India Connecting Continents (ICC)

Study materials for FMGE

www.indiaconnectingcontinents.com
indiaconnectingcontinents@gmail.com
(+86)15069629250 , (+91)93441 60131

OBSTETRICS

&

GYNAECOLOGY

Pregnancy Induced Hypertension (PIH)

* Hypertension in pregnancy is when BP ≥ 140/90 mmHg on two occassions 4 hrs apart.

2 conditions

- Hypertensive ♀ has conceived → Chronic HT in pregnancy
- Female BP was normal before conceiving. During pregnancy due to some of placental pathology her BP increases → PIH

Chronic HT in pregnancy	PIH
* Past history of (H/o) HTN present	* Past H/o HTN is absent
* Increase in BP will be seen before 20 wks of pregnancy	* Increase in BP will be seen after 20 wks (placental pathology occurs after 20 wks)
* BP does not come back to normal within 12 wks of delivery	* BP comeback to normal within 12 wks of delivery

PIH

- Pre-eclampsia
- Gestational HTN

- BP↑ after 20 wks
- BP comes back to normal in 12 wks after delivery

Pre-eclampsia:
* Proteinuria +nt (OR)
* Signs of end organ damage +nt

Gestational HTN:
* Protenuria -nt
* Signs of end organ damage also -nt

⇒ Now proteinuria is not essential to be present in pre-eclampsia. If signs of end organ damage is present then its enough.

* Proteinuria:
Defined as excretion of proteins in urine ≥ 30 mg/dl or ≥ 300 mg in 24 hours.

* Signs of end organ damage:
PIH is a multisystem disorder.
⇒ Serum creatinine levels > 1.1 mg/dl
⇒ Platelet count < 1 lakh
⇒ Liver enzymes raised > 2 times normal value
⇒ Pulmonary edema
⇒ Cerebral edema / visual symptoms.

Chronic HTN with superimposed Pre-eclampsia

* Hypertensive ♀ who conceives (BP will be raised before 20 wks)
↓
If suddenly at 20 wks of pregnancy
↓
- BP becomes uncontrollable
or
- Signs of end ~~of~~ organ damage
or
- New onset proteinuria
↓
Chronic HTN with superimposed pre-eclampsia.

Pre-eclampsia

Mild
↓
- BP ≥ 140/90 mmHg but < 160/110
- Signs of end organ damage are absent

Severe
↓
- BP ≥ 160/110
- Signs of end organ damage are present

Criteria removed are
* Proteinuria
* Oliguria
* IUGR (intra uterine growth ~~rate~~ retardation)

Pathophysiology of Pre-eclampsia

Placental pathology
↓
Resistance in maternal blood vessel is high / pressure in maternal blood vessel is high ($P \propto 1/V$)
↓
So the volume of blood which is going to placenta will decrease
↓
Placental ischemia
↓
Inflammatory mediators will be released
↓
Endothelial injury (capillaries become leaky)
↓
Intravascular fluid leaks in to the extravascular space. So
* Hemoconcentration
* Fluid collects in extravascular space - Edema.
↓
Due to hemocentration, there is multiorgan dysfunction
↓
MC organ involved is Kidney
↓
When mothers BP is high, renal blood flow decreases, GFR decreases → so filtering capacity is decreasing. (MCQ)

P ↓

Serum urea, serum uric acid & serum creatinine increases

↓

Also renal blood flow decreases → GFR ↓ → urine out ↓ → Oliguria.

↓

Most characteristic lesion in PIH & in kidney is glomerular endotheliosis (imp MCQ)

Risk factors of PIH (Important)

Remember

1) Primigravida females (exposed to placenta for the first time)
2) New paternity
3) Pregnancies:
   - → Molar pregnancy
   - → Rh-ve pregnancy
   - → Twin pregnancy
4) Syndromes:
   - → Metabolic X syndrome
   - → Antiphospholipid Ab syndrome

Others

- Obesity
- Past H/O HTN or PIH
- Family H/O HTN
- Diabetes
- Chronic renal disease

- Drug to prevent PIH / Pre-eclampsia

↓

ASPIRIN ± Calcium

- No role of:
  - * Bed rest
  - * Dietary salt intake
  - * Fish oil
  - * Antioxidants

Predictors of PIH

1) Giants roll over test:

Based on concept of supine hypotension syndrome.

↓

In late 3rd trimester if a pregnant ♀ lies supine

↓

Pregnant uterus presses against inferior venacava

↓

Venous return decreases, cardiac output decreases

↓

- In mother HR ↑, BP ↓
- Amount of blood going to fetus ↓ → Fetal distress

- So a pregnant ♀ is adviced not to lie supine because of chance of fetal distress
- Left lateral position > right lateral position

- Normally in all ♀, turn them from lateral to supine position – BP↓

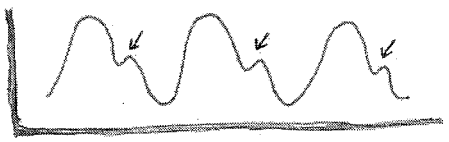
But if a female is about to develop PIH, instead of ↓ BP, BP↑ by ≥ 20 mmHg.

↓

This is called giants roll over test

- Time to do this test : 28-32 wks
- This test is outdated now.

⇒ The test which is done these days is Uterine Artery Doppler



In uterine artery doppler, normally a diastolic notch is seen

↓

This diastolic notch automatically disappears by 24 wks of pregnancy

↓

If it persists beyond 24 wks, means in future ♀ will have PIH

Management of PIH

* PIH / Pre-eclampsia is due to placental pathology
* So definitive management is termination of pregnancy / delivery
* Management of mild pre-eclampsia

↓

± Antihypertensive

↓

definitive mgt : Termination of pregnancy ≥ 37 wks

↓

Mode of delivery will be vaginal delivery

Eclampsia :

It is severe pre-eclampsia + Generalized tonic clonic seizures

* Signs of impending eclampsia in a case of severe pre-eclampsia
  (1) Headache
  (2) Visual symptoms like
    - Blurring of vision
    - Diplopia
    - Blindness
  (3) Epigastric pain, nausea, vomiting.
  (4) Oliguria.

* In Eclampsia, convulsions can occur during pregnancy

↓

Antepartum Eclampsia

* Convulsions during labor

↓

Intrapartum Eclampsia

* Convulsions after delivery

↓

Post partum Eclampsia (within 48 hrs of delivery)

mcq * MC variety of Eclampsia } Antepartum Eclampsia
mcq * Eclampsia with worst prognosis }

Management of severe pre-eclampsia

BP ≥ 160/110

* Two risks here

Due to high BP → Intracranial bleeding → Have to give Antihypertensive

Due to high BP, volume of blood going to brain ↓ (cerebral blood flow ↓) → Cerebral anoxia → Convulsions (Eclampsia) → Drug to prevent convulsion: $MgSO_4$

* So Antihypertensive + $MgSO_4$

mcq * Cerebral Anoxia is reason for convulsions / Eclampsia

* Definitive management in severe pre-eclampsia: → Termination of pregnancy at 34 wks → Mode of delivery: Vaginal.

Antihypertensive during pregnancy

Used in pregnancy

1) Labetalol
2) Methyldopa
3) Hydralazine
4) CCB (Ca^{2+} channel blockers)
5) Nifedipine
6) Nitroglycerine

Contraindicated

1) ACE inhibitors (Captopril, Enalapril) etc
2) Losartans
3) β-blockers
4) Diuretics
5) Diazoxide.

* Antihypertensive of choice for

mcq
- PIH during pregnancy
- chronic HTN during pregnancy
- Hypertensive crisis during preg

→ First choice: Labetalol

mcq * Second choice for chronic HTN during pregnancy: α-Methyl dopa

mcq * second choice for hypertensive crisis during pregnancy: Hydralazine.

Management of Eclampsia

* First line management:

mcq Management of airway (convulsion → tongue back → blocks airway)

mcq * Next step → Drug to treat convulsion → $MgSO_4$

mcq * $MgSO_4$ is not antiepileptic DOC during pregnancy

mcq * Antihypertensive DOC: Labetalol

* Definitive management:
Termination of pregnancy immediately irrespective of the gestational age.

* Mode of delivery: Vaginal.

$MgSO_4$

* MOA :- Blocks Ca^{2+} channels
- Also blocks release of ACh.

MCQ
* Therapeutic range:
4-7 meq/L

* Dose: PRITCHARD regime
↓
Loading dose: 4 gm i.v
+
10 gm i.m
(5 gm in each buttocks)

* Suppose Eclampsia patient comes at 12:00 noon
↓
loading dose $MgSO_4$
↓
Then every 4 hrly: Maintanence dose (5 gm im in each buttocks) → alternatively (5 gm at a time)
↓
continue till 24 hrs after delivery.

* At 4:00 pm → maintanence dose
→ check 3 things
1) Knee jerk +nt
2) RR ≥ 14 breaths/min
3) Urine output ≥ 30 ml/hr

(RR: Respiratory Rate)

* If any of these 3 things is ~~not~~ not present, it indicates magnesium toxicity.
↓
So dont give that dose at 4pm
↓
At 8:00 pm again check all these 3 things and give if normal.

MCQ
* Signs of $MgSO_4$ toxicity
1) Absent knee jerk / deep tendon reflexes
(when Mg ≥ 10 meq/L)
2) Decrease respiratory rate.
Respiratory arrest occurs when Mg ≥ 15 meq/L
3) Cardiac arrest (Mg ≥ 30 meq/L)

* DOC for $MgSO_4$ toxicity
Calcium gluconate

* Contraindication (C/I) of $MgSO_4$
1) Renal failure
2) Myaesthenia gravis

* DOC for status eclampticus:
(continous convulsion)
Thiopentone sodium

HELLP syndrome

H → Hemolysis
E, L } Elevated liver enzymes
L, P } Low Platelet count
(Thrombocytopenia)

* HELLP syndrome is a complication of severe pre-eclampsia

↓

Management is immediate termination of pregnancy.

⇒ Smoking is protective in PIH (releases tension in both ♂ & ♀)

HELLP syndrome

* Hemolysis
  - LDH ≥ 600
  - Bilirubin > 1.3
  - Haptoglobin will be less (most specific)
  - Peripheral blood smear have Burr cell, Helmet cells, Shistocytes

* Elevated liver enzymes:
  SGOT, SGPT ≥ 70

* Low platelet count
  < 1 Lakh

* For diagnostic criteria:
  1) LDH ≥ 600
  2) SGOT, SGPT ≥ 70
  3) Platelet count < 1 lakh

Tennesse Criteria for diagnosing HELLP syndrome

HIV in Pregnancy

mcq
* MC infection in pregnancy
  Cytomegalovirus (CMV)

mcq
* Most teratogenic
  Rubella

mcq
* Non teratogenic (no congenital malformation)
  HIV

⇒ HIV is
- Not teratogenic
- Universal screening is adviced for HIV
- Screening method of choice is ELISA
- According to NACO (National AIDS control Organisation)
  → 3 ELISA → if 1 or < 1 is +ve, ♀ is HIV-ve

  ↓

  if 2 are +ve, the result is equivocal. So do a confirmatory test which is Western blot

  ↓

  if all are positive, HIV is confirmed.

* If a pregnant ♀ is HIV +ve

  ↙ Was on anti retroviral (ART) therapy before pregnancy

  ↓

  continue the same during pregnancy

  ↘ was not ART

  ↓

  put her on ART irrespective of her CD4 count

* Timing to start ART depends on CD_4 count

↓

CD_4 < 500 → as soon as possible (in first trimester)

↓

CD_4 > 500 → start ART at 14 wks (after 1st trimester)

* ART of choice during pregnancy.

Tenovofir + ~~E~~favirenz + Lamivudin
(300mg) (600mg) (300mg)

depending on WHO ↓ ↓ ↓

Zidovudine + Nevirapine + Lamivudine

* With the use of ART, rate of vertical transmission (mother to child) can be reduced by 63%. (rate becomes < 2%)

MCQ

* ART should be continued throughout pregnancy.

* If a pregnant female → during labour → ELISA → HIV +ve → not on ART

↓

Give her Zidovudine/Nevirapine 4hrs before C-section or at onset of labour

+

Baby should be given Nevirapine within 72 hrs of p/ delivery

MCQ

* MC time of transmission of HIV Peripartum period (during labor + imm. after that)

Ways of ↓ vertical transmission

MCQ

1) ART
2) Cesarean section

* C-section in HIV patients:

According to ACOG (American College of Obstetricians & Gynaecologists) C-section should be done in HIV patient if viral load > 1000 copies/ml

According to NACO & WHO, C-setion should be done in HIV patients only for obstetrical reasons. (like contracted pelvis, fetal distress)

* C-section reduces risk of vertical transmission by 50%.

* For cesarean to be effective, it should be done 4 hrs before membrane rupture

* Contraindications in HIV pts

1) Instrumental delivery (forceps / vaccum)
2) Fetal scalp monitoring
3) Artificial rupture of membrane.

* Once the baby is born, immediately give bath to the baby

↓

Breast feeding is not contraindicated

↓

Give baby Nevirapine/ Zidovudine for 6 wks

Imp points on other infection

1) Rubella

* Most teratogenic
* MC time of transmission - 1st trimester
* If rubella affects before 20 wks of pregnancy to a pregnant ♀
  ↓
  leads to congenital Rubella syndrome in fetus
  ↓
  indication of doing MTP
* Rubella vaccination is contra indicated during pregnancy
* After rubella vaccine, patient do not conceive for 1 month.

2) CMV (Cytomegalovirus)

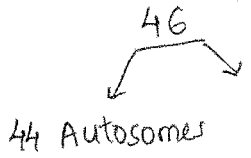
* MC infection during pregnancy
* Transmission is equal in all trimesters
* Severity of infection to fetus ↓ as pregnancy ↑.
* It does not lead to congenital heart disease in fetus

3) Toxoplasma

* By Toxoplasma gondii
* Max. transmission occurs in 3rd trimester (mc)
* Most severe fetal infection occurs if transmitted in 1st trimester.
* In mother → toxoplasma → cervical lymphadenopathy → in fetal leads to a triad of chorioretinitis, microcephaly, intracerebral calcifications.
* DOC for preventing fetal transmission of toxoplasma:
  Spiramycin
* DOC for treating fetal infection
  Sulfalazine + Pyrimethamine

Fundamentals

* Chromosome number → 46 → Diploid number



* Males → 44 + XY, 46 (XY)
* Females → 44 + XX, 46 (XX)

} Karyotype (genetic makeup of individual)

* Diploid - 46, Haploid - 23
* ~~Triploi~~ Chromosomal aberration
* Triploid → ~~2x~~ 23 × 3 = 69
* Trisomy → One extra chromosome
  46 + 1 = 47
  Chromosome 21, Trisomy 21
  ↓
  Down's syndrome
* Monosomy → 1 less chromosome - 45
  Monosomy X → Turner's syndrome
  45 (XO)
* Klienfelter's syndrome: 47 (XXY)
* Sex of an individual / fetus is decided by SRY region (sex related region of Y-chromosome) present on short arm of Y-chromosome
* If Y-chrom. +nt → Sex of fetus is male
* If Y-chr. −nt → Sex is female.
* Y chromosome is +nt in Klinefelter's syndrome → Male
* Turners → no Y → Female
* Genotype / Karyotype: Genetic makeup
* Phenotype: Physical appearance

Sex determination

* MC method → Looking at the ext. genetelia
* In some cases can not do that
  - Ambigous genetelia
  (by looking at genetelia, sex cannot be determined)
* Best method of sex determination is karyotyping
  Y +nt → Male
  Y - nt → Female
* Another method → No. of barr bodies
* No. of barr bodies = No. of X − 1
* So in females, BB = 2 − 1 = 1
  Males → 1 − 1 = 0
* But in Turners → 0 (but not male)
  Kleinfelters → 1 (but not ♀)
  So it is not a good method

Gonads / Germcells / Genatalia

* Initially in both males and females all these are bipotential → they can either develop in to female / male.
* Bipotential → Till 6 wks present
* By 6 wks, SRY region determine sex of baby.

	Gonads	Germcell	Genatalia
Males	Genital ridge ↓ Y+nt Testis	Ychr:+nt Spermatogonia (46XY)	Testosterone +nt
Females	Genital ridge ↓ Y-nt Ovary	Oogonia (46XX)	Testosterone -nt

* Testis formed by 6-7 wks
* Ovary formed by 7-8 wks
* For formation of ovary → Y chr -nt
* So in turner's syndrome, gonads are ovaries
* In males → if Y+nt → ectoderm of yolk sac → forms male germ cells (spermatogonia) → 46XY
* In females → Y-nt → ectoderm of yolk sac → forms female germ cell (oogonia) → 46XX
* In males → Chr No: 46XY

Males (46XY)
↓
Gonads (Testis)

Sertoli cells
↓
releases Mullerian inhibiting factor
↓
inhibits Mullerian duct (Paramesonephric duct)
↓
Mullerian duct regresses
↓
Remnant of Mullerian duct in ♂ - ~~Appendix~~
- Appendix of Testis,
- Prostatic utricle.

Leydig cells
↓
Releases Testosterone
↓
leads to development of Wolfian duct (Mesonephric duct)
↓
Wolfian duct forms
- Seminal vesicle
- ~~Epidy~~ Epididymis
- Ejaculatory duct
- Vas deferens (SEED)

↓
Male internal genital organs.

* Also Testosterone
↓
Dihydrotestosterone
↓
Male external genital organs

Key concept

- Male external genital organs are formed only when testosterone is present in intrauterine life
- If testosterone is not present in intrauterine life then male external genital organs are not formed & female external genital organs are formed

* If SRY-region is +nt (present)
↓
Gonads - Testis
↓
Leydig cells
↓
Testosterone
↓

MCQ

- Genital tubercles → Penis
- Genital swellings → Scrotum
- Genital folds → Penile urethra

No Testosterone
↓
Dihydrotestosterone absent
↓
Genetalia looks like female external genetalia
↓

- Genital tubercle → Clittoris
- Genital swellings → Labia majora
- Genital folds → Labia minora

* In females : 46(XX), Y-nt
↓
Gonads : Ovary

No Sertoli cells
↓
Mullerian inhibiting factor absent
↓
Mullerian duct will grow
↓

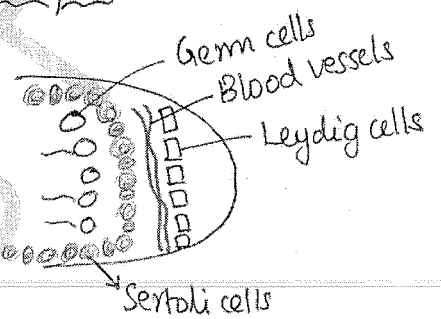
- Fallopian tube
- Uterus
- Cervix
- Upper vagina

↓
female internal genital organs

No Leydig cells
↓
No testosterone
↓
Wolfian duct regress
↓
Remnants of Wolfian duct in adult life

- Epo-opharon
- Para-opharon
- Gartner's duct
- Kobelt tubercle

Key concepts



MCQ
* Sertoli cells forms a blood-testis barrier b/w developing germ cells and blood
* They protect germ cells from the toxins which are present in blood

MCQ
* Leydig cells lies outside the blood-testis barrier.

MCQ
* For formation of ovary, absence of Y-chromosome is needed. But for proper development of ovary both X~~(XX)~~ chromosomes are needed (XX)

ie why in Turner's syndrome (45XO) → Y-nt (gonad-ovary) → Because 2X are not +nt → no proper developement of ovary → Streak gonads

Female internal genital organ development

↓

from the Mullerian duct

↓

develops fallopian tube, uterus, cervix, upper vagina (from MD)

↓

- Lower vaginal → Urogenital sinus (sinovaginal bulb) MCQ
- Vaginal epithelium → Endoderm of urogenital sinus (MCQ)

In a female if both mullerian duct is absent

↓

Mullerian agenesis / Mayer's Rokenstansky Kuster Hauser syndrome (MRKH)

- fallopian tube, uterus, cervix, upper vagina → Absent
- Lower vagina, Ovaries (from genital ridge) → Present (MCQ) → Ovulation also present

Homologous organs → organs which have the same embryonic origin

	(♂)	(♀)
* Genital tubercle →	Penis,	Clitoris
* Genital swelling →	Scrotum,	Labia majora
* Genital folds →	Penile urethra,	Labia minora

Males	Females
• Prostate gland →	Skene gland
• Cowpers gland →	Bartholin gland

Ambiguous genitalia

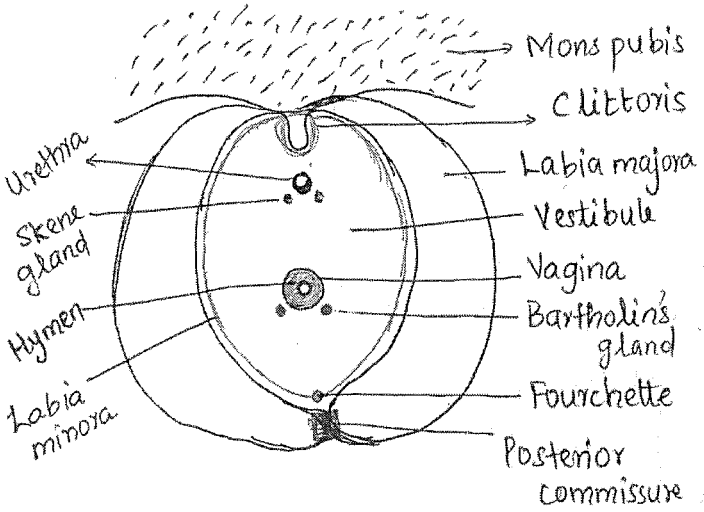
* In males, external genitalia looks like males because of presence of testosterone in intrauterine life.
* If in intrauterine life, in males, testosterone is -nt, their genitalia will start resembling females (ambigious genitalia)

MCQ
* MCC of ambigious genitalia in males : Androgen insensivity syndrome (Testicular feminising syndrome)

* In females, external genitalia are formed because of absence of testosterone in intrauterine life
* If in intrauterine life, if females exposed to testosterone, their genitalia starts resembling males (ambigious genitalia)

MCQ
* MCC of ambigious genitalia in females : Congenital adrenal hyperplasia

Female External Genitalia (Vulva)



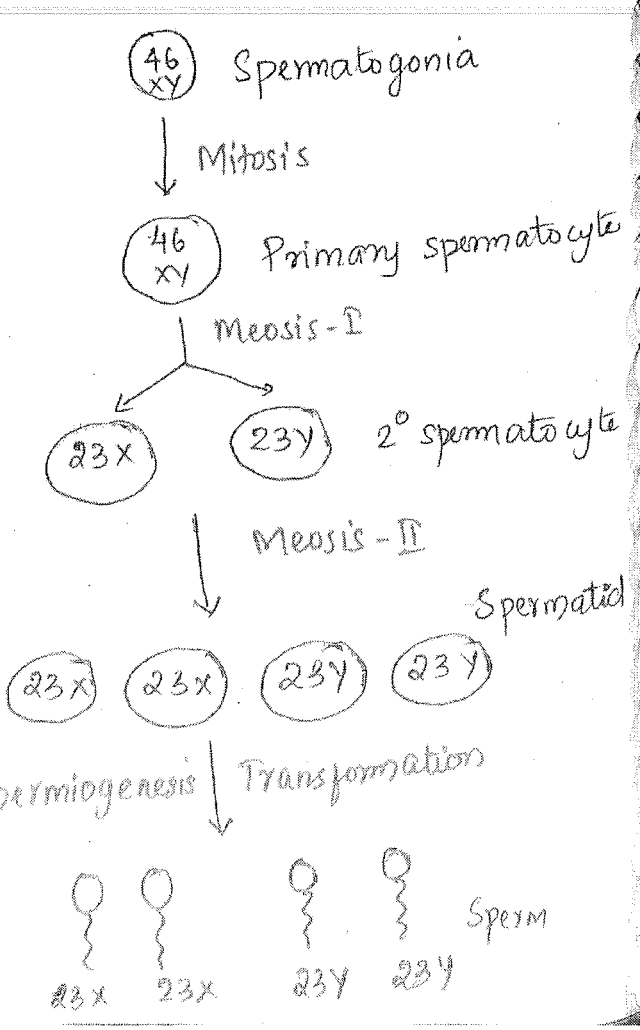
* Clitoris → Erectile, vascular
* Posteriorly labia majora meets at Posterior commissure
* Posteriorly labia minora meets at Fourchette
* Triangular area bounded by clitoris and labia minora is Vestibule
* Labia minora encircles clitoris
* Openings in Vestibule
  - Urethra
  - Skene gland
  - Vaginal opening / Introitus
* Vaginal opening is covered by a thin membrane → Hymen
* Small hole in hymen → to menstrual blood to comes out
* If no opening → Imperforate hymen

* Both sides just below urethra - Skene gland
* On either side of vagina - Bartholins gland

Differenciation of germ cells

Spermatogenesis

* In cell division
  1) Mitosis → Chromosome no: remains the same
  2) Meosis → I & II
     - I → Chromosome no: become half (reduction division)
     - II → Chromosome no: remains the same



* Spermatogenesis → Spermatogonia changes in to sperms
* It involves both mitosis & meosis
MCQ * Total time taken → 74 days (70-75)
* Spermatogenesis occurs at puberty and continues throughout the life
* Spermiogenesis → transformation of spermatids in to sperm.
* Here no mitosis & meosis
* Time taken → 14 days

