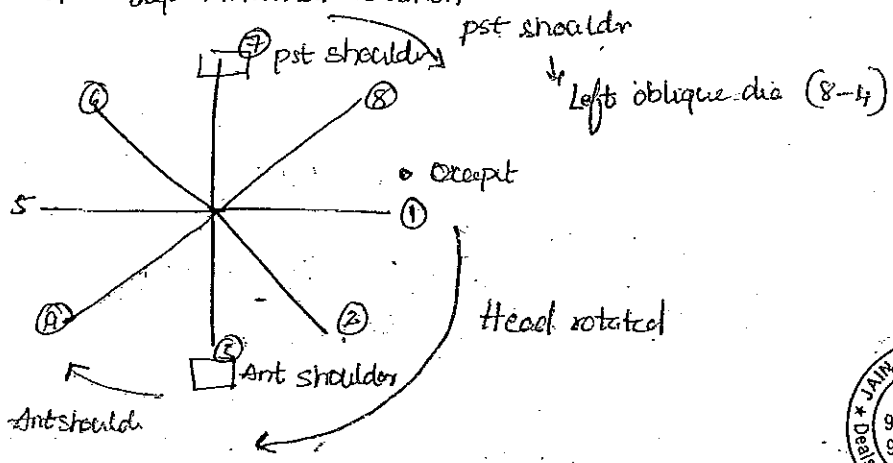
they occur simultaneously

1st step: Engagement

2nd Descent

3rd flexion → (Reduces the surface area of body to pass into the small pelvis)

4th Step: Internal rotation



Internal rotation: 2/8 head

1/8 shoulder (left oblique dia)

at the level of ischial spine

5) Extension :

6) External rotation (opposite to Internal rotation)

↓

after ER occiput goes back to orginal position and the shoulders are moving inside 1/8 shoulders attain the AP dia

1st external rotation of head & out rotation shoulder
it is called Restitution (Not a cardinal movement)
to remove the trosion of the shoulder

↓

2nd 1/8 causes shoulder rotation

Restitution and internal rotation = opp direction.
" " external " = same direction

7) Shoulders

8) Lateral flexion

Crowning: when BPD stretches the vulval outlet and does not receeded back
- Not a cardial movement

Normal labour to be normal

Passage

push

passenger

Passage = Pelvis

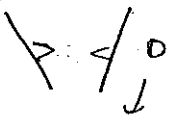
m/c Gynecoid : Seen in 50% female
Inlet is circular

Anthropoid : seen in 25% female
Inlet A Partly oval

Android : seen in 20% female
inlet Heart shaped (male pelvis)

Platypelloid → 5% female
→ transversely oval
→ Flat gynecoid

Difference b/w Gynaecoid & ~~Anthropoid~~ Android

Gynaecoid	Android
①  Side wall are parallel	①  → side walls are convergent m/c/c op → Android pelvis
② Ischial spine blunt	② Ischial spine sharpe  DTA ↓ m/c Android

③ subpelvic angle

obtuse : 90-100

④ shallow

⑤ Acute angle : 85°

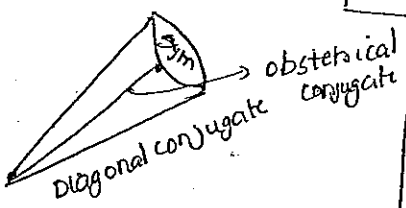
④ deep

deepest : Anthropoid

Inlet

smallest inlet are

A-P diameter



→ Diagonal conjugate : 12 cm

Obstetrical con : 9 cm

true conjugate : 11 cm

OC DC -

TC DC - 1 cm = 11 - 1

= 10

= 1-15

contracted inlet

(OC shortest) < 10 cm

Cavity

Interischial dia

IID = 10.5

↓

contracted = <8 cm

Outlet

Intertuberous

⑥ 11 cm

contracted < 8 cm

↓

four knuckle test

(should be fit easily)

Pelvic Assesment

In primi gravida = 37 wks
↓
contracted pelvis
↓
elective C.S

In multigravida = ~~at~~
onset of labour
↓
thu mean already delivery
a over = No abn pelvis

Cephalo pelvic Disporportion :

only (ro) best way to know CPD is
trail of labor

Next best MRI
↓
CT Scan
↓
Xray pelvimetry
} not done in preg women
↓
Pelvic examination (Manual)

Prev / cs → done for contracted pelvis → always cs should be done (Recurrent indication)

Prev / cs → CPD
↓
depends on baby
↓
trail of labour (VBAC)
Vaginal birth after caesarean

Plane of least pelvic dimension : Ischial spine
↑ obstetric outlet corresponds to least pelvic dimension

Greatest pelvic dimension:

pass through disc space b/w S_2 & S_3

Outlet — Anatomical outlet : passes through ischial spines tuberosities
— Obstetrical outlet

(Upper boundary)

Ant S. pubis — plane of least pelvic dimension — obstetrical outlet — coccyx (Pst)

Anatomical outlet

(Lower boundary)

BUSH

→ PUSH ←

Ut contraction: Pace maker is ~~cornu~~ cornu

Rt > Lt

→ @ 2cm/sec

→ depolarise 2 in 15 sec

→ At which IU pressure = 10 mm Hg

↓

the ut contraction palpable

= 15 mm Hg (pain contraction)

min P → cervical dilatation = 15 mm Hg

moderate contraction = 40 mm Hg

to know that moderate contraction = finger on fundus
for intendation
↓
Strongest contraction

Adequate contraction:

1) 3 contraction / 10 min, each contractn 45 sec duration

2) contractn which generates a pressu of 220-250 Monte Videounits

Tachysystole: >5 contractn in 10 min
↓
fetal distress

Hyperstimulation = that is done by Misoprostol
↓
so not used in augmentation
oxytocin is used in augmentation
↓
but this can also cause Hyperstimulation
↓
then stop the oxytocin
↓
women in left lat position (↑ the maternofetal circulation)
↓
oxygen to mother
↓
IVF (wash out the oxytocin from reception)
↓
after 30 min
↓

restart the oxitocine at half dose
at which hyperstimulation

Pressure → contraction 1st stage labour 50 mmHg

Pressure → " → 2nd stage 80 mmHg

Labour

- False (No change)
  - ↓
  - Mx : give sedation
  - ↓
  - Sedation : ↓ contraction, ↓ pain
- True labour (change of dilatation)
  - Latent phase 0-5
    - 0-3
    - ↓
    - 8am 1 cm EE, Vx at 3 mm (+)
    - 8pm 2 cm EE, Vx at mem (+)
    - ↓
    - sedation > w/w
  - Active phase 6 cm
    - 4 cm
    - Slow
      - ↓
      - 1 cm/1 hr
      - ↓
      - Slow (whether the contraction are adequate or)
        - Yes ~~Inade~~
          - adequate contraction
          - ↓
          - Membrane rupture is over
          - ↓
          - (1st reason) → CPD (or) OP → (2nd reason)
        - (No) Inadequate
          - ↓ (augmentation)
          - AR M → (PGS)
          - after 30 min gap → ↓
          - oxytocin
    - Normal

↓

If CPD (or) OP

↓

Intervention

↓

P/V exam to rule out

CPD / OP

↓

finding for CPD

- Scalp oedema ← caput
- Overriding of parietal bones ← Moulding
  - grade 1 : bone touch
  - grade 2 : overlaped, manually seperated
  - grade 3 : overlaped, cannot be seperated

finding for OP

- 6 PM 7cm 80% vx at 0
  - mem 0
  - 220 monte vedo units
- 10 PM : Scare

Arrest (or) delivered

If it is arrest

↓

then CS is Mx

If waited after arrest

- 9PS
  - dehydration
  - exhaustion
  - Acidic Breath
  - Tachycardia
- P/A :
  - pathological ring in P/A exam
  - BANDLE'S ring → (b/w upper & lower segment)
  - upper segment : Tonically contracted
  - lower segment : stretched & thinned

Schroeddors Rind

↓

Physiological contract ring

↓

In P/V exam

FHR distinct / absent.

Suprapubic bulge (Full Bladder)

P/V exam: Vagina hot & dry
caput / moulding

Catheterise: Hematuria

FHR may be present / Absent
↓
in obstructive labour
↓
only CS is done

Older destructive procedure → CRANIOTOMY
↓
but not Now-a-days

m/c/c of Vesicovaginal Fistula: in developing country
obstructed labour

presentation is delayed

In developed countries
(Surgeries for malignancy)

→ PASSANGER ←

(fetus)

Vx Non Vx

1) CDP

2) OP (Malposition)

(Mal presentation) breech inc malpresentation

↓

Risk factors: 1) Prematurity

2) Multigravida

3) abn pelvis

4) poly/oligo

5) Hydrocephalus

⇒ % of babies at the OP at the onset of labour: 15-20%

↓

m/c/c of OP is Android pelvis

↓

nxt m/c/c a deflexed head

↓

In OP the most common position: ROP

↓

OP is more commonly seen ī primi

↓

Engaging diameter: 11.5 deflex head

occipito frontal (o) 10

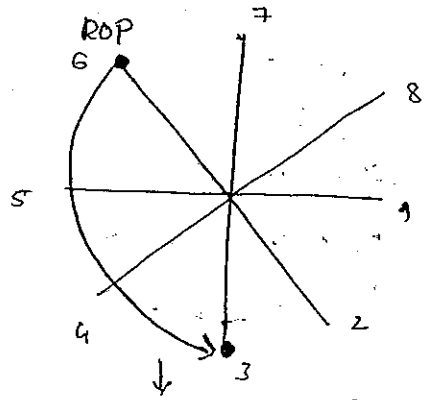
% op at the end of labour: 05%

↓

they come into OA

Outcome:

Good scenerio: — pelvis adequate
baby avg wt.
Contractn - good



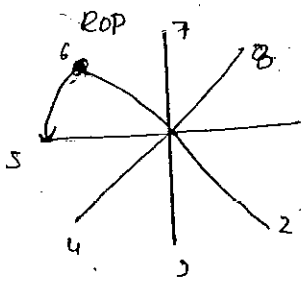
↓

3/8 rotation (slow labour)

↓

in this Mx in w/w to rotation should complete

Scenerio 2: — Adroid Pelvis



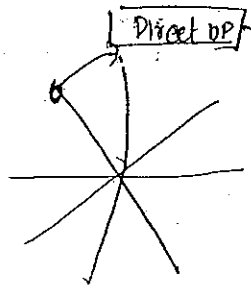
Deep transverse A

↓ Pelvis (N) — Pelvis abn

↓ 1) Manual rotation
2) forceps "
3) Vaccum extraction

↓ ① CSU only Mx
, forceps extraction

Scenerio: Direct OP



Direct OP → Anthropoid ← Posterior OP

= Face to pubes spontaneous delivery is in Anthropoid pelvis

spontaneous does not occur

↓

C.S > forceps

↓

cause more severe maternal perineum injury.

grade 3 & 4 injuries

→ BREECH ←

m/c/c Prematurity

at 28 wks → Risk is 25%

32 wks → 7-11%

term → 3%

Types of breech:

Franck breech: m/c type of breech

flexion at hip

extension at knees

Complete breech : flexion at hip & knees

Footling breech: also called incomplete breech

↓ one foot (or) two foot

↓ Highest risk of cord prolapse

Lowest risk of cord prolapse in breech: frank breech

Higest risk of cord prolapse: Transverse lie

least : Cephalic presentation

⇒ M/c c/c of death in breech: cerebral Haemorrhage

Indication of c.s in breech:

* Absolute Indication

1) footling breech

2) Stargazer baby
↓
due for delivery is more

* Relative Indications:

1) Primi breech

2) Previous Caesarian

3) Hydrocephalous (Ventriculo megaly) → V.P shunt
Ventriculo

4) Macrosomia

5). Prematurity (Pre term baby).

Zatuchni Anderson scoring → 0 to 4 do c.s

External rotation

for all non cephalic presentation the first Mx should be ECV if requirments are met

Requirments

External cephalic version

1) ≥ 37 wks
2) presence of adequate liquor
3) memb intact
4) ECV in singleton preg
5) FHR (N)
6) no CI for VD
7) labour - Latent phase
   ↓
   Ut should be relaxed : by Inj terbutaline
   FHR continuos monitoring
   ECV ⟶ if Fetal Distress ⟶ [Reversion]
   ↓
   then C.S

8) $G_2P_1L_1$ 37 breech FHR ⊕
   PI fundal
   PI uterus ad
   ↓
   Mx ?

   Ans) ECV if Leaking PV
   ↓
   Assisted breech Vaginal Delivery

① Lovset's Manoeuvre

↓

for delivery of extended Arms

↓

~~app~~ Hands

② PINARD'S → EXTENDED LEGS

③ BURNS - MARSHALL MANOEVRE

↓

after coming head in flexion delivery

④ MSV method MAURICEAU SMELLIE VIET

malar flexion and shoulder traction

(Best method to deliver after coming head)

⑤ PRAGUES MANUEVRE

* when baby is dorso posterior

in dorso ant < MSV
Burns marshall

⑥ DUHRSSEN'S INCISION

Incision on Cx

two incision: at 2'o clock

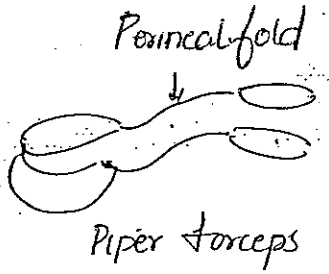
↓ 10'o clock

only preterm head

term head rupture the Cx

⑦ Symphysiotomy.

⑧ Forceps : called as PIPER forceps



(9) Last Manoeuvre: Zavarelli

Relative C/I to ECV:

1) prev CS
2) Pre eclampsia
3) IUGR

FACE AND BROW

- Extension < partial / complete
- m/c/c Anencephaly
- Risk factors: prevent flexion of head
  1) ant neck masses (multiple neck loops of cord)
  2) Platypelloid pelvis

Face < Mento Ant / Mento post

↓

delivery of head in Flexion

In mento ant → V.D is possible

mento post → No mechanism of labour

↓

In labour (C.S is done)

In early labour

↓

W/W (bcoz it has property to rotate to mentoant)

⇒ face presentation confused ∈ frank breech

↓

to differentiate

face : bony prominants △

frank breech : " " ○—○—○

Engagment Dia : in Brow mentovertical (14 cm)

↓

More length in dia

Q) Brow in Labour

↓

C.S is the M/x

Q) Brow in early Labour

W/W (brow can go into face (or) flexion)

Transverse lie : Highest risk of cord prolapse

↓

M/c/c → Prematurity

M/c/c at term = PP

Pelvis = Platypelloid

Shoulder presenta = rib cage grid iron

Military position: brow position

→ Transverse Lie at term

↓ Do ECV (provided criteria are met)

Neglected shoulder presentation in labour

Neglected shoulder c̄ FD
" " c̄ IUD } should do C.S

→ Decapitation

Hook c̄ knife → baby is taken out as a parts

→ Evisceration: scissors → cut the abd → organ expelled → baby expelled

→ Corpora Condoplicata:

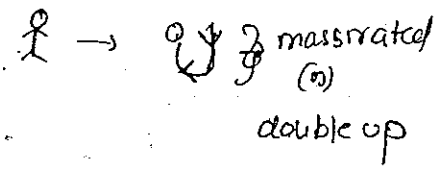
delivery in a double up manner

Seen in preterm and old IUD

↓

massiration

↓



→ INSTRUMENTAL DELIVERY ←

→ Cut short the 2nd stage of labour

Indication may be: Fetal Distress / Maternal exhaustion

Requirments:

F - Fully dilated cx

O - no obstrctn in the path

R - Ruptured memb

C - good ut contrctn

E - Engaged / Epsiotomy / empty bladder

P - Favourable presentation

Forceps	Vaccum
① training	① easier application
② maternal exhaustion ↓ doctrot need maternal effort	② Need maternal effort
③ Heart disease	> ③ Not CI
④ In fetal distress	④ Pressure has to be raised slowly
⑤ In preterm ↓ forceps protect the preterm head	⑤ C/I in preterm head

⇒ Face presentation: only forceps

No vaccum

→ ~~pos~~ mentoant (forcep)

mentoant (No VD only CS)

⑥ Full dilatation required — ⑥ Can be applied ≥ 6 cm (ideal - full)

⑦ Maternal injuries more — ⑦ More fetal injures

⇒ Which fetal injuries are m/c in forceps

a) Intravascular Haemorrhage

b) Facial N palsy

c) Brachial plexes

d) Injury to cornea to eye

Exclusive injuries in vaccum

a) Cephalo hematoma

b) 6th N palsy

c) Retina of eye injury

Forceps: station

Outlet ≥ +3

Low +2

Midcanty 0 – +2 } not in use

High above 0 }

Wrigley's outlet forceps → m/c used forceps
↳ it has a English lock

Sumpson's forceps → used in primi
Tucker Mc Lane → " " Multi

For Rounded and Moulded Heads
↓ Tucker Mc lane
↓ Sumpson's forceps

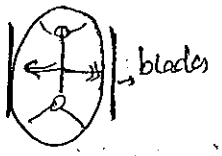
Keilland forceps : ① Sliding lock
② Rotation of head

longest forceps :

⇒ Wrigley's forceps is short so not used in aftercoming head

⇒ Left blade used 1st : becoz the position are (L)
if the position is (R)
↓
use (R) blade 1st

⇒ After application of blade should be equidistant from saggital suture

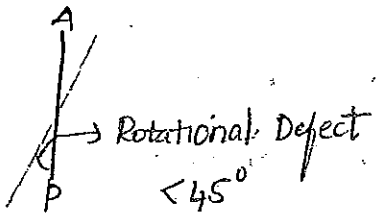


⇒ along which dia of head we apply
Occipitomental dia : 13.5 cm
↓
Second longest dia

Requirments of outlet forceps

1) Scalp visible of Introitus
2) head on perineum
3) ~~scalp~~ skull on pelvic floor
4) ⇒ preferably sagittal suture should be in A-P dia
5) In case sagittal suture is not in A-P dia

then



↓

use A-P dia

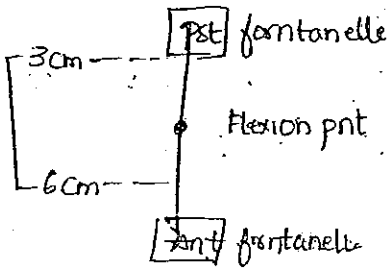
↓

if $>45^0$ → Do 1) Kelland forceps to reduce Rotational defect

2) Vaccum

Vaccum cups:

1) Made of silastic
2) Pressure 'P' is 600 mmHg (or) 0.8 kg/cm²
3) Flexion point: at sagittal suture point



- failed vacuum : 3 application
  ↓
  No descent

⇒ If any instrument forceps (or) vacuum fails then take to C.S.