Sperms (MCQs)

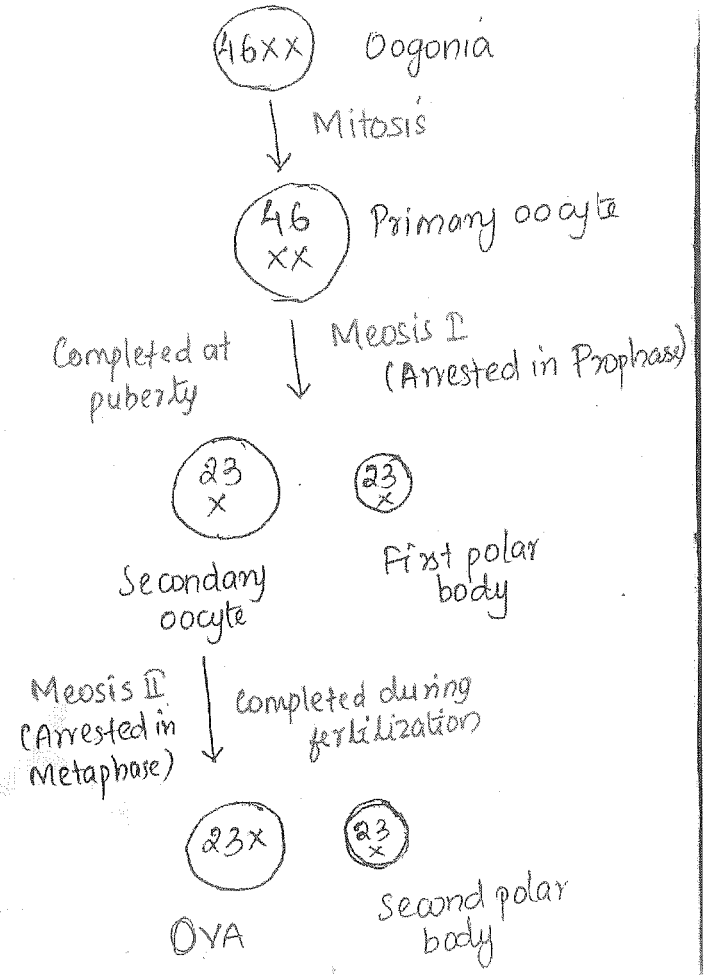
* Size of sperm : 50-60 microns
* Fertilisable span → 48-72 hrs
* It attains motility & maturity in the epididymis

Capacitation

* Ability of sperms to penetrate ova.
* It occurs in female reproductive tract (maximum at fallopian tube)
* Time taken → 7 hrs

Oogenesis

* Oogonia derived from the ectoderm of yolk sac.
* Oogenesis begins in intrauterine life
MCQ * Meosis I → arrested in prophase → completed division in puberty
* In new born ♀ → ovary → primary oocyte



MCQ * Meosis II → Arrested in Metaphase → completed during fertilization
* Ovulation is release of secondary oocyte from primary oocyte
MCQ * First polar body (23x) is released along with ovulation
MCQ * 2nd polar body (23x) is released at the time of fertilisation.
MCQ * Size of ova → 120 micron (Largest cell in body)
* In a new born ♀ ovary & up till puberty, 1° oocyte gets surrounded by follicular cells → Primordial follicle

mcq
* Size of primordial follicle just before ovulation : 18-20 mm
* Fertilisable span of the ova is 12-24 hrs

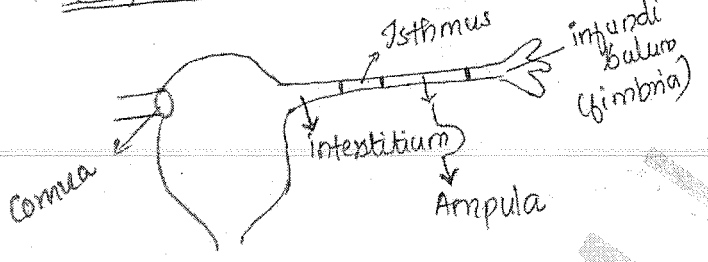
mcq
* Primitive germ cells ~~take place~~ → in ectoderm

mcq
* Meosis I → Actual reduction division → 1° spermatocyte to 2° spermatocyte

mcq
* Ovary & Testis → from genital ridge

Anatomy of female genital tract

1) Fallopian tube



* Medial to lateral :
  Interstitium → Isthmus → Ampulla → infundibulum
* Length of fallopian tube is 10 cm
* Lined by ciliated columnar epithelium
  ↓
  MC cancer is adenocarcinoma

mcq
* Fallopian tube also has Peg cells
* Blood supply of
  - medial 2/3 → Uterine artery
  - lateral 1/3 → Ovarian artery

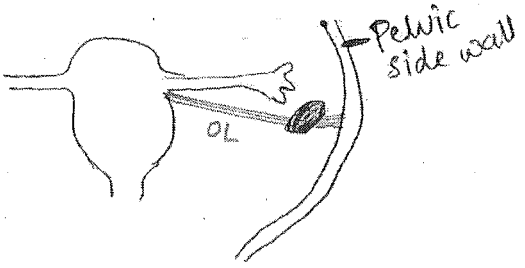
* Lymphatic drainage:
  - medial → to superficial inguinal lymph nodes
  - lateral → to paraaortic LN

Important MCQs

* Longest part : Ampulla
* Widest part : Ampulla
* Fertilisation occurs in Ampulla
* MC site of ectopic pregnancy : Ampulla
* MC site of doing tubectomy : Isthmus
* MC site of blockage due to tuberculosis → Cornual end of tube (uterine angle)
* MC site of blockage due to Gonococcus → Fimbrial end of the tube.
* Fallopian tube develops from the Mullerian duct.

2) Ovaries

*



* Ovarian ligament connects ovary to cornua of uterus
* Ovary to pelvic side wall by Suspensory ligament of ovary/ Infundibulopelvic ligament
* Developed from genital ridge

* Size of ovary → 3x2x1 cm
* In adult life, ovary is present in the lateral pelvic wall
* In intra uterine life → present at T_{10} vertebra → from T_{10} vertebra it descends down with the help of "Gubernaculum".
* Uterus divides gubernaculum in to two parts → Ovarian ligament & Round ligament.

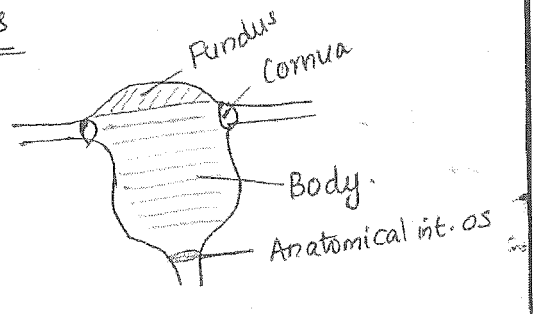
MCQ
* Ovarian ligament & round ligament are derived from gubernaculum.
* Ovarian ligament connects ovary to the uterus
* Infundibulo pelvic ligament connects ovary to the lateral pelvic wall.

MCQ
* Ovarian vessels are present in
  - Infundibulo pelvic ligament

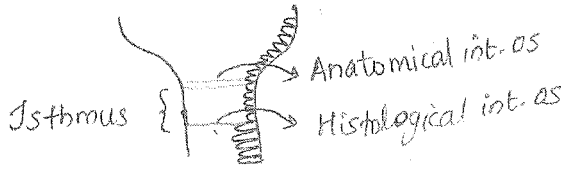
MCQ
* If during hysterectomy, ovary is not to be removed, which ligament should not be cut
  Suspensory ligament / Infundibulo pelvic ligament

* Lining epithelium of ovary is Germinal epithelium.
* Blood supply:
  Ovarian artery (branch of abd. aorta → at level of L2)
* Venous supply → left & right ovarian vein
* Left ovarian vein drains in to left renal vein
* Rt ovarian vein → inf venacava
* Lymphatics: Para-aortic LN

3) Uterus



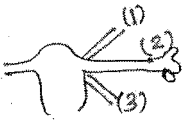
* Part above cornua: Fundus
* Part below cornua: Body
* Lined by columnar epithelium
* Anatomical internal os → where anatomically uterus becomes cervix
* Histological internal os → area where lining epithelium changes to ~~long~~ high columnar epithelium
* Always histological internal os lies below anatomical internal os



* Distance b/w ~~anatomical~~ anatomical int os to histological int. os
  - Isthmus

* Parts of uterus :
  - Cornua (angle of uterus)
  - Body
  - Fundus
  - Isthmus

* 3 structures attached to cornua of uterus from ant. to posterior:
  - R – Round ligament
  - T – Fallopian tube
  - O – Ovarian ligament



* MCC of failure of female sterilisation → Identification of wrong structure

* Lymphatic drainage of
  - Cornua → Superficial inguinal LN

* Lymphatic drainage of body in to External iliac LN

* Isthmus is the part of uterus which lies b/w anatomical internal os & histological internal os.

* It is 0.5 cm in length

* It forms the lower uterine segment during pregnancy.

* Length of lower uterine segment at term → 10 cm

* Identification of lower uterine segment
  ↓
  loose fold of peritoneum attached to the lower uterine segment.

* Uterus is pyriform shaped

* Weight of uterus
  - In non pregnant : 60-80 gm
  - Pregnant : 1000 gm

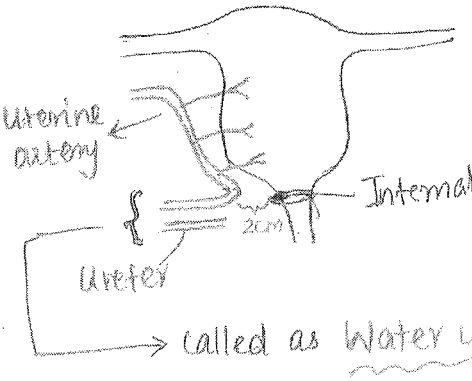
* Length of uterus
  - Non pregnant → ~~6-8 cm~~ 6-10 cm
  - Pregnancy → 35 cm

* Volume of uterus
  - NP → 10 ml
  - P → 5000 ml

* Lined by columnar epithelium
  ↓
  MC endometrial cancer is Adenocarcinoma.

* Blood supply :
  - Major → Uterine artery (branch of ant. division of internal iliac artery)
  - Also supplied by ovarian artery

* Uncontrollable PPH in which arteries can be ligated to stop bleeding
  1) Uterine A
  2) Ovarian A
  3) Ant. division of internal iliac artery.



→ called as Water under bridge

MCQ
* MC site of ureteric injury → The place where uterine artery crosses (2cm lateral to int. os)

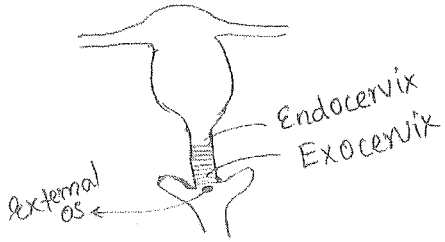
MCQ
* MC gynaec surgery causing the ~~ute~~ ureteric injury
Hysterectomy
(Removal of uterus + cervix)
↓
called as total abdominal hysterectomy (TAH)
or
Simple hysterectomy.

* Two important branches of uterine artery:
1) Descending cervicovaginal artery (supply cervix & vagina)
(MCQ) 2) Sampson artery (Supplies round ligament)

* Nerve supply: $T_{10} - L_1$ segments

(4) Cervix

* Has 2 parts → near uterus & vagina



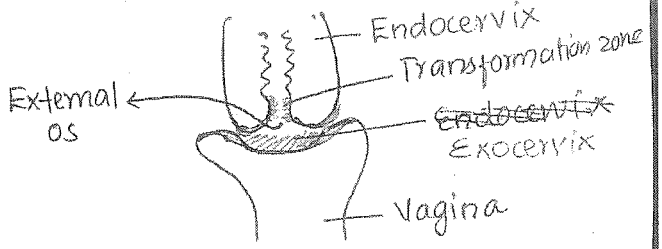
* Endocervix (~~Portio vaginalis~~) is lined by columnar epithelium
* Exocervix (Portio vaginalis) stratified squamous epithelium

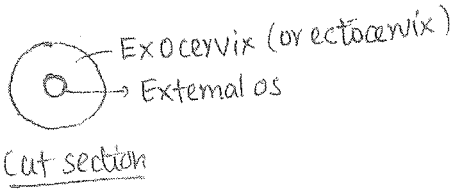
(cervix)
* Part where cervix opens in vagina
External os.

* Transformation zone / squamo-columnar junction
↓
columnar epithelium of endocervix transforms into squamous epithelium of exocervix. (gradually)

* Length of cervix:
- Non-pregnant → 2.5 cm
- Pregnant → 4 cm

* Parts of cervix:
1) Endocervix → part close to uterus (red) (columnar epithelium)
2) Exocervix → part inside the (pink) vagina (Portio vaginalis) (stratified squamous epithelium)





Cut section

* On perspeculum examination, exocervix appears pink in colour & endocervix appears red
* Surrounding them, whitish area will be transformation zone.

MCQ
* External os will be pinpoint / circular in shape ○
↓
in female who have never give birth to a child
↓
Nulligravida.

MCQ
* External os is ~~slight~~ slit like in ⬭
↓
♀ given birth
↓
Multigravida females

* Cervix : Corpus ratio (corpus = uterus)
  - Before puberty, cervix is bigger than the uterus
  - Before puberty → 2:1
  - At puberty → 1:2
  - In reproductive age → 1:3 / 1:4
  - After menopause → 1:1 (both undergoes atrophy)

* Blood supply of cervix
  - descending cervical artery (branch of uterine artery)

MCQ
* Lymphatics →
  I - Internal iliac LN
  H - Hypogastric LN
  O - Obturator LN
  P - Presacral / Ureteric LN
  E - External iliac LN

MCQ
* Cervix does not drains in to Superficial inguinal LN

MCQ
* Sentinel LN for cancer cervix
  Ureteric LN (Presacral LN)
  (first LN – Sentinel LN)

* Nerve supply : $S_2 - S_4$ segment
* pH : 7.8 (alkaline)

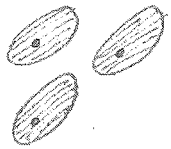
(5) Vagina

* Has an anterior wall & post. wall
* Posterior wall of vagina is 2 cm longer than the ant. wall.
* Lined by stratified squamous epithelium.
↓
has 3 types of cells
↓
1) Superficial cells
2) Intermediate cells
3) Basal / Parabasal cells.

* ~~superficial cells~~

Concept
* Study of vaginal epithelium can tell us which hormone is predominant in the body

MCQ
* For hormonal study, sample should be taken from which wall of vagina?
  Lateral wall of vagina

Cells	Drawing	Characteristic	Predominant
1) Superficial cells		* Eosinophilic (pink) * Pyknotic nuclei	• Predominant when estrogen is predominant ↓ eg: First half of menstrual cycle
2) Intermediate cells		* Basophilic (blue) * Little bigger nuclei	• Predominant when progesterone is predominant ↓ eg: Pregnancy, 2nd half of menstrual cycle
3) Basal & Parabasal cells		* Small basophilic cells (blue) * No distinct boundary * Big nuclei	• Predominant when no hormone is predominant eg: Menopause

mcq
* Vagina does not have any glands so vaginal secretions are actually from the:
  - → Cervical
  - → Endometrial
  - → Bartholin glands

* Bartholin gland, give discharge only at the time of intercourse.

mcq
* Vaginal has inhabitant bacteria called as Doderlin bacteria (lactobacilli) which appear at the time of puberty

* Doderlein bacteria converts glycogen present in vaginal epithelium to lactic acid. So acid pH in vagina

⇒ Doderlein bacteria disappears after menopause (leads to dry vagina)

Age	pH_vagina
• At puberty	• Changes to acidic 4-5.5
• Reproductive age	• Acidic 4.5
• Pregnancy	• Very acidic 3.5 (↑ no. of Doderlein)
• Before puberty • After menopause • During the menstrual cycle	6-8

* Lining of vagina → Stratified squamous epithelium
↓
MC cancer is Squamous cell carcinoma

* Blood supply
1) Descending cervicovaginal artery (branch of uterine A)
2) Internal pudental artery
3) Middle rectal artery

* Lymphatic drainage:
= Upper vagina → Same as cervix (IIHOPE)
= Middle vagina → Int. iliac LN
= Lower vagina → Sup. inguinal LN

6) Vulva

* Blood supply → Internal pudental artery
* Nerve supply → Pudental nerve
* Lymphatic drainage → First goes to superficial inguinal LN (Sentinel LN)
↓
then to deep inguinal LN

MCQ
* Clittoris drains in to lymphnode of Rosenmuller / Cloquet LN.

Key concepts

* Cervix has 2 parts
1) Endocervix (columnar)
2) Exocervix (squamous)

* MC cancer of cervix is squamous cell carcinoma.
* MC site of cancer cervix is Transformation zone
* MC site of adenocarcinoma is Endocervix

⇒ Pain during labour
✓ During early labour (due to uterine contraction pains)
$T_{10} - L_1$
✓ During 2nd half of labor, pain is due to dilatation of cervix
$S_2 - S_4$

MCQ
- Painless labor → epidural analgesia, DOC: Bupivacaine, given at T_{10} segment (from there to below it blocks)
- For caessarean → spinal anesthesia → at T_4 (because the peritoneum also have to be blocked)
- For applying forceps / for vacuum → block lower vagina and vulva → pudental nerve block.

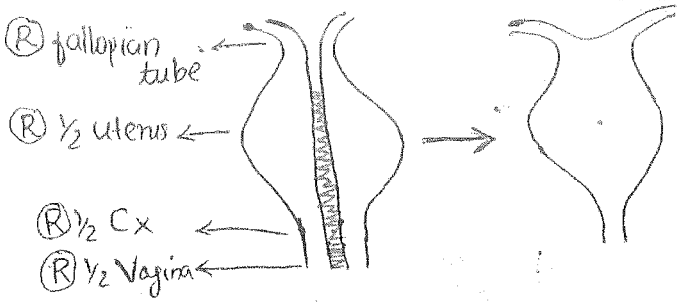
mcq
* Site of giving pudental block
Ischial spine

mcq
* Ligament pierced during pudental block is
Sacrospinous ligament

mcq
* Bartholin's cyst:
↓ Due to blockage of bartholin's gland
Management ⇒ Marsupilisation

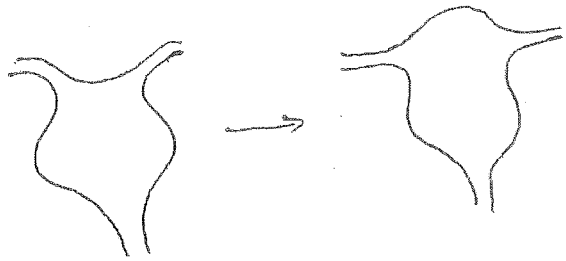
mcq
* Rx. of Bartholin abscess:
Incision & Drainage (I & D)

Development of female genital tract

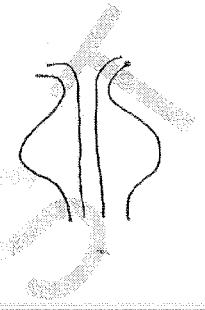
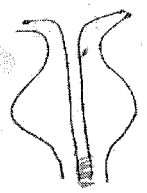
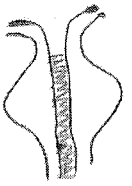


* Both side mullerian duct grows from above to down.
* Right MD → Ⓡ ~~half~~ fallopian tube, Ⓡ half uterus, Ⓡ half cervix Ⓡ half ~~vagina~~ upper vagina.
* Then they fuse in below upward direction
* Once they are fused, a septa is formed → then septa resolves in below upward direction at 20 wks of pregnancy
* Now two fallopian tube, ~~the~~ single uterus, single cervix (Cx) and single vagina.

mcq
* So uterine cavity is formed at 20 wks.
* After that fundus of uterus becomes dome shaped.



Mullarian Duct Anomalies

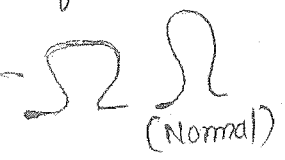
Condition	Called as	Diagram	Comment
1. If both MD are absent	Mullerian agenesis MRKH syndrome		* Ovary is normal * Ovulation is present
2. If one side MD absent	Unicornuate uterus		* 2 ovaries * 1 FT * Uterus Cx Upper vagina } ½
3. Both MD +nt but fails to fuse	Uterus didelphus		* 2 ovaries * 2 FT * 2 Uterus * 2 cervix * 2 upper vagina 2 canulas (LWC)
4. Both MD+nt, but incomplete fusion	Bicornuate uterus Bicornis Unicollis (1 Cx) Bicollis (2 Cx)		* 2 ovaries * 2 FT * 2 Uterus * 1 vagina (upper) * 1 Cx → Unicollis 2 Cx → Bicollis
5. If both MD+nt, fusion occurs, but septa fails to resolve	Septate uterus		* 2 ovaries * 2 FT * 1 Uterus * 1 Cx * 1 vagina But inside the uterus septa is present

LWC → Leech Wilkinson Canula

mca

* Normal uterus is dome shaped, if fundus of uterus is flat

↓

ARCUATE Uterus  (Normal)

MCQs

⇒ MC uterine anomaly
Septate > Bicornuate uterus

⇒ MC problem with them
Abortion

⇒ MC anomaly associated with abortion
Septate uterus

⇒ MC time of abortion
Second trimester

* ⇒ Repair of uterine malformations is not indicated unless they lead to 3 or >3 abortions

⇒ IOC is MRI > 3D USG

⇒ Gold standard : Laproscopy

⇒ Other investigation which can be done : HSG (Hystero salpingo graphy)

⇒ HSG cannot differentiate b/w septate & bicornuate uterus. So it is not IOC

⇒ In HSG, a radiopaque water soluble dye is passed in to the uterus using special canula called as Leech Wilkinson Canula.

⇒ The path of dye is seen during various X-rays.

⇒ Best time to do HSG is first half of menstrual cycle, especially day 10.

↓

Rule of 10

↓

All radiological investigations should be done in ♀ during first 10 days of menstrual cycle.

⇒ Contraindications of HSG :
1) TB of genital tract
2) PID (pelvic inflam. disease)
3) Pregnancy

mca

⇒ HSG cannot differentiate b/w septate & bicornuate uterus. But still we can identify it.

Bicornuate

- Angle b/w 2 horns of uterus > 60°
- Distance b/w 2 horns ≥ 4cm

Septate

- Angle < 60°
- Distance < 4

Management of mullerian malformations

↓

- Surgery (when ≥ 3 abortions)

↓

Repair the uterus K/a Metroplasty

↙ Abdominal

↘ Hysteroscopic

- Abdominal metroplasty
  1) Jones metroplasty
  2) Strausman M
  3) Thompkins M
  } outdated

- Nowadays, hysteroscopic metroplasty is preferred.

⇒ In septate uterus → hysteroscopic resection of the septa.

VAGINITIS

Bacterial vaginosis :

* Alteration in vaginal flora.
* Lactobacilli (Doderlein) replaced by cocobacilli
* MC organism → Gardenella vaginalis
* Earlier k/a Gardenella vaginitis
* Now it is seen that no inflammation so k/a bacterial vaginosis (not vaginitis)
* It is not an STD (sexual transmited disease)
* MC vaginosi vaginitis
* pH of discharge >4.5
* Symptoms : Foul smelling, dirty white discharge, no pruritis, (no inflammation, so no itching)
* IOC : Saline microscopy (clue cells seen → vaginal epithelial cells adhered by bacteria)

(continues...)

Candidiasis

* MC organism → Candida albicans (fungus)
* Usually it is not an STD because candida is a commensal in vagina
* MC vaginitis in pregnant ♀, diabetics, in OCP users, steroid users, antibiotic user, immuno-compromised states. (like HIV)
* pH < 4.5 (only one survive in acidic media)
* Curd / cottage cheese like discharge, intense itching
* IOC : Saline microscopy (hyphae seen)

(continues)

Trichomonas vaginitis

* MC → Trichomonas vaginalis (flagellated protozoa)
* It is an STD
* pH of discharge is 5-6
* Greenish white discharge, itching, dysuria (pain during urination)
* Signs → Red "angry looking" / strawberry like vagina.
* IOC : saline microscopy (motility +nt because of protozoa flagella)

(continues)

Bacterial vaginosis

* Gold standard ⟶ No gold stand.
  Dx → No.
  Diagnosed by Amsel criteria.

Amsel criteria

1) Foul smelling dirty white discharge thinly coating vagina
2) pH of discharge > 4.5
3) Clue cells > 20%.
4) When 10% KOH is added → fishy odour (Whiff test +ve)
   (Amine test +ve)

* Whiff test → Always +ve
* DOC: Metronidazole (500mg BD x7d)
  • Pregnancy
    = DOC → Metronidazole (250 mg TD x 7 day)
    Clindamycin
  • Drugs should be avoided in 1st trimester
* No Rx for male partner (Not STD)

Candidiasis

* Gold standard → Culture (Saberound media)
* Whiff test → Always -ve
* DOC: Fluconazole (150 mg stat)
  • Pregnancy → Fluconazole (but avoided in 1st trimester)
* Rx of partner done only if symptoms are present

Trichomonas

* Gold standard: Culture (diamond media)
* Whiff test → +/-
* DOC: Metronidazole (500mg BD x 7 days) or (2gm stat)
  • Pregnancy → Metronidazole (should be avoided in 1st trimester)
* Since STD, Rx male partner.

OVARIAN CYCLE (Menstrual cycle)

* At puberty → ovary has 1° oocyte surrounded by follicular cells called as primordial follicle.

Hypothalamus
↓ GnRH (pulsatile manner)
Anterior pituitory
↓
FSH

* At puberty hypothalamo pituitary ovarian axis (HPO) becomes functional & releases FSH
* FSH acts on follicular cells and converts them in to granulosa cells & theca cells.

HPaxis
↓
FSH
↓
Follicular cells
↙ ↘
Granulosa cells | Theca cells

* FSH acts now on granulosa cells and releases
  - Estrogen
  - Inhibin B

* Functions of Estrogen are:
  (1) Negative feedback on FSH
  ↓
  All follicles degenerate except one k/a dominant follicle

  (2) Acts on the uterus & proliferates its endometrium.
  (so excess estrogen causes endometrial cancer)

  (3) Positive feedback on ~~inhib~~ LH

* Inhibin B → Negative feedback on the FSH.

* Positive feedback of Estrogen on the LH
  ↓
  Levels of LH suddenly increase k/a LH surge.
  ↓
  For LH surge to occur, levels of estrogen should be 200 pg/ml for 48 hrs.

* Levels of LH suddenly rise,
  LH surge
  ↙ ↘
  Ovulation | Acts on theca cells & from theca cells
  ↓ | ↓
  • 1° oocyte to 2° oocyte | Releases androgens
  • follicle to corpus luteum (by LH) | ↓
  ↓ | converted to estrogen in adipose tissue. with the help of Aromatase
  Corpus luteum releases 4 hormones
  ↓
  1) Progesterone (main)
  2) Estrogen } Negative feedback on FSH.
  3) Inhibin A
  4) Relaxin (only during pregnancy)

Progesterone
↙ ↘
• Negative feedback on LH | • Support the endometrium
↓ | ↓
Corpus luteum degenerates. | k/a secretory action.
↓
↓ progesterone → ↓ progesterone
↓ Estrogen | ↓
↓ Inhibin A | • Support to the endometrium is lost.
(At pregnancy ↓ relaxin) | ↓
| Endometrium sheds – Menstruation.
| • Progesterone is a smooth muscle relaxant
| ↓
| ↓ preg, blood vessel constricts

↓ Progesterone
↓
Vessels contract
↓
Releases PGF 2-α
↓
Pain during menstruation

MCQ
⇔ Prostaglandin released during menstruation : PGF 2-α

⇔ ↓ Estrogen & ↓ Inhibin A
↓
Levels of FSH ↑
↓
New cycle begins.

⇔ Obese female → Androgen to estrogen conversion increases → Estrogen proliferates endometrium → so endometrial cancer (high risk) it can lead to.

⇔ Pain during menstruation indicates ovulatory cycles.

⇔ Anovulatory cycles are always painless

(Pain → due to PGF2α from progesterone influence → Proge. from corpus luteum → CL if ovulation occurs)

Important MCQs

* Hormone which initiates ovarian cycle → FSH

* Levels of Estrogen needed for LH surge → 200 pg for 48 hrs

* Size of follicle just before ovulation → 18-20 mm

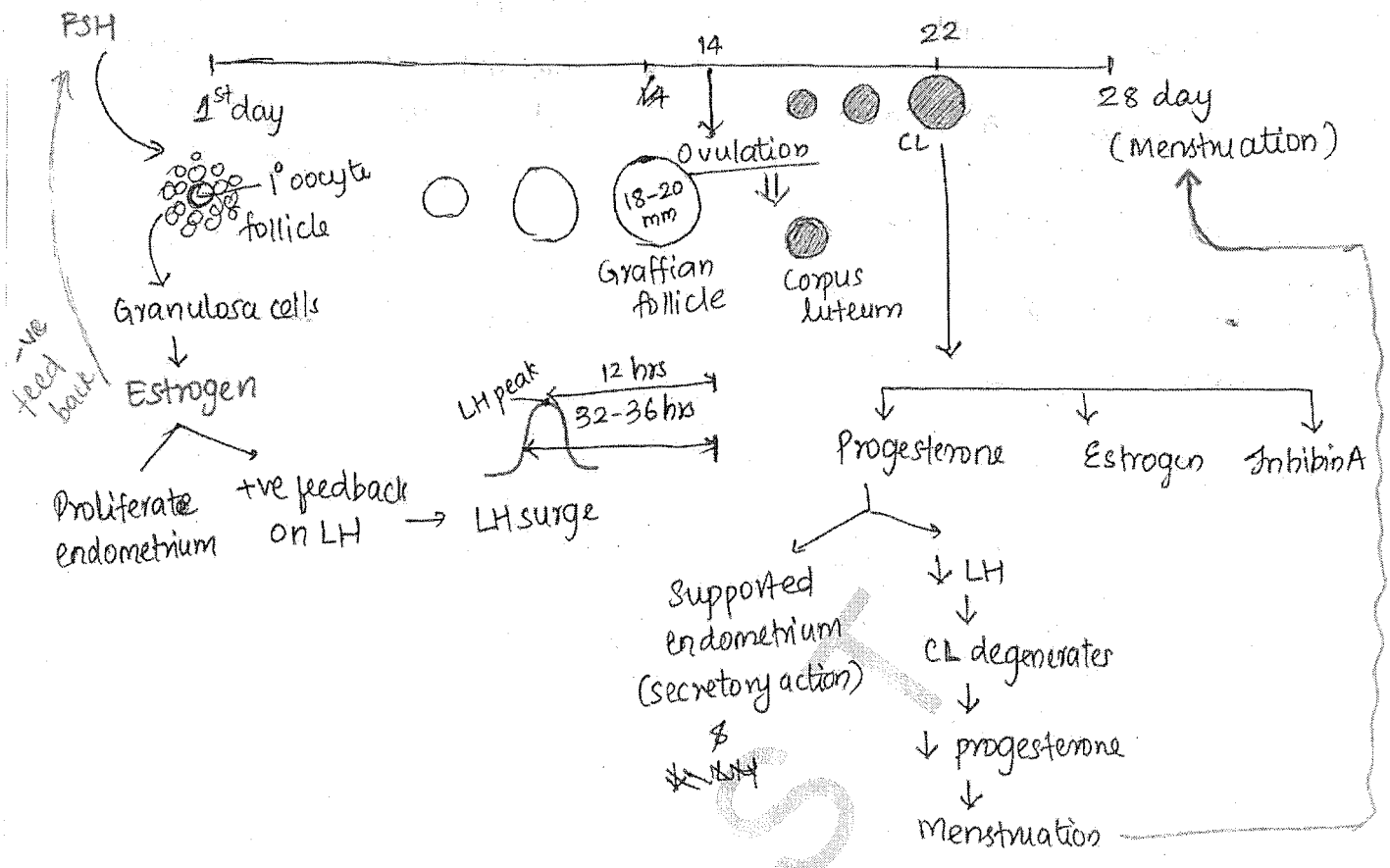
* Time interval b/w LH surge and ovulation is 32-36 hrs
(32-36 hr > 24-36 hrs)

* Time interval b/w LH peak & ovulation is 12 hrs

* 8 days after ovulation (22nd day) corpus luteum have maximum size & activity

Corpus luteum

- Corpus luteum in ♀ non-pregnant females is maintained by LH
- CL in pregnant females is maintained by hCG.
- So functionally LH & hCG are same
- Maximum activity & size of CL seen on 8th day after ovulation i.e, 22nd day
- Or maximum progesterone production is on 22nd day / 8 day after ovulation.
- Lifespan of corpus luteum in non-pregnant females is 12-14 days
- Lifespan of CL in pregnant ♀ 10-12 weeks.
(hCG has max. activity at 10 wks, after that it decreases)



MCQ continues

* Day of ovulation = Length of the cycle − 14
(Secretory phase always fixed, proliferative phase may vary)
(eg: 42 day cycle, ovulation occurs at 42-14 = 28 day)

* Menstrual blood is mainly arterial blood.

* Menstruation is shedding of the endometrium → mainly because of progesterone → but progesterone can only act on that endometrial on which estrogen has already acted.

* Estrogen proliferates the endometrium. So whenever estrogen alone is given to a ♀ it can lead to endometrial cancer.

* To prevent this estrogen should never give alone, always give it with progesterone.

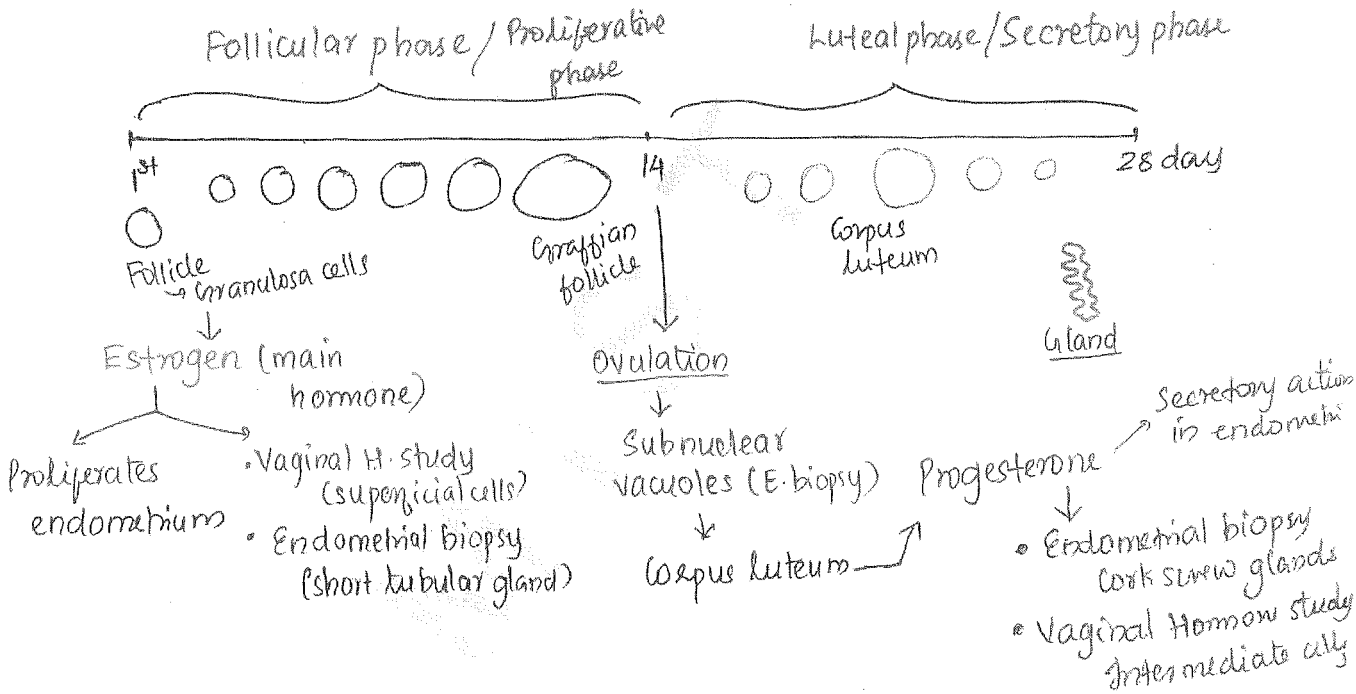
* Androgens are converted in to estrogen in adipose tissue.
∴ All hyperestrogenic conditions (endometriosis, endom. cancer, fibroid) are more common in obese females.

* Enzyme responsible for converting androgens to estrogen is Aromatase
* So any drug which inhibits Aromatase can be used for treating excessive estrogen conditions.

eg: Letrozole & Smoking

[Smoking is protective for endometrial cancer, endometriosis, fibroids]

* Endometrial biopsy on first half on menstrual cycle → short tubular glands
* On vaginal hormone study → in 1st half → superficial cells (pink)
* During ovulation → endometrial biopsy → Subnuclear vacuoles (First sign of ovulation)



* Endometrial biopsy on 2nd half → tortous glands k/a cork screw glands / saw tooth
* Vaginal hormone study during 2nd half → intermediate cells.
* Meosis I is resumed because of the hormone LH
* Meosis II is not hormone dependent

* Endometrium has 2 layer
  → Superficial layer
  → Basal layer.
* During menstruation superficial layer shed off, basal layer remains
* Basal layer is responsible for regeneration of endometrium in next cycle.
* Ovulatory cycle → Painful
* Anovulatory cycle → Painless

Painful menstruation

* k/a dysmenorrhea
* Can be 1° or 2°

Primary dysmenorrhea

* No cause of pain
* Seen in young ~~females~~ girls
* ♀ will complain the dysmenorrhea since menarche
* Management → NSAID's, OCP
  COCP → Makes cycle anovulatory

Secondary ~~dysmenorrhea~~ dysmenorrhea

* Pain due to pelvic pathology like endometriosis
* 30-35 age
* Earlier no dysmenorrhea but now it has started
* Mgt → Rx the cause.

Mittleschmerz syndrome

* In a few females pain occurs at the time of ovulation
* Complain of mid cycle pain
* This is k/a Mittleschmerz syndrome.

Characteristics of the menstrual cycle

* Length of menstrual cycle
  21-35 days
* Latest → 24-38 days
* Avg length → 28 days
* 28 day cycle present in
  15% females

* Cycle < 21 days → frequent menstruation → Polymenorrhea
* Cycle > 35 days → long cycle → Oligomenorrhea

↓

Regular cycle in polymenorrhea and oligomenorrhea.

* Amount of bleeding : 20-80ml
* < 20 ml → Hypomenorrhea
* > ~~80 ml~~ > 85 ml → Menorrhagia
* Average bleeding is -
  35 ml > 50 ml.

↓

Here also regular cycle.

* Hypomenorrhea is seen in Asherman syndrome
* Menorrhagia is seen in Fibroid

* Duration of bleeding : 2-7 days
* < 2 d → Hypomenorrhea
* > 8 day → Menorrhagia
* Irregular bleeding → Metrorrhagia
* Metrorrhagia is seen in Polyps.
* Menometrorrhagia → Irregular, excessive bleeding.
* It is seen in a fibroid polyp.

Hormones

Hypothalamus
↓ GnRH (pulsatile)
Pituitory
↓
LH & FSH
↓
Ovary
↓
Estrogen & progesterone

* All these hormones can be natural & synthetic.

GnRH

* Can be natural or synthetic

Natural	Synthetic
• Synthesised by arcuate nucleus of hypothalamus	• Preparations are Buserelin, Nafarelin
• Released in a pulsatile manner	• Always given intranasaly
↓ LH & FSH ↓ Estrogen ↑	• It can be given in a pulsatile manner/continously

* Synthetic GnRH, when pulsatile
↓
LH & FSH ↑
↓
Estrogen ↑
* Continous synthetic GnRH
↓
↓ LH & ↓ FSH
↓
↓ Estrogen.
* Uses of continous GnRH
1) Used in excessive estrogen conditions - like endometriosis, fibroids, precocious puberty

mia
* DOC for precocious puberty
Continous GnRH
* Pulsatile GnRH → used in ↓ estrogen in body → used in delayed puberty ovulation induction

LH & FSH

Natural	Synthetic
• By ant. pituitory	• Obtained from urine of postmenopausal ♀ & k/a HMG (human menopausal gonadotropin)

* HMG has 75 IU of LH & FSH
* Chances of multiple pregnancy with HMG → 30%

Estrogen (C_{18} compounds)

Natural (C_{18})

* E_1 - Esterone
  E_2 - Estradiol
  E_3 - Estriol
  E_4 - Estetrol
* Most potent is E_2
* Second - E_1
* 3rd → E_3
* Least potent → E_4

MCQ $[E_2 > E_1 > E_3 > E_4]$ (order of potency)

* E_2 is main homone during reproductive age.
* Source: Granulosa cells.
* E_1 is the estrogen produced in the adipose tissue when androgens are converted in to estrogen.
* E_1 is the main form of estrogen during menopause

MCQ
E_2 : Reproductive age
E_1 : Menopause.

* E_3 & E_4 formed by placenta during pregnancy.

MCQ * MC type of estrogen during pregnancy → E_2

MCQ * Most specific estrogen during pregnancy → E_3

Synthetic

* Ethinyl estradiol (MC in OCP)
* Diethyl stilbesterol (DES) ↓ (Not used these days) (Due to many congenital malformation)

DES
- Congenital malformation
- Ectopic pregnancy
- Abortion

Progesterone

Natural (C_{21})

Synthetic (C_{19})

* Androgens are also C_{19}
  ∴ Synthetic progesterone have androgenic side effects
* 4 generations of synthetic P.
  1) First
  2) Second
  3) Third
  4) Fourth
* As the generation of progesterone increases, androgenic s/e decreases (hirsuitsm, balding)

MCQ * Least androgenic s/e seen in Third generation progesterone
- ✓ → Desogestrel
- ✓ → Norgestimate
- ✓ → Gestodene

MCQ * Fourth generation progesterone are anti-androgenic.
- ✓ eg: Spironolactone
  Cyperoterone acetate

Estrogen

- Source : Granulosa cell, Theca cells, Corpus luteum, Placenta
- Effect on uterus:
  - Proliferates the endometrium
  - Responsible for growth of non-pregnant uterus
- Effect on cervix:
  - Cervical mucus watery, copious, elastic, can be stretched b/w fingers k/a Spinbarkeit
  - Under microscope, fern like appearance due to ↑Na, ↑Cl, ↑estrogen.
- Effect on vagina
  - Superficial cells
- Effect on lipid profile
  - Cardioprotective (~~↑LDL, ↓HDL~~) (↑HDL, ↓LDL)
- Other effects
  - ↑ bone mass
  - ↑ coagulation factors

Progesterone

- Source : Corpus luteum, placenta
- Effect on uterus:
  - Secretory action
  - Responsible for growth of pregnant uterus.
- Effect on cervix
  - Cervical mucus is thick, scandy, non elastic, cannot be stretched, breaks on stretching k/a Tack
  - No fern like appearance in microscope.

MLA

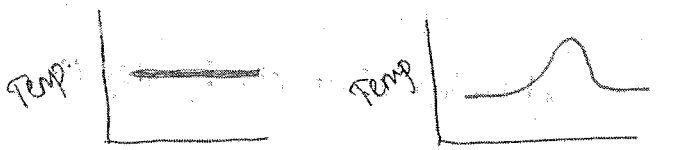
*Ferning disappears by day 18 of cycle

- Effect on vagina
  - Intermediate cell
- Effect on lipid profile:
  - ↑~~NO~~ LDL, ↓HDL
- Other effects:
  - ↑ basal body temp. by 0.5°C
  - Smooth muscle relaxant

Test for knowing whether ♀ is ovulating / not

* Easiest test → Serum progesterone level
* Best test / MC used → Follicular monitoring
* Every day follicle grows by 2 mm ~~per day~~ (2mm/day)
* In a female, ovulation has occured or not can be known by studying the follicle by doing TVS (trans vaginal USG) → Follicular monitoring
* Done from day 10 onwards
* Every day follicle will grow till they become 18-20 mm size
* Then size of follicle suddenly ① decreases

* Fluid is seen in pouch of
② Douglas.
* Endometrium appears triple layered
③ → Trilaminar appearance
* These 3 are signs of ovulation on follicular monitoring.
* Gold standard test for ovulation is Laparoscopy.
* Other tests → Basal body temperature.
(↑ temp. in mid of cycle.



(Non ovulated) (Ovulated)

* Cervical mucus study (♀20-22)

ferning	no ferning
↓	↓
non ovulated (estrogen)	ovulated (progesterone)

* Endometrial biopsy → done 1 wk before menstruation.
⇒ Coiled gland – ovulated
⇒ Tubular gland – Not ovulated
* Vaginal epithelial study (♀20-♀22)
⇒ Superficial cell – Not ovulated
⇒ Intermediate cell – Ovulated
(from lateral wall of vagina)

PCOD

* Polycystic ovarian disease / polycystic ovarian syndrome.
* Also k/a Stein Leventhal syndrome.
* There is excessive production of androgens from the ovary.
* In females → 2 sources of androgens → Ovary & Adrenal gland.
* DHEA sulphate is produced exclusively by adrenal gland
* Rest all androgens are produced both by ovaries and the adrenal gland.
*

PCOD
↓
Excessive androgens from the ovary
↓
Hirsuitism | Male pattern baldness | ↑LDL, ↓HDL
↓
future: Heart disease

* PCOD females are usually obese.
* In adipose tissue, these androgens will be converted in to estrogen (E_1) → ↑ E_1
* Normally $E_2 : E_1 = 2:1$
In PCOD, $E_2 : E_1 = 1:2$

Androgen
↓
In adipose tissue converted to estrogen (E_1)
↓
Excessive estrogen

-ve feedback on FSH (FSH ↓)

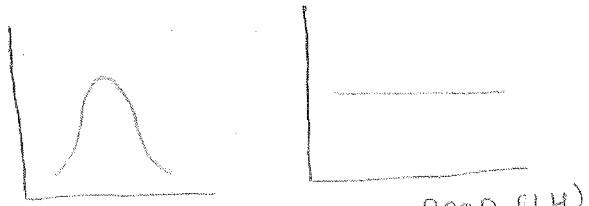
+ve feed back on LH (LH ↑)

↑ endometrial proliferation
↓
In future :
* Endometrial cancer
* Breast cancer
* Ovarian cancer ±

* Normally, FSH : LH = 2 : 1
In PCOD, FSH : LH = 1 : 2 / 1 : 3

* Levels of LH & FSH are checked on day 2 / day 3 of the cycle.

Excess estrogen
↓
+ve feedback on LH
↓
Levels of LH continously raised.



↓
in PCOD no LH surge
but levels of LH are raised

No LH surge
↓
Anovulation
~~Raised LH~~

* Raised LH
↓
Theca cell hypertrophy
↓
↑ Androgens

* Anovulation :
- Infertility (easily treatable or reversible)
- 1° oocyte not getting converted to 2° oocyte.
- Follicles gets converted to a cyst → Multiple cysts in the ovary

* Multiple cysts in ovary → On USG → arranged around the periphery of ovary → giving a necklace appearance.

* follicle converted to cyst → so no corpus luteum → ↓ progesterone levels → Oligomenorrhea / Amenorrhea

* But excessive estrogen → proliferation of endometrium → proliferated endometrium is weak & no progesterone to support → leads to menorrhagia / menometrorrhagia (irregular excessive bleeding) (or menorrhagia can be there) (endometrium keeps on proliferating but one day shedoff due to lack of support)

* Other features of PCOD:

- Insulin resistance is seen → darkly pigmented skin in nape of neck, axilla → K/a Acanthosis Nigricans
- Also due to insulin resistance → in future – Diabetes mellitus
- HAIRAN syndrome in PCOD

H, A	Hyper androgenism
I, R	Insulin resistance
A, N	Acanthosis Nigricans

- Metabolic X-syndrome can be associated with PCOD

Que: Can ovaries be normal in PCOD → Yes, because out of 3 only 2 need to be present. (R. criteria)

Que: Is LH/FSH ratio used for diagnosing PCOD → No only rotterdam criteria follows.

Que: Is obesity a criteria for PCOD → NO (Not in Rotterdam criteria)

Que: Can PCOD seen in thin ♀ → Yes

Criteria for diagnosing PCOD

ROTTERDAM CRITERIA

Out of following 3, any 2 should be present to diagnose PCOD

1) Ovulatory dysfunction manifested by ~~hypo~~ oligomenorrhea / amenorrhea.
2) ↑ androgen levels, manifested by clinically – hirsuitism, or biochemically – ↑ testosterone.
3) If USG criteria says
   - NO: of follicles ≥ 12
   - size of follicle/cyst < 10 mm
   - Volume of ovary ≥ 10cc.

   (Important)

These should be present in one or both the ovaries.

Hirsuitism

* 4 major causes of hirsuitism.

(1) Idiopathic hirsuitism → female is having hirsuitism but androgen levels are normal.

(2) PCOD → Rotterdam criteria → Also androgen levels are raised but always < 200.

(3) Testosterone / Androgen producing tumor of the ovary. Androgen levels are raised but always > 200.

(4) Late onset congenital adrenal hyperplasia. (CAH) Normally in CAH → ambigious genitalia → but in late onset CAH → genetalia are normal, ♀ have hirsuitism.

↓

Test: ↑ level of 17 hydroxy progesterone.

mcq

* MCC of hirsuitsm in young ♀
  Idiopathic hirsuitsm

mcq

* MC pathological cause of hirsuitsm in females:
  PCOD

mcq

* MCC of rapid onset hirsuitsm is Masculanizing tumours of ovary (Androgen secreting tumor)

Management of PCOD

* 1st step → Weight loss
* Management depends on problem which the patient is coming.