⇒ C/I for instrumental delivery

1) Contracted pelvis
2) CPD

⇒ In any place where V D is C/I No instruments should be used

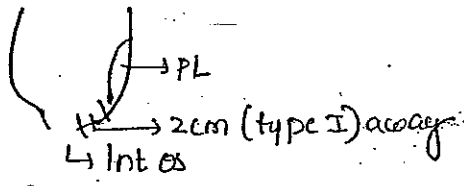
3) HIV (+ve)
4) osteogenesis imperfecta
5) Coagulopathy defect in baby

→ Anti partum Haemorhage ←

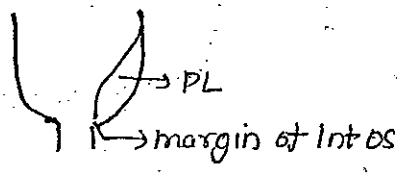
any blood loss from (or) into genital tract beyond period of viability (28 wks)

Placenta previa	Abroptio placenta
• PL lying in the LUS	• Premature seperation of (n) located placenta
• 1 : 300	• m/c 1 : 200

type 1 :

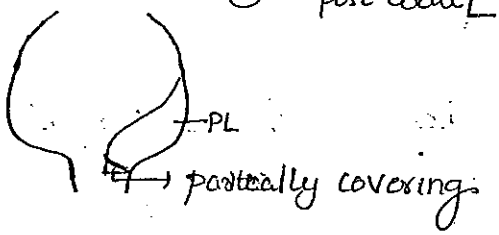


type 2 :

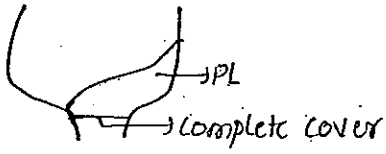


type 1 & 2 — (A) Ant wall
(B) post wall [m/c]

type 3



type 4



type 1A, B, 2A } minor

type 2B, 3, 4 } major

⇒ Dangerous PL Previa : type 2 B

marginal → pl compressed c̄ head
↓
fetal distress

Stall worthy sign : push the head into pelvis and check for FHR

Abruption

Revealed → Blood expelled

Concealed → Blood concealed

Mixed → M/c

PAGE'S CLASSIFICATION

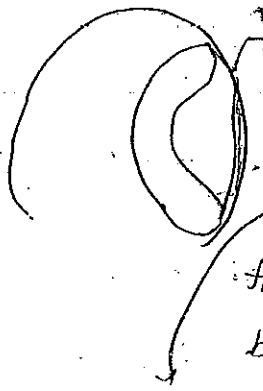
type 0 → Retrospective Asis
↓
after delivery

type 1 - BPV + Pain + FHR (n)

type 2 - BPV + Pain + Fetal distress

type 3 - BPV + Pain + IUD + shock (of mother)
± DIC

⇒ In PLPG Abruption : the blood lost is Maternal blood



decidua
↓
Breaks / tears
↓
decidua starts releasing tissue thromboplastin into maternal blood → initiates DIC

Uterotonic, less BA

⇒ In which kind of women more risk of DIC is ?
↳ women who release more thromboplastin

⇒ So only ↑x in abruption is termination

Risk factors of = PLP : Abruption

Highest risk: Previous h/o upto 5% — Previous h/o risk upto 15%

- ↑ maternal age
- Smoking
- Multiparity
- Prev C.S

↑ No of C.S ↑ risk

⇒ When there is any type of scar by any condition (D&C / Hysterotomy scar) prevent PL migration

- Endometriosis
- Ut anomalies

- ~~Placenta~~
- Pre eclampsia
- Trauma to abd
- APLA synd
- Thrombophilias of pregnancy
- polyhydromnios
- prolonged rupture of memb
- Folic acid Def
- Presence of fibroid in Ut

m/c factor V Leiden mutation
Prothrombin gene mutation
Deficiency of Activated Protein C and Protein S

Clinical Features: PL previa : Abruption

Chief Complaint: Bleeding ↓ painless bleeding — Bleeding ↓ painful bleeding ↓ pain by thromboplastin

- fresh, bright Red
- Clots

- Altered blood

- Pre eclampsia
  (or)
  Trauma

- Hypotension is seen
- ~~ht~~ Unlikely (No Hypotn)
- ht = POG
  < POG
- fundal ht is more than POG
- tense / tender ut
- Relaxed
  notense / nontender
- Warning Hge
- Acute
- Fetal distress L/c
- FD M/c
- Malpresentation M/c
- L/c

P/s (or) P/v are not done in a pt of APH till we rule out PL previa

⇒ APH : IOC : USG
↓
$\boxed{\text{Transabd USG}}$

Can TVS : safe
↓ better to locate pst PL previa (missed in TAS)
2 things should be looked
① distance of PL P from int os
② Retroplacental area

APH (PL previa)

Indications for TOP

1) Unstable Haemodynamically
2) Foetal distress
3) Mother is continuously bleeding < fetal (n), Mother (n) } vitals stable → But do TOP
4) Mother safe, not bleeding, baby safe } but ≥37 wks → DO TOP
5) active labour
6) IUD
7) Gross congenital anomaly

order of Importance

Indication for CS in APH

1) Haemodynamically unstable
2) Fetal Distress
3) Major degrees of PL previa
4) far from term (i.e. <32 wks)

Abruption
↓
TOP
↓
route: VD
↑
Risk of DIC — thrombolastin → uterotonic → fast in labour → ppt labour → Em 3 hrs

Conservative Mx in Placenta previa

↳ Macatte's Regime

1) Bed Rest

2) Maternal monitering
   a) Vitals
   b) Blood loss
   c) fundal ht & circumference
   d) Baseline Ix

3) fetal monitering FHR

4) Cervical cerclage Not done in PLP

4) <34 weeks → steroids (for foetal lung maturity)
   ↳ Dexamethasone 6 gm Inj IM every 12 hrsly for 4 doses
   Betamethasone 12mg IM 24hrly 2 doses

5) <34 weeks → Ut irritable, mild contrctn
   ↳ tocolytics
   TOC : Nifedipine (safest)
   ↳ in chr HTN
   atosiban > nifedipine
   ↓
   Oxy R antagonist

In DM → Nifedipine
↳ give nifedipine
β agonists are not given (cause hyperglycaemia)
cause no of side effects
tremors
R ↓ effected
- Hypokalemia
- Arrythmia / MI
- Pulmn oedema

Indication of tocolytics:

- as long as steroid are given
- while giving steroid Mother should not have labour

Preterm Labour

<34	>34
steroids + tocolytic	w/o

Indomethacin : < 32 weeks
beyond 32 wks : premature closure of DA

Halothan: anesthetic drug

Alcohol :

Diazoxide

$MgSO_4$: → Action only in large dose level 9-10 meq
this level: toxic signs

progesteron is not a tololytics

Prevention ← Mx of Preterm labour → Tt of Preterm labour
↓ ↓
Previous h/o Preterm labour Rx c̄ tocolytics
↓
progesteron

Clinical Q) G3P2L2 c̄ 34 wks preg c̄ APH BP = 160/70
PR = 100 USG placenta type 4 and FHR (+)
liquor Adequate
↓
conservative Mx up to 37 and the C.S

Conservative Mx in Abroption → No Conservative Mx
↓
only TOP

Abroption care ~~c~~ ~~Rh~~ DIC
↳ In DIC Sx should not be done
so do VD
FFP → V.D
↓
to replace

⇒ During C.S → Red ot (abroption)
↓
Couvelaire — Bleeding into myometrium
↓
not a Indication for Hysterectomy
↓
but it is a risk factor of PPH

⇒ For PL previa there is Double set up examination

APH → PL previa → term → 37 wks

then taken her OT and do P/V

↓
OT
↓
arrange 2 table

2nd table
↓
Sx ~~sho~~ Should be done
↓
C.S done

I table for P/V
↓
bleeding ← major type (or) degree
bleeding → No bleeding
↓
ARM
↓
shift to labour room

Pt G3P2L2 comes c̄ APH, Vitals stable,

FHR (n), ut 34 wks, tense & tender, on USG → PL is fundal and not low lying and there is no RPC

(retroplacental clot)

So this is (released type) of Abruption

ARM is both ⚠stic & therapeutic

↓ Blood stained AR (dar)

↓ TOP

↓ PG

↓ initial labour

→ ADHERENT PLANCENTA ←

"the pl is attached to decidua basalis"

in adherent pl : there is no "

↓

pt is attached to myometrium

↓

depth of attachment

m/c → Acreta : surface of myo (only attached)

Increta : Invades into myo

L/c → Percreta : upto serosal layer

Histopathological Findings → absent / incompletely developed

↓

Nitabuch's layer

(b/w trophoblast & decidua)

Risk factors:

1. Highest Risk: ① PL previa + Prev C.S

PP > Prev C.S

② Multiparity

③ Fibrous bands in conditions: D&C scar

④ * Incidence: 1 in 730

IOC: ① USG

② USG + doppler (better)

↓

Heterogenous (black & white color in USG)

↓

(N) PL is Homogenous

this is labelled by Radiologist as: ① Intrapl lakes

② obliteration of Retropl clear space

③ Gold stnd is MRI

Clinical ⊕) k/c/o Adherent PL

↓

Post them for elective C.S

↓

at time of C.S and do Hysterectomy

Uk/c/o Adherent PL

↓

comes in Labour

↓

delivery

↓

No pl seperation (during delivery)

↓

So obs Pg's put Hands into ut (Manual)

↓

and take small pl tissues

↓

Sinus are opened

↓

PPH

↓

Refractory PPH

↓

Hysterectomy

known and unknown case difference in Morbidity

Cant do a Hysterectomy but k/c/o Adherent pl

↓

then do V.D

↓

cut the cord

↓

leave the pl inutero

↓

Autolysis of PL (6-8 months)

↓

but pt will have red blood loss

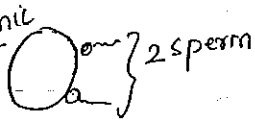
~~GESTATION~~

GESTATION TROPHOBLASTIC DISEASE for NEET PG1

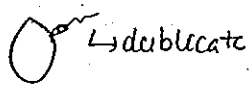
MOLOR PREGN	GTNeoplasia
1) Partial mole	1) Invasive mole
2) Complete mole	2) chorio Ca
	3) PSTT (placental site trophoblastic Tumour)
	4) ETT (Epitheloid Trophoblastic Tumour)

Partial Mole	Complete Mole
→ Genetic Makeup of the conceptus	
① Triploid	① Diploid
② 69XXY 90%	monospermic 80% 46XX
10% tetraploid	Dispermic 20%
	- all genetic material Paternal

Dispermic



Extra set of chromosome is paternal



↓ genetic mat of ovum is degenerated

↓ degenerated genetic mat

↓ duplicated

Risk factors:

1) Recurrence 1% (1-1.5%)

2) extremes of age of men <15 (or) >15

anytime >35 RF ↑ed

**3) Vit A def

(or)

def of carotene

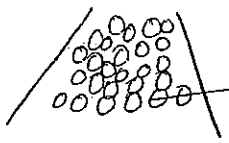
4) ↑ parity

IOC :

1) USG : very good for complete molar

↓

ⓐ { Snow strom apperance
Honey combing apperane



→ black (Hydrophic degeneration)

ⓑ No felus in USG (rrul)

ⓒ Hydrophic degeneration

⇒ Partial Moles are missdiagnosed on ultrasound

↓

in this fetus is present

↓

Report come as missed abortion

a) focal Hydrophic change

b) Transverse dia > AP dia

⇒ Molar pregn is m/c diagnosed in 1st trimester
bcoz USG is routinely conducted in 1st trimester

⇒ Diagnostic & ~~therapeutic~~ confirmatory
↓
Gold standard : HPE

Complete Molar

① Extensive trophoblastic proliferation
② Complete hydrophic degeneration
③ Villi → ~~[illegible]~~ avascular
④ No fetal tissue

Partial Molar

① Missmatched
② Focal Hydrophic degeneration
③
④

M/c presentation

1) present as bleeding, and in 2nd trimester
2) passage of grape like vesicles
3) Fundal Ht > POG (in partial : FH = POG)
4) HCG in Very High = 10^5 (in partial : $> 10^5$)
↓
only in complete Molar
a) Hyperemesis gravidarum
b) Thyroid storm
c) Pulmn embolism

d) Early onset of PE (<20 wks)

e) Theca lutein cyst (in ovaries)

Partial Mole	Complete Mole
↓ 3-5%	↓ 15-20%
GTN	Neoplasia
Partial Into	Complete
↓ <1% (neglesable)	↓ 4%
chorio Ca	Chorio Ca

TOC : Suction and evacuation

↓

change the answer when

↓

≥40 yrs complete family

↓

Hysterectomy → to reduce morbidity & Mortality

Suction and evacuation: Pulmn embolism

Thyroid strom

Anesthestist

↓

oxytocin ready

↓

Start suction

↓

Start oxytocin (after suction)

↓

+/b sharp curettage

↓

Send tissue for Histopathology

↓

follow up the pt

↓

[illegible] convert into malignancy

↓

follow up

① weekly HCG till 3 (n) Values

monthly → 6 months (3 mnths)
Period of surveillance → partially 3 months

② Most Imp advise in surveillance period is not to conceive bcoz it raise HCG ↓ mis ∆sed as Neoplasia

↓

contraception → OCP is choice
↓
condoms
→ avoide : IUD

⇒ avg time HCG returns to (n) is 9 wks
complete : 9 wks
partial : 7 wks

~~High Ris~~

~~Rist~~ High Risk for GTN

1) ≥40 yrs
2) HCG ≥ 10^5
3) FH > POG (more molar tissue)
* 4) B/L large thecolutin cyst (>6cm)
↓
Prophylactic chemotherapy
methotrexate
Actinomycin D

→ GTN Asii ←

Day 1 :

1) 4 consequtive HCG value shows platues

Day 1 to Day 7 D14 D21 ±10% of prev values

2) 3 consecutive value show a rise

D1 D7 D14 [>10%]

3) HCG remain elevated even after 6 months

(N) value of HCG <5

4) HPE → report → GTN

Clinical Presentation

1) BPV even after suction/Evacuation

2) Shock (typical seen in Invasive mole) → B into peritoneum

3) Uterine subinvolution

4) presutant thecalcentine cyst

(N) S/E disappear c̄ in 2-4 months

5) Presents c̄ Metastasis : Lungs m/c

Vagina → Bluish → vascular, suburethral, Nodule

In x ray : Charrio ca

① Cannon ball apperance

② snow strom apperence

m/c GTN : Invasive mole

m/c GTN after Full term pregn : Chorio Ca

Chorio Ca occurs m/c after which preg : Complete mole

Invasive Mole	Chorio Ca
HPE : Villi present	Villi absent

chorio Ca	PSTT
① m/c	① rare
② m/c after C mole	② Full Term pregn
③ Malignant	③ Benign
④ Tumour Markers - Hcg	④ Tumour Markers HPL > PL Alkaline phosphate
⑤ dimorphic cells	⑤ Mononuclear monomorphic cells
⑥ More Haemorrhage and necrosis in HPE	⑥ less
* ⑦ chemosensitive ↓ TOC	⑦ chemo resistant ↓ Hysterectomy

→ Mx of GTN ←

Staging

Stage 1 : limited to ut

2 : outside ut & in pelvis (vaginal meta)

3 : Mets to lung

4 : Mets to elsewhere

→ Scoring system (WHO) ←

↓

Modified by FIGO

↓

Modified scoring System

	Low	High
1) Age	≤ 39 yrs	≥ 40 yrs
2) Hcg	$< 10^3$ (good progno)	$\geq 10^5$ (poor Progno)
	>40,000 [old criteria] (poor prognosis)	
3) type of Antecedent preg →	Molar	FTP
4) duration from antecedent preg	< 4 months	>12 months
5) Size	< 3 cm	>5cm
6) no. of mets	<4	>8

7) sites of met → Lungs, Liver, brain

8) prognostic H/o chemo → single agent chemo, Multi agent chemo

final score is ≤ 6 → Low risk GTN → Mx is: Single agent chemo → Methotrexate → if resistant to Mtx → then give Actinomycin D

≥ 7 → High risk GTN → Mx is Multi agent chemo

E etoposide
M Mtx
A Actino D
C Cyclophosphomide
O oncovin

↓ Resistant

E → D_1
M → D_2
A → D_3
E → D_4 etoposide
P → cisplatin

Weekly → till 3 ⓒ

Monthly period surveillance 12 months (low risk)

High risk: 24 months

→ TWIN PREGNANCY ←

M/c type of twins: Dizygotic 70%

Monozygotic 30%

M/c type of twins: Dichorionic Diamniotic (DCDA)

Depends on time of cell division

1st 72 hrs: DCDA

4th & 8th D: MCDA

8-12 Days: MCMA

>12 Days: conjoined Siamese

M/c type of monozygotic: MCDA

Fraternal → Dizygotic twins → prevalence is variable

- geographically variable
- age
- parity
- obese / Tall women
- ART (Assisted Rep Twins)

Identical → Monozygotic → prevalence is constant 3-5

High Risk

Fetus

1. prematurity
2. IUGR
3. ↑ GCA
4. monochorionic twins
   ↓
   a) TTTS (most characteristic)
   b) TAPS (Twin anemia polycythemia synd)
   ~~c) Atrial per~~
   c) TRAP (Twin reversed atrial Perfusion)
   ~~d)~~
5. Monoamniotic twins
   a) ~~chorecter~~ characteristic
   ↓
   cord entagilment
   conjoined twins

Mother

1)
2) PE
3) GDM
4) obstetric cholestasu
5) AFLP
6) Malpresentation
7) PPH

Markers of DC	MC
1) USG 1st Trimester 2 seperate PL	
2) Twin peak sign (or) lambda sign ↓ Ideal time 10-14 weks	2) T-sign (or) Inverted T sign

Opposite sex twins

How many layer in the dividing memb

4 layers
↓
2 Chorions
2 Amnions

thickness of dividing Memb
≥ 2 mm

4) 2 layers
↓
Amnions

5) 6

⇒ cord entaglement
↓
delivered by CS at (32–34 wks)

⇒ mk conjoined twins
↓
Thoracopagus twins

⇒ TTTS a) MC (DA, MA) → seen in MCDA
↓
Cause → b) Vascular Anastomosis
↓
DA { deep artery of one baby connecting to deep vein of another baby
MA { have Many superf Ar–Ar Anastomosis is present

Deep artery (Donor)	Deep Vein (Receipient)
↓	↓
Intravascular Vol (↓)	cardiac overload
↓	↓
(↑) Vasopressin	ANP, BNP
(↑) renin angiotensin	↓
↓	Polyuric
oligouric	↓
↓	Polyhydrominous
oligohydrominos	

at criteria
↳ DVP Deep Vertical Pocket
↑
Simultaneous presence of both
↓
Δsis of TTTS

Prognosis by Quintero Staging

Stage 1 : Poly/oligo Bladder of oligo - visible doppler
Doppler (N)

2 : Poly/oligo bladder invisible
Doppler (n)

3 : Poly/oligo , Doppler abn

4 : either (both hydrophic)

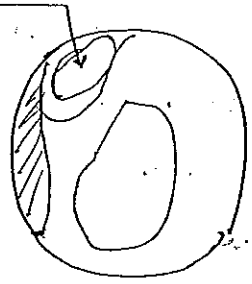
5 : either (both death)

Treatment TTTS :

<26 → { laser ablation of vascular anastamosis

>26 amino reduction

Stuck twin



Vanishing twin : Spontaneous loss of one gsac

~~fetus papyraceus~~ :

fetus papyraceus : one twin dies
↓ paper
↓ Mummified
↓ compressed (papro)

Fetal Reduction: Multifetal preg

↳ ① Do it at 10 – 13 wks

② By inj kcl into the fetus

③ Reduce it to twin

but never to singleton

Superfecundation

Superfetation

Blood Chemerism, two sets of stem cell types

in monochorionic & Dizygotic twins

→ Outcome of twin pregnancy ←

outcome depends on:

Lie of 1st twin : m/c combination → Vx – Vx
1st 2nd twin

2nd m/c Vx, breech
1st 2nd twin

⇒ as long the 1st twin is longitudinal we can do VD

if 1st twin is non longitudinal " " " CS

① 1st Vx
2nd breech } VD

② 1st breech
2nd Vx } V.D can done but CS is better

↓

these twins have twin interlocking

↓

So do C.S

③ 1st Vx → delivered VD

2nd breech → Assisted VD

④ 1st Vx → IPV

2nd Transverse → Internal podalic Version

↓

breech extraction

↓

Indication : 2nd twin Transverse lie and non scarred uterus

6/07/2017

A case of prevs C.S

High risk for pregn

Elective Repeat C.S

↓

Absolute Indication

1) Prev classical C.S

↓

weak scar

Rupture risk 4-9%

↓

LSCS

2) Prev H/o rupture of

Once of rupture is always a rupture

3) ≥ Prev 3 LS.CS

4) H/o of Sx in which ut cavity opened

↓

Hysterotomy / sometime myomectomy

↓

+ a dark on upper segment

Trial of VBAC

Impending Rupture

↓

Can be happen any time

Dehiscence

↓

when all layer of myometrium have given way but the overlying serosa is intact

↓

(fetus will not be expelled)

Rupture

↓

all layer of myo including serosa have given way

↓

(fetus is expelled out into Abd cavity)

5) Any c/I for VD

6) CPD in current preg

→ $G_2P_1L_1$ c̄ prev LSCS done for CPD, now 38 wks preg cephalic present, V_x at −3 pelvis seems adequate

→ $G_2P_1L_1$ prev LSCS, 40 wks preg & C-S done for FD no cephalic px and head is (free floting) the pelvis is sufficient

Relative Indications:

$\boxed{CS > VD}$

1) Prev C.S & currently the baby is in breech

2) Post term pregn (>42wks)

3) Macrosomic baby

→ Signs / symptoms of impending Rupture:

1st: changes in FHR
↓
(Non reassuring FHR)

1st change in FHR is Tachycardia

m/c: bradycardia

2nd: Maternal tachycardia
3rd: Scar tenderness / pain

c̄ FHR change

↓

Mx in this condition is

(Emergency C-Section)

↓

In this: Scar dehisence

no a clinical finding

Intra op finding

Signs / symptoms of Complete Rupture in VBAC

1) Maternal tachycardia
2) ± " Hypotension
3) generalised abd pain
4) FHR < severe bradycardia / FHR absent
5) Fetal parts → supf palpable
6) Ut contour → once rupture the contour is lost

7) Ut contrctn ⇒ sudden stopage

8) Rupture ⇒ fresh BPV

9) Loss of station
↓
Characteristic to Rupture

10) ± gross Haemoturia
↓
Involves bladder
↓
Mx is emergency laprotomy
↓
Goal: a to repair
↓
if can't repair (or) Haemostasis
↓
Hysterectomy

Prognostic Marker for VBAC is :
↓
Prev Vaginal Delivery

Q) $G_3P_2L_2$ - Both prev CS

$G_3P_2L_2$ < 1st VD, 2nd LSCS

→ VBAC ←

Can we do Induction of labour in prev C-S

Yes - Not CI

Prefer. spont labour

drug	not to be used
Oxytocin	Misoprostol (PGE1)

→ Induction of Labour (IOL) : ←

Contraindication :

1) Contracted pelvis
2) prev classical CS
3) H/o ut rupture
4) Transverse lie
5) Cat 3/4 FHR bracings
6) Major degrees of PP

Pre induction score / ~~Bishop's~~ Bishop's score

Cervix	score 0	1	2	3	4
Position					
Consistency					
Effacement					
Dilation					
Baby's station					

Score ≥9 is considered has favourable → successful Labour

≤5 is considered has unfavourable score

↓

In this case

↓

Do: cx ripening

Medical method
① Misoprotol (Tab)
② Cervidprime (Gel)

Mechanical method
① Bulb of Foley's Catheter
② Laminaria tents

laminaria tents
↓
(Hygroscopic osmotic dilation)

Misoprost (Tab) — Cervidprime (gel)

↓ PGE — ↓ Dinoprostone

↓ PGE2

a) 25 mcg

b) P/V every 4 hourly for a max of 6 doses

↓ used in IDL

a) 0.5 mg (sml gel)

b) 3 applications (ideally in cx canal but can also be kept in post fornix)

c) Ideally 6 hourly

Better as Misoprost

But clinically Cervidprime is used more

Ripening + Lit contractn (4 hrs waitn) ↓ oxytocin ↓ Not in Prev C.S

only cx ripening ↓ 6 hrs waitin ↓ oxytocin ↓ Prev C.S → (single application)

Can you augment labour

↓

after contractn appear to ↑ in frequency, intensity & duration

↓

can be done → (but ↑ risk of rupture)

↓ Partically not Done ↓ continuous FHR monitoring

3) ECV (Prev C.S is a Relative C/I)

IPV (prev C.S is a absolute C/I)

4) Ideal time for conception after C.S

↓

ⓐ 18 months

ⓑ Min duration : 6 months

TOPIC → Rh -ve ←

Rh-ve

↓

HBg +ve

↓

FMH (Fetomaternal Haemorrhage)

↓

It brings the sensitization of mother

↓

Amount of Ab (Critical titre)

↓

Ab cause destroytion of baby RBC

↓

Anemia in fetus

↓

Significant Anemia (Present / not)

↓

If sig : Heart failure

↓

Hydropic baby

↓

baby will die

⇒ 1st preg is safe becoz

① Baby is delivered out

② IgM Ab are produced (Does not cross pl.)