① * Insulin resistance → Metformin
  * Metfomin:
    - MC s/e: GI upset
    - Most dangerous s/e: Lactic acidosis
    - No teratogenecity → so can be used in pregnancy (no congenital malformation)

② * Obesity → Life style modification

③ * Irregular cycles → OCP
  (No E & P in body → OCP have both → take for 3 wks → i.e, 21 days → stop → sudden fall in P → menstruation)

④ * Hirsuitism → OCP in which 3rd & 4th generation progesterone
  (P has -ve feedback on LH)

Management of infertility in PCOD

* It is due to anovulation → give ovulation inducing drugs.
* First line drug is Clomiphene citrate (SERM → Selective estrogen receptor modulator)
* Second line drug:
  1) Clomiphene + Metformin (for obese ♀)

     Clomiphene + Bromocryptine (if prolactin ↑ in ♀)

  2) HMG (human menopausal gonadotropin) (Synthetic LH & FSH) (obtained from urine of postmenopausal ♀)
* Third line drug:
  Synthetic GnRH
  ↓
  In a pulsatile manner

Que: Nulliparous cervix : body uterus ratio

a) 50:50
b) 60:40
✓c) 30:70 (1:2)
d) 70:30

Que: When is the time for ovulation

a) 1 & d after menstruation
b) ~~1 & d~~ Along with LH surge
c) ~~1 & d~~ 1wk before menstruation
✓d) 2 wk before next menstruation

(LH surge duration: 32-36 hr)

Clomiphene

* It is an SERM (Selective Estrogen Receptor Modulator)
* It inhibits estrogen
  ↓
  -ve feedback on FSH lost
  ↓
  FSH ↑
  ↓
  ↑ No. of follicles grow
  ↓
  leads to multiple pregnancy.
* This means for Clomiphene to act HPO axis should be normal.
* It is given D_2-D_6 / D_5-D_9 (D → day)
* Initial dose → 50 mg/day
* Maximum dose → 100 mg/day
* Then from D_{10} start monitoring follicles by doing follicular monitoring
* When follicle size reaches 18-20 mm → give injection hCG (similar to LH (ovulation trigger)) → after 32-36 hours → Ovulation occurs. (MCQ)
* S/e of Clomiphene
  1) Multiple pregnancy (< 10% ≈ 5-8%) (MCQ)
  2) Most dreadful s/e is ovarian hyperstimulation syndrome. (< 1%) (MCQ)
     (huge follicles in USG)
  3) ↓ estrogen → menopausal symptoms (eg: osteoporosis)
  4) ↑ ovulation → ↑ ovarian cancer
  5) Due to which complication, its use should be immediately stopped - Visual symptoms.
* Maximum Clomiphene should be used for 12 months.

Long term complications of PCOD or Anovulation

1) Heart disease
2) Endometrial cancer
3) Breast cancer
4) Ovarian cancer (±)
5) Diabetes mellitus
6) Sleep apnea syndrome } (due to obesity)
7) Psychiatric problems
8) Metabolic X syndrome
9) Non alcoholic steatohepatitis

Note

* Can osteoporosis be a long consequence of PCOD (MCQ)
  No (it ↑ estr → ↑ bone mass)
* Chances of multiple pregnancy with HMG > 30%. (MCQ)
* Chances of ovarian hyperstimulation syndrome with HMG 5% (MCQ)

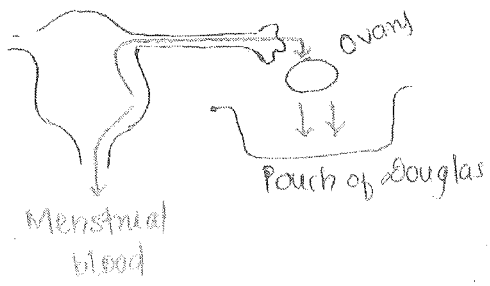
Endometriosis

* Occurence of endometrial tissue (glands + stroma) outside the uterus
* Sites :
  - MC in ovary (in the form of chocolate cyst / endometrioma)
  - 2nd MC site is pouch of Douglas
  - Can occur anywhere in the body
    - Uterosacral ligament
    - Lungs
    - Pleura
    - Colon.
  - Can occur in scars of certain surgeries, then it is k/a scar endometriosis
    eg: In C-section scar
    Myomectomy scar

Theories related to endometriosis

MCQ

* MC accepted theory is Sampson theory of implantation / Theory of retrograde menstruation.



* Other theories are
  - Coelomic metaplasia theory by Meyer & Ivanoff
  - Metastatic theory given by Halban

Risk factors for endometriosis

- It is due to ↑ estrogen → so MC in obese females & also in nulliparous females
- Age group : 25-35 years
- Higher socio-economic ~~factor~~ status.

(Note ⇒ During pregnancy endometriosis is relieved → bcoz ↑ progesterone → decidualization of endometrial tissue.) ∴ Progesterone can be used in its management.

- Early menarche & late menopause

Protective factors

* Exercise
* Multiparity
* Pregnancy
* Smoking (Inhibit Aromatase)

Symptoms

MCQ

* MC is pain (pain in endometriosis is proportional to depth of lesion)

↓

Pain

- MC in 2° dysmenorrhea > chronic pelvic pain > dyspareunia (pain during intercourse)

* 2nd MC — Infertility
* Ovarian cyst (chocolate cyst / Endometrioma)

Management of pain

* Depends on whether mild / severe
* If mild / minimal disease → give symptomatic Rx → by NSAID's & OCPs
* If moderate / severe pain → reverse the disease pathology (due to excessive estrogen, so should ↓ estrogen ↓)

↓

Rx :- GnRH in a continuous manner
- Aromatase inhibitors (Letrozole)
- Danazol (anti-estrogenic) (But has androgenic s/e, so should not be used in young females)
- Gestrinone (is like Danazol)

(OC (but costly))

* First / Second line of drug → Progesterone (mostly used) (cheap)

(Decidualization by progesterone)

* 2nd MC complaint → Infertility
  → Because in endometriosis
  - Adhesions are formed which leads to tubal blockage

(mgt)
* Management → IVF (invitro fertilizat^{n})

* 3rd MC complaint is ovarian cyst (chocolate cyst) (or Endometrioma)

↓

On USG, ground glass appearance

* Mgt of chocolate cyst is Laparoscopy
* 4th MC presentation is menstrual problem. (excessive endometrial tissue → excessive bleeding)

mca
* IOC in endometriosis

Laparascopy

↓

- See chocolate cyst of ovary
- Blue / black lesions on the peritoneum k/a Powder burn appearance / Gunshot appearance.
- Nodules on uterosacral ligament k/a Cobble stone appearance.

Some other important points on endometriosis

* Endometriosis has genetic predisposition
* CA 125 levels are increased here
* Triad of symptoms in endometriosis
  - Dysmenorrhea
  - Dysperunia
  - Infertility

(No solid area → means cyst
if solid → tumour)

* Always in endometriosis, treat the symptom which she is coming.

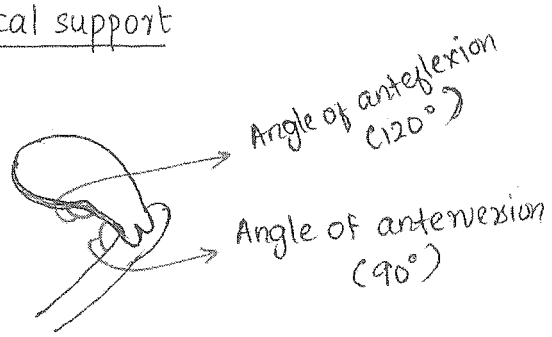
⇒ Danazol can be used but due to androgenic s/e not usually used

⇒ Mesoprost is not used.

Supports of uterus

1) Mechanical support
2) Ligaments which support uterus
3) Muscles which support uterus.

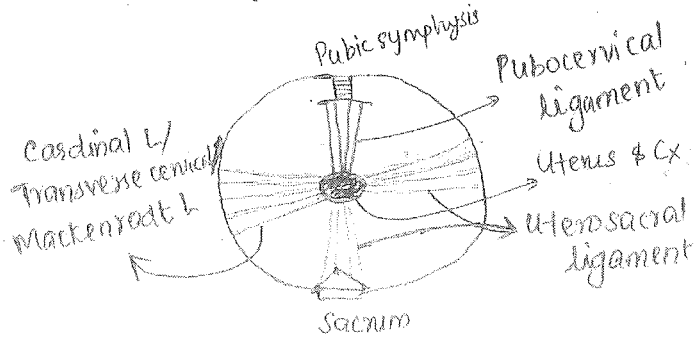
Mechanical support



* Angle of anteversion (b/w vagina & Cx) 90°
* Angle of anteflexion (b/w Cx & uterus) 120°
* Infront of uterus, bladder is there (acts as a support)
* But behind, no support
* So first step of ~~spx~~ prolapse is Retroversion

Ligaments

* Anteriorly pubic symphysis
* Posteriorly - Sacrum



1) Pubocervical L
2) Uterosacral L
3) Cardinal L / Transverse cervical L / Mackenrodt L

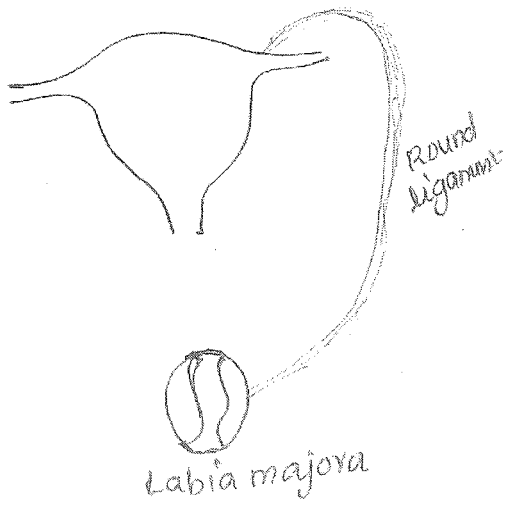
* Together called as triradiate ligament.

⊛ Most imp ligament support to uterus
Cardinal L > Uterosacral L

⊛ Ligament which prevents retroversion of uterus
Uterosacral ligament

⊛ Back pain in pt with prolapse is due to stretching of Uterosacral ligament.

⊛ Ligament, which does not have any role in supporting uterus
Broad ligament (actually peritoneum)



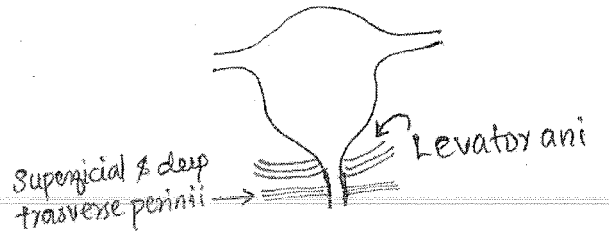
MEQ

⊛ Ligament which helps to keep the uterus in anteverted position
Round ligament
↓
Thus it indirectly supports the uterus

Que: All are supports to uterus except
a) Cardinal ligament
b) Uterosacral L
✓ c) Round L
d) Pubocervical L

Que: All are supports of uterus except
a) Cardinal L
b) Uterosacral L
c) Round L
✓ d) Broad L

Muscles



* Most imp muscle supports uterus
Levator ani
(which forms the pelvic diaphragm)

* Superficial & deep transverse perenii muscle which forms the urogenital diaphragm.
(major support)

* Other supports of uterus:
1) Bulbospongiosus
2) External urinary sphincter
3) External anal sphincter.

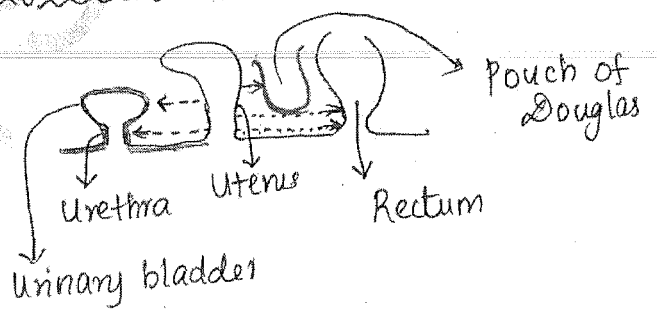
* Prolapse occurs when these muscles & ligaments become laxed
↓
It occurs in
- After menopause
- Repeated child birth

* So prolapse is MC in elderly multiparous females.

* If prolapse occurs in a young and nulliparous female k/a
↓
Congenital prolapse
↓
Risk factors for it are
- Spina bifida
- Ehler Danlos syndrome.

* In prolapse, it can be a vaginal prolapse or utero cervical prolapse

Vaginal prolapse



Vaginal wall prolapse
↓
Anterior wall — Posterior wall
↓
upper 2/3 (Cystocele) — Lower 1/3 (Urethrocele)

* Repair of both cystocele & urethrocele is done by
Anterior colporaphy

Posterior wall

- Lower 1/3 → Laxed Perineum
- Middle 1/3 → Rectocele
- Upper 1/3 → Enterocele

* Repair of laxed perineum & rectocele together k/a
  Posterior colpoperineorraphy
* Repair of enterocele is k/a
  Moscowitz repair / Halban repair

Uterine prolapse

* Earlier the classification used for uterine prolapse → Shaw classification
* Newer → POP-Q classification
  (Reference point is Hymen)
  (whether uterus lying above or below the hymen)

- Management : depends on
  - Age
  - Parity (No. of children)

- Young, nulliparous (Congenital prolapse) → Sling surgery / Cervicopexy.

Sling material ⇒ Mersilene tape.

Eg: of Sling surgery are
- Khanna sling Sx
- Shirodkar sling Sx
- Purandare sling Sx

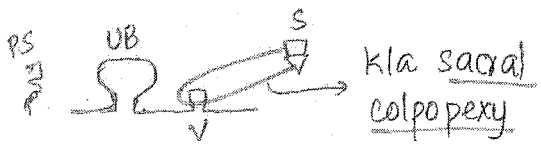
- Reproductive age female & she wants future child bearing
  Sling surgery.
- Female < 40 yr, who does not want children in future but wants to retain her uterus
  Fothergills repair / Manchester surgery.
  ↓
  * 2 important steps
    (1) Amputation of cervix [all complications are due to this step → it can lead to cervical stenosis & injury to internal os k/a cervical incompetence → so it is not done in ♀ who want child in future]
    (2) ~~Ligation~~ Plication of cardinal ligament
    In shirodkar modification of fothergills repair only this 2nd step is done (cardinal lig tied in front of uterus here)
- Female > 40 yr, don't want children, don't want uterus
  Ward Mayo Hysterectomy
  (Vaginal hysterectomy)

- Female ≥ 60 yr, Diabetes, HTN
  (cannot give spinal anesthesia)
  (or general anesthesia)
  ↓
  Le fort's colpocleisis
  (done under local anesthesia, stiching of ant. & post. wall of vagina)

- During pregnancy or immediately after child birth

  Ring pessary at the level of internal os.
  ↓
  - Temporary mgt
  - Should be changed every 3 months
  - s/e ⇒ Vaginitis
  - If it is forgotten can lead to vesicovaginal fistula.

⇒ Some times after hysterectomy (ut + Cx) the vaginal stump left behind also gets prolapsed, k/a Vault prolapse.



k/a sacral colpopexy

Note
* Colpo suspension is not a Sx for prolapse.
* It is Sx for stress urinary incontenence (SUI)

Other important points

- Decubitus ulcer if it is present on a prolapsed uterus → it is due to venous congestion.
  ↓
  Mgt - Acriflavin (antiseptic) + Glycerin packing (hydroscopic agent)
  (stuff this in to uterus, & return it to normal position)

⇒ Best method to prevent prolapse is advising perineal exercises/ Kegel's exercises during pregnancy and after child birth.

Menopause

* Age of menopause worldwide is 51 years.
  India → 47 yrs
* Menopause is defined as amenorrhea for > 12 months

Pathophysiology

* In menopause → ovarian failure → no more follicles → ↓ estrogen, ↓ androgens, no ovulation - so no progesterone → no menstruation
* Since anovulation → infertile.
* No hormonal predominance, so vaginal study → Basal & Parabasal cells.

* ↓ Androgen → ↓ libido
* ↓ progesterone → Amenorrhea.
* ↓ estrogen → negative feedback on FSH is lost → FSH level ↑ →
* ↑ FSH ≥ 40 IU is the diagnostic test for menopause.

* ↓ Estrogen → ↓ bone mass → Osteoporosis
* Diagnostic test for Osteoporosis is DEXA scan (Dual Energy X-ray Absorptiometry)
* ↓ Estrogen → ↓ cervical mucus → dryness of vagina k/a Senile vaginitis (not an infection)

* ↓ Estrogen → ↓ HDL & ↑ LDL → more chances of heart disease.

⊛ Most characteristic symptom of menopause is Hot flushes.
(sweating, tacchycardia, palpitation)

* During menopause → ↓ estrogen & ↓ androgen from ovary → but adrenals are normal → they secrete aadrogens → which in adipose tissue converted to estrogen (E1)
* So most common estrogen in menopause is E_1.
* Mgt : Hormone replacement therapy
↓
If uterus of female is intact
Estrogen + Progesterone
(Estro. alone leads to endometrial (a)
↓
If uterus is removed then
Only Estrogen.

Management according to symptom

* Osteoporosis
  - 1st line : Non homonal. Weuse Bisphosphonate (Alendronate, Pamidronate)
  - 2nd line : HRT
    - Uterus +nt → E+P
    - No uterus → Only E
  - In 2nd line also can give
    1) Tibolone (E+P+Androgen)
    2) Raloxifene (SERM) [s/e - Hot flushes]

* Hot flushes
  - 1st line : HRT
    - Uterus +nt → E+P
    - No uterus → E
  - 2nd line :
    - SSRI (Selective Serotonin Reuptake Inhibitor) Fluoxetine
    - Tibolone
    - But never use Raloxifene as s/e is hot flushes

* Senile vaginitis
  - Topical estrogen cream.

* ↓ Libido
  - Androgen

MCQ *

⇒ HRT is not useful for cardio-vascular diseases (CVS)

(it will ↑ CVS complication)

MCQ

⇒ Premature menopause:

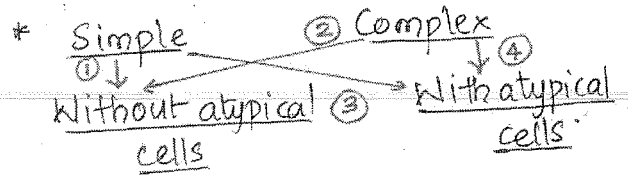
Menopause < 40 yrs age

⇒ ~~Cut~~ cut off level of estrogen for diagnosis of ovarian failure

20 pg/ml

⇒ Estrogen → hypercoagulability → so absolute C/I for thrombosis

ENDOMETRIAL CANCER



MCQs

* Simple* hyperplasia without atypical cells (1%)* (1) (Least chance for malignancy)

* Complex hyperplasia without atypical cells (3%)* (2)

(3) Simple HP with atypical cells (8%)*

(4) Complex HP with atypical cells (30%)* (Maximum chance of malignant)

* Simple HP without atypical cells also k/a Cystic glandular hyperplasia

* Patient complaint of excessive bleeding

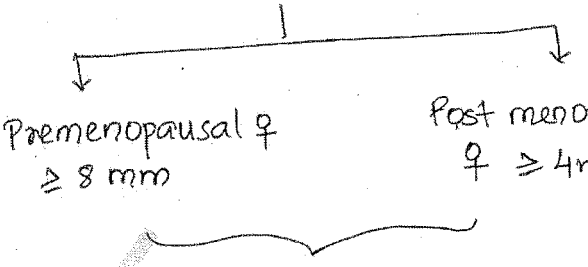
⇓

1st investigation (Ix) done

TVS (Transvaginal sonography)

↓

On TVS look at endometrial thickness



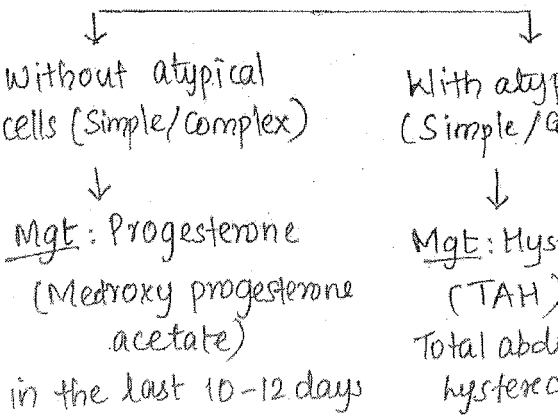
Suspect endometrial hyperplasia / cancer

↓

Endometrial biopsy and send it for histopathological examination.

↓

On report, endometrial hyperplasia



Types of hysterectomy & structures removed

(1) Simple hysterectomy:

(Total abdominal hysterectomy -TAH / Type I hysterectomy)

* Remove Uterus + Cervix
* MC done hysterectomy
* Done for all benign causes like fibroids.
* If ♀ >40 yr, along with TAH, both the ovaries & tubes are removed (Bilateral Salpingo oophrectomy)

TAH + BSO

↓

k/a PAN HYSTERECTOMY

* If ♀ <40 yr, ovaries are not removed unless ovaries are diseased

(2) Type II hysterectomy / Wertheims hysterectomy / Modified radical hysterectomy.

* Remove → TAH + BSO + 1 cm vagina + medial half of cardinal ligament + medial half of utero-sacral ligament + half of uterine artery (after it gives origin to the ureteric artery)

* MC done hysterectomy for female genital tract malignancy

(3) Type III hysterectomy / Radical hysterectomy

* Do TAH + BSO + 2cm of vagina + entire cardinal ligament + entire uterosacral ligament + entire uterine artery.

Mcqs

⇒ ~~Uterer~~ Ureteric injury is MC with which type of hysterectomy

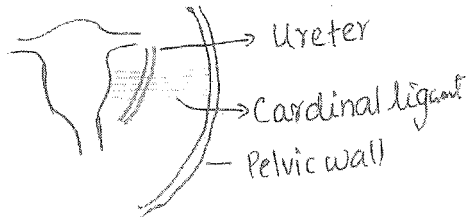
TAH (MC done hysterectomy)

⇒ Maximum risk of ureteric injury

Type II / Wertheim's H

⇒ M/C site of ureteric injury while doing hysterectomy.

- 1st → where it is crossed by uterine artery (water under bridge)
- * 2nd → Pelvic brim.



Risk factors of endometrical cancer

(Due to excessive estrogen)

Family → Familial risk

Has → Hypertension

OOO

O → Obesity

L → Late menopause & early menarche

D → Diabetes

A → Atypical endometrial hyperplasia

U → Unopposed estrogen

N → Nulliparous females

T → Radiation Therapy & Tamoxifen therapy

I → Infertility

* Familial risk of endo. Ca is associated with HNPCC (Hereditary Non Polyposes colon cancer)

* Unopposed estrogen → like HRT, estro. secreting tumour of ovary, PCOD.

* Since more common in diabetes, HTN, obese & → there are ↑ chances of endometrial cancer → k/a Corpus Cancer Syndrome.

Protective factors

1) Exercise
2) Smoking
3) Multiparity
4) OCP
5) Pregnancy

Note

* 2 cancers which are estrogen dependant:
  1) Ovarian cancer
  2) Endometrial cancer
* So both will be common in
  → Obese &
  → Nulliparous
  → Early menarche & late menopause
  → Familial inheritance
* Cancer cervix is MC in
  ✓ Multiparous
  ✓ No role in early menarche, late menopause
  ✓ No role of familial inheritance

x——————x

* MC age group of endo. Ca
  5th – 7th decade
* MC variety of endo. Ca
  Adenocarcinoma / Endometroid variety
* MC malignant variety
  Clear cell carcinoma > Papillary serous tumour
* MC route of spread
  Direct
* MC complaint is
  Irregular vaginal bleeding > Post menopausal bleeding.
* Most specific complaint is
  Postmenopausal bleeding.

Patient complains about post-menopausal bleeding

↓

1st Ix: TVS

↓

On TVS if endometrial thickness in postmenopausal ♀ ≥ 4 cm

↓

Suspect endometrial cancer

↓

IOC: Endometrial aspiration Biopsy

(OPD procedure, no anesthesia is needed for this)

↓

Gold standard Ix: Fractional curettage

↓

Once cancer is confirmed do staging.

*

* In case of both postmenopausal bleeding & endometrial cancer

↓

have same Ix, IOC, Goldstandard

MCQs

* MCC of post menopausal bleeding - Endometrial Atrophy / Senile endometritis
* MC cancer causing post. menopausal bleeding - Endometrial cancer
* MC cancer causing post. MB in India Cancer Cervix.
* % of post. MB patients have endometrial cancer → 10%

Staging

* In endometrial cancer, it is surgical staging.
* ie, remove the uterus + cervix + tubes + ovaries (>40yr) + enlarged lymphnodes and send it for histopathological examination (TAH + BSO + LN)
* If while doing surgery, cancer is seen to spread to cervix, then instead of doing TAH + BSO, we do Werthime's hysterectomy.
* In Obs & Gynae, staging followed is FIGO staging (Federation of International Obstetricians & Gynaecologists)
* Acc
  - = Stage I → Cancer limited to uterus
    - Ia: <50% myometrium involved
    - Ib: >50% myometrium involved
  - = Stage II → Cancer to uterus + cervix (stroma of cervix is involved)
  - = Stage III → Spread beyond uterus & cervix
    - IIIa: + Tube & Ovary (Uterine serosa also)
    - IIIb: Vagina involved
    - IIIc: LN involved
    - IIIc1: Pelvic LN
    - IIIc2: Para-aortic LN

= Stage IV : Metastasis

IVa → Urinary bladder or rectum

IVb → Distant metastasis or inguinal LN involved.

* Each stage has 3 grades :
* Grade I → Well differentiated tumor
* Grade II → Moderately differentiated
* Grade III → Poorly differentiated.

■ Mgt of endometrial cancer

* Already Sx is done for staging
* So mgt is post operative / post surgical management.
* Post operative mgt of choice is Radiotherapy

mcq Except in stage IA, Grade I & II where no post-operative Mgt is needed.

* So stage & management :

(1) Stage IA, G I & II → TAH + BSO, no post. Sx mgt

(2) Stage IA, G III → TAH + BSO, Radiotherapy (RT)

(3) Stage IB → TAH + BSO + RT

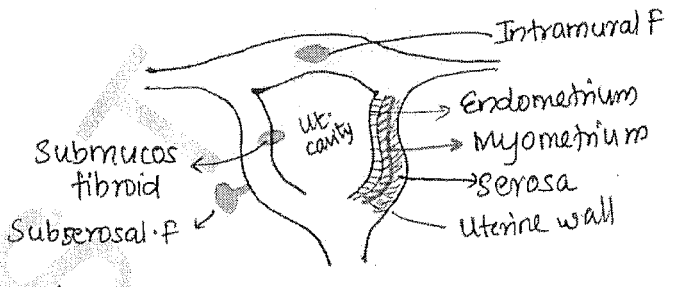
(4) Stage II (Cx) involved → Wertheim's H + RT

(5) Stage III → Wertheim's H + RT

(6) Stage IV → Palliative Rx.

FIBROID UTERUS

* Fibroid is mc pelvic tumor in ♀
* It is MC solid, benign tumor in ♀
* A fibroid can be uterine or extra uterine
* MC → Uterine
* In extra uterine → Cervical F & Broad ligament F
* Cervical F → can be anterior or posterior cervical F



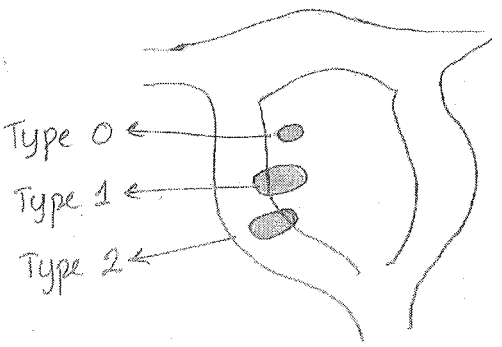
* Intrauterine fibroid :
* MC variety is Intramural F.

Submucous F

→ Type 0 : Entirely inside the uterine cavity

→ Type I : >50% inside uterine cavity, <50% inside uterine wall

→ Type III : <50% inside ut. cavity, >50% inside ut. wall



* Type 0 + Type 1 submucous F can be removed hysteroscopically.

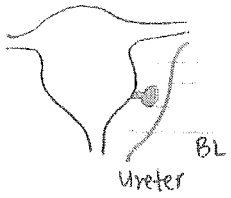
* Type 2 → Removed perabdominally. (cannot be removed by hysteroscopically)

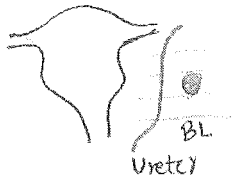
MCQ
que: Broad ligament fibroid is related to
✓a) Ureter
b) Uterine artery
c) IVC
d) Gartner's duct

Broad ligament F

Pseudo BL fibroid
[Uterine F grown in to the layers of broad ligament]
↓
Ureter is lateral to it

True BL fibroid
(Fibroid which originates from the broad ligament)
↓
Ureter is medial to it





MCQs

* MC variety of intrauterine F
Intramural / Interstitial F

* To begin with all F are intramural
→ it goes to uterine cavity
k/a submucous
→ it goes to peritoneal cavity
k/a subserous

* Fibroid is E+P dependent condition (E: Estrogen, P: Progesterone)

* Because it is Estrogen dependent M/C in nulliparous ♀ & the protective factor is smoking

* Age group: Reproductive age (35-45 yrs)

* Fibroid is MC in black ♀

* Familial inheritance is present

* OCP have no relation to fibroid

* MC presentation of F is asymptomatic.

* MC symptom of fibroid
→ Menstrual symptom
↓
ie, Menorrhagia

* If a ♀ has menometrorrhagia it means it is fibroid polyp.

* MC F to cause symptoms
Submucous F

* MC chance of abortion
Submucous F

* MC F to cause infertility
Submucous F

* MC F to undergo torsion is
Pedunculated subserosal F

* MC F to show calcification is
Subserosal F

* MC F to show urinary symptoms
Cervical F

Cervical F

Anterior CF → ↑ frequency of urination

Posterior CF → Urinary retention

* MC F to show inversion of uterus — Fundal fibroid
* MC symptom of fundal F is Menorrhagia (Not inversion)

* A fibroid is covered by a pseudo capsule.
* All the blood vessels which supply fibroid, they lie in that pseudo capsule.
* So most vascular part of fibroid is periphery of fibroid
* Least vascular part of fibroid is centre of fibroid.
* MCQ Because periphery is most vascular, calcification begins from periphery
* MCQ Since least vascular, degeneration begins from centre of fibroid.

Degeneration of Fibroid

* MCQ MC degeneration is Hyaline degeneration
* Least common change Malignant transformation

MCQs

* Malignancy of fibroid is rare (0.5%)
* MC fibroid to undergo malignancy Submucosal
* Fibroid malignant → Leomyosarcoma → ≥ 10 mitosis per high field.

Degeneration of fibroid

Red degeneration of fibroid

* Specific to pregnancy
* MC in 2nd trimester
* Pathology → Aseptic thrombosis in blood vessels which supply the fibroid → aseptic necrosis of the fibroid.
  (aseptic → no infection)
* Abdominal pain, nausea / vomiting, fever
  ↓
  Reactionary ↑ in TLC & ESR
* Fibroid appears salmon pink in colour.
* Mgt → Conservative Mgt (Analgesics, antiemetics)

MCQ Never do
- Give antibiotics
- Never do myomectomy
- Never terminate pregnancy

* IOC in fibroid → USG
* Differential diagnosis is:
  Adenomyosis
* Management (Mgt)

Mgt
- Mostly asymptomatic
  - Do nothing
  - But suspecting malignancy or
  - Pedunculated fibroid likely to undergo torsion
  - Surgery on the asymptomatic fibroid.
- Symptomatic
  - Mgt of choice is Surgery
    - Young, want pregnancy in future, complains abortion, or infertility → Myomectomy.
    - Elderly, completed her family → TAH
  - Refuses surgery or C/I to Sx

* If patient refuses surgery or C/I to Surgery then
  - Young → Medical Mgt
    - Drug which ↓ blood loss (no effect on size)
      - OCP
      - Progesterone
      - Tranexemic acid
    - Drug ↓ size of the fibroid
  - Old → ↓
    * If female is old → Uterine Artery Embolization → It is done by a radiologist via femoral artery.

Drugs ↓ size of F (E+P dependent)

* So ↓ estrogen:
  (1) GnRH continously
  (2) Danazol
  (3) Letrozole (Aromatase inhibitor)
* ↓ progesterone:
  - Progesterone Antagonist
    Mifepristone RU 486
  - Selective progesterone receptor modulator
    Ulipristal

Pneumonic
- U - Ulipristal
- Are - Aromatase Inhibitor
- Gyn - GnRH (continously)
- M - Mifepristone RU 486
- D - Danazol

Myomectomy
* Remove the fibroid not uterus
* Can be done
  - Laparoscopically
  - Abdominally
  - Vaginally
  - Hysteroscopically
    - Type 0 / Type 1 submucous F < 5cm

* Instrument used in myomectomy
  - ✓ (1) Bonney's myoma screw
  - ✓ (2) Bonney's myomectomy clamp.

Fibroid	Adenomyosis
	* Growth of endometrium inside myometrium * k/a endometriosis interna. * MC in elderly ♀
* symptom is Menorrhagia	* Menorrhagia & Dysmenorrhea
* Irregularly enlarged uterus	* Symmetrically enlarged uterus
* Size of the uterus ≥ 12-14 wk pregnant uterus (can reach uptill 20 wk preg. size)	* Size of uterus < 14 wk pregnant uterus size.
* Non tender uterus	* Tender uterus (k/a Halban sign)
* IOC : USG	* IOC : MRI
* ~~Gold standard~~	* Gold standard - histopathological examination
	* Mgt - TAH + BSO

PUBERTY

In females

* First sign is growth sput (↑ skeletal growth)
* First visible sign is breast budding ~~Fetarche~~. (Thelarche)
  ↓
  Then appearance of pubic hair (Pubarche)
  ↓
  Peak height velocity
  ↓
  Menarche
* Breast budding : Secondary sexual characteristics
* Main hormone responsible for puberty in females is Estrogen

  Estrogen
  ↓
  - ↑ breast development
  - ↑ height

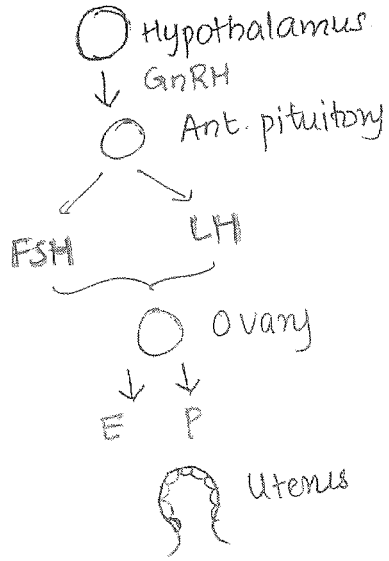
* In case of ♀ → for development of pubic hair & axillary line
  ↓
  hormone responsible : Androgens
* Breast development & pubic hair deve. occurs in stages which is defined by Tanner.

Stage 1 } Less development
Stage 2 }
Stage 3
Stage 4 } Properly developed
Stage 5 }

* Age of puberty :
  ♀ → 10.5 yr
  ♂ → 11.5 yr
* Precocious puberty :
  ♂ → < 9 years
  ♀ → < 8 yrs
* Precocious puberty is MC in ♀
* MCC of precocious puberty in ♀ is idiopathic.
* DOC for precocious puberty
  Continous GnRH.
* In males, first sign of puberty is Testicular growth
* Delayed puberty in
  - ♂ → No testicular growth by 14 yrs
  - ♀ → No secondary sexual characters by 13 yr
* Delayed puberty
  ↓
  MC ⟹ Boys.
  ↓
  MCC ⟹ Constitutional delay.
* Delayed puberty, DOC in ♀
  Pulsatile GnRH

Amenorrhea

* Absence of menstruation.
* Can be 1° or 2° amenorrhea
* 1° amenorrhea → patient has never experience menstruation
  ie, no menarche by 13 yr in absence of secondary sexual characters (OR) (by 13 yr = ⩾ 14 yr) by 15 yr age irrespective of presence or absence of 2° sexual characteristics.
* For menstruation to occur :



* for normal menstruation, need normal HT, P, O, U.
* Any problem in them → Amenorrhea

Uterus (related causes)
- Mullerian agenesis (46 XX)
- Testicular feminizing syndrome or Androgen insensitivity syndrome (46 XY)

Ovary

- Turners syndrome (45XO)
- Pure gonadal dysgenesis (46XX)
- Swyers syndrome (46XY)

Ant. pituitory

⇒ Craniopharyngioma

Hypothalamus

* Kallman syndrome.

⇒ MCC of 1° amenorrhea
Turner's syndrome (45XO)

⇒ 2nd MCC of 1° amenorrhea (46XX)
Mullerian agenesis (MRKH)

⇒ 3rd MCC (46XY)
Testicular feminising syndrome

⇒ Best Dx for 1° amenorrhea
Karyotyping

⇒ Best Dx for 2° amenorrhea
Hormonal study.

Mullerian agenesis / MRKH syndrome

* Karyotype is 46XX
* Gonads – Ovary (developed from genital ridge)
↓
So normal ovary
↓
Normal Estrogen
↓
Normal 2° sexual characteristics.

* They cannot menstruate because both Mullerian ducts absent
(FT, U, Cx, upper V absent)
* Since no uterus → 1° amenorrhea
* ♀ with normal 2° sexual characteristic will complain of 1° amenorrhea
* If along with Mullerian agenesis, patient has renal anomalies in which
MC – Renal agenesis > ~~Horses~~
Horse shoe shaped kidney
↓
Also have skeletal anomalies
(MC – cervical spine
↓
Condition is called MRKH syndrome
(Mayer Rokitansky Kuster Hauser syndrome)

Mgt of Mullerian agenesis

* Can never menstruate (no uterus)
* Make them capable of getting married by doing a vaginoplasty
* Vaginoplasty (MCQs)
  - Best time → Just before or just after marriage
  - Technique → Mc Indoe vaginoplasty
  - Latest → Laproscopic Vecchetti surgery

MCQs

* They cannot become pregnant
* But can have their own biological child because normal ovulation

↓

Take ova by IVF

↓

Surrogacy

MCQ

* If ♀ comes with C/O 1° amenorrhea with ~~absence of~~ & normal 2° sexual characteristics.

(1) If uterus is present then
Imperforate hymen / Cryptomenorrhea

(2) If uterus is absent then
- MRKH syndrome
- Androgen insensivity syndrome.

Cryptomenorrhea

* Or imperforate hymen
* Patient menstruates normally but menstrual blood fails to come out as there is no opening in the hymen. → blood collects in vagina & cervix k/a hematocolpos → blood collects in uterus k/a hematometra.
* Patient C/O cyclical abdominal pain but no bleeding.
* On examination → hymen is bluish, bulging and tensed.
* Per-abdominally, a mass is present → it is hematometra (U+blood)
* Per-rectally (because per vaginal examn should not be done in virgin ♀ → uterus is present and is bulky.
* Mgt. ⇒ Give an incision on the hymen.

* ~~If 2° sexual absent~~

Androgen insensitivity syndrome.

* Also k/a Testicular feminizing syndrome.
* Inherited X-linked recessive disorder
* 46 XY → so gonads: Testis → but it will be inside the abdomen → so ↑ chances of malignancy & present as inguinal hernia →

Testis

↓

Sertoli cells → Mullerian inhibiting factor → MD absent, so no U, FT, Cx, upper V.

Leydig cells → Testosterone

* These ♂ have complete insensitivity to testosterone.
* So Wolfian duct structure absent (SEED -nt)
* ~~In adipose tissue~~, testosterone to estrogen.

* No testosterone → so female genatalia → so parents consider them as ♀ babies
* Spermatogenesis – nt.
* At puberty : Resistant to testosterone
  ↓
  (1) So 2° sex. chara. of ♂ will not develop
  (2) Testosterone → Estrogen in adipose → so proper breast development (Tanner's stage 4,5)
  (3) ♀ also need androgen/testosterone for pubic & axillary hair growth So they are less developed (Tanner's stage 1, 2)

MCQ
* ♀ c/o 1° amenorrhea, breast development ≈ Tanner stage 4/5 but pubic hair are absent, inguinal hernia +nt,
  ↓
  Complete Androgen Insensitivity syndrome.
* Differential diagnosis is MRKH

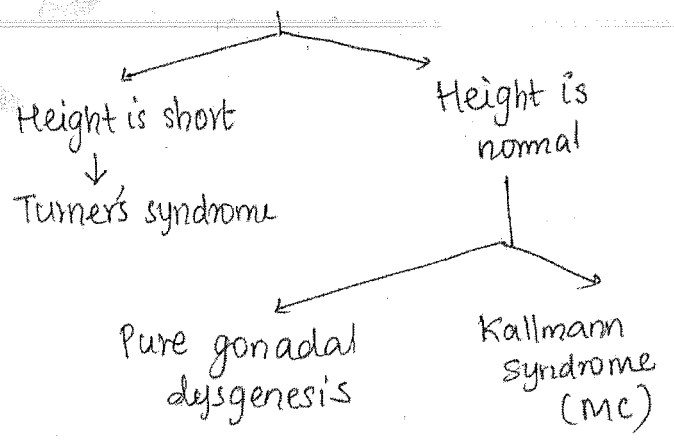
MRKH
- 46 XX
- Ovaries
- Testosterone N
- Barr body = 1 (no. of X – 1)
- Renal anomalies (in IVP)

AIS
- 46 XY
- Testis
- ↑ Testosterone
- Barr body = 0
- No renal anomaly

* Best test to differentiate both Karyotyping.

Mgt
* Let them be female
* Intraabdominal testes is removed → Gonadectomy (time: after breast development is complete = 14–15 yrs)
* Estrogen Replacement therapy
* Since no ovaries, cannot have their own biological child.
* Vaginoplasty is done just after/ before marriage.

MCQ
* ♀ c/o of 1° amenorrhea with absent 2° sexual characteristics



Turner's syndrome
* MCC of 1° amenorrhea.
* 45 (XO) → So gonads : Ovary
* But both X not +nt → so no proper dev. of ovary → streak gonad
* Streak gonad → ↓ estrogen and 1° amenorrhea, short stature.

* ↓ Estrogen → secondary sexual characters absent, less growth of uterus, C_x & vagina k/a hypoplastic (Hypoplastic uterus, HP Cx, HP vagina)

* Additional features:
  1) Webbing of neck
  2) Low post. hair line
  3) Widely placed nipple (as breast is absent)
  4) Shield chest (shaped)
  5) Short 4th metacarpal
  6) Cubitus valgus
  7) CVS anomalies (coarctation of aorta)
  8) Renal anomaly (MC- horse shoe shaped kidney)
  9) ↑ risk of autoimmune disorders (Diabetes / Hashimoto's thyroiditis)
  10) Normal IQ
  11) FSH ↑ as estrogen is less (-ve feedback lost)

* Mgt
  * Initially low dose estrogen to promote breast development for 1yr
  * ~~Followed 1 yr later by cyclical E+P~~
  * Then later on by cyclical E+P

Pure gonadal dysgenesis

* It is like Turners except
  1) Karyotype 46 XX
  2) Normal height
  3) Additional features of Turner's syndrome are absent

Kallmann syndrome

* Hypothalamic failure
* So ↓ GnRH → ↓LH, FSH → ↓ Estrogen → absent 2° sexual characteristics → ♀ c/o 1° amenorrhea → infertility.
* Along with all these they have anosmia (inability to smell)
* Also normal height.

Secondary Amenorrhea

* ♀ has been menstruating normally earlier but now for past 90 days (3 months) she have amenorrhea
* MCC → Pregnancy.
* Other systemic causes
  1) Thyroid disorders
  2) ↑ prolactin levels
  3) Renal failure
  4) Chronic anemia

* DOC for hyperprolactinemia
  Cabergoline
* DOC for ↑ prolactin in infertile ♀
  Bromocryptine
  (Also induce ovulation)

* Causes of 2° amenorrhea in
↓

Uterus
- Ashermann syndrome.

Ovary
- PCOD / Anovulation
- Premature menopause (< 40yr)

Pituitory
- Sheehan's syndrome
- Pituitory tumor - Prolactinoma

Hypothalamus
- Stress
- Anxiety
- Excessive diet & exercise (Anorexia Nervosa)
- Excessive eating - Bullimia nervosa

Ashermann syndrome

* Presence of intrauterine adhesion & endometrium is very thin (damaged)
* MCC → Post partum currettage,
* Other causes → Dilatation & Currettage (D & C), TB, Shistosomiasis
* C/o hypomenorrhea (less bleeding), 2° amenorrhea.
* DOC : Hysteroscopy
* Other Dx → HSG (Histo salphino graphy) ↓ (graphy)
Honey comb appearance

* Mgt ⇒
1) Hysteroscopic adhesiolysis +
2) Insert CuT to prevent + adhesion
3) Estrogen & Progesterone. (to proliferate endometrium)

Sheehan's syndrome

* Due to PPH → excessive bleeding → necrosis of ant. pituitory gland → k/a Sheehan's syndrome
* All hormones from ant. pitui. ↓
↓
- ↓ LH & FSH ⇒ So 2° amenorrhea
- ↓ prolactin ⇒ Failure to Lactate

Prolactinoma

* Prolactin secreting pituitory tumor.
* ↑ prolactin → ↓ LSH & FSH
- ↓ LSH & FSH ⇒ 2° amenorrhea
- ↑ prolactin ⇒ Galactorrhea (excess milk)
* Pituitory is near to optic chiasma → so tumor → visual symptoms & headache.
* Best Dx for 2° amenorrhea is Hormonal study

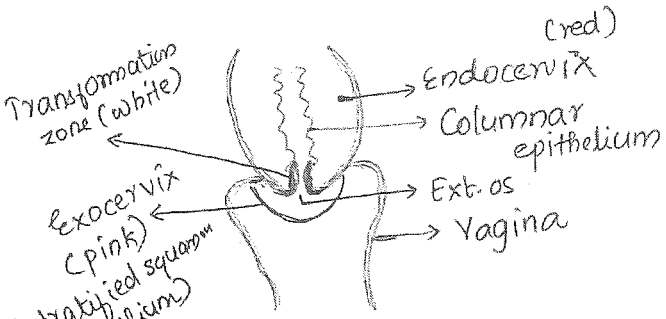
Homonal test

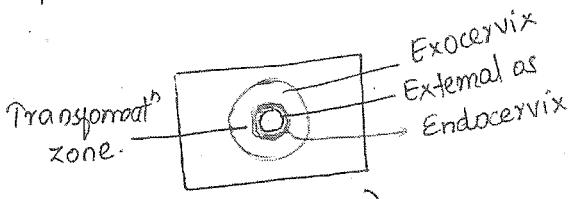
- TSH
- Prolactin
- UPT (urine pregnancy test)

* If 3 are normal
↓
then progesterone challenge test
(give progesterone for 7-10 days and withdraw)

- if bleeding +nt → PCOD / Anovulation
- if no bleeding → E+P challenge test.
  - give E+P like a normal cycle.
    - if bleeding absent → Asherman's syndrome (because we did work of HT, P & O by supplying hormones)
    - Bleeding +nt → FSH ≥ 40 IU then premature menopause → if FSH N/↓ → MRI → Pituitory / Hypothalamus.

CANCER CERVIX





(Trans-section)

* All ♀ the columnar epithelium of endocervix, it changes in to squamous epithelium of exocervix
↓
k/a metaplasia

* Metaplasia is
  - Physiological
  - All females
  - Not precancerous

* If infection occurs → most of the time the infection will clear → sometimes it persists → then it leads to disorderly metaplasia k/a Dysplasia.

* Dysplasia is
  - Pathological
  - In few ♀
  - Precancerous

* Dysplasia leads to CIN (Cervical Intraepithelial Neoplasia)

* CIN → to carcinoma insitu → cancer cervix (Ca Cx)

* Dysplasia → CIN → Ca insitu → Ca Cx

Took 15-20 years

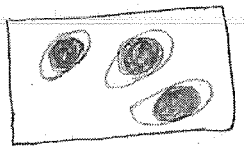
↓

So screening plays an important role in cancer Cx.

* No screening test for endometrial Ca & ovarian cancer.

Dysplasia

* Characteristics of dysplastic cells are



* ↑ nucleus : cytoplasm ratio (Big nucleus)

* If < 1/3 cells become dysplastic the k/a CIN I

* 1/3 - 2/3 cells dysplastic → CIN II

* > 2/3 cells dysplastic → CIN III

(Cells means cervical epithelium)

* If entire cervical epithelium becomes dysplastic

↓

if overlying epith. is intact → Cancer in situ

if overlying epi. broken → Invasive Ca Cx.

* Age group → ~~20-30 yrs~~

CIN : 20-30 yr

Ca insitu : 30-35 yr

Invasive Ca Cx ← 35-40 yr, 50-60 yr

* CIN : 20-30 yr (So screening by Papsmear begins at 21 years)

* Invasive Ca Cx → 35-40 (if it is continuation of Ca insitu)

* Inv. Ca Cx → 50-60 (if occurs not as a continuation)

CIN

* Age group : 20-30 yr

* Newer classification for CIN is Bethseda classification

↓

- CIN I k/a LSIL (Low squamous intra-epithelial lesion)
- CIN II, CIN III, Ca in situ, Ca Cx k/a HSIL (High squamous intra-epithelial lesion)

* Risk factors for CIN : Same like cancer cervix

* Screening method of choice

Pap smear

- Should do an universal screening (all ♀ should be screened)
- Age started → 21 yrs
- From 21 yr should do pap smear every 3 yr till she become 30 yr
- From 30 yr → do Pap smear + HPV DNA testing every 5 years till 65 yrs of age.

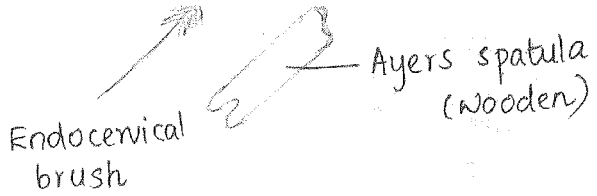
* If after 30 years, only Pap smear is being done after every 3 yrs
* HPV DNA testing begins from 30 years.

Some important points

* In HIV +ve females, annual screening is done.
* If a ♀ has been given cervical cancer vaccine → still screening should continue
* If in a ♀ hysterectomy has been done for a benign cause like fibroid → then screening can be stopped

Methods of doing Pap smear:

* Instrument used:



* Transformation zone is near to the external os
* Ayers spatula have a concave end → take sample from transformation zone.
* From the other end → take sample from posterior wall of vagina
* From the endocervix → sample is taken by endocervical brush

TZ	1st slide
EndoCx	2nd slide
PWV	3rd slide (control slide)

MCQs

* For cytological study/pap smear, sample is taken from
  Post. wall of vagina
  (Hormonal study: Lateral wall ✓)
* Fixative used:
  95% Ethyl alcohol + 5% ether
* Pap smear was initiated by
  Georgis Papinacolaou.
* For liquid based cytology, use
  Cervical brush / Cervical broom
* Fixative used here is ethanol or Methanol.