③ Ab produced are in less No.

⇒ ① Gene for Rh location: Short Arm of chromosome

② Min fetomaternal Haemorrhage that can bring about sensitization is 0.1 ml

③ Anti-D [effective, only when mother is not sensitized]

↓

Prevent Sensitization

Anti D — <12 wks 50 mcg
— >12 wks 300 mcg

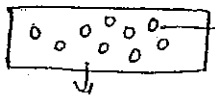
(*) mcg in India & US
IU in UK
1 mcg = 5 IU

300 mcg = can neutralize
15 ml of Red cell of fetal
30 ml of Mother Red cells

⇒ If FMH is more then how do we know the dose

Dose : KB test

↓



→ Red cell of fetus

↓

formula = no. of vials of Anti D

⇒ Anti D inj ; IM inj in Deltoid Muscle

=> Most imp it has to be brought in a cold chain (or) else it is not given as it cause anaphylactic reactions

Case: Rh-ve
HBsAg +ve
↓
Seen for sensitization

- ICT (-ve)
  ↓
  {Not yet sensitized}

- Rpt ICT at 28 weeks
  [Ideal is every 4th week]
  ↓
  not affordable

Give Inj Anti D of 300 mcg
(Prevents sensitization at 28-40 weeks)
↓
Mother delivers at term
↓
BBg +ve → (Ideal) DCT
→ Another Dose of Anti D
↓
Ideally given c in 72 hrs but given upto 28 days

Case 2:

Rh -ve

Husband BG +ve

↓

ICT +ve (sensitized)

↓

Critical titer 1:16

↓

ICT +ve < critical titer

↓ (look for ICT titer) 4 weekly

Maintain ICT +ve < critical titer

↓

Maintain upto term

} Monitoring of titer

↓

Deliver the baby

Case 3:

Rh -ve

HBG +ve

↓

ICT +ve > 1:16

↓ Next

look for Fetal Anemia

↓

by MCA doppler

PSV (peak systolic velocity)

[multiple of median (mom)]

- ≥ 1.5 mom
  - ≥ 34 weeks
  - < 34
- ≤ 1.5 mom → ↓ insignificant anemia

≥34 wks → Delivery

< 34 wks → Cordocentesis, Hb < 8 gm%

↓ keep monitoring of MSC & PSV

↓

strat IUT (Intraut Transfusion)

Hydrops = Hb < 5 gm%

⇒ △sis (if any of 2 mets)

↓

ⓐ Pleural effusion

ⓑ pericardial ,,

ⓒ Ascites

ⓓ subcutaneous odema

⇒ Placentomegaly & Polyhydromnios also seen in Hydrops fetalis but they are not △sitic criteria

⇒ Buddha sign (Halo around Head)

↓

sign of Hydrops fetalis, Not sign of IUD

* m/c/c of Non immune Hydrops → CVS anomalies
* Parvovirus B19 can cause ↑
* m/c/c of Haemolytic dis of the New born : Rh incompatability

TOPIC → HEART DISEASE ←

⇒ Heart dis in pregn : Means HD in mother

s/s in underlying Heart Disease

Symptoms
ⓐ orthopnea
ⓑ PND
ⓒ Exertional Dysponea (physiological)

Progressive Exertional Dysponea (pathological)
↓
[Coarsening of NYHA grade]

Signs :
ⓐ Cyanosis
ⓑ Clubbing
ⓒ Systolic mormor ≥ grade 3
ⓓ Diastolic murmour
ⓔ Cardiomegaly
ⓕ persistent distended Neck veins (raised JVP)
ⓖ Arrhythimias / CCF
ⓗ PAH → loud P_2
ⓘ Wide split S_2

m/c Heart dis in preg = Mitral stenosis

m/c Congenital Heart Dis in preg = ASD

m/c congenital valvular Heart disease = Mitral vascular prolapse (MVP)

Heart Disease

Poor Prognosis	Good prognosis
→ High mortality Rate 50%	1) ASD
	2) VSD
1) marfan's synd c̄ aortic root	3) corrected TOF
2) Eissenmengers	* 4) Ebstein
3) Coarchation c̄ aortic valve	5) PDA
	*6) MVP (Mitral valve prolapse)

→ Advise TOP [MTP]

↙

4) EF (Ejection fraction <45%)

5) NYHA 3/4

6) Severe MS Valve area <1.5 cm²

Severe AS " " <.1 cm²

⇒ Which HD in preg is m/c/c maternal mortality is "Mitral stenosis"

HD is Indirect cause

↓

Managment

Severe MS → Undergo corrective Sx befor conception

→ After pregnancy = Balloon valvotomy

↓

20 weeks

=> Valve replacement is C/I in pregn than it is High mortality ≥30%

Important points:

1) V.D
2) C.S reserved for obstetrictic Indications
3) IOL not C/I
4) preffered → spontaneous labour
5) HD pt's in Labour: Pain relief → cause tachycardia ↓ H compensate the tachycardia ↓ slowed ↓ HR↓
   ↓ by epidural analgesia (or) Nouraxial analgesia
6) propped up / LLP
7) O_2 by mask 5as
8) Restrict IV fluids @ 75 ml/hr in AS ↓ they.
9) Restrict No of P/V examinations
10) ARM can be done
    Spon rupture > ARM
11) Memb rupture = prophylatic Antibiotics
    IV Ampicillin Gentamicin
12) the most imp is cut short the 2nd stage of labour (forceps > vacuum)

13) Methergine is C/I in HD

14) Hang her legs down from delivery table
→ Inj Lasix

Highest risk CCF

Immediate PP > 2nd stage of labour > 28-32 wks

15) Metallic Heart Valves
↓
She need life long anticoagulants

① 1-12 weeks : Heparin → warfarin in 1-12 (feto-toxic) → chrondrodysplasia Punctata (Stippled epiphysis)

② 12-36 weeks : warfarin

③ 36 - onset of labour : Heparin

④ Stop during labour

Restart the drugs after VD = 6 hrs
after CS = 24 hrs

DOC after Delivery = warfarin (more potent)
(Heparin + warfarin)
↓ slow in onset
to compensent the warfarin
↓
then withdrawal

⇒ which HD in preg is a indication for C.S ?
1) marfan's c̄ aortic root
2) coartation c̄ " valve

3) aortic dissection

4) S.AS

⇒ HD → CS → Anesthesia
↓
Epidural > Neuraxial

In which HD, epidural is C/I =

① S. AS
② S. AR
③ HOCM
④ Synotic Heart Dis

These ④ have Reduced Ejection fraction → Give (General Anesthesia)

Epidural cause Hypotension

⇒ Aortic dissection = Medical emergency = General Anesthesia

TOPIC → FETAL MONITERING ←

→ for fetal healthlyness in preg

1) fetal movements < Primi - 18 weeks / multi - 16 weeks

DOG (↑) - FM(↑) → Reach a peak

Max at 32 weeks and then they plateau
↓
Before onset of Labour (Reduced)

Normal fetal movements = 10 FM in a 2 hrs period of Rest

10 FM in a 12 hrs period of Non rest

Reduced Fetal movement

↓

Very reliable feature

⇒ if No FM for (12 hr) = Baby is dead
(any time after 12 hr)

Next

Modified Biophysical Profile (BPP)

Non stress test (NST) → tells Acute injury to baby

Amniotic fluid → tells chronic injury to baby → [before 15 days of UPI]

Non stress test

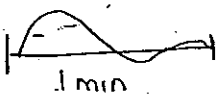
NST Machine: Graph paper

1) Baseline FHR: 110 - 160 Beats P Min

Brandycardia <110

Tachy >160

2). Beat to Beat variability: 5 - 25 bpm (n)

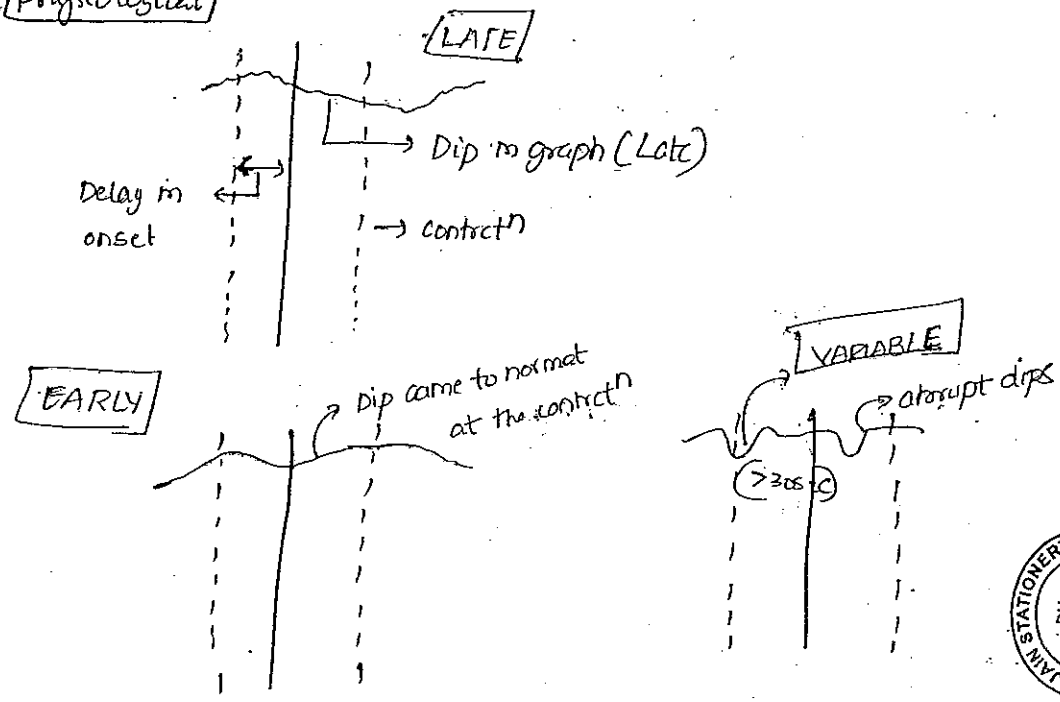


3) Acceleration: (↑) in FHR by 15 bpm above baseline for atleast span of 15 sec

4) Decelerations:
- early
- late
- variable

Early deceleration: Caused by Head compression
Late deceleration: utero placental insufficiency
Variable " : Cord compression

Physiological



Reports: Reactive
↓
2 acceleration x 20 mins
↓
Reassure the baby is safe

Non Reactive
↓
<2 acceleration x 40 mins
↓
Management
↓
Monitor further

Category 3 tracing:-
↓
Immediate TOD

1) Sinusoidal HR pattern
Causes < SF Anemia
S.F hypoxia

2) absent beat variability

(a) bradycardia

(b) late decelerations

(c) persistant variable

⇒ Variable deceleration become significant

→ ≥50% accompany of ot contractn

→ <70 bpm x 60 sec

Complete/BPP : Manning's score

* BPP is done by USG
* Done for 30 min
* Components of BPP

5 components

(1) Breathing movements ⇒ 1 x 30 sec ⇒ +2

(2) Fetal Body " ⇒ 3 x ⇒ +2

(3) Fetal tone ⇒ Flexion Exten F ⇒ +2

(4) Amniotic fluid ⇒ 1 Pocket of 2 cm ⇒ +2

(5) Non stress test ⇒ Reactive ⇒ +2

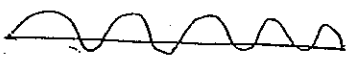
8/10 , 10/10 ⇒ (n)

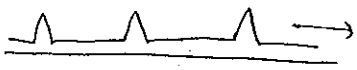
6/10 ⇒ Equivocal

≤4/10 ⇒ Delivery

→ Fetal Hypoxic Injury
↓
loss of Acceleration > loss of Breathing Movement > Loss Gross body movement > tone loss

→ Doppler ←


Reversal of End diastolic flow
↓
TOP


REDF

TOP if pog ≥ 34 wks

Ⓐ S/D beyond 30 weeks

Utefetal Insufficient <3

MCA Doppler ~~UPI~~ ~~in early~~

UPI in early stage: Retribution

Middle cerebral Artery → brain sparing effect

Ⓐ in MCA doppler

Best use in MCA Doppler is in = Fetal Anemia

PSV ≥ 1.5 mom

→ Intrapartum Monitoring ←

M/c Intermittent FHR auscultation

Ideal:

	Low risk	High risk
1st stage of Labour:	every 30 min	every 15 min
2nd " " " :	every 15 mins	every 5 min

⇒ Best way of Fetal monitoring = Fetal scalp ph testing
↓
Scalp blood ph

(n) fetal ph = 7.25 - 7.35

7.25 - 7.20 (borderline) → Rpt test after 30 min
↓
If it comes Acidosis
↓
Delivery

<7.20 (Acidosis)
↙
Delivery

Fetal scalp stimulation :- Stroke scalp
↓ ↓ (n)
Rule out Acidosis ← FHR(↑) (10bpm)

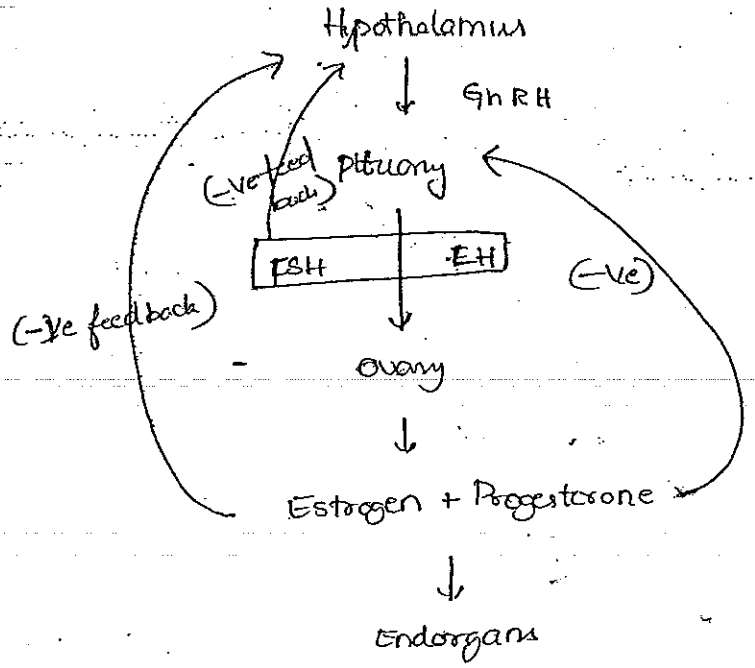
VAST (Vibro acoustic stimulation test)

take a artificial ~~larym~~ larynx
↓
Maternal abd
↓
High intensity sound waves
↓ (n)
(↑) 15 bpm above baseline τ in 15 sec of stimulation

Fetal Pulse oximetry

fetal oxygen sat < 30% × ≥ 2 min
↓
Hypoxia (abn)

GYNAECOLOGY



Estrogens → steroid: Cholesterol ← Progestorgens

C18
- N → E_1 Estrone, E_2 Estradiol, E_3 Estriol
- Synthetic → Ethinyl Estradiol, CEE → OCPs component → m/c progesterone in OCP; u Desogestrel

C21
- N → Progesterone
- S → progestins → C19 compounds (Androgens) → Generations

Q/ZA

OCP
- Dose classification → depend on oestrogen
- Generation classtn → depend on progestins

1st ⇒ norethindrone

2nd ⇒ LNg (Levonorgestrel)

3rd ⇒ Desogestrel / gestodene, Norgestimate ← Least androgenic

4th ⇒ spironolactone (Drospirenone) ← Anti androgenic

Potency order

$E_2 > E_1 > E_3$

⇒ Most potent: Ethinyl estradiol: usually in mcg

Estrogens:

→ source E_1: predominant in post menopausal ♀

↓

Androstenedione

Aromatase
↓
inhibitor estrogen

↓

E_1 → Fat (Adipose tissue)

Source of E_2: 2 cell 2 gonadotropic theory

↓

theca cell

granulosa cell

Theca cell	Granulosa cell
↓	↓
Activated by LH	Activated by FSH
↓	↓
Androgen	estrogens
↓	
Aromatase	17 OHLase

+ve

-ve

E_3: placenta
specific to preg

E_2: throughout the reproductive life

Progesterone source:

↓

(N) Corpus Leuteum.

FSH → gc → Estrogen

LH → gc —(luteinization)→ Progesterone

Estrogen	Progesterone
(1) Bound form → mainly SHBG (a) albumin → 1% is free	(1) Binds to albumin small amount : CBG → 2% free form
(2) SHBG : Syn in Liver estrogen ↑es the SHBG production	(2) Receptor : Intracytoplasmic (in the absence of Ligand Binding)
(3) Repectors: Intranuclear	(3) End product: Pregnanediol
(4) End product: Glucuronides (some sulphanides)	(4) Effect at ut Ut Non preg: protective for Endometrium stops proliferation
(5) Effect at ut Ut Non preg: proliferation of endometrium (R.F of Endo Ca is estrogen)	{E+P is not Risk factor for Endometrial Ca
(6) Effect on preg ut Growth	(5) Effect on preg ut a) Growth + smooth muscle (b) relaxation (maintain preg)

⑦ Effect on Cx

Cx Mucus: * thin copious watery

* Spinbarkiet (streatched)

* Ferning ⇒ oestrogen
↓
(E + ↑ Nacl)

Day 8 + Day 18
appear disappear

⑥ Effect on Cx

Cx Mucus: * Thick
scanty
viscous

* TACK
↓
If stretched it breaks

Porrovulatory Cx Mucous ↓ oestrogen ⇒ fertile phase

Cx Mucous ↓ used as natural conceptions = Billings natural Methods

Best Natural Method = Symptothermal
↓ Cx Mucous + Basal body temp

Mechanism of action of POP's : change mucous
↓ → Natural contraception
stops sperm to enter
Infections impermeable
↓
Natural defence

⑧ Falloplain tubes
↑ motility
↓ secreations

⑦ Falloplain tubes
↑ secretion
↓ motility
↓
R.F for ectopics

⑨ Effect on Vagina
Supficial cell
↓
Pyknotic nucleus
more cytoplasm

⑧
Postmenopausal women → Intermediate cells
↓
Parabasal cells } predominant
basal cells
(more nucleus less cytoplasm)

* Supt cells : more pink (cytoplasm)
less blue (nucleus)
↓
High karyopyknotic Index

* Intermediate cells
more blue (nucleus)
Less pink (cytoplasm)

(10) Breast :

Ductular development

(9) Breast :-

Glandular development

Breast cancer =

(11) Water & Salt

Retension

(11) Water & Salt

Excreth

(12) cholestrol ↑ HDL
↓ ↓ TG
↓ total ↓ LDL

(12) ↓ HDL
↑ LDL

⇒ Coronary dis are more in men bcoz of lack of oestrogen

(13) Bones [mineralisation
closure of epiphysis

[Percocious puberty
short statur]

(13) Bones

No effect

(14) Coagulation prolifi

Hypercoagulable
(7, 8, 10) ↑ed
C/I in VTE, CAD, Stroke k/c

(14) Cagulation prolifi

E – upregulation of P receptors in endometrium

P – downregularation of E Receptors on endometrium

Estrogen $\xrightarrow{-ve}$ on FSH

Estrogen $\xrightarrow{\text{low amount}}$ on LH
$\xrightarrow{-ve}$ → Neuroendocrinial phenomena
$\xrightarrow{\text{High amounts}}$ on LH
$\xrightarrow{+ve}$

Progesterone $\xrightarrow[+ve]{\text{Low}}$ LH/FSH

progesterone $\xrightarrow[-ve]{\text{High}}$ LH/FSH

E and P $\xrightarrow{-ve}$ Hypo (a) GnRH

→ Testosterone ←

↓

Source: (major)

50% → peripheral conversion (Androstenedcol)

25% → adrenal

25% → ovary → (*) Theca interna

From ovary = max androgen

↓

Androstenedione > DHEA > testosterone

↓

from ovary

DHEAS: from adrenal gland

* C_{19} compounds

* steroid receptor (intracellularly) intracytoplasmic

* Bound = SHBG
albumin
1% free in ♀
2% " " ♂ → main source
[Leydig cells]

Sertoli cells
↓
a) Spermatogenesis
b) BTB Blood testies barriers
Sertoli - Sertoli
c) MIS
d) TBP (testos pirindine protein)
e) Inhibin
f) Relaxin
g) estradiol
h) temp sensitive cell (Sertoli)
i) Main Hormone
FSH + testosterone
↓
main for spermatogenesis

j) Gene for Androgen Receptor
↓
located on Long arm of X Chromosome

testosterone $\xrightarrow{(-ve)}$ SHBG synthesis

↓

End products → oxosteroids / ketosteroids

Androgens in ♀ — Functions:
1) Pubic / Axillary
2) control of libido
3) Indirectly for production of E2
4) Intra ovarian environment is androgen predominant → arrest the follicular growth

→ Pituitary ←

↓

* Gonadotropins
FSH / LH

* Ant pituitary
- Pulsatile
- Basophils
- Polypeptide Hormone
- LH $t_{1/2}$ = 20 min
  FSH $t_{1/2}$ = 3-4 hrs

* Receptors: Memb bound receptors
G protein coupled receptors

FSH receptors: Granulosa cell
Sertoli cells

LH receptors: ♂ Leydig cell
♀ theca / granulosa cells

FSH

* Functions: ① selection of the a cohort of the follicles
② selection and growth of dominant "
③ Has role in ovulation:
↓
collagen breakdown & release of ovum

LH

* Functions ① ovulation
② formation and maintainance corpus luteum
③ Final growth of follicles.

LH surge $\xrightarrow[(24-36)]{36\ hrs}$ ovulation

LH peak (75 mg) $\xrightarrow[12\ hrs]{}$ ovulation

⇒ Initiation of LH surge: High levels of estrogen
min (200 pg × 48 hrs)

Maintainence of LH surge: E + P

⇒ True for ovulation (co) befor ovulation
↑ LH and ↓ed FSH
LH peak > FSH peak
↓
brought by small progesteron peak

P syn begin $\xrightarrow[36\ hrs\ before\ ovulation]{}$ Before ovulation

⇒ Resumption of meiosis I happen 36 hr before ovulation by LH

→ Hypothalamus ←

* Release of GnRH
↓
released from arcuate nucleus

Neuron : secrete GnRH derived from
↓
olfactory Placode
migrate → olfactory N → Hypothalamus
↓
In kallmann synd
↓
no GnRH + Anosmia

GnRH : decapeptide t½ 3-4 min

⇒ Dormant Hypothalamus of Puberty (after birth)
↓
pulsatile release (due to neurosal transmitted)
& brings puberty

↓ GABA
↑ Leptin
↓ NPY
↑ kisspeptin → m/c
↑ glutamate

↓

↑ GnRH (at night)

LHRH — other name for GnRH

↓

↑ LH (at night time)

↓

↑ LH / FSH (Night / Day)

GnRH

(High) ↓ LH — pulse frequency — FSH ↓ (low)

GnRH Agonists

→ Continuous → initial flare response → ↑ LH / FSH → ↓ → E+P → -ve feedback → suppress the ovary

→ Pulsatile → ↓ → ovary stimulated

Is: Precocious puberty

2) endometriosis

3) fibroid ut

4) Hirsutism

5) DUB

6) Breast Ca

7) Prostate Ca

m/c used in Infertility

goserelin, Naferelin, Buserelin — FDA approved drugs

GnRH → Not orally
→ only S.C, IM, IV, Nasal spray
→ > 6 months (Hyperoestrogenic Bone loss)

Pulsatile
→ ovulation induction

GnRH Antagonists : m/c cetrorelix
↓
given in oral formulation

Luteal phase → Secretory
Follicular → proliferative
↓
ovary

corresponding changes in endometrium

Luteal PHASE

* onset of LH surge → ovulation
→ Corpus luteum → ↑ Progesterone
↓
maintained

(-ve) feed back → ↑↑ Peak level
↓
↓ed LH (Late luteal phase)

8th day after ovulation

C.L. regresses
↓
corpus albicans

fall of progesteron
↓
called as withdrawal

↑ levels of
PGF2α

Blood vessels of endometrium
↓
Vasoconstriction of spiral arteries
↓
Avascular necrosis & shed off
↓
menses

myometrium → 1° Dysmenorrhea

corpus luteum:

Life span: 14 days

⇒ all girls have constant luteal phase but have variable follicular phase

⇒ max activity of CL = 8th Day post ovulation

Progesterone
estradiol
Inhibin A & B
Relaxin

Granulosa cell: Inhibin B

luteolysis: ① HCG rescues the corpus luteum
↓
in pregnancy

⇒ Corpus luteum maintained by LH

⇒ " " " " HCG in pregn

⇒

	Textbook	FIAD
② N length of menstrual cycle	21 – 35	24–38
③ (N) amount of blood loss	= 80 ml	
avg blood loss =	35 ml	
	35 – 50 cc	

④ no 2–7

→ Abnormal Ut Bleeding ←

① Menorrhagia: Heavy blood loss (or) More no of Days

Amount >80 ml

Durath > 8 days

Cycle length is Normal

② Hypomenorrhea: <2 days

③ Polymenorrhea:

④ Oligomenorrhea:

⑤ Metrorrhagia:

Modern classification

PALMCOIN

→ Puberty :

"apperance of secondary sexual character"

" Hypothalamus – Pituitary – ovarian axis "

↓

pulsatile Release

Puberty	Female	Male
avg age	~~10-15~~ 10.5 yrs	11.5 yrs
precocius	<8 yr	<9 yr
Delayed	13 yrs	14 yrs
1st sign	Growth spurt	testicular enlargment
1st visible sign of puberty	Thelarche → apperance of breast buds → Tanner stage 2	testicular enlargment

⇒ Precocious : m/c in Girls

↓

So girls should go Neuroimaging

m/c/c is Idiopathic [80%]

Brain tumour [20%]

↓

Hypothalamic Hamartoma

(gelastic seizures)

↓

laughing spells

Precocious Puberty

- **Central**
  - ↓
  - Brain [Hypothalamus]
  - ↓
  - Levels can differentiate central (or) peripheral
  - ↓
  - LH / FSH ↑
  - Iso sexual

- **Peripheral**
  - ↓
  - [Sex hormone producing tumours]
  - ↓
  - MCQ Mc Cune Albright synd
  - ↓
  - * Precocious puberty
  - * Polyostatic fibrous dysplasia
  - * Cafe Au lait
  - ↓
  - LH / FSH ↓
  - Depends on what type of Hormone secreting tumour ← { Hetero sexual ← Androgen secreting tumour; Isosexual }

→ Delayed Puberty : m/c in Boys

m/c/c constitutional delay

↓

unlikely in girls

MCQ → Order of Puberty

Growth spurt → Thelarche → Pubarche / Arcarche

Peak Ht velocity ← Breast (Tanner 3)

Peak Ht velocity ← Pubarche (Tanner 3)

Peak Ht velocity → 6 months → Menarche

↓

avg age 12.5 yrs

Precocious < 10 yrs

Anovulatory : in Adolescent girl

(80%) ↑ m/c/c ← irregular cycles

(20%) ↓ coagulation abn ⇒ DOC : OCP

other drugs → only P

- → only E

Heavy blood loss → metchimic acid

& unstable vitals

→ MENOPAUSE ←

"Absence of mensus for 12 consequtive months in respective age groups."

avg age : 51 yrs

avg age in India : 47 years

→ " All the follicles undergo Atresia "

Premature menopause	Delayed menopause
• ≤ 40 yrs	• 55 yrs
• Primary ovarian insufficiency	↓
• Premature ovarian failure	Needs Assessment

• Asic of premature

S. FSH levels ≥ 40 IU on 2 consecutive occasions × 1 month apart

- Hormone
  - E_2 (<20 pg)
  - FSH (↑) → raise is due (Inhibin ↓)
  - LH (↑)
  - ovarian androgen production (continous)

Menopausal transition

- m/c Symptom: Vasomotor
  - Hot flashes → High body temp and sweating more during night
  - cause estrogen withdrawal

Hot flashes coinside c̄ LH surge

Moderate- severe Hot flashes: HRT is given

Given when

1st line a) ut intact → E+P
b) post hysterectomy → E

a pt cannot take estrogen i.e estrogen is C/I then SSRI is given.
↓
only given in PMS [DOC]

2nd line

3rd Line: antiepileptic

Pregalbin / gaba pentin / clonidine

ut intact: E + P (cannot be given)
↓
then use mirena

SERM

tamexifen
Raloxifen
ilomiphine
Ormeloxitene
cloniphene

Bazedoxitene + estrogen = Protects endometrium

Bazedoxitene → SERM / SERD

SERM m/c side effect = Hot flash

So SERM should not used in Rx of Hot flash

only (Bazedoxiten + estrogen) can be used

→ Perimenopausal Women ←

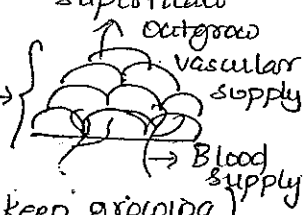
→ Anovulatory

" period of Amenorrhea t/b Heavy bleeding "

" They have ~~up~~ unapposed estrogen "

↓

Ted proliferation →



↓

but not shed off (keep growing)

↓

Superficial outgrow vascular supply (No blood supply)

↓

So they shed off

↓

So called estrogen breakthrough bleeding

Pt. present with

- Acute blood loss
  - ↓
  - 2 months 15 days
    - Stable pt.
      - ↓
      - High dose oral estrogens ($\bar{c}$ in 24 hrs)
      - ↓
      - It scale the breaks in layer by proliferation
      - ↓
      - $\bar{c}$ in 24 hrs
      - ↓
      - then put on Progesteron
      - ↓
      - No single Progestron but in OCP
    - unstable pt.
      - ↓
      - take to OT
      - ↓
      - immediately stop bleeding through doing D and C
      - ↓
      - ⇒ D&C u ~~not~~ c/I in a unmarried women
      - ↓
      - after D&C
      - ↓
      - estrogen IV
      - ↓
      - P/OCP
- Amennorhea

  2 ½ months UPT → (-ve)

  ↓

  thick endometrium

  ↓

  Rx : P/OCP

→ Metropathica Haemorrhagica ←

- 40-45
- 2m amenorrhea → heavy bleeding
- anovulatory
- Endo. → absence of progesterone (absence of secretory phase)
  → Swiss cheese pattern

  Cystic granduler Hyperplasia
- Ovaries – cystic
- Painless bleeding
- Toc : progesterone therapy

Case: ≥ 45 yrs ♀ - AUB
↓
1st thing do: [Endometrial sampling]
and then medical Mx

→ AMENORRHOEA ←

Primary amenorrhea

→ "absence of mensis by 13 years of age in the absence of 2° sexual characters."

[OR]

absence of mensis by 15 years of age but this time in presence of 2° secondary sexual characters

→ m/c/c is gonadal dysgenesis
↓
Turner's synd

Secondary amenorrhea

→ "absence of mensis for 3 consecutive months (90 days) previously menstruating female"

→ m/c/c is preg [physiological]

PCOS [pathological]

Primary Amenorrhea
↓
Thelarche
[Secondary sexual characters]

- Present
- Absent
  ↓
  Absent Thelarche:
  1. Gonadal dysgenesis
     - a) turner
     - b) pure
     - c) mixed
  2. Kallmann's synd
  3. Constitutional delay

Gonads
- ovary (Y absent) → SRY absent
- testis (Y chromosome) ↓ (should have) Functional SRY

Ovary: N (or) abn depends on XX chromosomes

Internal genitalia:
- Male (MIS, tutosterone)
- Female (absence of androgens)

External Genitalia
- Male (Tutosteron → DHT)
- Female (absence of androgens)

Turners dygenesis	Pure dysgenesis	Mixed dysgenesis
① 45XO	46 XY ↓ Normal karyotype ↓ pure (SLOYER's synd)	mosaic 45OX / 46XY ↓ ② Streak ovaries ↓ testes but abn
② ovary appear ↓ ③ fibrotic streaks (streak ovaries)	② even Y is present it is not functional SRY mutation ovary appear ↓ streak ovaries	

③ Ut is present (Hypoplastic)	③ Ut is present	③ Mullerian structures ↓	Wolffian structures ↓
④ Ext genitalia ↓ will be female ↓ but the structure donot grow further they remain as if **Infantile**	④ Ext genitalia ↓ female	④ They have ambiguous ext genitalia	
⑤ Pubic / Axillary hair ↓ from adrenal androgens ↓ from adrenal perpheral conversion ↓ **Present** →	⑤ Present	⑤ Present	
⑥ Most characteristic feature : Short stature → ↓ XO ↓ No X chromosome ↓ In this X there is ↓ SHOX gene absent ↓ Mandatory for epiphysial growth	⑥ N / Tall	⑥ N / Tall	

⇒ Turner's

- Webbed neck
- Low Post hair line
- m/c Bicuspid aortic Valve
- cubitus valgus
- Streak ovaries
- Short 4th metacarpel
- Shield shaped chest
- Nipple widely placed

⇒ IQ in turner

- Normal
- If one extra x is present then IQ is sublevel

⑦ Male pseudohemophodite
↓
phenotype is female 46XY
female pseudohemophodite
XX ↓ phenotype female
ext genitalia
→ as soon as Δsu done should do (Gonadectomy)

↓

m/c/c CAH (Congenital Adrenal Hyperplasia)
m/c 21OHLase

True Hermaphodite
karyotype mosaic
ext genitalia → ambiguious
→ gonads : ovotestis
↓
H is same as Mixed
should be differenciated c̄ Mixed by Histopathology

Eg: klinfelter's synd | XXY
↓
One gonad (female) one gonal (male)
↓
Mixed.
Both in Both gonads (syngyer's)

Turner's	Kallman's
① 45 XO	① 46XY
② Streak gonads	② (N) gonads (ovary)
③ Short stature	③ N/Tall
④ XLO anosmia	④ Anosmia
⑤ LH/FSH ↑	⑤ LH/FSH ↓
→ Hypergonadotropic	→ Hypogonadotropic
→ Hypergonadism	→ Hypogonadism

(6)

(6) M/c mode of inheritance

X Linked Recessive

F : M = 1 : 5

M/c gene KAL-1 (Anosmin)

↓

TOC : pulsatile GnRH

→ Constitutional Delay ←

(1) Diagnosis of exclusion

(2) only in male

(3) Family History

(4) Present c̄ short stature at birth but later on gain height

(5) IOC is karyotyping

→ Primary Amenorrhea c̄ 2° sexual characters and Thelarche +ve ←

1) Anullerian Agenesis

2) AIS

3) Imperforate hymen

4) Trans vaginal septum

Mullerian Agenesis

(MRKH synd ⊛
Rokitansky synd ⊛
Kustner Hauser synd)

↓

1) Triad of
- MA
- Hemivertebrae
- Skeletal anomalies

Mullerian absent

B/L renal agenesis
Horse shoe kidney — Renal anomalies

Characterised by

- Absent vagina
- absent ut
- FT proximal absent distally present

agenesis / atresia / aplasia — Absent

2) Gonad ovary 46 XX

3) Breast (Normal)

Androgen Insensitivity Synd

TFS : Testicular Feminisation Synd

1) Androgen Insensitive Receptor

Vagina → Blind ending vagina / small vaginal pouch

↑

so ut is absent

↑

produce MIS

↑

2) Gonad ~~ovary~~ testis 46XY

3) Breast (Present)

↓

testis

↓

produce testosterone

↓

at periphery — testosterone → estrogen

↓

breast (2° sexual characters)

4) Ext genitalia : Female

Androgen insensitive

↓

so no male genitalia

clitoris and L. majora Hypoplastic

⑥ genotypic XY

phenotype female

Male Pseudo hermophodite

m/c/c AIS

Inguinal Hernia

↳ undescended testis

↓

So Mx is : Gonadectomy

↓

But after puberty

↓

to attain full female phenotype

⑦ "Gonadoblastoma" is m/c tumour in undescended testis

"seminoma" is m/c malignant tumour in undescended testis

⑧ Worst Reproductive outcome

↓

No children

↓

Adoption

⑨ Vaginoplasty

MA AIS

m/c vaginoplasty by : MC INDOE

But test for both MA & AIS is = karyotyping

↓

(conformatory)

S. testosterone
- MA (N)
- AIS (↑) (male level Hormone)

→ Partial AIS ←

① Reifenstein synd

② Receptors are slightly sensitive to androgens

③ Clinical Features: of Partial AIS

* Clitoris : clitoromegaly

$\boxed{\text{Mullerian Agenesis} > \text{AIS}}$

⇒ In MA: the pubic & Axillary hair is $\boxed{\text{Normal}}$

In AIS: the pubic & Axillary hair are $\boxed{\text{Absent (or) spares}}$

→ Imperforate Hymen ←

1) (n) XX

2) Ovaries +ve

3) 2° sexual characters +ve

4) Hormones (N)

5) obstruction of outflow

cyclical abd pain

↓

Menstrual blood collected in vagina & ut

Hematometra

Hematocolpos

6) present to emergency in urinary obstructn

⑦ Do examination

Bluish bulging Hymen

↓

give Incision : Cruciate Incision

⑧ Cribriform Hymen : multiple small openings

seive like hymen

⑨ Transverse vaginal septum : m/c upper 1/3 site

⑩ Treatment : Excision

→ Secondary Amenorrhea ←

① UPT should be −ve

② Next IOC : Hormonal Assessment

↓

① TSH — Hypo / Hyper } can cause Amenorrhea and oligomenorrhea

Next

② Prolacto → Hyperprolactenemia

↓

Have −ve feedback on GnRH

m/c/c, microadenoma (<1cm)

Next

③ FSH

If PrL = <50 { Rpt PrL test after 1 month }

PrL = >50 { stress induced then MRI }

④ M/c/c presention in Hyperprl is Infertility

Hyperprol : DOC is

Cabergolin > Bromocriptine
↓ (Dopamine agonists)
twice a week

③ FSH (D2 to D4 recent menses)

then FSH (or) otherwise do
ammonerrhea random sample

FSH

- N / Low
  ↓
  FSH → N/Low
  [Serum E2 / PCT]
  ↓
  SE2 (Normal)
  PCT (+ve)
  ↓
  she is having
  withdrawal bleed
  ↓
  that she is def in progesterone
  ↓
  def in progesterone seen in
  ↓
  anovulation
  ↓
  Now a day anovulation is PCOS

- High
  → POI (1° ovarian insufficiency)
  Premature (or) failure
  ↓
  [Serum E2 / PCT progesterone challange test]
  - Serum E2 → Non reliable → should not do alone da. ē Progesterone
  - PCT → MPA medroxy P acetate 10mg for 5-10 days
    ↓
    If (-ve) report
    ↓
    It means No withdrawal Bleeding

→ $SE_2 \downarrow$, PCT -ve, E+P +ve } Estrogen is def
↓
E_2 def u in Higher centor
↓
Do MRI
↓
[trauma having infection Intradh]
↓
[Normal]
↓
Functional Hypothalamic Amenorrhea
- Anorexia Nervosa
- Stress induced
- excessive induced
- chronic malnutrition

→ SE_2 Normal, PCT -ve, E+P -ve } Everything Normal which means the endo is problem
Endo must have E+P has food
↓
Ashermann's synd

→ ASHERMANN'S SYND ←

"Development of Intraut adhesion (or) Synechiae"

① m/c/c : vigorous curettage ② H/o Instrumentation
(in PPH
MTP
DUB
tuberculin, schistosomiasis

(3) m/c/ presentation : Menstrual irregularities > infertility
↓
[Amenorrhea ⊽/b Hypomenorrhea]

(4) Screening test: ~~SMG~~ HSG

Multiple small irregular filling defects

(5) IOC: Hysteroscopy
↓
Diag + theurapeutic

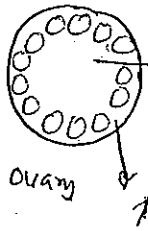
Adhesiolysis →
Recurrance
↓
Prevention : High dose E
CuT insertion.