Report of Pap smear

1) Normal → Continue screening as usual.

2) Infections → Pap smear can detect a no: of infections like
   - HPV
   - Herpes
   - Trichomonas
   - Candida
   - Bacterial vaginosis (Gardnerella)

* But it cannot detect:
  - Gonorrhea
  - Chlamydia
* Give antibiotic & repeat Pap smear after 3-6 months.

3) ASCUS → Atypical Squamous Cell of Unknown Significance.

* Have 3 options
* (i) → Do HPV DNA testing
  ↓ if positive
  ↓ Do Colposcopy
  (ii) Directly do Colposcopy (Best)
  (iii) Papsmear after 6 months
  → if report is ASCUS or more than it → Colposcopy.

4) LSIL (Low squamous
5) HSIL

In both, if papsmear show LSIL/HSIL → Papsmear is only a screening test → So do a confirmation test: Biopsy
↓
In biopsy, call the patient & examine the cervix.

- if visible ulcer or growth +nt → Punch Biopsy
- if no visible lesion → Colposcopic guided biopsy / Colposcopy

* If there is an LSIL on Papsmear report:
  Mgt → Punch biopsy (visible growth)
  or
  Colposcopy (invisible growth)
  ±
  Endocervical curretage
* If there is an HSIL on Papsmear
  ↓
  Mgt: Punch biopsy or Colposcopy + Endocervical curretage

MCQ
- If MCQ nothing is mentioned, growth is visible or not → take it as "not visible" → answer it as colposcopy.

Colposcopy

* Colposcope is a magnifying instrument
* Minimum magnification → 5 times
* Maximum " → 30 times
* Focal length → 30
* With colposcope → Ectocervix, Vagina & ~~Vag~~ Vulva can be seen.
* But endocervix is not visualized by colposcopy.
* So only endocervical curretage is done for endocervix
* Colposcopy is an OPD procedure
* No anesthesia is needed.

Steps of Colposcopy

* With colposcopy → Cx is seen.
  Biopsy is taken from the –
  1) Any irregular area
  2) Nomal – pink in colour, so any pale area.
  3) Abnormal blood vessels like
     - ✓ Masaic pattern
     - ✓ Reticular pattern
     - ✓ Punctate

* Then apply 5% acetic acid to the cervix.
* Area with dysplasia appears white after applying 5% acetic acid → Acetowhite areas
* Entire ectocervix appears pink except dysplasia cells.
* White → because dysplastic cells have ↑ nucleus → ↑ nuclear protein → these proteins they coagulate in presence of acetic acid and appear white → Acetowhite areas
* From acetowhite area → take biopsy

⇒ Colposcopy report comes as CIN1, CIN2, CIN3, Ca insitu, Ca Cx

Clinical case

Pap smear report shows HSIL, but colposcopy says normal.

↓

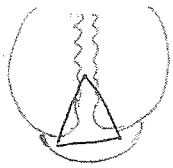
Colposcope cannot visualise the ~~endoscope~~. So endo cervix.
So the area of cancer will be in endocervix

↓

In this case do a Cone biopsy.

Cone biopsy

* It is both diagnostic as well as therapeutic
* Cone shaped area of cervix is cut.



* The cone should include endo Cx, TZ, Ecto Cz
* OT procedure done under the general anesthesia.

Indications of cone biopsy

Diagnostic

- Discrepency in report of Papsmear & colposcopy
- Unsatisfactory colposcopy → entire Tz is not visible on colposcopy
- Suspecting an adenocarcinoma of Cx
- Endocervical curettage is positive for cancer

Therapeutic

- In treating stage IA1 Ca Cx and cancer in situ in young females

Management of CIN:

* CIN 1 → Simply follow-up patient for 2 yrs. Normally CIN 1 regresses automatically by 2 years.
  ↓
  If it persists ≥ 2yr
  ↓
  Cryotherapy
  [Cold N_2O (nitrous oxide) or CO_2]

* CIN 2 / CIN 3 → At any age
  ↓
  Mgt is LEEP (Loop electro excisional procedure)
  or
  LLETZ (Large loop excision of transformation zone)

Cryotherapy

* Passing CO_2 / N_2O at very low temperature
* It is an OPD procedure
* Intracellular H_2O freezes and cells get destroyed.
* Long term s/e is watery discharge
* No bleeding.

LEEP / LLETZ

* Loop Electro Excisional procedure
* Large loop excision of Transformation Zone.

* Principle → Cut & coagulate simultaneously
* OPD procedure.
* No anesthesia needed
* No training needed to do this
* Both diagnostic & therapeutic
* Very less time (< 2 min)
* Very less / minimal bleeding.

Clinical cases

* 40 year old ♀ P_2L_2 (pregnant twice, 2 living children) on Papsmear - CIN 3. What will be the management?
  Since Papsmear only done
  ↓
  confirm with colposcopy

* 40 yr ♀ P_2L_2 on colposcopy, CIN 3. What is the Mgt?
  LEEP

* 45 yr ♀ P_3L_3 on colposcopy CIN 3. What is the Mgt?
  LEEP

* 45 yr old P_3L_3 on colposcopy CIN 3. What is the best Mgt?
  LEEP

(Always ans: to Mgt for CIN 2 & CIN 3 of colposcopy at any age group or any best or other Mgt
↓
Ans: is LEEP)

* Hysterectomy is never done for CIN except:
  - Recurrent CIN
  - Adenocarcinoma
  - Associated with any other pelvic pathology for which hysterectomy has to be done ~~except prolapse~~ like prolapse.

Cancer Cervix

* MC in low socio economic ~~stages~~ status.
* MC cancer in ♀ in India / Worldwide
  Breast cancer.
* 2nd MC in ♀ in India is
  Cancer cervix.
* MC ♀ genital tract cancer world wide.
  Ca Cx
* MC ♀ genital tract cancer in the developed countries
  Endometrial Ca.
* MC ♀ genital tract Ca in the developing countries:
  Ca Cx.

Risk factors for Ca Cx:

* Early age of 1st intercourse
* Early age of 1st child birth
* Multiparity
* Multiple sex partners
* Low socio economic status.
* All STDs
* Pre-invasive lesions like CIN

Not seen for Ca Cx

- Early menarche & Late menopause
- Familial inheritance.

⇒ Smoking is a risk factor of squamous cell carcinoma of Cx
⇒ OCP is risk factor for the adeno carcinoma of Cx.

* Most important risk factor is HPV infection.

* HPV infection (subtypes)
  - High risk subtypes
    - HPV 16, HPV 18 } 70% Ca Cx
    - HPV 31, HPV 33, HPV 45, HPV 52, HPV 58
    - ↓
    - Leads to CIN, Ca Cx, Vulval Ca, Vaginal Ca
  - Low risk subtypes
    - ↓
    - HPV 6, HPV 11
    - ↓
    - Leads to Genital Warts

Important questions on HPV

* MC HPV associated with CaCx
  HPV 16
* Most specific HPV associated c CaCx
  HPV 18
* MC HPV asso. c squamous cell Ca of Cx
  HPV 16
* MC HPV asso. c Adenocarcinoma of Cx
  HPV 18
* Viral proteins needed for the malignant transformation
  E_6, E_7
* Viral proteins needed for the replication of HPV
  E_1, E_2.
* Prevention of HPV / Ca Cx
  HPV vaccines
  (Gardasil & Cervirax)
* Gardasil ⇒
  - Quadrivalent
  - Active against HPV 6, 11, 16, 18
  - Protect against CaCx and Genital warts
* Cervirax ⇒
  - Bivalent
  - Active against HPV 16, 18
  - Protect against CaCx
* Dosing Schedule
  - G ⇒ 0, 2, 6 months
  - C ⇒ 0, 1, 6 months
* G → Can be given to both boys & girls (penile cancer)
* C → Only to girls.
* ~~Age group~~:

  Common for both G & C
  - Age group
    Ideal = 11-12 yr
    Can be given = 9-26 yr
  - C/I in pregnancy
  - s/e is syncopal attack.
    (ask to rest for 15 min)

Imp points on Ca Cx

* MC variety of Ca Cx
  Squamous cell carcinoma.
* MC site for Ca Cx
  Transformation zone
* Adenocarcinoma of Cx
  - Endocervix is site
  - OCP is a risk factor
  - Seen in young ♀
  - Associated c HPV 18
* MC symptom of Ca Cx
  Irregular vaginal bleeding
* Most specific symptom for Ca Cx
  Post coital bleeding.

Clinical case

1) 35 yr ♀ P_2L_2 with post coital bleeding. What is next step in Mgt?

↓

Next step will be Pap smear

↓

Only if Pap smear +ve then do colposcopy.

2) 35 yr ♀ P_2L_2 with post coital bleeding and 3×4 cm mass is seen on the anterior lip of Cx. Then next step in mgt?

Punch biopsy

(P.C bleeding + something visible)

* MC route of spread of CaCx
  Lymphatics
* MC site of metastasis
  Lymphnodes
* MC LN involved in Ca Cx
  Obturator LN
* Sentinel LN of Ca Cx is
  Presacral LN / Ureteric LN
* All LN which are involved in Ca Cx

  I
  H
  O
  P
  E

* LN not involved in CaCx
  Superficial inguinal LN

(so whenever Sup. Ingu. LN has involved → metastasis → stage 4)

* MC site for hematogenous spread of Ca Cx is
  Lungs
* Ca Cx rarely involves the ovaries
* So when hysterectomy is being done for young ♀ (30-35 yr) ovaries can be left behind.

Staging of Ca Cx

FIGO staging

* Clinical staging → do Ix & then stage Ca Cx.
* Many Ix recommended by FIGO for staging Ca Cx.
* Ix not recommended by FIGO
  - USG
  - CT
  - MRI
  - PECT
  - Laparoscopy

FIGO staging of Ca Cx

- Stage I → Cancer is limited to Cx
- Stage I_A : Microscopic
  I_B : Visible
- Stage I_{A_1} – < 3 mm deep
  I_{A_2} – 3–5 mm deep
- Stage I_{B_1} : Size of tumour < 4cm
  I_{B_2} : Size ≥ 4 cm.
- Stage II → Cancer has involved upper vagina (2/3)

(Collective name for ligaments of uterus: Parametrium)

- Stage 2A – without involving parametrium
  2B – with parametrium
- Stage $2A_1$ → Size of Tm < 4cm
  $2A_2$ → Size of Tm ≥ 4cm
- Stage 3 : Lower vagina involved.
- Stage 3A – Pelvic side wall not involved
  3B – Pelvic side wall is involved / hydroureter / hydronephrosis (kidney)

⇓

* MCC of death is due to Renal Failure (involves kidney before metastasis has occured) or Uremia

- Stage 4 : Metastasis
- Stage 4A – Bladder / Bowel
  4B – Distant metastasis / sup. inguinal LN are involved.

* Ureter & Kidney involved stage III B
* Bladder involved is Stage IV A
* Sup. inguinal LN involved is Stage IV B

Management of Ca Cx

Principles for managing Ca Cx

* Radiotherapy can be used in all stages of Ca Cx (Stage I → IV)
* Whenever RT is done in squamous cell Ca, to make the cells more sensitive, a drug is used k/a Radiosensitizer.
* Radiosensitizer used in CaCx is Cisplatin.
* So instead of RT it is better to call it chemoradiation.
* Surgery can be used in CaCx from Stage I → stage IIA.
* But it is used in Mgt of stage IA_1 & IA_2 and IB_1
* IA_1 → Simple hysterectomy
  IA_2 → Werthime's hysterectomy
  IB_1 → Radical hysterectomy.

Complete Mg of Ca Cx

* If cancer in situ or stage IA_1
  - Young ♀ → Conization (cone biopsy)
  - Old ♀ → Simple hysterectomy
* In stage IA_2 :
  - ~~Young ♀~~ →
  - Old ♀ → Werthimes H + Pelvic LN dissection

- Young → Remove the Cx & stitch the uterus to vagina k/a Radical Trachelectomy + Pelvic LN dissection.

* Stage IB_1 : Radical H + Pelvic and para-aortic LN dissection.

* Stage IB_2 – Stage IV
Chemoradiation.

* In stage IA to IB_1 Sx is preferred because :
1) In young ♀ ovaries can be left behind.
2) Radiotherapy leads to vaginal fibrosis & that interferes with coital function

INFERTILITY

* Inability of a ♀ of reproductive age group to conceive even after 1 year of unprotected intercourse.

* It can be
- Primary
- Secondary

* Primary → ♀ has never conceived in her life.

* Secondary → Earlier she had conceived but now unable to become pregnant.

Cause

* It could be a male factor (30-40%)
* Female factor (40-50%)
* Combined (10%)
* Unexplained (10%)

* In case of female factor :
1) = Ovarian cause
↳ Anovulation / PCOD
↳ Ovarian failure / Premature menopause.

* Anovulation / PCOD ⟹ Most easily treatable infertility.

2) = Tubal cause (blockage)

3) = Uterine cause
↳ Mullerian malformation

* In tubal block → TB, PID, Endometriosis, tubectomy

4) = Cervix
- Antisperm antibodies

⟹ Basic investigation to be done in all infertile couple :
1) Semen analysis
2) Tests for ovulation
3) Tubal blockage (HSG – hysterosalphinography)
4) TVS

⟹ HSG : D_{10} of cycle
⟹ Ovulation :
- Follicular monitoring – D_{10}
- ottness – D_{22} – D_{24} of cycle

* TVS & Semen analysis anytime.

Female infertility

Ovarian causes :

(1) Anovulation –

- Tests for ovulation (dealt earlier)
  - * Best → Endometrial biopsy
  - * Easiest → Serum progesterone levels
  - * MC done → Follicular monitoring.
- Mgt of anovulation :
  - * 1st line drug → Clomiphene
  - * 2nd line → Clomiphene + Bromocriptine
    - (i) Clomiphene + Metformin
    - (ii) HMG
  - * 3rd line → GnRH (pulsatile)

(2) Ovarian failure –

- Tests for ovarian reserve (follicles)
  - (*) Day 3 serum FSH levels
    - If no follicles → less estrogen → -ve feedback on FSH lost and FSH ≥ 40 IU → ovaries failure
  - (ii) Best test can be done on any day of the cycle ↓ Measure Antimullerian (AMH) hormones (from puberty → granulosa cells secrete some AMH)

↓

Decreased AMH levels.

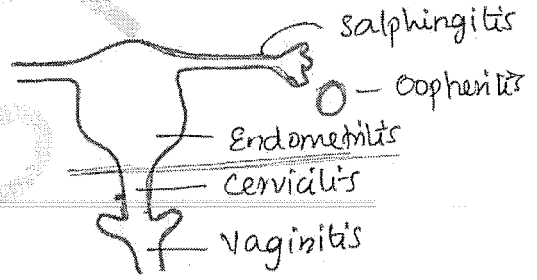
(iii) Quantitative test is with the help of TVS → count follicle → ↓ antral follicle count if ovary failure.

- Mgt : IVF with donor eggs.

Tubal causes :

* It is due to blockage of tubes due to

(1) PID (Pelvic inflammatory disease)



* PID includes all except vaginitis & cervicitis
* MC route of spread : Ascending infection along with sperm except in genital TB.
* In genital TB → MC route of spread Hematogenous

MCQ

* MC PID in virgin ♀ : Genital TB

Genital TB

→ 2° infection
→ MC 1° site is Lung > Lymph node
→ From lung / LN., it comes by hematogenous spread
→ In genital part, MC site affected
Fallopian tube > Uterus
→ MC symptom is (tubal blockage)
Infertility
→ MC site for fallopian tube block
Ampulla.
→ MC site of blockage on HSG
Cornual end of tube
→ In patients of genital TB
HSG is C/I (leads to spread)
→ But HSG, if done → specific appearance on HSG
1) Golf stick appearance of tube
2) Leadpipe appearance of tube
3) Beaded appearance of tube
4) Tobacco pouch like app. of tube
5) Bilateral cornual block.
→ Endometrial TB ⟶ mode of spread is direct spread.
→ Endometrial TB can manifest as Asherman syndrome. or pus inside uterus k/a Pyometra.
→ MCC of Pyometra:
Senile Endometritis
→ MC cancer causing Pyometra
Ca Cx > Ca endometrium
→ MCC of hematometra
Imperforate hymen.
→ Diagnosis of genital TB
Endometrial biopsy (1 wk before menstruation)
or
Menstrual blood.
→ Mgt
- Genital TB : ATT x 6 months
- Infertility due to TB : IVF

→ % of genital TB patients who are infertile are
70%.
→ % of infertile patient who has genital TB
- Worldwide - 10%
- India - 7%

Tubal infertility

* IOC: HSG
* Gold standard Dx:
Laproscopic chromo pertubation
* Mgt: Depends on site of tubal blockage.
(1) Cornual / Proximal block
(2) Midsegmental block
(3) Distal block / Fimbrial block
* Rx is needed if only all the tubes are blocked.
* In unilateral block → NO Rx.

Cornual block

MCQ
* MCC of bilateral cornual block is physiological spasm.
↓
* Tubes undergo spasm while doing HSG which appears as a blockage.
* Mgt : Pass a guide wire in to the tubes via vagina under hysteroscopic guidance k/c
MCQ Hysteroscopic tubal cannulation
↓
MCQ followed by Laparoscopic chromopertubation
* If the block is relieved, it means physiological spasm
* If block not relieved → some other pathology
MCQ
* MC pathological cause of bilateral cornual block is
Genital TB
↓
(Mgt by IVF)

Distal block

* If mild → do fimbrioplasty
* If severe → IVF.

Mid segmental block :

* Patient has undergone sterilisation in the past and now coming for reversal of sterilization

Sterilization procedures

Females :

* Criteria in India → ♀ should have atleast 1 child, age : 22-45 years, married.
* Consent of husband not needed.

MCQs
* Most cost effective method of contraception :
Vasectomy.
* Most effective method of contraception
Implants (Implanon®/ Nexplanon®)

Tubectomy

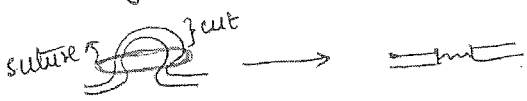
* Can be done (cannot on 8th/9th day)
1) After delivery → within 7 days of delivery → Post partum sterilization.
2) Non pregnant ♀ / after 42 days of delivery → interval sterilization
* MC method of post partum S is
Minilaprotomy
* MC method of interval steril
Laparoscopic sterilization
* Methods of sterilization (in tubectomy)
→ Laproscopically
→ Minilaprotomy (small incision below umbilicus)

* In laparoscopic
  - Clips (Hulka clips)
  - Fallope ring (in India)

MCQ
* Part of fallopian tube we do sterilization is Isthmus

* In minilaprotomy → various techniques → MC done is Pomeroy's method.

* Pomeroy method ⇒



* MCC of failure in ♀ sterilization
  Identification of wrong structure

* Modified pomeroy method :-

- 2 sutures
- Cut end send for histopathological exam to confirm tube is cut.

Important points

* MC method of female sterilisation
  Laparoscopic
* But laparoscopy cannot be done in post partum period (uterus upto below umbilicus - rupture)
* So MC method in post partum
  Minilaprotomy
* Least failure rate is with
  Unipolar cautery > Modified Pomeroy
  (not used)

* Highest failure rate / order of reversal
  Clips > Fallope rings > Pomeroy > Modified Pomeroy > Cautery

Laparoscopy

* MC used gas : CO_2
* Pressure : 10-12 mm Hg
  (Never > 15 mm Hg)
* Needle used - Verres needle

Midsegmental block
↓
Patient wants reversal
↓
- Reversal will be best if clip > fallope rings was used for sterilization
- Reversal will be best if can reconstruct > 4 cm of tube.
- Reversal will be best if MCQ Isthmo-isthmic anastomosis

Cervical cause

* In a few females, antisperm Abs are present in cervix
* Test for diagnosing
  Post coital test / SIMS test
* Mgt : IUI (intra uterine insemination)

Male ♂ sterilisation

* Infertile couple → always first do semen analysis
* Volume > 1.5ml
* No semen → Aspermia
* pH : ♂ 7.2 - 7.8
* Viscocity < 3
* Semen analysis should be done on liquefied semen
* Sperm concentration - 15 million/ml
  - < 15 → Oligospermia
  - < 5 → Severe oligospermia
  - No sperm in semen
    Azoospermia
* Motility should be > 32%
  - ↓ motility → Asthenospermia
* Morphology - > 4% should be normal
  - Morphologically abnormal k/a Teratospermia
* Viability: > 58% should be viable
  - < 58% → Necrospermia
* Mgt of oligospermia:
  - Sperm count ≥ 15 million/ml
    ↓
    Mgt - IUI
    (Intra uterine insemination)
  - Sperm count : 5-15 million/ml
    ↓
    Mgt - IVF
  - Severe oligospermia / asthenospermia / azoospermia
    ↓
    Mgt: ICSI (Intra cytoplasmic sperm Injection)

IUI

* Intra uterine insemination
* Semen from male partner (husband : IUI-H)
* Semen from donor (IUI-D)
* Semen is washed & processed
* 0.5 ml of semen is taken, with the help of a catheter it is put in ♀ uterus
* Day of insemination → Day of ovulation (18-20 mm follicle on follicular monitoring)
* Cost : Rs. 5000/-
* Indications for IUI :

  Male

  1) Sperm count = 15 million/ml
  2) Ejaculatory disorders like hypospadias, epispadias, retrograde ejaculation
  3) Has inherited disease and risk of transferring it to offspring (use IUI-D)
  4) Unexplained infertility

Female

1) Cervical factor infertility (Antisperm Ab) -
2) Unexplained infertility.

IVF

* Invitro fertilisation
* Male partner → semen → wash & process it.
* Female partner → Clomiphene from D_2-D_6 or D_5-D_9 → No. of follicles starts growing → From D_{10} do follicular monitoring → when follicles reach 18-20 mm size → inj. hCG (acts as ovulation trigger) → after 36 hrs, under USG guidance do ovum pick up → ova is put in a petridish → add sperms to it (3 to 5 lac/oocyte or ova) → fertilisation occurs in petridish
* When embryo become 8 celled stage (~day 3) → transfer back inside the female uterus (2 cm below the fundus as this is the site for implantation)
* Maximum no: of embryos which you can transfer in IVF → 4
* Success rate : 25 - 30%
* Cumulative success rate : 50%
* Costly (1.5 - 2 lakhs)

* Indications :-

Male

- Oligospermia
- IUI failed 3 times

Female

- Tubal infertility
- Donor egg with IVF is done in ovarian failure.
- IVF with sarrogacy is done in Mullerian agenesis

ICSI

* Intra cytoplasmic sperm injection
* The procedure is like IVF.
* Male → sperm → process
* Female - - - - → oocyte pick up → in to each oocyte, injeet a single sperm under microscope →
* So can be used in Mgt of severe oligospermia.
* Also used in Asthenospermia
* So indications are :
  - Oligospermia
  - Asthenospermia
  - Azoospermia (sperms are absent in semen but present in the testis → sperms are retrieved from testis by
    - TESA (Testicular sperm aspiration)
    - TESE (Testicular sperm extraction)
    - PESA (Percutaneous epididymal sperm aspiration)

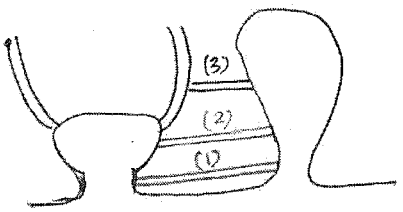
- MESA (Microsurgical epididymal sperm aspiration)

↓

then ICSI is done.

FISTULA

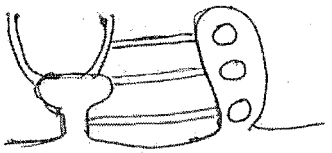
Urinary fistula



(1) Urethro vaginal fistula
(2) Vesico vaginal fistula
(3) ~~Uterco~~ Uretero vaginal fistula

* If a ♀ complaints of continous dribbling of urine from vagina + normal urination.
  Uretero vaginal fistula
* If a ♀ complaints of continous dribbling of ~~cat~~ urine from vagina but no normal urination
  Vesico vaginal fistula.
* If a ♀ complaints of no continuous dribbling of urine from vagina but when she sits for urination, urine from both vagina & urethra
  Urethro vaginal fistula.

* Methylene blue 3 swab test



- Place 3 cotton swabs inside vagina
* Now with the help of ~~methylene blue dye~~ foleys catheter, methylene blue dye is inserted inside bladder & urethra.

* Observation & type of fistula.