→ POLY CYSTIC OVARIAN SYND ←

* " Stein Leventhen synd "

* Problem lie at the level of ovary

Adipose tissue (Ei) → Persistently elevated LH and ⊽ FSH
↑ Androgens
↓
Anovulation (contributes)
Initates theca cell for more androgen



→ stromal Hyperplasia → Thecal cells Hyperplasia

↑ in follicles to some extent and stopped
↓
but no Dominant follicular selection
↓
anovulation

* Upto 50% → Obese

upto 75% (50-75%) → Hyperinsulinemia

Insulin resistance

HAIRAN

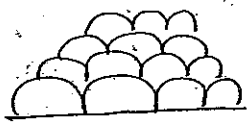
- H → Hyperandrogenisen
- I → Insulin resistent
- A → Acanthosis Nigricans → cutaneous marker for ~~Hyperins~~ insulin resistance → m/c site : nape of neck; other sites : breast, Axilla

* Rotterdam's Criteria

If any 2 of 3 criteria are met we will make a provisional diagnosis of PCOS

1st criteria ① Amenorrhea and (or) oligomenorrhoea → Anovulation

⇒ In some cases PCOS have menorrhagia



thick layer → bleed → heavy bleed

2nd criteria : Hyper~~gene~~androgenemia and/or Hyperandrogenism

- Hyperandrogenemia → Blood level ↑
- Hyperandrogenism → Clinical feature

Total testosterone

(n) ~~28~~ <7 ng/dL

PCOS mild 70-150 ng/dL

Moderat ~~Severe~~ <200mg

Severe >200mg → Androgen tumour

~~Hirsuta~~ ⇒ Hirsutism : m/c/f Now a days

~~to~~ ① terminal hair → coarsce thick
↓
male pattern distribution
↓ upper lip
Jaws
Axilla
chest

② Acne
↓
Scarring Acne
Non reponse to ~~Rx~~

③ Allopecia

Ferriman Gallway scoring
≥ 8 ⇒ hirsutism

Virilization : Never seen in PCOS
↓
Seen when blood testosteron severely ↑ed
↓
only in tuticular tumour

Not PCOS ←

clitoromegaly : Clitoral index : ≥35
Clitoral length : >1cm

* Hoarseness of voice
* ↑ muscle mass
* temporal recession (male pattern Baldness

USG Criteria of Rotterdams

1) ≥12 follicular each < 10 mm
and / or

2) Ovarian volume >10cc

⇒ Necklace pattern of follicular arrangement
↓
Is a specific finding

⇒ % of (N) women we can seen Poly cystic ovary is 15-20%

⇒ In obesity women = 50% PCOS
also present in = thin & lean girls

⇒ Diagnosis

Oligomenorrhea + Hirsutism = Do S. testosterone

Provisional Dx is : Non classical CAH
is PCOS only when ↑ [Adult onset CAH]
↓
Rule it out c̄ Non classical CAH

Rule out by screening : S. 17OHP
↓
≥200 ng/dl
+ve
Diagnosis : ACTH stimulation

D/D: ① Cushing Synd
⇓
Dexamethosone suppression test
⇓
24 hr- urinary cortisol

② Androgen Produced tumour

Lab tests in PCOS

① Total serum testosteron
- free testosteron is better but there is no single universal test
- We check for only total s.testosteron

② $\frac{LH}{FSH}$ > 2:1

③ T. E_1 ↑
E_1 / E_2 reversed
T. E_2 → (n)
Free E_2 → ↑
} More true for obese PCOS

④ SHBG ↓ Sex Hormone Binding Globulin
Testo $\xrightarrow{-ve}$ SHBG
(IR) — Hyper Insulin

⑤ IR 75gm OGTT
$\frac{F\ glucose}{F\ Insulin}$ < 4.5 - IR

6) Lipid profile ⇒ dyslipidaemia

7) USG findings ⇒ { Necklace
Stromal Hyperplastic
thick tunica }
but not Rotterdam criteria

8) TSH
9) PRL } but turn into (n) in majority PCOS pts

10) 17 OH Progesteron (confirmatory)

Clinical Presentation

Short term complication

1) Hirsutism
2) Menstrual irregularity } coexist
3) Infertility

Long term complication

1) type 2 DM
2) HTN
3) CAD
4) endo ca
5) ovarian ca
6) Sleep apnea in obesity
7) Nonalcoholic steatohepatitis
8) Mood disorder – both extremes
9) High risk for: Abortion
GDM
PE
PTL
Still birth

Management of PCOS

→ Hirsutism and Menstrual irregularity

- 1st is wt loss (5-10%)
- Medical Mx of hirsutism

↓

DOC: OCP → 3rd and 4th generations

↓

In these: Low dose pills → 30-35 mcg of ethinyl estradiol

Very low dose pills → ≤ 20 mcg " "

2nd line drugs: spironolactone

3rd line drug: Cyproterone Acetate

Others → Flutamide

Finasteride

Ketoconazole

Metformin

GnRH agonist

Eflornithine (topical)

Danazole: It has severe androgenic side effect and are dose dependent: Not used in Hirsutism

→ Rx for Infertility ←

TOC: ovulation induction

1st: wt reduction (5%) → can reduce resume spontaneous ovulation

DOC: OI in PCOS

CC clomiphene citrate (SERM)

close antogonism

2u + En

enclomiphene — Central action

↓

bind the c Receptors in

Hypothalamus

↓

↑ LSH and FSH (released)

Starting : 50 mg (D5 to Day 9)

max approved dose = 100 mg

what % ovulate c̄ CC = 80%

% conception = 40% (Not v good)

↓

bcoz it acts as antogonist

so it thickens the mucosa of Cx

⇒ It u not a teratogenic drug

⇒ Not ↑ the risk of ectopic

⇒ m/c side effects : Hot flashes

2nd m/c S/E : formation, ovarian cyst

⇒ Risk of multiple preg : 6-8%

↓

but only twins

⇒ Ovarian Hyperstimulation Synd

↓

Risk of OHSS c̄ clomiphene = ≯ <1%

⇒ max approved time = 12 months

women who are not ovulating & are obese

then use **CCT + Metformin**

MCQ obese PCOS infertility
↓
DOC: Letrozole : it is a aromitase inhibitor
↓
Starting dose: 2.5 mg (D3 – D7)
max 7.5 mg

11 JUNE 2017

2nd line Rx in PCOS infertility

Injection: gonadotrophins > laparoscopic ovarian drilling
↓
(LH/FSH)
↓
Source: urine of monopausal women (and also in blood)

If drilling overactively:
S/E → premature ovarian failure occurs
wedge Ressection (Not done Nowadays)

Inj: HMG (Human menopausal gonadotrophin)
↓
They have many impurities
↓
So Now we use Recombinent FSH/LH → expensive, effective
S/E ↑
a) multifetal preg 30%
b) OHSS – 15%
c) Higher order gestations

Drugs for ~~vulat~~ ovulation induction

3rd line: ① GnRH agonist (pulsatile manner)
↓
they become 1st line drugs for ovulation induction when cause is

Hypothalamus: Eg: kalmen's disease

→ Multifetal preg:

gonadotrophins > CC > GnRH agonist

→ OHSS:

gonadotrophins > GnRH agonist > CC > GnRH antagonists

→ Bromocriptine (ovulation induction) in case of Hyperprolactenemia

→ Aromatase inhibitors (Not FDA approved)

↓

used in obese

acts ↓

OHSS: a) enlarged follicles

b) ovaries overactivated and release inflammatory cytokine into circulation

c) Cause: ① Inj HCG (ovulatory trigger in IVF cycle)

d) Mediator: VEGF

e) Early OHSS [c̄ in 9 days of ovulation trigger] → m/c

Late OHSS >9 days

f) High Risk for OHSS ① early age: young female have more follicles

② High E2 levels >1500pg

③ Inj gonadotrophin

④ Large size follicles

⑤ Pregnancy (natural endogenous HCG ↑ed in preg)

↓

Preventions of OHSS

1) Coasting : delaying the Inj HCG (when we see the follicles are enlarged)

2) Cryopreservation: embryo preserved

3) GnRH antagonist: to prevent OHSS

4) Bromocriptine: prevent ovarian hyperstimulatory synd

5) Albumin infusion

→ INFERTILITY ←

" Inability to conceive even after 1 yr unprotected intercourse

(or) called subfertility

Both male & female contribution

↓ ↓

40% 40% - 55%

Unexplained: both 10% contribution

→ Female factor infertility

Ovarian 30 - 40%

tubal 30%

ut 15%

cx 5%

unexplained 10%

→ Ovarian factor : Anovulation

whether the cycles are ovulatory or Not

tests for ovulation:

1) Cx mucus E ⟶ P
thin ..... thick
coprous scanty

2) Vaginal cytology E → superficial cells
↓ P → Intermediate
smear → (Hormonal assessment)
→ lat vaginal fronix
→ upper 1/3 of lat wall

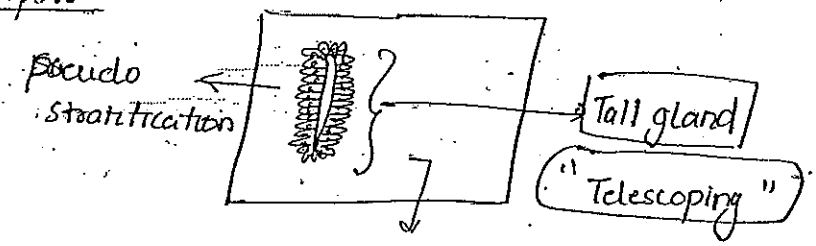
3) Basal body temp
Progesteron: ↑BBT (0.5°F)
[0.5 to 0.8°F]
↓
A graph plotted [Biphasic Graph]

4) Simplest test of ovulation
S. Progesteron level on Day 21 → ≥3 ng → Indicate cyclear ovulatory
(7 days before expected date of menses)

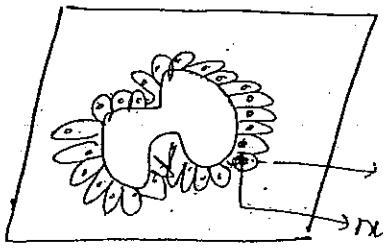
5) Best test: Endometrial Biopsy (tissue diagnosis)
↓
a) Invasive
b) done in premenstrual phase (endo is proliferated & excess)
↓
2 days before the expected date of menses
↓
Day 26
↓
If report comes has secreatory endo: ovulatory cycles

if proliferative endo: Anovulatory cycles

Report:



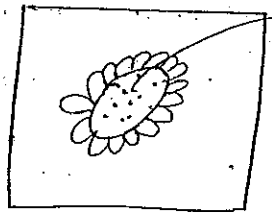
endo ↑ in Estrogen effect: Proliferative endo



P effect: Secretory endo

→ Subnuclear vacuolation

→ nucleus



→ secretions into the lumen of gland

Corkscrew glands on Day 20

Max stromal edema = Day 22

Leucocytic Infiltration of endo

↓

Premenstrual Day 26

6) Luteal phase defect: a) S. Prog <15 ng

b) Best endo biopsy

c) More than 2 days lack b/w the Histological and Menstrual dating

7) Transvaginal Scan: follicular monitoring

a) m/c used

b) call women every alternate day

○○○○○ } follicle size Ted

18-20mm

@ rate of growth 2mm/day

c) when follicle become [18-20 mm size]

this is antigrade fat

↓

the follicle is ready to rupture.

↓

Mature follicle — Antrum and fluid

↓

Timed intercourse

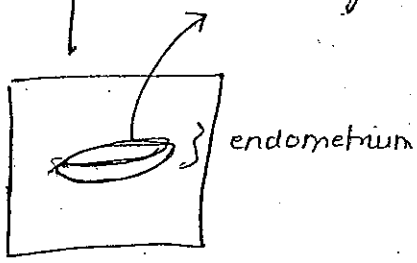
→ ovulation: 1) sudden reduction in size of follicle

2) apperance of fluid in POD

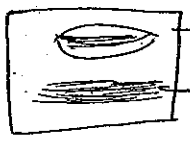
d) Check for endometrium

↓

In radiology Trilaminar endometrium / Three (or) layer endo } Peri ovulatory phase



↓



→ Single hyper echoic Line } secretory phase

→ * C pst enhancement

⇒ Only single hyper echoic line can also seen in Peri ovulatory phase

8) LH urinary kits: a) urinary LH surge

b) if +ve [it has to be C in 24 hrs]

↓

the women is going to ovulation

LH surge : in Serum

Serum LH surge : 36 hrs (ovulation)

⇒ if only LH surge asked : it means S.LH surge

9) Test for ovarian Reserve
↓
follicular pool in the ovary

A) Serum FSH : a) Done in Day 2 - Day 4

b) Day 3 FSH m/c done

↓

1-10 IU → (n)

10-15 IU → Borderline reserve

>15 IU → poor

≥20 IU → premature ovarian failure
(or)
POI

≥40 IU → diagnostic of Premature failure

B) CCCT Chemiphin citrate challenging test

100mg D3 - FSH
D5-9 → CC
D10 → FSH
} Rising values indicate poor Reserve

C) Serum Inhibin B : D3 <45 pg (poor)

D) AFC Antral follicle count :

D2 - D4

Combined score of both ovaries <10 (poor)

E) Best : S. AMH (antimullerian hormone)

S. AMH → <0.5ng (poor reserve)

AMH shows no falcuations in a menstrual cycles and that u why no False Tone Values done in any day (Making it best tut)

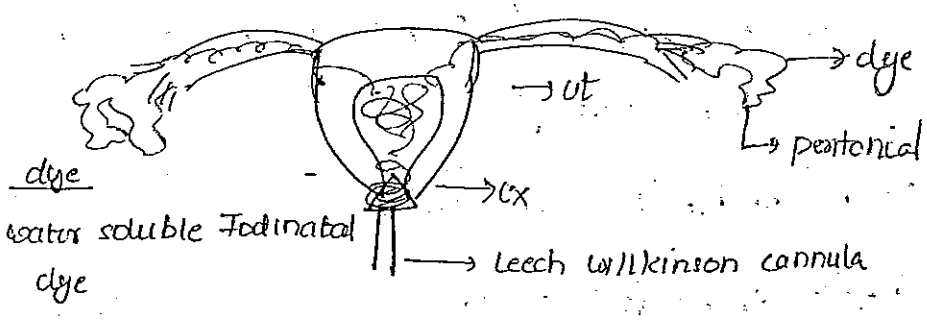
F) Serum Estradiol (not a tut for OR).

FSH + S. E2 → (N) / Low

→ TUBAL FACTOR INFERTILITY ←

* patent tubes
↓
IDC for tubal patency: HSG (Hysterosalphingography)
↓
① done in pst menstrual phase.
② D5 – 5/11 (m/c D10)

ensure that the ♀ u not preg
os dilated (easier / painless)



C/I : ① Preg
② actively Bleeding / Infections
③ Current pelvic Infection
④ K/c/o genital TB
⑤ K/c/o contrast allergy

⇒ The tubes in the x ray graphs will not be straight they are Narrow, folded.
↓
(N) tubes.

⇒ Any thing that distrubs the ut cavity HSG can peak up that.

⇒ HSG is a screening test : So, we have to use goldstndrd for tubal defects.

Goldstndrd : [Laproscopy + chromopertubation]

Abnormal : Tubal Block < U/L
B/L → No conception

Best prognosis ← ① B/L cornual block : proximal block
↓
Blog majority of time it is physiology Block

② B/L fimbrial block (or) B/L distal block
B/L hydrosalphinx (distended tubes)

③ B/L midsegmental Block (tubal Ligation block)

physiology block : ① while screening & cannula the pain generated cause spasm of the circular muscle at ostia

② while done in DS the mucus is ~~thick~~ more and block ostia

Best procedure Laproscopy + Hysteroscopic cannulation + chromopertubation
↓
quite like HSG but no xrays

↓
therapeutic
↓
put a guide wide and push it through the cannula which is visualised & Hysteroscope

⇒ If cannulation not effective than we can DO

→ Cornual micro Sx < IVF

→ they heal by fibrosis — only when cornual micro sx does not respond

→ ectopic can be formed

↳ dangerous ectopic : if they Rupture they rupture both tube & ut

→ Δstic Lap

— Mild disease — Severe distortion of pelvic anatomy

frozen pelvis seen in:
Genital TB > Severe endometriosis > Severe PID

↓ Tubal corrective Sx

① Adheriolysis

② fimbryoplasty

↓ IVF bcos of land in ectopics

3) Mid segment Block (tubal Ligation)

↓

Recannulisation : only when there is good prognosis for preg

a) Types of Ligation :

→ clips > falope rings

→ (poor) cautiry > modified pomeroy

b) type of anatomosis

→ Isthmo isthmic

→ Isthmo ampullary

c) tubal length : >4cm

d) only reason for infertility is Ligation

Husband Semen Analysis: It should be good
↓
before we do recannalization

→ GENITAL TUBERCULOSIS ←

* Not a primary infection
* 2° infection from m/c 1° in Lung
* m/c route Haematogenous spread
  direct / Lymphatic / ascending infection
* m/c site : Fallopian tube [>90% cases]
  ↓
  bcoz Haematogenous spread

  Endometrium [50-60% cases]

  Least / common site Vagina / Vulva [<2%]

B/L tubes

m/c site in tube is 1st Ampulla → (distal part)

L/c Interstitial

⇒ Salpingitis Isthmica Nodosa (same as genital T.B)
↓
proximal part of tube

Acute stage : edematous / inflammed

Chronic stage : thick walled / adhesions

① cobble stone appearence of tubes

② tobacco pouch " " "

③ peritubal Halloo

④ Lead pipe apperance (or) pipe stem apperance
↓
Straight tube

⑤ Beads on string apperance

Endometrium TB: only superficial endo
but not myometrium

Acute: Red endo [we may think (N) endo]
(Grossly)

Chronic: ulcers / granulomas / Adhesions

★ Route of spread: Direct spread

Cx TB: apperance as exophytic growth [TB] ↓
↓
send D/D for Cx Ca → send for bacilli

★ m/c presentation:

Infertility > Pain > Menstrual Irregularity

→ Menstrual Irregularity
- 1st – menorrhagia ↓ endometritis
- 2nd → amenorrhea > oligomenorrhea

On examination of Genital TB :

m/c normal pelvic exam : pt feels tender.

4/c (Reproductive age)

B/L adrenal mass

↓

In Adolscent girl m/c ~~gist~~ B/L adrenal masses

Δsu: ① Menstrual Blood PCR

↓

1st Day of Menstrual Blood sample becoz bacteria are superficial layer of endo

Endo Biposy

HPE — AFB (cultures)

Treatment: ATT x 6 months

↓

It can be given empirically

If the tubal damage is severe, ATT is not effective but ATT should be given and also other methods for Infertility, If, dt all not responding do IVF

IVF ⇒ tubal factor disease

↳ Test tube baby (earlier, called like that)

① Severe tubal disease

② B/L Hydrosalphinx

↓

Before IVF { poor outcome in IVF
fluid is toxic to embryo

↓

do

salpingectomy (or)
tubal clipping

③ Conual + Distal block = combination

Steps of IVF

1) stimulate the ovary → m/c drug to stimulate: Inj gonadotrophine

2) Egg retrieval → Ovulation trigger: Inj. HCG 5000-10000 IU
→ Ovum pickup: after 36 hrs

3) fertilize c̄ sperm → test tube; special: ICSI
Intracytoplasmic sperm Inj
↓
m/c done in male factor infertility

4) Embyonic transfer
↓
M/c Day 3
8 cell stage
2cm below the fundus (USG guidance)

Day 5: transfer: PIGD → pre implantation genetic Δsis
↓
any genetic disorders

Genetic material → Blastomeres
→ Polar Bodies
→ Blastocyst trophoectoderm (Best)

→ usually 1 cell is taken out for genetic Δsis
→ to take pierce the zona pellucida
→ in Ca/Mg free media

→ UT causes of Infertility ←

① fibroid = submucosal

② Endometrial polyp

③ Endometritis

④ acute retroversion

(It cause malalignment of ut & Cx)

⑤ Diethyl silbestrol → in ut
(DES)

↓

m/c Hypoplastic ut

m/c characteristic : T shaped ut cavity

Cx → Anatomical = colon (cs) bowel

CIN (cs) adenoCa

Vagina : Clear cell Ca

Vaginal adenosis

Tubes : paratubal cysts

⇒ In typical of fetus : DES cause renal anomalies in male fetus

" do not cause " " " female fetus

Male fetus : Hypospadias

cryptorchidism

testicular Hypoplasia

→ MULLERIAN ANAMOLY ←

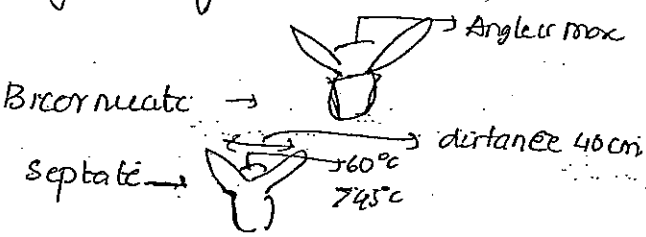
m/c MA : Septate

2nd Bicornuate unicollis

abortions - septate

Infertility - Septate

Screening test for MA is : HSG



IOC : MRI

[3D/4D USG → peak of type of anomaly
Saline infusion sonography]

Gold standard : Laproscopy (+ Hysteroscopy)
↓
In/imp.

Good Reproductive outcome : Arcuate uterus

Arcuate ut > didelphius > Bicornuate
↓ Sx correction is not required

cause : Preterm Labour / abortion (unlikely)
↓
operate : Unification surgery
↓
Strassman metroplasty
↓
Join two cavities into one

Worst : Septate →
TOC : Hysteroscopic Resection of septum
(Tompkins / Jones)

⇒ All MA assc urinary tract Anomalies
↓
must undergo IVP / Renal USG → Highest c unicornuate ut

Unicornuate ut → well developed
→ Rudimentary
→ c̄ Ectopic ovary

non communicating c̄ main Horn
↓
Pain
↓
cyclical dysmennorrhea

→ Cx FACTOR INFERTILITY ←

* problem c̄ Cx Mucus

Mucus — Impermeable
— Anti sperm Antibodies (Immunological Infertility)

Mucus and its characteristics Insler's Score

Score 10-12 (♀ is about to ovulate)

→ Anti sperm Ab : sperm show forward mobility (or) progressive motility

* PCT (Post coital test) (SIM's Huhner) test
(No longer done)
↓
♀ come after immediately coitus
↓
→ If ASA present — Circulatory motility
— Shaky motility

→ Done at Day 10-12th menstrual cycle

→ m/c/c of abn. PCT is wrong timing

Other tests: ① Immunobead Assay
② sperm immobilization test
③ sperm Agglutination test

These are not done usually becoz there is No treatment for ASAb

⇒ Treatment for ASA / immunological infertility

Intrauterine inseminization [IUI]

↓

Semin in ut directly (bypassing cx mucus)

simplest form of ART (Assisted reproductive tech)

We taken the semen sample

↓

process the semen

↓

insemination

⇒ ASA also present in males

→ semen sample (max 0.5 ml)

semen + liquid media → (very good quality of motility)

on the day of ovulation

to reach the site of fertilization i.e. in 30 min

(n) time is 30 min

Indications of IUI: 1) Immunological

2) Mild male factor infertility

3) unexplained infertility

4) use it i.e. CC

for 1st 3 months only CC is given
↓
Anti estrogenic effect.
↓
If she only ovulating and the
↓
CC + IUI

5) Sexual dysfunction
- → vaginismus
- → failure of erection

→ MALE FACTOR INFERTILITY ←

→ Husband's semen analysis ① Abstinence for 2-7 days
② Best by mastrubation
③ after liquifaction & mixing
④ avg time liquefaction = 30 min (20-80 min)
⑤ sample Reach the lab τ in 60 min

→ ⑥ WHO semen analysis

the vol > 1.5 ml
the pH > 7.2
Sperm count > 39 million/ejaculate
Sperm conc > 15 million/mL
avg sperm count = 100 million/ejaculate
T. motility > 40%
Progressive forward ~~[illegible]~~ > 32%

mortality > 4%

Vitality > 58%

WBC < 1 million/mL

Aspermia → absence of ejaculate

Azoospermia → absence of sperm in ejaculate

Oligospermia → < 15 million/mL

Asthenospermia → Reduced motility

Necrospermia → dead sperms

Teratospermia → abn morphology

globospermia → Sperm c̄ round heads they lack acrosomial cap

Mx :

abn in the semen sample
↓
Repeat 4 weeks apart

→ Azoospermia
↓
LH/FSH and testosteron

Normal

Cause: obstruction
↓
Obstructed Azoospermia
Sites: Vasectomy
EDO (Ejaculatory duct obst.)

Abn → LH/FSH ↑ & Testosteron ↓
↓
testis problem
↓
known obstructive Azoo
↓
pathology at testis
↓
abn spermatogenesis

Obstructed Azoo

- Vol and fructose (N) → Scrotal USG
- Vol and fructose is reduced → Vol ↓, fructose ↓ → TRUS (Tran rectal USG)
  - dilated seminal vessicles → EDD
  - Absent seminal vesicle → ⇒ Cystic fibrosis → CFGR gene analysis
    - → [Viscosity is ↑ed, Vas impalpable]

→ treatment of Azoospermia → Abn spermatogenic → microsx TESE (tuticular sperm Extraction) → But it has no motility → bcoz it is not from epidydimis → so TESE + ICSI

Testicular biopsy is done for obstructive Azoospermia (has more role)

· b/c we need to see spermetogenesis is (N)/not

Mild male factor infertility: (15 million/mL) IUI

5-15 million/mL – IVF (Imp parameter is motility)

<5 million/mL – ICSI (Imp parameter is morphology of sperm)

→ ENDOMETRIASIS ←

Presence of ectopic endometrial tissue (glands + stroma)

Theories ← FG1

Most accepted theories: ① Sampson's theory

Theory of retrograde menstruation

② Coelomic metaplasia

Bone marrow stem cells, mullerian rests

③ Immuno mediated

cell mediated } deficiency
Humoral Immunity

④ Genetic Kras

one 1st degree realative → Risk in the pt 7 times higher

⑤ Lymphatic / Haematogenous theory

Umbilical endometriosis

⑥ Hormone theory

⇒ dependent on ovarian Hormone, E/P → ovarian steroids

Reproductive age group = 25-35 yrs

postmenopausal / Adolescent girls = rare

↓

if seen do endometrosis = (MRI)

↓

to see mullerian anomaly

* In preg stage the endometrial tissue Regress

i.e Improve

↓

bcoz: Progesterone (decidualisation)

↓

continous exposure → ↑ ↓

atrophy

* Diagnosis of Endometriosis

① Atic laproscopy = ③ Stage the disease

↓

② IOC

Red flame lesion: New implants (or) New lesion

Powder burnt area: Chronic implants

↓

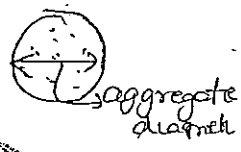
white and black

through laproscopy take biopsy to Histopath

American society of Reproductive Medicine.

Minimal: Isolated peritoneal and superficial implants

Mild: multiple superficial in aggregate <5cm diameter



Moderate: Superficial + Deep >5mm

Severe: Superficial + Deep + Adhesions (pelvic distortion of anatomy)

* Chocolate cyst of the ovary = endometrioma
  It is severe endometriosis

* USG is not useful in endometriosis
  only chocolate cyst can be picked up which has a characteristic appearence: ground glass appearence
  ⇓
  endometrioma

* MRI is also only in endometrioma

* 9 % endometriosis have raised CA 125
  ↓ (n) value of CA125 is < 35
  (non specific test)
  In endometriosis > 150

* Clinical features:
  a) Pain
  b) Infertility / subfertility
  c) Have adnexal mass
  d) Menstrual irregularities in form of menorrhagia
  e) Bowel & Bladder complaints
  f) Catamenial Pneumothorax (Thoracic endometriosis)

* On Examination

  on P/V : tenderness in POD (pouch of douglas)
  tenderness and Nodularity on uterosacral ligaments.
  tenderness adnexal, adnexal mass
  fixed retroverted uterus

Pain

Dysmenorrhea > Chronic pelvic pain > dyspareunia > low backache

causes: ① chronic inflamation processes

② they have sensory (or) sympathetic nerve ending in the implants

③ Severity of pain: Depends on the depth of implants.

Depth more pain red

dyspareunia: Deep retrovaginal implants

* 2° dysmenorrhea ① to a disease process

→ ② Endometriosis > PID ⑧ Less responsive to NSAIDS

③ congestive ④ Premestrual (7 days)

⑤ No change in menstrual blood flow

1° dysmenorrhea ① progesterone withdrawal

② spasmodic

③ 1st day of menses

④ is not seen beyond 72 hrs

⑤ Very responsive to NSAIDS

treament:

Minimal / Mild / Moderate

→ OCP + NSAIDS

↓

cause inhibition of ovulation

→ if ♀ want to conceive don't give OCP give only NSAIDS

→ if poor response then NSAIDS + change combination of OCP (or) give only progestiron

Injectable DMPA or PDD

[Norethindrone, MPA, dienogest] m/c decidualization → Atrophy

Severe

Non responsive to prev txt

→ start GnRH agonist

↓

continuosly

↓

suppress the ovary

→ last resort is Aromatase inhibitors

↓

till not responding

↓

taken up for Sx Management

Surgical Managment:

1) Destroy the Implants → by ablation
2) Adhesiolysis
3) LUNA laproscopic ~~uterosacral~~ uterosacral Nerve ablation
4) Presacral neurectomy
5) Hysterectomy [If the family is complete]
   ↓
   we should put them on GnRH suppression therapy

→ ADNEXAL MASS ←

"Chocolate cyst": Does not Respond to Medical Mx

Managment:

cyst ≤5cm: wait & watch

>5cm and complications: TOC = cystectomy

No I/D (Highest recurrance = 80%)

→ Managment of Infertility ←

↓

Sub-fertile

↓

cause: ovarian dysfunction (single best Ans)

↓

Destruction of follicle

poor quality of ovum

ovum pickup defective

ovarian + tubal

Minimal / Mild:

① Superovulation c̄ CC + IUI × 3 months

[Never mark GnRH has tx of Infertility]

CC X IUI = fail
⇓
then IVF is done

Moderate - Severe and infertility: IVF ± Sx

During IVF ⟶ she has chocholate cyst
↓
Never touch this
↓
if removed : it reduces the developing follicles
↓
which help in IVF to be sucess
↓
If Does not removed Improves the IVF sucess

Long term: Ass ⊼ certain ovarian Ca
(epithelial) clear cell cancer > endometriod cancer

⟶ FIBROID ⟵

* Smooth muscle tumour
* m/c benign pelvic tumour in female (Reproductive age group)
* It is estrogen dependent mainly = proliferation
  progesteron slightly - inhibits apoptosis
* Types of Fibroids: Submucosal = L/c
  Intramural = m/c
  Subserosal

All Fibroids are begin as Intramural

Submucosal Fibroid:

Further divided by Hysteroscopic appearance

type 0 : completely intracavitary

type 1 : >50% Intracavitary

type 2 : <50% Intracavitary

↳ cannot be Hysteroscopically removed

M/c presentation:

① Asymptomatic

(>35 yrs >50% fibroid)

② Symptoms

a) Bleeding: Submucosal

↳ Menorrhagia

↓

Blood is more but Menses is Regular

* Metrorrhagia: Pedunculated Submucosal fibroid

↓ fibroid polyp

can cause inverted Uterus

* Inversion: Fundal Fibroid polyp

↳ m/c presentation: Menorrhagia

b) Pain:

Dysmenorrhoea ⊕ → not a 1° complaint

Dyspareunia → Not a finding of fibroid

- Torsion : m/c → Pendunculated subserosal Fibroid.
- Degeneration : m/c degeneration is HYALINE

  L/c " is SARCOMATOUS CHANGE
  ↓
  Rare malignant change = <0.1%

  Calcareous degeneration : Calcific degeneration
  ↓ → subserosal fibroids undergo
  popcorn kernel appearance

  Red degenaration : Specific to pregn
  2nd trimester

  aseptic condition ← present c̄ acute abd < Vomiting, nausea / Abd pain / Fever
  ↓
  conservative txt
  (Analgesic, IV fluids, + NPO)

  ↓
  Investigation : leucocytosis

c) Pressure → Bowel / Bladder

m/c subserosal

Ant wall → Ped urinary frequency

Pst wall → urinary Retention

Ligament fibroids < True : a raise denovo b/w layer of BL
False : ss uterine fibroid which are outgrows of BL

Ureter : lies medial to Fibroid : True

" Lat to " : False

D) Infertility
E) Recurrent Pregn Loss } Submucosal

F) Pregn Mostly no change – It may cause preg complications

Red degeneration
preterm Labour
PPH
malpresentation
IUGR
Postpartum H
abruption
dystosia of Labour

Fibroids on USG are Hypoechoic : have capsule

→ Same echostructure as myometrium

→ Fibroid : board basement attachment

poly : peduculated narrow attachment

But Investigth of small submucosa Fibroid :

Hysteroscopy > Saline Infusion sonography

Fibroid are well seen in USG

MRI is a preoperative investigation

Fibroid	Adenomyosis
Smooth muscle	* Endometrial glands & Stroma in myometrium
Menorrhagia	* menorrhagia + Dysmenorrhea
Reproductive age group	* 40-45 yrs
Irregular growth : lumpy pumpy	* Symetric growth of Globular Ut

I Love You
Love you so ..... much

* 18-20 weeks pregn size
* Non tender
* IOC = USG
* TOC:

Medical Managment

2 types of Drugs

- 1 reduce the blood loss but not size
- 2). reduce both < blood loss / size

DOC: ↓ blood loss
- OCP's [only Low dose pills]
- Merena [LNg containing IUD]
- Tranexamic acid

↓ size → ① GnRH agonist
↓
continuos manner
↓
give it preoperatively 6 months before operative
↓
reduce myometrium and reduce blood loss
↓
Majority of times it is not used beoz plane of dissection are lost

② GnRH Antagonist

③ mifepreistone, ulipristal } SPR Modulators

④ Aromatase inhibitors

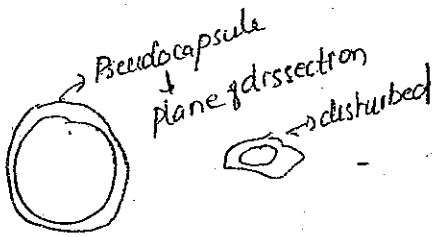
* 10-12 weeks
* Tender
* IOC = MRI
↓
Junctional Zone thickness

(Endo-myo) ≥12 mm likely ΔSU
Nonconformatory
< 8 mm unlikely ΔSU

on USG:
- Venetian blind apperance
- poorly defined Junctional zone
- salt pepper appearance
- Myometrial cyst
- subendometrial cyst

conformatory ΔSU : HPE
↓
Post Hysterectomy confirmation
↓
Depth of glands in myometrium
↓
≥5 mm Deep (or) one high power field deep to the Junctional Zone

* TOC = Hysterectomy
↓
(beoz of age)



↓

Not responding to Medical Mx

but refusing to do Sx

↓

Then do minimally invasive

UAE : Uterine artery Embolisation : ↓ blood flow to fibroid

MRg HIFU : Magnetic resonance guided High intensity USG

↳ Done if only family is complete

↓

Surgical Txt of Fibroids

- Conservative Sx
  - ↓
  - Only fibroid tissue is destroyed
    - a) Laser probes } Mydrysis
    - b) Cryo
    - c) Myomectomy
      - → Abd myomectomy
      - → Laproscopy myomectomy
      - → Hysteroscopic myomectomy
        - ↓
        - Submucosal type 0 & 1
- Radical
  - ↓
  - Hysterectomy
    - ↓
    - ① M/c : Total Abd Hysterectomy of Fibroid
      - ↓
      - removing ut & Cx
    - ② Subtotal Abd Hysterectomy
      - ↓
      - removing only ut
      - ↳ Supracervical Hysterectomy
    - ③ TAH + B/L Salphingo oophorectomy
      - ↓
      - Panhysterectomy

⇒ Laproscopic has slightly higher recurrence
↓
Not significant

Seedling myoma : small myoma
↓
they grow after

⇒ Hysteroscopic myomectomy

complication: m/c 1) perforation

2) fluid absorption electrolyte Imbalance

3) Bleeding

→ MALIGNANCIES ←

m/c cancer Amoung Indian women = Ca breast

2nd m/c = Ca Cx

Ca Cervix

HPV Infection: → if persist →

mostly encountered

Pre invasive lesion

Long History

Avg time from inf to invasion 10 yrs may take upto 30 yrs

→ regress

→ Invasive ← Progress

Very common cancer

universal screening: ACS / Acog

→ Start screening = 21 yrs

→ By PAPS (a) → every 3 years

PAPS + HPV → every 5 years

→ stop screening → 65 yrs

provided the PAP smear in last decade are not suspicious

High sentivity: HPV test → so done on ≥ 30 years

High specificity: PAP Smear

persistant HPV infection

WHO:

* Start ≥ 30 yrs

Importance to 30-49 years

(most/c prone for Ca)

& Screen and treat

↓

For screening : ① HPV - Screen (Highly Sn)

② VIA Visual inspection c̄ acetic acid

V > VIA > PAPS

③ PAPS

PAPS → abn → confirm → treat

HPV test : every 5 yearly

VIA test : } 3-5 years
PAPS test : }

After screening and then treat

Treatment

Cryotherapy > LEEP > conization
(not eligible) (not eligible)

Screening :

1) HPV DNA testing → 16, 18 types
High risk viruses

2) VIA → 5% acetic acid

abn area : Stained areas are abn } Dysplasia areas
acetowhite areas are abn }

↓

More Nuclear material

↓

made up of chromatin

↓

made of protein

↓

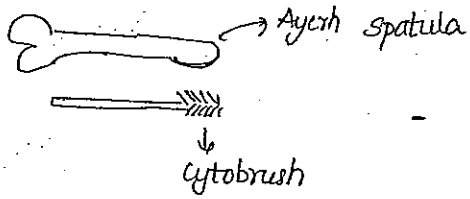
form coagulum

3) VILI Visual inspection of Lugol's Iodine
3-5%
Schiller's Iodine
ab

abn - unstained cells → no glycogen

(N) Stained cells → normal cells have glycogen → stained

4) PAP smear



Transformation zone > SCJ > endocx

* coe collect the secretions and make slide

No air drying

↓

keep slide in fixative → 95% ethyl alcohol, 5% ether

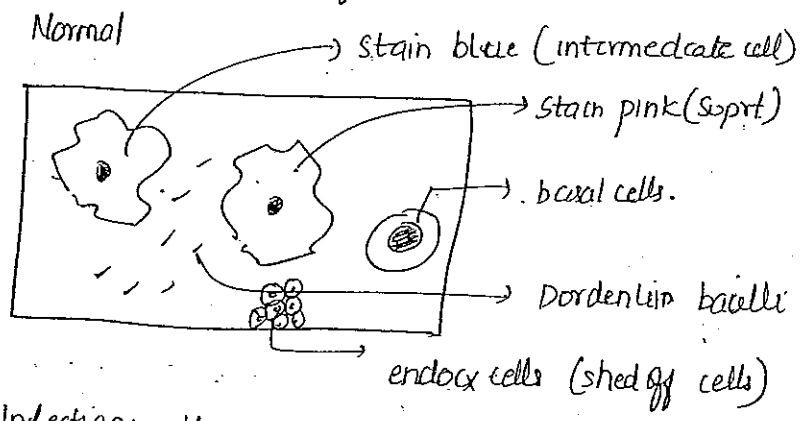
↓

Report = cytology

Hx = activity Bleeding pattern

Betherda classification of Reports

1st Normal



2nd Infection: the infections on the endocx cells cannot be picked up like chymidial, gonorrhea

3) Reactive and reperative change

Inflammatory change → Healing

4) ASCUS

Atypial sq cells of undetermined significance

5) LSIL:

Low grade sq intraepithelial lesion

6) HSIL

High " " " "

7) Frank cancer

Abn PAPS

↓

conformation by biopsy

↓

taken up by the colposcopy directed biopsy

↓

colposcopy [c̄ inbuilt microscope which show only ectoderm]

↓

max magnification 30 x

and focal length 30 cm

Abn areas on colposcopy

1) Irregular mucosa
2) Mucosa which is pale
3) Acetic white area (by acetic acid)
4) Abn vascular pattern
5) ↳ Green filter ⇒ Green light on blood vessels

Reticular pattern

mosaic pattern

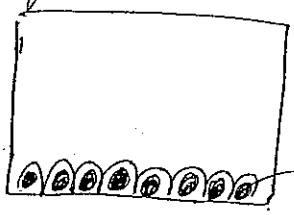
punctate

Colpo ⇒ Histopath Report

In HSIL → colpo Biopsy + ECC (endocx curettage)

Reports of Colposcopy

CIN 1	CIN 2	CIN 3
Mild dysplasia	Moderate dysplasia	Severe dysplasia
↓	↓	↓
Lower 1/3	Lower 2/3	> Lower 2/3
↓		
HPV infection		
infects basal cells first		



→ Large nuclei less cytoplasm

LSIL → CIN I —1%→ Ca
(80% → spontaneous Regress = 2 yrs)
10% ↓
HSIL → CIN 2 —5%→ Ca
10% ↓ CIN 3 (60% Regress)
Ca 12% ↓ 40%
Ca (40% Regress)

treatment:

1) CRYOABLATION
↓
Eligibility criteria
① SCJ should be visible
② if there is lesion ~~on the lesion ca~~ → completely visible
③ lesion on Cx → Lesion should occupy <75% of ectoCx

* Done in OPD < No anesthisia
ablation
↳ destroy only abn cells

Cryoprobes : These touch the Cx at abn areas
↳ uses cold gases < CO_2
Nitrous oxide
↓
they cause crystalization of intra cellular water
↓
desiccation of cell (dehydration)
↓
cell die

⇒ It cause mild pain so we give pre analgisia

Longterm side effect:

Persistant watery Discharge
Cx stenosis
" Incompetence } Rare

In CIN I → 1% chance of cancer
↓
Initial txt
↓
Observation x 2 years
Repeat → (PAP/ HPV)

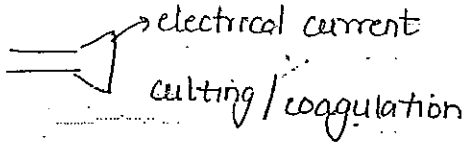
If Not eligible then do LEEP

⇒ LEEP → loop electrosurgical excisional procedure
LLETZ → large loop excision of ~~excision zone~~ transformation zone

LEEP / LLETZ

Loop = large loop

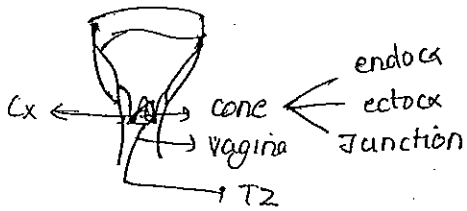
Wire loop



* OPD procedure
- quick
- < 2 min
- No training

CONE (Astic & therapeutic)

Indication of cone / C/I of Leep



1) Colposcopy is unsatisfactory
↓
entire T2 is not visible

2) HSIL and has +VE ECC (endocx curatage)

3) when cytology and HPE discrepency

4) Suspicion of micro invasion

5) when Histology is like adenocells
↓
columnar glandular cells
↓
endocx

Hysterectomy for preinvasive lesion

1) recurrent CIN

2) adeno family is complete

3) If the pt II could not follow up

CANCER CERVIX

Risk factors:

1) Multiple sexual partner
2) Early age at 1st intercourse
3) Early age at 1st childbirth
4) Multiparity
5) STD's
6) presence of preinvasive ca
7) Dis of Low socioeconomic status
8) smoking → Squamous cell cancer
9) OCPs ↑risk (≥5yr)
   reversible risk factor

⇒ early menarche (or) late menopause (or) family H/o are not risk factors

Etiological factor:

HPV: It can cause Cx, vaginal, vulval
Penile, Anal & oral Ca

Cx: parabasal & basal koilocytosis

High risk: types 16, 18 (70%)
31, 33, 35, 45, 52, 56, 58 (30%)

Low risk: 6, 11, 42, 44 → cause genital warts
↓
Condylomata Acuminata

Laryngeal papillomatosis

$\underbrace{E_1 \quad E_2}$ E_6 E_7

↓

viral protein

↓

to replicate the virus

↓

It has to integrate c̄ host genome

↓

once integrates the E_2 is called (Altered E_2)

↓

causes upregulation of E_6 & E_7

↓

It binds P53 ← E_6

↓

It mutates P53

↓

cell become immortal

↓

Now there will be second hit

↓

E_7 binding to Rb gene

↓ abn cell

complete cell cycle

↓

[E_7 is the single cell for malignancy]

m/c type-16 → Sq cell cancer

m/ specific type 18 → adeno cancer

HPV vaccines:

Gardasil	Cervarix
Quadrivalent	Bivalent

use inactivated capsid protein of virus $\underbrace{6, 11, 16, 18}_{\text{Quadri}}$ & $\underbrace{16, 18}_{\text{Bivalent}}$

9 valent Gradasil

6, 11, 16, 18, 31, 33, 45, 52, 58

Dose of Vaccine → 0.5 ml (given as IM inj)

Schdule 0, 2, 6 month (No booster dose after this)