(1) Upper most cotton swab wet but not blue in colour
  Uretero vaginal fistula
(2) Middle cotton swab wet and blue in colour
  Vesico vaginal fistula
(3) Lower most cotton swab wet and blue in colour
  Urethro vaginal fistula

Vesico vaginal fistula (VVF)

* MC variety of fistula
* MCC of VVF in developed countries
  Hysterectomy
* MCC of VVF in developing countries
  Obstructed labour
* IOC: Cystoscopy
  (no:, size, site)
* Mgt: Surgical repair

* Surgery in VVF

↳ Time : If it is detected within 24 hrs then do immediate repair

- If detected after 24 hrs then wait for 3 ~~wks~~ months (inflammation subsides)

MCQ ↳ Technique: Chassar Moir technique (or Layer technique)

* Post-operative precautions

- Bladder drainage (foleys catheter) x 2 wks
- No intercourse, no per vaginal examination, no per speculum examination x 3 months
- No pregnancy x 2 years (if pregnant → delivery by C-section)

OVARIAN CANCER

Risk factors

* ↑ estrogen
* Theory of incessant ovulation, ie, more a female ~~ovu~~ ovulates more are chances of ovarian cancer.
* Early menarche
* Late menopause
* Nulliparity
* Obesity
* Diabetes
* PCOD
* Familial - gene : BRCA-1, BRCA-2 and HNPCC.

Protective factors

* Physical exercise
* Smoking
* Multiparity
* Anovulation
* ~~OCPS~~ - OCP
  - Pregnancy
  - Breast feeding

Ovarian cancer types

1) Epithelial (90 %)
2) Germ cell tumour (5-8 %)
3) Sex cord tumor (3 %)
4) Metastatic tumour

Epithelial cancers

- Serous → 75 %
- Mucinous → 10 %
- Endometrial → 10 %
- Brenner, Clear cell } 5 %

MCQs

* MC variety of ovarian tumour — Epithelial
* MC benign tumour of ovary — Dermoid cyst
* MC benign epithelial tumour — Serous cyst adenoma
* MC malignant tumour of ovary — Serous cyst adenocarcinoma.
* MC ~~epithelial~~ malignant epithelial tumour of ovary — Serous cyst adenocarcinoma

Imp. points on ~~Endo~~ Epithelial tumors (serous type)

* In epithelial tumours CA 125 ↑ except mucinous variety.
* Age group → Post menopausal → 6-7th decades.
* Non specific signs & symptoms
* Diagnosed at ~~last~~ late stage
* Chances of malignancy = 20 %.
* Bilateral in ~~80~~ 20 %.

Mucinous variety

* Mostly unilateral (Bilateral only 10%)
* Do not have marked rise in CA 125 levels
* Seen in perimenopausal ♀
* Very large
* Associated with Pseudomyxoma Peritonei

Pseudomyxoma Peritonei

* Condition where peritoneum is filled with gelatinous substance
* MC seen with appendix cancer
* Other conditions:
  - Mucocele of appendix
  - Mucinous variety of ovarian Ca.

Endometriod variety

* Associated with endometrial cancer and endometriosis

Brenner's tumour

* Solid ovarian tumor
* Have bladder like transitional epithelium.
* On HPE, characteristic cell → Walthard cell nest
* Mostly benign
* Can be associated with Pseudo Meig syndrome.

Hereditary Ovarian Cancer

* % of ovarian cancers are hereditary → 10%
* Hereditary ovarian cancer associated with:
  - BRCA-1 → Upto 45%
  - BRCA-2 → Upto 25%
  - HNPCC → Upto 15%
* In these conditions to prevent ovarian cancer - best method is to "Bilateral salphingo-oopherectomy" after patient has completed her family.
* 2nd method - Put her on OCPs

Route of spread of epithelial tumours

* MC → Tumour exfoliation (Transcoelomic)
* 2nd MC → Lymphatic
* Uncommon → Hematogenous
* Tumour marker of epithelial ovarian tumours → CA 125 levels.
* In postmenopausal females CA 125 > 35 IU is diagnostic
* In reproductive age females - CA 125 levels can be raised in a no: of conditions like fibroid, endometriosis etc.
* Hence it is not a useful investigation. Only if levels are very high >200 then it may indicate ovarian cancer.

Staging of ovarian cancer

* Surgical staging
* steps: TAH + BSO + Infracolic Omentectomy + Pelvic & Para-aortic LN sampling

* Figo staging:
  - ⇒ Para aortic LN involvement → III A
  - ⇒ Pleural effusion → IV A
  - ⇒ Liver & Spleen capsule involved → III C
  - ⇒ Liver & Spleen parenchyma involved → IV B
  - ⇒ Inguinal LN involved → IV B

* Management:

Post operative management
↓
Choice is Chemotherapy
(Carboplatin & Paclitaxel)
↓
except stage I A & B, grade I & II
(No post. operative mgt)

Germ cell Tumors

Varietes

Thank → Teratoma < Mature dermoid cyst / Immature malignant

You → Yolk sac tumor (Endodermal sinus tumor)

C → Choriocarcinoma

D → Dysgerminoma

E → Embryonal cell Ca

* MC Germ cell tumor
  Dermoid (Benign)
* MC ovarian tumor in pregnancy
  Dermoid (benign)
* MC ovarian Ca in pregnancy
  Dysgerminoma
* MC malignant germ cell tumor
  Dysgerminoma

Dermoid cyst important points

* Age group → Reproductive age
* Has derivative of ectoderm/ endoderm / mesoderm
* Bilateral in 10% cases
* Risk of malignancy < 2%. (Sq. CCa)

Common points on all germ cell tumor

* Unilateral tumors (Only dysgerminoma is bilateral, that too only in 15-20% cases) (Imp)
* MC age : 10-20 yrs
* Rapidly growing unlike the epithelial cell tumor.
* Produces hCG so can present c precocious puberty.
* Better prognosis.

Endodermal sinus tumor

* Highly malignant
* Germ cell tumor c worst prognosis
* Rapidly growing
* Histopathological examination
  Schiller Duval bodies
* Tumor marker : Alpha feto protein & Trypsin (Imp)

Dysgerminoma

* GCT c max incidence of bilaterality
* GCT c best prognosis
* most radiosensitive GCT
* Tumor marker : LDH & Placental alkaline phosphatase

Sex cord tumors

* Least common
* Unilateral
* Best prognosis

Classification

- Estrogen secreting
  - Granulosa cell T
  - Thecoma
  - Fibroma
- Androgen secreting
  - Sertoli cell T
  - Leydig cell T
  - Hilus T

Granulosa cell tumor

* MC before puberty or after menopause
* Secrete estrogen so patient can present with precocious puberty or post menopausal bleeding
* Histopathological examination :
  Coffee bean nucleus & & Call exner bodies
* 25-50% are associated c endometrial hyperplasia or Ca.

Fibroma

* Seen asso. c Meigs syndrome
* meigs syndrome ↓

  Rt side pleural effusion } + Fibroma
  Rt side ascites

* When these features are seen c any other ovarian tumor like Brenner tumor or Thecoma it is ↓
  N/a Pseudo Meig Syndrome

Krukenburg tumor

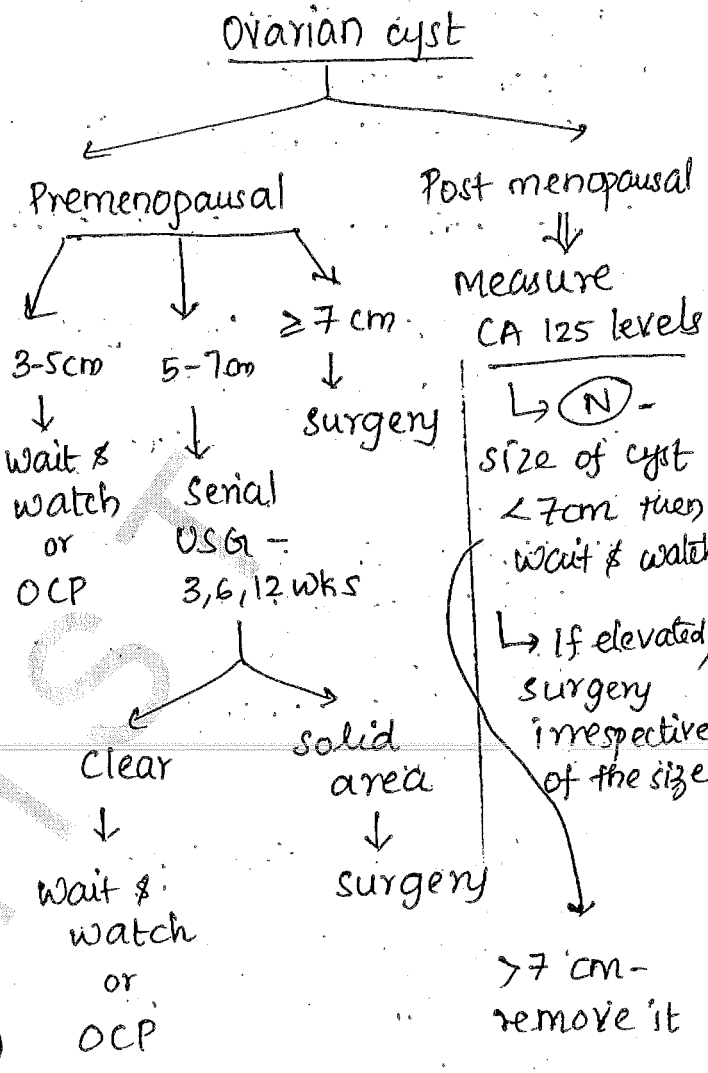
* Metastatic tumor to ovary
* From Ca stomach it reaches ovary via lymphatic spread
* Characteristics
  - Always bilateral
  - Freely mobile
  - No adhesions
  - Shape of ovary is "N"
  - Histopathological examination → Signet ring cells
  - Waxy consistency

Clinical case 1

Whenever a ♀ presents c ovarian mass & is non pregnant then

Mgt

- Principle → Surgery for ovarian mass is done if
  - ≥ 7 cm mass
  - ≥ 10 cm adnexal mass (cant diff. b/w ovary & tube)
  - CA 125 ↑ in post menopausal females
  - USG shows features of malignancy like
    - Solid areas in mass
    - Ascites
    - Bilateral
    - Septa present inside the mass
- Removed irrespective of size of tumor if malignant mass



Ovarian mass

* 1st investigation is CA 125 levels, TVS

Clinical case 2 :

Management of ovarian cyst in pregnancy.

- MC ovarian tumor in pregnancy
  Dermoid cyst
- * MC ovarian tumor to undergo torsion in pregnancy
  Dermoid cyst
- * MC time for ovarian cyst to undergo torsion in pregnancy
  End of 1st trimester / puerperium (after child birth)

* If the cyst is symptomatic, ie, torsion had occured, then cyst is removed immediately irrespective of time of pregnancy
* If any cyst is detected during puerperium it should be removed immediately (MC time for torsion)

Size of cyst : Mgt of asymptomatic cyst

- < 5 cm → Wait & Watch
- 5-10 cm → USG / MRI
  - Solid areas → Operate in 2nd trimester (14-20 wks)
  - Clear → Wait & Watch
- ≥ 10 cm → Operate in 2nd trime.

Best time : 2nd trimester.

OBS

NIST
1980

* After fertilisation zygote starts undergoing cell division.

2, 4, 8 celled zygote
↓
16 celled zygote
↓
k/a Morula
(Mulberry shaped)

* Morula covered by zona pellucida (which prevents multiple sperm entry)

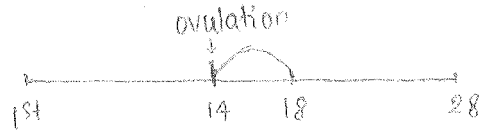
MCQ
* In which stage - zygote enters the uterus?

16 celled stage (Morula)

(a. 2-4 cell
b. 4-8 cell
c. 8-16 cell
d. 16-24 cell)

* Morula enters uterus via
- Peristalsis of fallopian tube
- Movement of cilia

* Morula enters uterus 4 days after fertilisation / day 18 of the cycle.



↓
fertilisable span of ova:
↓ 12-24 hrs
fertilization (D14)

* Zona pellucida is lost → k/a zona hatching → 5 days after fertilisation

* zona pellucida → function: prevents polyspermy.

* When morula enters uterine cavity → fluid enters in to it → converted to blastocyst.

* Blastocyst has to attach itself to the endometrium → process is k/a ~~endometriosis~~.

Implantation.

* ~~Bla~~ Implantation can be superficial or blastocyst can go deep

* Superficial → Not seen in humans

* Deep inside it go to implant → k/a interstitial implantation
↓
occurs in human beings

MCQs
Important points on implantation

* Implantation occurs in which form:
Blastocyst

* Implantation begins by 6th day after fertilisation
δ_{20} of cycle.

(D_{14} - Fertilisation
D 18 (after 4 days) - Morula enters uterine cavity
D_{20} (after 6 days) - Implantation)

* Implantation is completed by 10 days after fertilisation

* In a few %, bleeding occurs at the time of implantation
  Hartman sign

* After implantation → endometrium is k/a
  Decidua

* Which hormone is responsible for converting endometrium in to ~~endometrium~~ decidua
  Progesterone
  ↓
  k/a decidual reaction

MEQ
* The limit of penetration by the blastocyst in to decidua is decided by Nitabuch's layer

* If Nitabuch's layer is absent, blastocyst will penetrate
  ↓
  Myometrium
  ↓
  then serosa

* If this happens in
  ⇒ superficial myometrium
  Placenta accreta
  ⇒ deep to myometrium
  Placenta increta
  ⇒ serosa
  placenta percreta

* Placenta Accreta, placenta increta, placenta percreta
  ↓
  together k/a Morbidly Adherent placenta

* Normally placenta is attached to the endometrium (decidua)

* In placenta accreta, placenta is attached to myometrium superficially

* In placenta increta, placenta is attached deep in to myometrium (it invades ~~en~~ myometrium)

* In placenta percreta, placenta attached to serosa

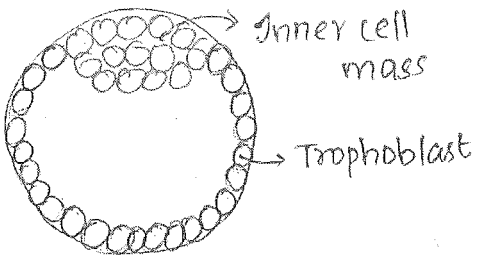
* In all the 3 cases, placenta will not deliver after the delivery of baby & will present as
  Retained Placenta

* Highest risk for this condition is, if there is present placenta previa
  ↓
  2nd : Previous Caeserrean section

* Placenta previa → when the placenta attaches to lower uterine segment

* Pathology here : Nitabuch's layer is absent

* Management : Hysterectomy



* Blastocyst has 2 parts
  - Inner cell mass
  - Trophoblast

* Inner cell mass forms all the 3 germ layers

  ie, entire embryo is formed by inner cell mass

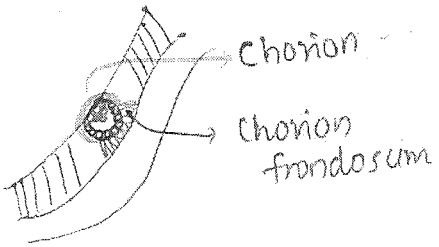
mcq

* 8 days after fertilisation, trophoblast divided in to
  - Cytotrophoblast
  - Syncitiotrophoblast

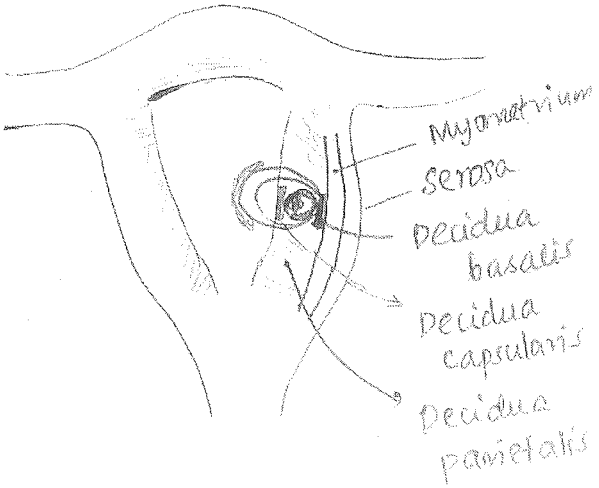
Functions of trophoblast

* Syncitiotrophoblast will form all the pregnancy hormones
  eg: hcg, HPL (human placenta lactogen)

* Cytotrophoblast :
  - At the site where future placenta is formed → it will form a number of chorionic villi
    ↓
    k/a Chorion frondosum
    ↓
    forms the fetal side of the placenta
  - Rest of cytotrophoblast will form the chorion.
  - Prevents PIH in pregnancy



Decidua



* Decidua capsularis is the part of decidua which seperates blastocyst from uterine cavity.
* Decidua parietalis is the entire decidua which lines the uterus
* Decidua basalis is the part of decidua which lies below the blastocyst where future placenta has to be formed.

Note

* Decidua basalis forms the maternal site of the placenta.
* As the embryo grows, it grows inside the uterine cavity such that finally one day it occupies the whole of uterine cavity
* And the uterine cavity gets obliterated. (14-16 wks gestation)
* The decidua capsularis fuses with decidua parietalis
* This happens b/w 14-16 wks of gestation.

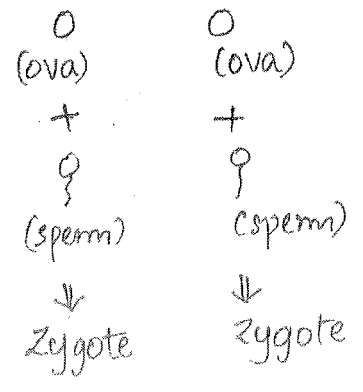
Fetal Membranes

* Two imp. fetal membranes
  - Amnion
  - Chorion
* Innermost fetal membrane is Amnion
* Amnion develops from fetal ectoderm
* It is avascular
* It is formed 10th day after fertilisation.
* Chorion is attached outside to the amnion
  ↓
  formed by cytotrophoblast
  ↓
  formed 8th day after fertilisation

Twins

* It can be of two varieties

1) Dizygotic twins



Dizygotic

* More common
* Same sex / different sex
* Incidence of dizygotic twins varies from country to country.

(2) ○ (ova) + ♀ (sperm) ⇓ single zygote → ○ (divides) → ○ ○ → Monozygotic twins

Monozygotic

* Same sex
* Also k/a identical twins
* Less common than dizygotic twins
* Incidence of monozygotic twins remains same throughout the world.

*

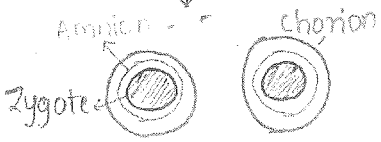
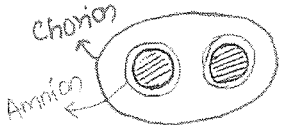
⇒ In dizygotic twins → 2 zygotes → each zygote forms its own amnion & chorion → So always

Dichorionic Diamniotic

MCQ

⇒ Because dizygotic twins are more common ↓ MC variety of twins is **Dichorionic Diamniotic**

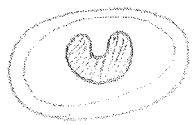
⇒ In monozygotic twins → single zygote → it divides in to two → division occurs at

- **< 4 days** ↓ ○ ○ zygote ↓  → **Dichorionic Diamniotic**
- **If b/w 4-8 day** ↓  ↓ Monochorionic Diamniotic

MCQ

↓ MC variety in monozygotic twins

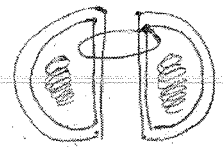
If division occur at

- **B/w 8-12 days** ↓  ↓ Mono chorionic Mono amniotic
- **> 12 days** ↓  ↓ Conjoint twins

Monozygotic Twins

DCDA

- Good prognosis
- In DCDA twins on USG
  ↓
  sign k/a Twin Peak sign +ve
- Time: 10-14 wks
- Time of delivery 37 wks

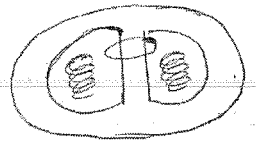


4 layer of membrane

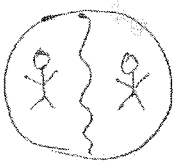
(thick)

MCDA

MC complication
↓
Twin to twin transfusion syndrome
↓
Time of delivery 36 wks



2 layer of membrane

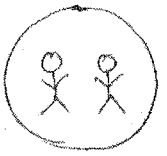


(thin)

MCMA

Bad prognosis
↓
MC complication
↓
Cord entanglement
↓
MCQ ★ Time of delivery 32-34 wks
MCQ ★ Always do cesarean section

No layer

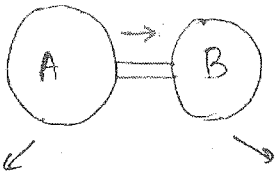


Conjoint

MC part fused is thorax, k/a
↓
Thoracophagus
↓
Always do cessarean section

Twin to Twin transfusion syndrome

* Seen in MCDA twins
* There is vascular connection b/w the twins, such that one twin becomes donor twin & other becomes recepient twin



Donor
↓
- Anemia
- Less growth

Recepient
↓
- Polycythemia (leads to thrombosis)
- More growth

↓

Discordant growth.

⇒ Fetal urine forms amniotic fluid normally.

Donor
- Giving blood
↓
Renal flow ↓, GFR ↓
↓
Oliguria
↓
Oligohydramnios
- Heart failure

Recepient
- Receiving blood
↓
Polyhydramnios
- Congestive heart failure

* Management:
Fetoscopic laser ligation of passage.

Hellen's rule

- Incidence of twins is 1 in 80 pregnancies
- Incidence of triplets is 1 in $(80)^2$
- Incidence of quadraplets 1 in $(80)^3$

MCQ
⇒ Why is incidence of twin pregnancy ↑ these days.
1) IVF
2) Clomiphene citrate (<10% → 5-8%)

⇒ Sign of pregnant female: ♀

MCQ
⇒ If ♀ has quadraplets but she wants only 2, then do Fetal reduction
↓
inject KCl in to the heart of fetus (in the intrauterine life)

⇒ In India, MTP is legal upto 20 wks (Medical Termination of Pregnancy)
⇒ > 20 wk → k/a delivery

Amniotic Fluid

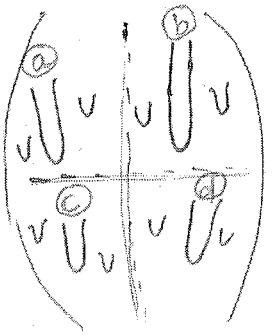
* Specific gravity
  1.008 to 1.010
* Osmolality:
  250 masm /L
* It is completely replaced in 3 hrs
* Rate of amniotic fluid turn over is 500 cc/hr
* Volume of amniotic fluid is maximum b/w (1L)
  36 – 38 wks
* Then it decreases such that at term, it is roughly 700 ml
* pH: 7.2 to 7.4
* Normally it is colourless.

Color	Seen in
* Green color (meconium)	• Fetal distress • Breech presentation or transverse lie • Listeria infection
* Golden color (Bilirubin)	– Rh incompatibility
* Tobacco juice	Intra uterine death
* Saffron colour, yellowish green	Post dated pregnancy.

* Source / major contributors of amniotic fluid.
  - Early wks (1st trimester) → Maternal plasma
  - 12–20 wks → Fetal skin
  - ≥ 20 wks → Fetal urine

Overall : Fetal urine

* Besides producing amniotic fluid, fetus also swallows amniotic fluid ie, how balance is maintained
* Composition of amniotic fluid
  99 % H_2O
* Functions → performs a no: of functions except nutrition
  (shock absorber, space for fetal growth etc)
* Measurement of amniotic fluid:
  Done by USG



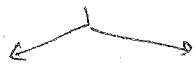
(divide to 4 quadrant)
Measure the largest vertical AF pocket in each quadrant
↓
add them
a+b+c+d = AFI

- AFI (Amniotic fluid index)
- Normal : 5 – 24 cms

* Oligohydramnios
if < 5 cm

* Polyhydramnios → > 24 cm.

* Best / Most sensitive marker
↓
Measure the single largest vertical pocket (b in diagram)
↓
Normally it is 2-8 cm



< 2 cm → Oligohydramnios

> 8 cm → Polyhydramnios

* If volume of A.F
< 200 ml → Oligohydramnios
> 2 L → Polyhydramnios
(Normal volume: 200ml - 2L)

Oligohydramnios	Polyhydramnios
• Volume < 200 ml AFI < 5cm Single largest pocket < 2 cm	• Volume ≥ 2L AFI ≥ 25 cm Single largest >8 cm.
• MCC of mild oligohydramnios ↓ Idiopathic	• MCC of mild polyhydramnios ↓ Idiopathic
• MCC severe OH Congenital defect ↓ Renal anomalies	• MCC of severe PH CNS defect

⇒ Severe polyhydramnios
↓
CNS defect
(Neural tube defect)
↓
1) CSF leaks in to A.F
2) Swallowing disorder.

Other causes of Oligohydramnios

D → Drugs : Indomethacin

I → IUGR (Brain sparing effect, so blood flow to kidney ↓, Renal BF ↓ → GFR ↓)

L → ~~Leanin~~ Leaking after amniocentesis

Mein → ↑ Maternal BP (P∝ ½)
so renal BF ↓ → GFR ↓ fetus.

P → Post term pregnancy

(Y) P → Premature rupture of membrane (PROM)

A → Amnion nodosum & chromosomal anomaly triploidy

R → Renal anomalies like Renal agenesis & horse shoe shaped kidney.