Ideal age of vaccine → 11-12 yrs

can be given as early as 9 yrs

as late as 26 yrs

Gardasil for ♀ & ♂

Cervarix for ♀ only

<u>Side effects</u> :

m/imp S/E → syncopal attack

① given only when ♀ accompain by someone

② observation after nxt 15 min

Vaccine → not used in ↑xt HIV +ve

C/I in preg

→ Cx cancer ←

m/c primodal Peak

m/c 3-4 th decade

2nd peak 5-6 th decade of life (postmenopausal ♀)

sq cell Ca : account 70% Ca

adeno cell Ca : 25%

rare Histology : 5%

Sq cell Ca : Large cell non keratanizing

m/c/route of spread : Lymphatic
direct spread

m/c/presentation : Irregular vaginal bleeding

m/specific complaint : PCB (post coital Bleeding)
↓
clinical examination
↓
PAP smear

Q. 35 yr ♀ P_2L_2 PCB 3x4cm growth on Ant. lip of cx
↓
Do a punch biopsy

m/c/cause of death : Renal failure (obst of ureter)

m/c/metastasis : Lungs

⇒ the risk of ovarian involvement in Ca cx is <0.5%

Most Imp prognostic factor is stage and lymphnode

Stage I 90%

Stage > Lymph node status

STAGING OF CX CANCER (FIGO)
↓
clinical staging

↓
certain Ix should not be used to changed the stage of cancer

a) USG
b) CT → Hydroureter : change the stage
c) MRI
d) PET

STAGING

1A [Microscopic]

1A1 1A2 → [Depth of spread <3mm
Horizontal of spread <7mm

1A2 [Depth 3-5 mm
Horizontal <7mm

Stage IB : [Macroscopic spread]

B1 : ≤4cm

B2 : Gross Ca >4cm

Stage II < IIA
IIB

IIA : Ca invaded upper 2/3 of Vagina

* ⊼ not involvement of parametrium

IIB : Ca upper 2/3 of vagina

* ⊼ parametrium

Stage III < IIIA
IIIB

IIIA : Ca invaded lower 1/3 of vagina

IIIB : Lat pelvic wall involvement

Stage IV < IVA
IVB

IVA : Ca spread to Bladder/ rectum

IVB : distant spread

⇒ Inguinal Lymph nodes involvment in FIGO is Stage IV B

Que) k/c/o CaCx 4cm growth invading upper 2/3 vagina & parametrium, Cystoscopy shows Bullous edema of Bladder mucosa
↓
Lymphoid are obstruction but not invasion into Bladder

treatment

upto stage IB_1 : 1° txt is surgery

stage ≥ IB_2 → 1° Chemoradiation

Stage IA_1
- → pt family complete → radicle : simple extrafascial Hysterectomy
- → pt desire future fertility
  ↓
  fertility preserving Sx
  ↓
  Conization

Stage IA_2
- → family complete → type 2 Hysterectomy
  Werthine's "
  Modified radicle "
  * m/c Sx for Ca Cx is
- → future fertility
  ↓
  Radicle Trachelectomy
  ↓
  Remove Cx and spare ut
  ↓
  * parametria
  pelvic lymph nodes
  small part of Vagina

Stage IB_1
- → family complete → type 3 Hysterectomy
  = Meigs "
  = Radicle "
- → Not complete
  (≤ 2cm) → radicle Trachelectomy
  (> 2cm) Hysterectomy

k/c/o Ca Cx 2cm 38 P2L2

↳ a) conization

b) type 1

c) type 2

d) chemoradiation

⇒ HPE finding on follow up Require post op Adjuant chemoradiation

↓

1) +ve LN (pelvic para aortic)

2) parametrial Invasion

3) +ve margin

Chemoradiation

chemotherapy + Radiotherapy (concurrent therapy)

↓ giving simultaneous

Radiation sensitizer

↓

Cisplatin > 5 FU

Radiotherapy — Brachytherapy (Intracavitary)

EBRT

Ext Beam RadioTherapy

Tele therapy

(EBRT & Brachytherapy)

→ Cesium

↓

to pelvis ≅ 50 Gy 5d/week for 5 weeks, 25#

also give it to abd (extended field radiotherapy)
≅ 30 Gy

→ Brachytherapy
↓
⇒ Ir 192
can also use cerium

Point A

① 2cm lat to cx
② 2cm above the ext os
③ this pnt corresponds to ureter and Paracervical(or) Parametrial node
↓
* sentinel LN for CaCx
[only gynaec Ca for sentinel LN is vulval ca]
④ Radiation dose 8000 centiGy
80 Gy

Point B

① 8cm lat to pnt A
② 2cm above ext os
* ③ the pnt corresponds to obturator L.N (m/c)
④ Radiation dose 6000 cGy
60Gy

Radiosensitive
Ovary > Rectum > Bladder > vagina

High Dose Radiotherapy
↓
the @ 12 Gy/hr
↓
only advantage: Course is finished little early

Recurrent Cancer:

Chemoradiation → (Recurrence)

- Operable
  Central recurrence in pelvis
  ↓
  Pelvic excentration
- Inoperable
  Reaching the lat pelvic wall
  ↓
  Palliation (Chemotherapy)
  ↓
  Cisplatin

Type 1, 2, 3 = Piver Rutledge Classification
Hysterectomy

Type 2	Type 3
Modified Radicle	Radicle
1) Uterosacral / cardinal ligament cut midway	1) close to pelvic wall
2) Ligate uterine artery after it gives the ureteric Branch	2) Ligate uterine Artery at its origin
3) remove 1/3 of vagina	3) remove 1/2 of vagina

→ OVARIAN CANCER ←

Not very common
non specific
No universal screening

Adnexal Mass

Benign	Malignant
* Reproductive age group	* Extremes of age < Postmenopasal, Adolescent
* Pain	* initial no pain
* O/E : Mass u U/L	* O/E : Mass u B/L
* Cystic consistency	* Variable in consistency (Cystic + solid)
* Ioc : TVS → completely Benign unilocular, anechoic [black]	* Malignant : Solid area + Cystic area and color doppler red vascularity

High risk for malignancy

① Solid masses

② Septae → thick septae
↓
2-3 mm thick

③ papillary excresences

④ Red vascularity on Doppler

⑤ Ascites

⑥ Ass c̄ presence of matted bowel loops & enlarged LN

SURGERY

1) USG → High risk factors
↳ laprotomy

2) ovarian mass > 7 cm

Adenexal mass > 10 cm

3) Raised CA 125 → criteria for Post menuposal women

⇒ In repro women many benign conditions causes ↑CA125
like menses
PID
fibroid
Endometriosis
genital TB
itis of all abd organs

4) Pt c̄ acute Abd

the mass torsion (or) Rupture
↓
m/c mass ovarian tumour is Dermoid
↓
mature cystic teratoma

Benign Masses in Reproductive Age

3-5 cm → wait & watch

5-7 cm → follow up c̄ serial USG

m/c cyst of the ovary = Follicular cyst

If the cyst does not resolve (or) causes other symptoms like pain and irregular menses use OCP's

Benign in Extremes of Age

Postmenopausal Women	Prepubertal Women
↓	
Tumour Markers	Tumour markers
① CA 125 (epithelial ov Ca)	① AFP & HCG (Germcell tumour)

Adnexal Mass in Pregnancy

Adnexal Mass in 1st Trimester: Wait and watch

↳ mostly corpus luteum

↓

Regresses at 12 weeks

2nd Trimester: a) High risk factors on USG
b) >10cm
c) Acute abd
} operate

FRANK OVARIAN CANCER 12 JUNE 2017

Risk factors:

Higher the E more is the risk (E = proliferation of epithelium)

" ↓ ovulatory cycles more the Risk

1) Early menarche
2) late menopause
3) Nulliparity
4) Infertility
5) PCOS
6) Obesity
7) Genes BRCA1, BRCA2, HNPCC (Lynch)
8) Smoking (mucinous)

Protective:

1) OCP = Reduce by 50%.
2) Breast feeding
3) Anovulation
4) Sx like sapingectomy, tubal ligation & Hysterectomy

prevent Ascending mitogens

ART is no longer risk factor for ovarian tumours

Ovarian tumours: 1) Epithelial → 90%
2) Germ cell →
3) Sex cord → } → 10%

Epithelial ovarian tumours: Serous → 75%
Mucinous 10%
endometroid 10%
Brenners } 5%
clear cell }

↓

General principles

m/c ovarian tumour: serous cystadenoma

m/c ovarian cancer: serous cyst adeno carcinoma

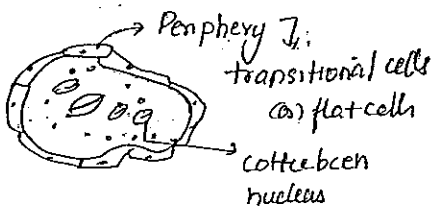
1) m/c age group: 6 & 7th decade [peak at 60 yr]
(decade of postmenopausal women)
2) m/c BL
3) non specific signs/symptoms (IBS)
4) Usually diagnosed in late stage
5) m/c route of spread: Tumour exfoliation
6) Sporadic 90%
familial 10%

Mucinous:

1) decade earlier
2) U/L tumour (only 10% B/L)
3) Grow to huge size [upto 20cm]
4) More symptomic and peaked up earlier
5) Better prognosis by early Asy
6) Serous: CA125
6) Mucinous CEA (carcino embryonic
7) Pseudomyxoma Peritonii
   * abd is filled with loculated mucinous collections
* Highest assoc appendicial tumour

Endometroid: ① 10%
② U/L
* ③ High ass c̄ endometrial ovarian cancer
2nd high ass c̄ Granulosacell tumour of ovary.

MCQs
Brenners:
① Benign
② Bladder like epithelium (Transitional epithelium)
③ Solid and U/L
④ HPE < Solid & U/L
Wall thorid cell Nest



Clear cell: ① Highest ass c̄ endotriosis
② 2nd high " " endometriod
③ familial 10%
④ BRCA1 (45%) HNPCC (15%) → life time risk for Ovarian Ca
BRCA2 (25%)

5) occur a decade earlier

6) Risk keep rising beyond 35 yrs of age

7) K/c/o genetic mutations

↓

a. ↑s the risk of ca by 3 times

b. absolute risk is 5%

c. 1st degrees relative affect the risk of ca by 3 times

d. Risk reducing Sx

Prophylactic BSO, B/L salpingo oophorectomy

↓

- ↓ OV ca by 90%
- breast ca is ↓ by 50%
- Ideal time to do BSO = 35 yrs

(or) As soon as family is complete

8) pt who have significant family H/o Breast and ovarian cancer + who refuse RRSx

① Screening should be done ↓ started at 35 yrs ↓ TVS and CA125 ↓ every 6 monthly / 12 monthly

② OCP

GERM CELL TUMOURS:

1) 5-8%

2) 10-30 yrs

3) Peak 2nd decade → what % are the germ cell (60-70%) germ cell

4) Teratoma
a) mature cystic teratoma (Dermoid)
Immature

d. Chorio ca

b. Dys germinoma

e. Mixed germ cell

c. Embryonal ~~cell~~ Sinus tumour (EST) YST

5) m/c germ cell tumor Dermoid

6) m/c germ cell cancer

Immature teratoma > Dysgerminoma

7) GCT has highest Risk Bilaterality

Dysgerminoma > Dermoid
b/l 15% BL in 10%

8) m/c specific s/s → Rapid growth
Acute abd → EST / Yolk sac Tumour
↓
precocious puberty (HCG) worst prognosis

→ GCT has best prognosis : Dysgermenoma

YST / EST → Schiller duval bodies
↓ (HPE finding)
Perivascular and look like glomerulus

9) peaked up in early stage

10) Better prognosis

11) Managment is usually c̄ conservative Surgery

Serum Marker for dysgerminoma → LDH
→ It also secretes (HCG & PLAP)
→ will not secreate AFP

EST / YST → AFP
→ small amount of LDH
→ will not secreate HCG

Embryonal → AFP & HCG
Choriocarcinoma → HCG

Dermoid does not produce any tumour marker (HCG rarely)

DERMOID (V. Imp for MCQ)

⇒ m/c ovarian tumour in Reproductive age women = Dermoid

⇒ m/c ovarian tumour in pregn women = Dermoid
↓
Reproductive age group

⇒ m/c ovarian cancer in preg = Dysgerminoma

⇒ m/c ovarian cyst = follicular cyst

Dermoid = mature cystic teratoma
↓
Imp

Rokitansky appearance : white in color
↓
presence of tooth & bone

Dermoid : Benign

Risk of malignancy (0.2-2%) <2%
↓
Sq cell ca

All the ovarian tumours are : ① Solid
② lobulated
③ Yellow (cut section)
④ Tane color → feature of dysgerminoma

Dysgerminoma : Radiosensitive (Moderately radiosensitive)

SEX CORD STROMAL TUMOURS OF OVARY [3%]

1) Granulosa cell
2) Thecoma
3) Sertoli-Leydig
4) Leydig cell ca
5) fibroma

1) These are seen in any age group

peak incidence : Peri menopausal women

2) Unilateral

3) Non specific S/ symptoms

+ AUB (Abn ut. Bleeding) estrogens

+ virilization (androgens)

4) Early stage

5) Do not show LN meta

6) Best prognosis

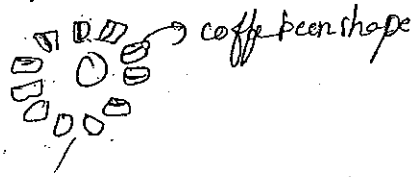
Granulosa cell tumour :

↓

① Res estrogen production ⇒ high risk ass c̄ endometrial Ca

coffee bean shaped nucleus = call Exner bodies

→ follicular arrangement



⇒ which ov tumour shows meta to Granulosa opposite ovary

② tumour marker is Inhibin

③ Rinky's crystal = in leydig cells tumour

KRUKENBRUG TUMOUR

m/c 1° gastric Ca of pylorus

m/c route of spread : Retrograde Lymphatics

- usually B/L = 80%

  may be U/L

- The capsule of the ovary intact

  ↓

  so the shape is maintained

  signet Ring cells

FIBROID (Meigs synd)

Triad: fibroma + Ascites + Pleural effusion

↓ Removed

↓ Spontaneously regressed

⇒ Pseudo meigs : Ascites + effusion

Granullosa, Thecoma, Mucinoma, Brenner } +

STAGING = FIGO

Stage 1
- A U/L ovary
- B B/L ovary
- C_1 Intraop Capsule Rupture
- C_2 Preop " "
- C_3 Malignant Ascities

Stage 2
- A : Ca spread to ut (or) (and) Falloplan tube
- B : other pelvic structures (pelvic LN)

Stage 3
- A_1 : +ve retropertoned LN (paraaortic)
- A_2 : microscopic extrapelvic pentoneal spread

B: Macroscopic [<2 cm]

extrapelvic peritoneal spread

C: Macroscopic [>2 cm]

Extrapelvic peritoneal spread or/And

Capule liver / spleen

Stage 4 A: Malignant PL effusion

B: → parenchyma of abd organs
→ Inguinal LN
→ distant metastasis

Procedure called as Staging Laprotomy
↓
Staging + treatment

1) Midline vertical incision

[Do not give pfannenstiel / Horizontal incision in Malignancy]

2) Ascitic fluid sample
(or)
saline lavage (100ml)

3) Inspection and palpation of all abd organs

4) Random peritonial biopsy
↓
a. paracolic gutters
b. POD
c. surface of diaphragm
↳ surface scarpings

5) Total Abd Hysterectomy c̄ BSO [Panhysterectomy]
→ type1 [simple extrafascial Hysterectomy]

6) Infracolic omentectomy

7) pelvic and paraaortic area LN sampling

8) Closure

Stage 1 & 2 → Early stage

Stage 3 & 4 → Advanced stage
↓
In this case do Sx → Debulking Sx
↓
↓ tumour size

Post operative:

Epithelial Ca : all stages except stage 1A & 1B Grade 1 need post op chemo

→ I/V (intravenous)

→ Carboplatin + Paclitaxel

→ Cisplatin + Paclitaxel

→ 6 cycles of chemotherapy

Germ cell

① all stages given postop chemo
→ Bleomycin, Etoposide / Cisplatin

Sex cord

① only advanced stage need chemo

Conservative Sx in Ovarian cancer

→ U/L Salpingo oophorectomy

Cystectomy / Aspirat → both are not Conservative Sx

1) Germ cell tumour

2) Stage IA_1 & family not completed

3) Borderline epithelial ovarian tumour

(Epithelial ov tumours of low malignant potential)

decade earlier

U/L usually

Good prognosis

lack stromal invasion

Which Ix is most Imp in ov ca is CT Scan

in follow up

there is CA 125

It indicates recurrance

if recurrance then do

PET scan

CT Scan cannot Do staging

ENDOMERIAL CANCER

m/c Histology : Endometroid Adeno carcinoma

M/Imp

type 1	type 2
① Endometroid (grade 1,2) → type 1	① Serous papillary clear cell (endo grad 3) → type 2
② 80% type	② 20%
Oestrogen Responsive	③ Oestrogen nonresponsive

(4) Preinvasive lesion	(4) No preinvasive lesion
(5) Good prognosis	(5) Poor prognosis Worst: Clear cell Ca Prgn Hobnail apperance → Nucleus is pushed out
(6) Obese women	(6) thin women
(7) Post menopausal Early menopausal	(7) increased age
(8) Pten → Imp MCG k Ras Microsatellite deletions Gatekeeper of Endometrial Cancer	(8) PS3

Risk factors :

1) ↑ed Estrogen (or) Unopposed estrogen
2) ↑ed mentrual cycles
1) Early manarche
2) Late menopause
3) nulliparity
4) Infertility
5) PCOS
6) BRCA1/2/HNPCC
7) tamoxifen
8) HRT (only E)

a) obesity
10) HTN
11) DM
→ causing endo ca is called as Corpus cancer syndrome

HRT is a Risk factor most significant = Breast Ca

Protective factors:

1) OCP ↓ by 60%

they cause endometrial atrophy (Long term use)

2) Smoking → ↑sed breakdown of Estrogen

3) physical activity → Green tea

Preinvasive Lesions:

1) Endometrial Hyperplasia

↑ ↓

Used by Histopathologist

Hyperplasia is divided into 4 — Risk

1) Simple Hyperplasia: c̄ out Atypia 1%

2) Complex Hyperplasia: c̄ out Atypia 3%

3) Simple c̄ atypia 8% → Neoplastic changes

4) Complex c̄ atypia 29%

→ proliferation of both glands & stroma

↓

cystic glandular hyperplasia

→ More glands (Back to back arrangment of glands) and less stroma

Hyperplasia treatment

Hyperplasia c̄ out Atypia — Simple / Complex

TOC: progestron

↓

decidualization

↓

Atrophy

m/c progesteron a: MPA

Medroxy Progesterone Acetate

(1M DMPA / Mirena)

MPA for 6 months (continuous)

[Intermittent dosage] ↓

PShow protective / 12-14 days

Hyperplasia c̄ Atypia

TOC: Hysterectomy

2nd line Txt: Progesteron

↓

Any formulation can given

↓

Megestrol Acetate (strongest)

⇒ m/c age group 5-7th decade

peak: 60 yrs

m/c 1st complaint: Irregular bleeding

Most specific: PMB (Post Menopausal bleeding)

m/c/c PMB is senile endometritis

(atrophic endometritis)

m/c/c PMB in India: Ca Cx

what % of PMB have endo ca is 10%

m/c/c of pyometra world over is Ca Endometrium

↓

In India: Ca Cx

⇒ Ca endomitrium is not usually screening except

the pt. c̄ BRCA1 A2 / HNPCC

↓

Risk of Ca = Ca of the colon

Breast ca does not comes under part of HNPCC
↓
colon
ovarian
endometrial

HNPCC Mutations
↓
prophylatic TAH $\bar{c}$ BSO
(family is complete)
↓
Screening at 35 years
↳ Fractional curretage
(Gold standard for endo ca is) -
done in OT under Anesthesia

Collect ECC (endo cx curettage)
↓
dilate Int os
↓
Endo curettings

PMB should be rule out the Endca

PMB (post menopausal bleeding)
↓
Next step
↓
TVS – look for endo thickness
↓
≤4 mm (No ↑ in risk of ca) → senile endoatrophy
≥5 mm (red Risk of ca)
↓
For all these Pt we do another screening
↓
IOC: endometrial biopsy

Endo Biopsy
↓
OPD (Pipalle / Vabra aspirator)

Karmane cannula
↓
they aspirate
Endometrial aspiration cytology
↓

Fractional curettage + Hysterectomy
- If the women has persistent symptoms
- Cx stenosis
- ino tissued u obtained

↓
Next step

PAPS + TVS + EB

If PCB (pericortubreedw

≥45 AUB ↓

1st

~~rule out~~ rule out ~~Sado Ca~~ endo

Staging

Stage 1A: only endometrium
(or)
<50% myometrium

1B: involve ≥50% of myometrium

Stage 2 : spread to ex ↓
(into the stroma)

Stage 3 A. : Spread to Adnexa & serous

3B :

3C : 1 If pelvic LN are involved

3C2 : Paraaortic LN

Stage 4 A : Spread to Bladder and Rectum

B : spread to distance metastasis

Inguinal LN → 4B

Staging → Surgical

m/c route → direct spread

Staging laprotomy : steps remain the same

Staging laprotomy

1) TAH & BSO (txt) type 1
↳ ex stage 2 —— type 3

2) In endo ca Routine omentectomy not done
↳ endo type 2 (Histology)

3) Pelvic / Paraaortic LN sample when the Histology type 1
type 2 (Histology) dissection

Stage 1/2 → early stage cancer

Stage 3 & 4 → Advanced stage
↓
txtis Sx
↓
Sx (Debulking Sx)

Post op treatment

Low Risk: 1) Endometroid ~~graded~~ Ca grade 1 tumor limited to endometrium } Need No further txt only follow up

High Risk: 1) type ~~II~~ (histopatology) 2) Stage 3 & 4 Ca } chemo + Radio
↓
Cisplatin + Paclitaxel

Intermittent Risk: pelvic - Radiotherapy (EBRT)

⇒ ERPR +ve status is a good prognosis

→ PROLAPSE ←

Disease of older age group and multiparous women
↓
prolonged labour
obstric labour
difficult delivery
Instrumental "

Multiparous

Congenital elongation of cx

Connective diseases

Prolonged raised intraabdomal pressure

Smoking

H/o of spinal cord injury

Delancey's level of supports

level 1 Utrosacral
mackenrudt
↓
weak = Apical problems
→ utex descent
→ enterocoele (bowel loops into POD)
→ vault prolapse (post hysterectomy pt)

level 2 supports: paravaginal tissue and their attachment to the Levator ani

weak = cystocoele (prolapse of the bladder through the upper 2/3 of vaginal wall)
↓
[Lower 1/3 wall urethrocoele]

level 3: Perineal body & Muscles attached

weak = Rectocoele (Lower 1/3 post vaginal wall)

1st degree prolapse: descent of the organs but they lie above Introitus

2nd degree: at the level of introitus

3rd degree: outside the Introitus

Procidentia:- entire ut has to lie outside (fundus - outside)

POPQ system: pelvic organ prolapse Quantification
↳ Referance point is HYMEN

Baden walker halfway system: Referance pnt HYMEN

Prevention: Kegel's exercises (pelvic floor muscle strengthening)

Que) 60 Y old lady P_3L_3 3rd uterocx descent + cystocoele + Rectocoele

3rd degree uterocervical:

Ans) Hysterectomy → Vaginal Hysterectomy
- If trained
- Lap
- not trained (n)

↓

Cant taken out by Vaginal Hysterectomy

↓

in
- > 12 weeks size
- Obese women
- Adhesions

size is imp

size upto 12 weeks of size

↓

can be removed

Uterosacral → mackenrodt

↓

UtA ligation

Utero ovarian pedunde ←

→ Abdominal Hysterectomy

pedicle

↓

UtA ligation

↓

mackenrodt

↓

Uterosacral

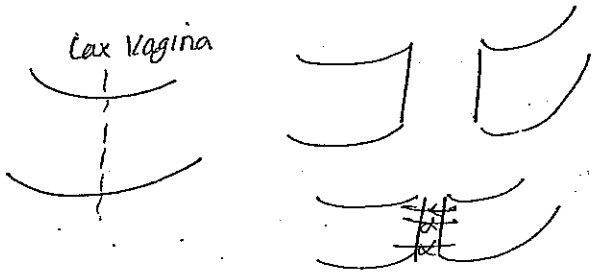
→

Urinary tract injuries:

Laproscopic > abd > Vaginal Hysterectomy > C.S

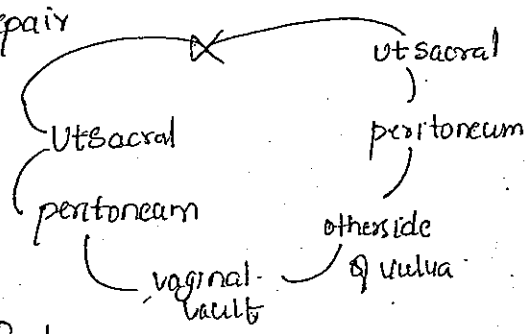
Cystocoele: treatment

Anterior Colporrhapy
↓
means vagina



Rectocoele: Posterior colpoperinorrhaphy

Ques) VH c̄ PFR (pelvic floor repair) + Mc.Calls culdoplasty
(Nowadays) ↓ POD
Prevention + ↓
treatment of ① prevents Enterocoele later on
Enterocoele ② prevents for vault prolapse
txt
Moschowitz
Halbain repair



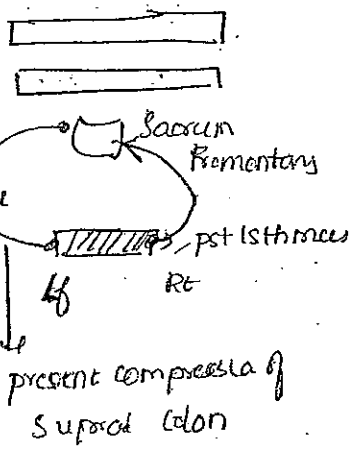
→ Nulliporous Prolapse ←
↓ desires Child bearing = Hysterectomy
m/c Sx ass Sling Surgeries: Ant sling, Pst sling

Ant Sling	Post sling
① Slins attached to Ant Isthmus ①	– Shirodkar's Abdominal Sling
② Purandre → one end of slins attached to isthmus and another to rectus muscle	– merselene tape

khannas

↓

In women after delivery the rectal muscle is weakened the slins fails

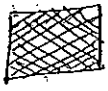


VI KRUD-

Best Surgery : 2) Sacrocervicopexy

↓

mesh



difficult surgery and higher complications

3) Shirodkar's Modification of Manchester Repair

Manchester repair : Fothergill's Repair

Cervical amputation

↓

healing c̄ fibrosis

↓ Cx stenosis ↓ Cx incompetence

⇒ cannot be done in nulliparous

can be done : family is complete (preserve the uterus)

2) Congenital cx elongation

↓

after completing child bearing

If we don't do Amputation : Shirodkar's Modification

of Manchester repair

↓

– Also done in Nulliparous women

Ques) 70 P_3L_3 + comorbidities

↓

PAC risk person for operation

↓

Should be done in short time

Lefortel's colpocleisis

↓

closure

↓

sexual inactive women

↓

but rule out Cx Ca

endo Ca

Denude the ant vaginal walls mucosa

and then pst vaginal wall mucosa

Ques) Old women + comorbidities

PAC → unfit for operation

↓

In this case we do or keep a Vaginal Pessary

space occupying device

Ischial spine (ext os)

① Early preg-i prolapse

Remove at 16 – 18 weeks

③ puerperium

④ diagnosis → SUI (stress urinary incontinence)

Decubitous ulcer: ulcer cannot give a clear anatomy of pelvic structure

~~AG pack: tampon Aortcline glycerol~~

→ Vault prolapse: ←

Prevention: By Mc call culdoplasty
└ Uterosacral suspension

treatment:

Abdominal > Vaginal
└ outcome difficult

1) Sacro colpopexy
↓
Best Sx for,

2) Uterosacral suspenchmission

1) sacrospinous fixation
(suture the vaginal wall to to sacrospinous ligament

2) Uterosacral suspenction

→ Colposuspension = Sx for SUI (stress urinary suspension)

- Abd sx for SUI
- proximal part urethra
  1. Burch (gold standard)
  2. MMK (not done)

- Burch: the urethra will be suspended to cooper's ligament

MMK: " " " " " periosteum of symphysis pubis

TOC of SUI: TOT — Transobturator tape

TVT — Transvaginal Tape
↓
Midurethral sling Sx

* Both are vaginal Sx
* Day Care Sx
* a effective a Bruch
* Mid urethra
* TOT > TVT
  ↓
  complication are less

Space of Retzius
↓
Retropubic space
↓
As to be entered blindly

we need to reach the Retzius space so complications are more.

→ SUI : Q tip test, Bonney's test → both are not done Nowadays → They check Urethral hypermotility

⇒ Urodynamic studies are not required

⇒ Urge incontinance → urodynamics are required

→ Duloxetine : Drug in Incontinance

→ Latzko ←
↓
m/c Sx for Vesicovaginal Fistula

* M/c urinary Fistula is : VVF
↓
Causes : in developing countries : Obs. labour
developing countries : Gynaecological Sx
↓
malignant
↓
Highest risk for VVF is Wertheim's Hysterectomy

Obs labour ⇒ 7-10 day after the C.S

Obstructed labour

→ Site: Bladder Base
Juxtacervical

Westheim's
Site
→ Vaginal vault

Ques) Continous dribbling of urine and no desire to Micturation

Ans) VVF

Ques) Uterovaginal Fistula + 0 acts of micturation (or) desire to pass urine

Urethro Vaginal Fistula = During micturation the urine comes out by 2 openings
↓ postural change
urethral, vagina

Ques) Cyclical Menuria
↓
Blood in urine only to a somedays in a month
↓
Uterovesical fistula (or)
it could be Bladder endometriosis

H/o operative intervention

3 swab test:

Bladder – dye

In ut
0 → upper two swabs are wet & stained (VVF)
0
0
only upper swab is wet but not stained (ureterovaginal Fistula)
only the lower swab wet & stained urethro vaginal Fistula

m/c Imp test of VVF = Cystoscopy
- site of Fistula
- No of "
- size " "

m/c test for VVF = Latsko Sx
↓
Vaginal Sx

Complicated Fistula:
- Ass c̄ malignancy
- combination of bladder and bowel fistulas
- Fistula which involve in ureteric opening

Abdominal

→ PELVIC INFLAMMATORY DISEASES ←

Acute/ subacute infection of upper reproductive tract
- Ut
- FT
- Ovaries

Risk factor:

1) Sexually active

Highest Risk: Multiple sexual partner

2) Age 15-25 (<25)

3) H/o STD

4) H/o intrumentation

5) Preg is a unlikely factor

Reduces Risk;

Barrier contraceptive

PID in IUD users:

1) Monofilament thread

2) 1st 21 days of IUD

m/c organism: Chlamydia > gonorrhoea
↳ subclinical

m/c organism to cause Acute PID: Gonorrhoea

m/c organism in IUD: Actinomyces

m/c organism in a virgin girl: Tuberculosis

Asy: Clinical Asy

1) lower abd pain
+
any one of the following
- ut tenderness
- Cx motion tenderness
- adnexal tenderness

2) AUB (in form of menorrhagia)

3) Vaginal D/s

4) Urinary frequency

5) Fever

⇒ VIOLINE STRING adhesion in pt of PID
↓
Fitz hugh curtis
→ PID
— ~~thy~~ Chlamydia > gonorrhoea
— Perihepatitis

D/D :- Ectopic pregn
↓
UPT +ve
ut size :- enlarge

Finding which support the PID

1) fever
2) ESR ↑
3) CRP ↑
4) purulent discharge
5) leukocytes in the microscopy of P/s
6) Lab test +ve chlamydial (or) gonorrhoeal test

Conformation:

① Gold stnd : Laproscopy
↓
① take biopsy
② prognostic scoring [Bocr mesel score]
↓
a) Prognosis for conception

② Endometrial Biopsy : chronic endometristis
↓ Plasma cells
(chlamydia)

③ Ultrasound : 3 Signs
① cogwheel sign
Coait sign
Beads on string sign

PID has longterms effects:

a) Recurrent PID
b) Infertility

3) Ectopic preg
4) Chronic pelvic pain

→ PID ←

Pts of mild to moderate cases

Inj ceftriaxone (250 mg IM)
+ doxycycline (100 mg BD X 14 days)
+ metronidazole (500 mg BD X 14)

Severe cases

Admit to Hspt

cefoxitin 2gm IV every 6 hrly

	m/c → ① Bacterial Vaginosis	② Candida	③ → Trichomonas
organism:	Gardenella vaginalis	Albicans ↓ glabrata	Trichomonas vaginalis
Ph:	>4.5	<4.5	>4.5
m/c presentation:	foul smelling D/S (off white (or) greyish)	Pruritis D/S is scanty ↓ it stick to vaginal wall curdy white color SPLASH DYSURIA	D/S → in green colour (or) it may be yellow ± pruritis ± dysuria dyspareunia

* IOC

Saline microscopy
↓
we see (clue cells)
↓
they are epithelial cells of bacteria in a piggy back

→ Pseudohyphae → Flagella organism

* EC : PMN's
= >1
↓
(No inflamation)

→ <1 (inflammation)

→ <1 (inflammation)

* Best

Gram stain
↓
look for Nugent score
↓
gram variable coccobacilli

Score : 7-10 Δtic

* Best

Culture
↓
Sabarauds culture media

* Best

Transports media
↓
Stuart's media

* AMSEL'S CRITERIA

3 out of 4 are met Δ
Δsu of Bacteria

① Off white foul smelling D/S
② Ph < 4.5
③ Clue cells (amin of 20% of Epithelial cells)
④ +ve whiff test
↓
Add 10% KOH to D/S
It will give a amine or ea

* Recurrent Vulvo vaginitis [VVC]

≥4 episodes / year

* Strawberry of cervix
↓
punctate haemorrhages

* Not a STD	* Majority: Not a STD commensal of Vagina and a over growth	* STD
* partner txt Not done	* Partner txt is Not done (partner will be txted only if he is symptomatic)	* Partner should be txted
* Metronidazole (or) clindamycin	* Oral fluconazole 150 mg OD	* Metronidazole
* In pregn Metronidazole	* In pregn topical azole like	* In pregn Metronidazole

→ ***** NBA Pal CONTRACEPTION ←

Oral combined Pills: → low dose → < 50 mcg
→ High dose → ≥ 50 mcg
} according to estrogen

low dose: avg 30-35 mcg

Very low dose: ≤ 20 mcg

lowest possible dose: 10 mcg (Lolocstrin)

Mala N/D →
✓ 0.03 mg EE + 0.15 mg LNg + 7 le tabs

No money ↓
free of cost

main OCP action: Inhibition of ovulation

m/c S/E = Break through bleeding

Missed 1 pill → take 2 pills

Missed ≥2 pills → Back up method of contraception

→ taking pills and then PID has set in. then it is by chlamydia (or) candida

→ Return of fertility: ≈ in 90 days after stopping

→ Ca Cx → ↑sec Ca Cx

Ca ovary → protective ↓sec

Ca Endo → " ↓sec

Ca colon → " ↓sec

Hepatic adenoma → ↑sec

HCC → (no action)

Ca breast → OCP are not Risk factor but HRT is a risk factor

Absolute contraindication

1) Stroke
2) VTE
3) Coronary artery disease
4) E/c/o breast ca
5) HTN ≥ 160/110
6) Migraine c̄ aura
   ↓ c̄ focal Neurological deficit
   complicated migraine
7) DM is not a absolute C/I but c̄ vasculopathy is a C/I

8) Age : Never a $c/_I$

but > 35 yrs + smoker

9) suspected pregn

10) Undiagnosed bleeding.

11) Acute liver disease

Vaginal Ring (or) Nuva Ring :

↓

Inhibition of ovulation

S/E remain same as OCP

absolute $c/_I$ are same as OCP

⇒ EE + Etonogestrel

Release rate = EE 15 mcg/day

Etonogestrel 120 mcg/dl

1 Ring for 3 weeks, 1 week is ring free

and then New ring should be used.

Patch → 1 patch - 1 week

↓

for 3 weeks

1 week, free patch

P = norelgestromin

Patch has higher Risk of Venous Thrombo Embolism (VTE)

so not used more

Progesteron Only pill = Minipill

- MOA · Cx mucus
- Advise : use at same time every day
  ↓
  bcoz they have very short safe period = 3 hrs
- Minipill → Cerazette
  ↓
  has longer t1/2 and longer higher levels in plasma and inhibition of ovulation
  ↓
  so long safe period

Safe : bcoz no Estrogen component

Lactating Females ⇒ POP > IUD

→ POP will (↑) acne

POP (↑) ovarian cyst formation

⇒ OCP and POP are used in txt of dysmenorrhoea

m/c S/E of POP : Irregular Bleeding (Metrorrhagia)

→ Absolute C/I

1) Any pt k/c/o breast ca
2) Suspected Pregn
3) Undiagnosed Vaginal Bleed
4) POP (↑) the risk of Ectopic
   so prev ectopic is not C/I

Return of Fertility: Immediate Return.

→ INJECTABLE CONTRACEPTION ←

DMPA : depot MPA
(depo provera)

→ Injectable

150 mg IM x every 3 months

→ Mechanism of Action [MOA]
↳ Inhibition of ovulation

→ Safe period = 3 weeks

Good → Good for pt of sickle cell anemia
(DMPA ↓es the crisis)

→ ↑ seizures thersold

Bad → ① Bone Loss

② Delay in the return of infertility
avg delay = 11 months
@ long as = 18 months

→ DMPA (↓) PID
↓
Make mucus thick and imperable to infections

→ IMPLANON ←

only subcutaneous implant nowadays

Implants : lowest failure Rate (FR) 0.05%

Most effect contraception method

M/cost effective contraception : barriers

→ Vasectomy : FR 0.01%

Implanon : 1) Single Rod device

2) 68 mg etonogestrol @ 67 mcg/day

3) 4 cm long Rod

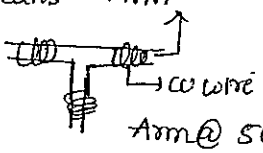
4) 3 years effective

5) FR 0.01%

6) Inhibition of ovulation

7) m/c S/E : Irregular bleeding

8) Upper Arm, Medical aspect, non dominent arm

Copper T (CuT)	Mirena
CuT 380A	
① Also called as : Paragard	① Also called : LNG-20
② 2nd generation	② 3rd generation
1st generation = Lippes loop	
③ A means = Arm	③ LNG-20 ↓ @ 20 mcg/day
 Cu wire Arm @ 50 mcg/day	LNG = 52 mg
④ Free of cost	④ Not free of cost
⑤ Radioopque → coated ↑ $BaSO_4$ ↓	⑤ Radioopaque
⑥ Life span 10 yrs	⑥ Life span 5 yrs

7) Cu → can be 12 yrs	7) Can be release 7 yrs
8) Emergency contraception	8) Not a EC
9) m/c S/E: Bleeding	9) ↓ser menstrual blood Loss
10) m/c c/c of Removing of CUT. is pain	10) C̄ in 6 months = amenorrhoeic
11) ~~↓ser the menstrual blood loss.~~	11) It has no role in PMS Premenstrual Synd DOC: SSRI

Mechanism of Action in IUD:

Inhibition of fertilization > Inhibition of Implantation

Dont act by inhibition of ovulation

IOC for a Missing IUD: Ultrasound.

Que) Preg + Cu T (insitu)

↓

on examination the theards can be seen

↓

Remove them

If ~~not~~ visible: continue preg C̄ not removing

Max infection: 1st 21 days

m/c infection: Actinomyces

→ * Absolute C/I ←

1) Undiagnosed Bleed
2) Suspected Preg
3) Current Pelvic Infection

4) Severe mullerian anomalies

5) Wilson's → CoX mirena

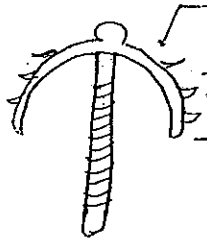
6) Breast Ca → ~~c~~

7) Post partum IUD: ① kept $\bar{c}$ in 48 hrs of delivery

② m/c used are CuT

③ upto 6 weeks [WHO recomendation]

Multiload : Bent Arms



→ less expulsion Rate

→ preloaded on the Inserter

→ effective for 5 years

→ Emergency Contraceptive ←

① Single m/Imp indication : unprotected sex

② Over Line counter

③ m/Effective emergency conception : CuT

④ m/Effective Hormonal emergency contraception :

Uhiprestal > mifepristone > LNG tablet

m/c used – LNG tab

Mirena
Minipill
Misoprost } Not a Emergency contraceptives

~~Abortion~~ Abortion

Ulipristal 30 mg SD

Mifepristone 10-50 mg

LNG tab → total dose = 1.5 mg

→ 2 tabs 0.75 mg (12 hrs apart)

→ 1 tab 1.5 mg (Better)

↓

↑ compliance

OCP's are used as Emergency contraceptive

200 mcg of EE + 1 mg LNG

↓

2 divided doses 12 hrs apart

E alone is not a EC

→ All EC's are effective upto 5 days

→ Hormonal are ideally ≤ in 72 hrs

On 5th day of unprotected act: CuT

MOA of Hormonal: delaying ovulation

↓

by delaying LH surge

Single best Ans

Delaying ovulation > Inhibition of Implantation

→ Laproscopic tubal ligation:

⇒ Liprocator

→ ESSURE: Hysteroscopic method

→ Female sterilization ←

→ " m/c used method of contraception "

- only done b/w 22-49 yrs
- only for married women / never married
- Child none : one child one year old
- only consent of the only the clint

(consent of the spouse is not mandatory)

*- Post partum sterilization

* Done τ in 1st 7 days
* Method: Minilaprotomy
  3 cm long incision
* Modefied pomeroy
* Laprosecopy is not used to post partum sterilization
* Ligation at ≥42 days : Not called as postpartum sterilizatio
  ↳ it is called Interval sterilization
  ↓
  non pregn state

Menstrual cycle = 1st 7 days

* Most/Effect Female sterilization =

Unipollar cautery > Ioving > Modified Pomeroy (or) (Parklend method)

↓ Not done ↓ Not done

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Unipollar cautory > Ioving > Modified Pomeroy (or) (Parklard method)

↓ Not done ↓ Not done

Least effective :

Clips > Bipolar cautery
(Hulka elemens)

least Risk of ectopic = modified pomeroy
Highest Risk of ectopic = cautery

→ ESSURE ←

Hysteroscopic method of Female sterilization

(coil)
↓
Intramural part of FT

⇒ m/c site for Female sterilization in tubal method is
Isthmus

inner part (stainless steel)
outer part (Nitinol)
↓
Nickel / titanium

⇒ onset of action is after 3 months
conform the block after " "
↓
conform by HSG

⇒ FR 0.5%

⇒ pomeroy = 0.4% FR
Modified " = 0.2% FR