(Mug up causes Amnion nodosum and triploidy)

DIL Mein PYAR
↓
P

Other causes of Polyhydramnios

(Urine ↑ by fetus)

1) Multifetal pregnancy
2) Maternal diabetes (maternal glu↑ → to fetus → ↑ fetal glu → polyuria)

(swallowing defect in fetus)

3) Esophageal atresia
4) Intestinal obstruction
5) Duodenal Atresia
6) Cleft lip / Cleft palate.

(CSF leak in to A.F)

7) Neural tube defect

Mug up causes

- Chorangioma of placenta
- TORCH infections
- Rh incompatibility
- Trisomy

Oligo H → Triploidy
Poly H → Trisomy

Effects of Oligohydramnios

- Occurs early in pregnancy:
  - Less space for fetus to grow
  - MC complication
    - (i) Lung hypoplasia (Pulmonary hypoplasia)
    - (ii) Limb reduction defect
- Occurs late in pregnancy

By this time organogenesis is complete

But still less space

↓

Cord compression

↓

Cord has umbilical Art. & Vein

↓

Less blood to fetus

↓

Fetal distress

↓

Fetus passes meconium in to amniotic fluid

↓

fetus swallows meconium stained amniotic fluid

↓

Meconium Aspiration Syndrome

* Management:

Amino infusion with normal Saline.

Effects of Polyhydramnios

* Excessive A.F.
  ↓
  Overstretching of membranes
  ↓
  PROM → Preterm labour

* In polyhydramnios → membrane rupture → AF comes out suddenly → size of uterus will suddenly shrink → placenta can get seperated → Abruptio placenta

* The uterus is over distended, so it loses it's tone
  ↓
  ∴ After delivery
  ↓
  Post partum hemorrhage.

PROM: Premature rupture of membranes

⇒ Management:

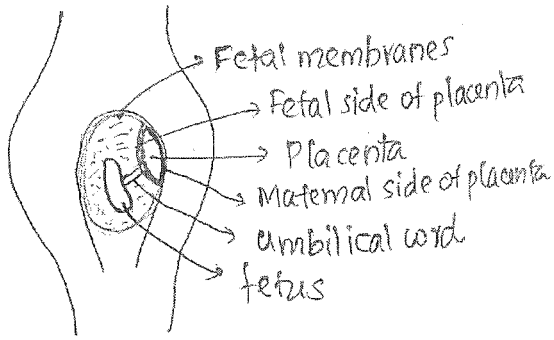
Serial amniocentesis
↓
Indication: when mother is having lot of respiratory discomfort.

⇒ Which drug can be used to manage polyhydramnios

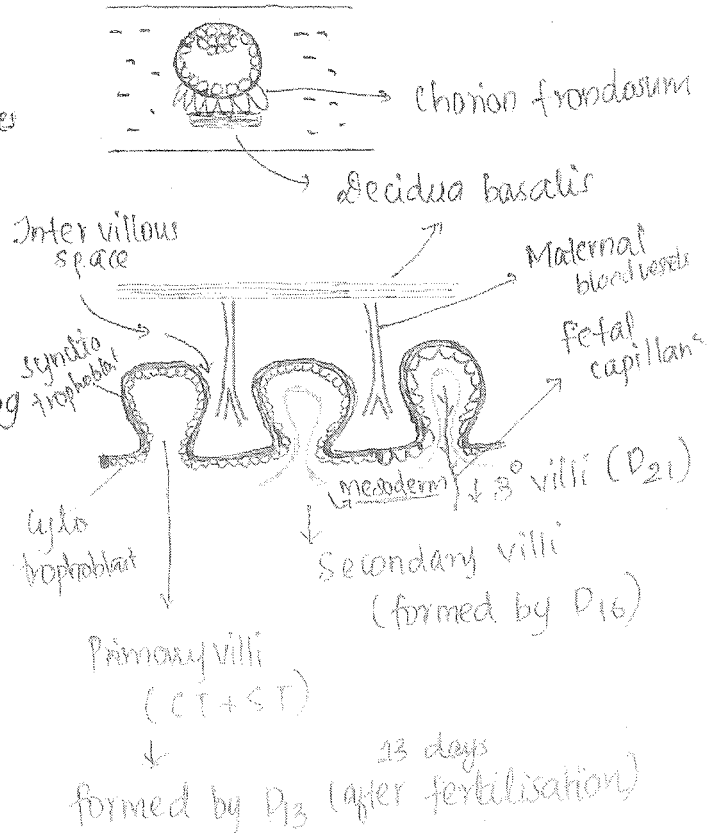
Indomethacin

* But it leads to premature closure of ductus arteriosus.
  So it should not be used beyond (>32) 32 wks of pregnancy.

PLACENTA



Formation of placenta



* From decidua basalis, maternal blood vessels comes to the inter-villous space.
* Fetal side of placenta
↓
Chorion frondosum
↓
Villi – 3 types
↓
1° villi (formed by cytotrophoblast & synctiotrophoblast
↓
2° villi (formed by CT + ST + mesoderm of fetus)
↓
3° villi (2° villi + fetal capillaries)

* 1° villi → D_{13}
* 2° villi → D_{16}
* 3° villi → D_{21}

Questions MCQs

* Villi of placenta have fetal blood which is = 350 ml.
* Intervillous space has maternal blood = 150 ml.
* Volume of placenta at term 500 ml.
* In placenta, both maternal & fetal blood are present. But they do not mix each other. They are seperated by
  1) Syncitio T
  2) Cyto T
  3) Mesoderm
  4) Endothelium of fetal capillaries

  } Placental barrier

*
* Fetal circulation is established by day 17 – 21.
$D_{21} > D_{17}$ – ~~D_{24}~~ D_{21}
* When maternal spiral arteries open in the intervillous space
↓
cytotrophoblast replaces its lining and converts them from high pressure vessel to low pressure vessel
↓
$P \propto 1/V$, hence V ↑

↓

k/a trophoblastic invasion

* If this action is incomplete. ie, incomplete trophoblastic invasion → pressure in maternal arteries remain high → leads to PIH

* Placenta at term

* Human placenta is

DISCOID → ↓ disc like shape

DECIDUATE → ↓ shed off after delivery

HEMO CHORIAL → ↓ lies in contact c maternal blood

Maternal side	Fetal side
* It is 1/5th in thickness	* It is 4/5th in thickness
* formed by- Decidua basalis	* Formed by- Chorion frondosum (Cytotrophoblast)
* Dull red in colour	* Shiny, grey in colour.
* Has polygonal areas k/a ↓ LOBES ↓ further divided to Lobules or cotyledons	* Has umbilical cord & fetal membranes attached to it-

* Weight of placenta @ term : 500gm
* Ratio of weight of placenta : fetus (3kg)
  1:6
* When is wt. of placenta = wt of fetus
  17 wks of pregnancy.
* Cells present in placenta (phagocytic cells)
  Hoffbaver cells
* Mature placenta has two sides
  - Maternal side
  - Fetal side.

MCQs

* Functional unit of placenta
  Cotyledon
* Normally umbilical cord is attached to centre of placenta

Normal

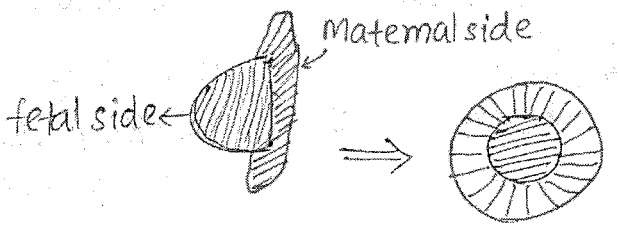
* If the cord is attached to the margin of placenta → k/a Battle dore placenta

* Normally fetal side of placenta and maternal side of placenta have equal area

MCQ ★
⇒ Uteroplacental blood flow
450 - 650 ml/min

MCQ
⇒ Uterine blood flow @ term
750 ml/min

* If fetal side is smaller than maternal side



fetal side is gets surrounded by maternal side in the form of a ring

↓

Circumvallate placenta

↓

fetal side small

↓

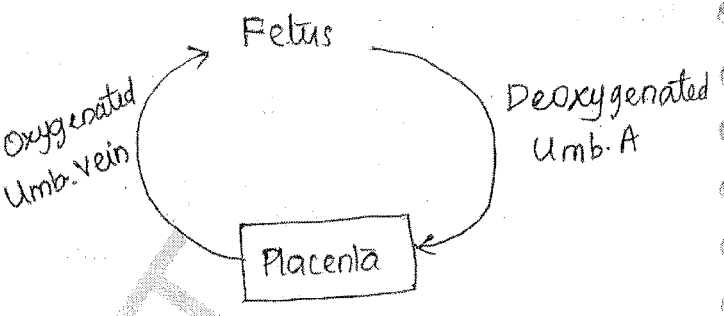
less blood to fetus

↓

IUGR

APH (Ante partum Hemorrhage)

Fetoplacental circulation



Umbilical vein
- Oxygenated blood

↓

pO_2 = 60-70% (Oxygen saturation)

- Pressure = 10-20 mmHg
- Remnant is Ligamentum teres

Umbilical artery
- Deoxygenated blood

pO_2 = 50-60%

- P = 60 mmHg
- Remnant is Medial umbilical ligament

⇒ Artery forms Medial umbilical ligament:

AMUL

MCQ
* Medial umbilical ligament is a remnant of Urachus

Placental circulation

Feto placental circulation

↓

Villi

↓

- Established b/w D_{14} - D_{21}
- Established by 21 days after fertilisation
- Blood in villi - 350 ml

MCQ

Uteroplacental circulation

↓

Intervillous space

↓

- Established by D_{12}
- MCQ Uteroplacental blood flow: 450 - 650 ml/min
- MCQ Uterine blood flow at term: 750 ml/min

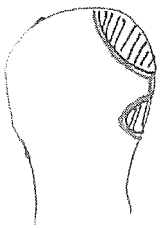
MCQ

* For localisation of placenta → Best time to do USG
  3rd trimester

* Normally placenta is attached to upper uterine segment.

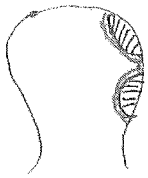
MCQ

* If placenta is attached to the lower uterine segment k/a Placenta previa
  (best identified at 3rd trimester)



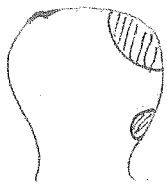
Placenta Succenturiata

(Small part of placenta seperated from main placenta. But both connected by blood vessels)



Placenta bilobata

(2 equal parts seperated and connected by blood vessels)



Placenta spuria

(small part seperated from main part but not connected by blood vessels)

* Placenta succenturiata, P. spuria, P. bilobata → can leads to PPH after delivery.

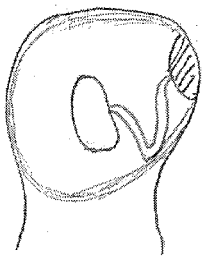
Umbilical cord

* Develops from connecting stalk
* Length of umbilical cord is 30–100 cm
* Average ⇒ 55 cm.
* If < 30 cm ⇒ k/a short cord
* Connective tissue of cord k/a Wharton's jelly
* Coils of cord k/a Folds of Hobokon
* In early intrauterine life umbilical cord has total 4 { Rt. umb A + Lt umb V and Rt umb V + Lt umb V

* Later on right umb. vein disappears and left is left behind
* At the time of birth → 3 vessels
* pH of cord is 7.2
* Umbilical cord shrinks at the moment it is exposed to atmosphere

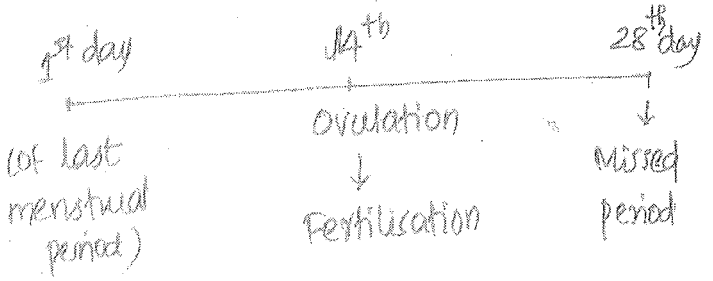
Anomalies of the cord

* If the cord is first attached to the membrane and then to the placenta k/a
  Velamentous insertion of cord.
  ↓
  It can lead to VASA PREVIA



Period of pregnancy

* Or period of amenorrhea / period of gestation.



* Pregnancy is always calculated from 1st day of last menstrual period (LMP) except in the growth periods.

MCQ
* If an event occurs 3 wks after fertilisation then
  5 wks of pregnancy.

Growth period
* Here calculation is done from the day of fertilisation.

Embryonic period
* It is 3–8 wks after fertilisation (ie 5–10 wks of pregnancy)

MCQ
* It is the most teratogenic period

Fetal period
* ≥ 9 wks after fertilisation uptill delivery.

Time	Event
• 8 wks	→ Gonads are formed
• 10-12 wks	→ Swallowing begins
• 12 wks	→ External genitalia starts forming
• 14 wks	→ Sex of baby can be identified by USG
• 11 wks	→ Fetal breathing movements begins
• 12 wks	→ Urine formation begins (Rate of urine product^{n} at term is 27 ml/hr) ie, 650 ml/day.
• 16-18 wks	→ Eye movements begin
• 24 wks	→ Fetus starts hearing
• 28 wks	→ Light perception

⇒ Fetal blood formation : Hematopoesis

Site	Time of pregnancy	Main Hb
Yolk sac	Till 6 wks	Gower 1 Gower 2 Portland Hb
Liver (spleen)	Till 20 wks	HbF (fetal Hb)
Bone marrow	≥ 20 wks	HbA (adult Hb)

HbF	HbA
• Less 2,3 DPG	• More 2,3 DPG
• Higher affinity for O_2	• Less affinity for O_2
• Less carbonic anhydrase	• More carbonic anhydrase.
• Resistant to acid and alkali	• Sensitive to acid & alkali

⇒ At term, fetal Hb (HbF) is 18 gm %.

⇒ At the time of birth, 75-80% of Hb is fetal Hb (HbF) and the rest is adult Hb (HbA)

⇒ By the time child is 6 months HbF is < 1%.

⇒ Fetal RBCs are larger than the adult RBCs.

⇒ Life span of fetal RBC is 90 days

⇒ Adult RBC → 120 days

VASA PREVIA

* It is a condition in which there is bleeding from the fetus in the antenatal period.
* It is seen in case of velamentous insertion of the cord.
* In velamentous insertion → cord is attached to the membranes.
* During labor → membrane will rupture → cord will also rupture
  ↓
  umbilical artery and umbilical vein rupture
  ↓
  Fetal bleeding.
* This fetal blood comes out through mother's vagina and is so confused with maternal blood (placenta previa)
* Diagnosis
  - In antenatal period → by Doppler
  - MCQ At the time of bleeding
    ↓
    To differentiate b/w fetal blood and maternal blood →
    Test: Apt test / Singers Alkali denaturation test:

    Based on principle → fetal blood is resistant to acid & alkali (HbF)

Maternal blood – HbA → which is sensitive to acid & alkali.
↓
Blood coming from maternal vagina → Add NaOH / KOH
↓
- If vasa previa → fetal blood → HbF → resistant to alkali → colour of blood remains same
- If placenta previa → maternal blood → HbA → sensitive to alkali → colour changed in to brown.

* Management of vasa previa:
  Cessarean section
* Prognosis → It leads to increased perinatal mortality not maternal mortality.

*Note

* There is another test based on the same ~~test~~ principle:
  HbF → Resistant to acid/alkali
  HbA → Sensitive to "

Kleihauer Betke Test
- Reagent used: Citric acid PO_4 buffer
- originally used to calculate dose of anti-D in Rh-ve ♀

Apt test
- KOH / NaOH
- To detect vasa previa or differentiate VP with placenta P.

* Kleibaur Betke ~~test~~ Apt test
Test

• Quantitative test	• Qualitative test
• Differentiate b/w fetal ~~blood~~ RBC and maternal RBC	• Differentiate b/w fetal blood & maternal blood

⇒ MC vascular anomaly of cord is
Single Umbilical artery (SUA)
↓
- MC in pregnant, diabetic, black ♀
- Finding of SUA is not insignificant
It is associated with many congenital malformations in fetus (cardiac & renal malformation)
- If SUA + Congenital malformation in fetus → check the karyotype → get trisomy.

Basics about pregnancy

* Total duration of pregnancy is
9 months + 7 days
or
40 wks or 280 days.

* If delivery (from LMP calculated)
1) 37 wks → 38 wk + 6 days
Early term delivery
2) 39 wks → 40 wk + 6 days
Term delivery
3) 41 wks → 41 wk + 6 days
Late term delivery
4) ≥ 42 wks
Post term delivery.

* Expected date of delivery (EDD) is calculated by
Naegle's formula

* EDD = 1st day of LMP + 9 months and 7 days.
- LMP (Last menstrual period)

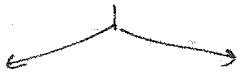
eg: LMP = 1st July
Missed period = 1st Aug
EDD = July $\xrightarrow{9m}$ 8th April 2018
(1+7)

* Only 4% ♀ deliver on their exact EDD
* 50% females deliver either 1 wk before or 1 wk after EDD
* Theoretically for delivery wait till 42 wks.
* Suppose a ♀ comes at 42 wks → post term pregnancy → first step to do - review her menstrual history
* Congenital anomaly leading to post dated pregnancy is
Anencephaly

Complications of post dated pregnancy

1) Amniotic fluid ↓ → ↓ in late pregnancy → cord compression → meconium aspiration syndrome

2) Wt. of baby ↑ → Macrosomia (excessive fat gets deposited in the shoulder area)



Vaginal delivery → Difficulty in delivery of the shoulder → Shoulder dystocia

Cesarean section

3) Placental ageing → fetal distress.

⇒ Management of post dated pregnancy

Induce labor.
(to initiate labor)

Gravida & Parity

* Gravida → used for pregnant ♀. It means total no: of times a female has become pregnant.
* Parity → No. of pregnancies which have gone beyond the period of viability

(Period of viability)

Earlier = 28 wks
Developed = 20 wks
India = 24 wks
WHO = 22 wks

If not mentioned ↓ 20 wks

* If a ♀ pregnant now for 2nd time, 1st baby – full term live baby then

G_2P_1

** Gravida & parity do not refer to the no: of babies

MLQ * If a ♀ pregnant now for 2nd time, 1st time → had twins at 34 wk

G_2P_1

* If a ♀ pregnant now for 3rd time, 1st time - abortion at 14 wks, 2nd time – abortion at 11 wks (period of viability 20 wks)

G_3P_0

* Second way of representing gravida & parity is

G_xP_{a+b}

x → No. of times pregnant
a → No. of pregnancies beyond the period of viability
b → No: of abortions.

* If a ♀ pregnant now for 3rd time, 1st time - abortion at 14 wks, 2nd time - abortion at 11 wks

G_3P_{0+2}

* 3rd way of representation: (GTPAL system)

$G_x P_{a+b+c+d}$

T P A L

T : Term pregnancy
P : Pre-term pregnancy
A : Abortion
L : No. of living children

MCQ
* If ♀ – 16 wks pregnant presently, H/O twins 2 yr old @ 39 wks, 5 year old @ 40 wks

$G_3 P_{2+0+0+3}$

Hormones in Pregnancy

1) Estrogen
2) Progesterone
3) HCG
4) HPL (Human placental lactogen)

* All synthesised by synctiotrophoblast

Estrogen

* Placenta cannot synthesize estrogen using precursor from mother. It is dependent on fetus for precursors

Fetal adrenal gland produce
↓
DHEA sulphate
↓
used by placenta to synthesize estrogen.

* Estrogen synthesized by placenta signals the uterus at term to start contracting
↓
labor is initiated.

⇒ For initiation of labour, need
a) maternal ACTH
✓b) Fetal ACTH
c) Prolactin
d) Progesterone

⇒ Which of following is produced exclusively by adrenal gland.
a) Testosterone
b) Androstenedione
c) DHEA
✓d) DHEA sulphate

MCQ
⇒ MC form of estrogen during pregnancy is
E_2 (estradiol)

MCQ
⇒ Most specific form of estrogen during pregnancy
E_3 (Estriol)

Progesterone

* Placenta can synthesize progesterone using maternal precursors

↓

Maternal LDL

↓ (placenta)

Progesterone.

* 2 main sources of progesterone during pregnancy are

(i) Corpus luteum

- Hormone maintains CL of pregnancy

HCG

- Life span of CL in pregnancy

12 wks [CL maintained by LH]

(Non pregnant ⇒ 12 days (life span))

(ii) Placenta

- It takes over function of CL : 8–10 wks.

* Progesterone is a smooth muscle relaxant.
* Due to progesterone:
  1) Decidual reaction
  2) Arias Stella reaction.
* Lack of progesterone during pregnancy : Abortion

(Luteal phase defect)

↓

MCQ DOC : Giving progesterone

HPL

* Also k/a HCS (Human Chorionic Somatotropin)
* Synthesised by syncitiotrophoblast
* It is responsible for insulin resistantce during pregnancy
* Also tells about placental well being.

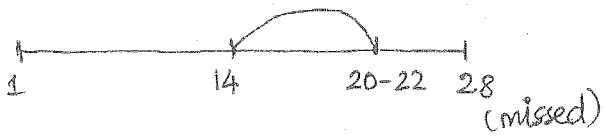
MCQ * Max. insulin resistance during pregnancy : 24–28 wks

MCQ * Which hormone is responsible for growth of fetus during pregnancy

Insulin & Insulin Like Growth factor.

HCG

* Synthesised by syncitiotrophoblast
* It has 2 subunits
  - α (non specific)
  - β (specific)
* α subunit (HCG) is similar to LH, FSH, TSH (anatomically)
* But functionally similar to LH
* Doubling time of HCG is 48 hours



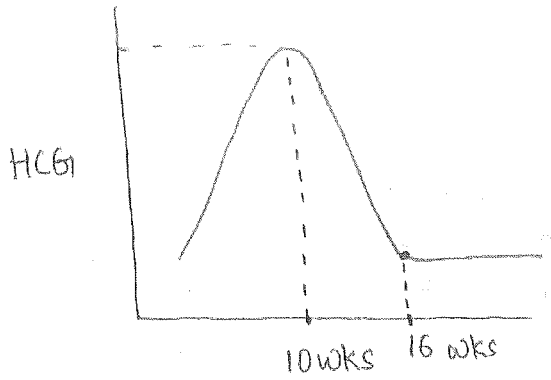
* HCG can be first detected on 6-8 days after fertilisation / Day 20-22 of cycle / 6 days before missed period.

* Most sensitive test : FIA > RIA

FIA → Fluoroscent Immuno Assay.

RIA → Radio Immuno Assay

* Maximum levels of HCG seen at 10 wks of preg. (70 days)



- Maximum at 10 wks
- Minimum at 16 wks which remains throughout pregnancy.

* HCG disappears 2 wks after delivery

* ↑ HCG levels in
  - Twin pregnancy
  - Molar pregnancy (H. mole / Chorio carcinoma)
  - Down syndrome (Trisomy 21)

* HCG level ↓ in
  - Abortion
  - Rest of trisomies

* In ectopic pregnancy, the level of hCG ↑ but not that much as they increase in normal pregnancy.

* In a non pregnant ♀, hCG is -ve

* Ectopic pregnancy : +ve (less)

* Normal pregnancy : +ve (more)

Preterm Labor

* Labour begins < 37 wks

* MCC of preterm labor (PTL)
  Idiopathic > Infections

* Infections :
  - Bacterial vaginosis
  - UTI
  - Asymptomatic bacteruria

* Lungs of fetus starts maturing at 34 wks and maturation is completed by 37 wks

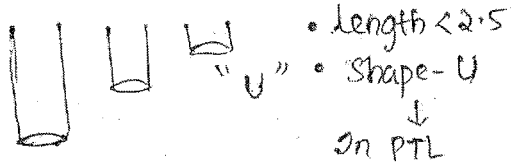
* Problem with PTL → Lungs of fetus are not mature

PTL
- ≥ 34 - 37 wks → Lungs of baby almost mature
- < 34 wks → Lungs not mature

Diagnosis of PTL

1) TVS

* Normal length of cervix during pregnancy is 4 cm
* When contraction occurs, as the cervix dilates, length of cervix keeps on decreasing



* Cut off length of Cx to predict PTL is 2.5 cm.

2) Fetal fibronectin protein

If it is present in amniotic fluid at < 37 wks → indicates PTL

⇒ MC risk factor of PTL
Previous H/O PTL in the ♀

⇒ Prophylatic DOC to prevent PTL
Progesterone
(smooth muscle relaxant)
↓
But cannot be used to Rx. PTL
It is not tocolytic

⇒ Other methods to prevent PTL
1) Quit smoking
2) Apply cervical Cerclage (used in abortion) → only in PTL & in cervical incompetence.

Management of PTL

1) PTL → b/w 34-37 wks
↓
Lungs of baby mature
↓
No risk

Mgt : Do nothing, wait & watch

New concept says by ACOG

Btw 34-37 wks
↓
planning vaginal delivery
↓
Do nothing

B/w 34-37 wk
↓
Planning cessarean (eg: Placenta previa)
↓
Have to give corticosteroid

2) If PTL < 34 wks
↓
Lungs of baby not mature, so respiratory distress syndrome
↓
Also chances of neurological damage in baby.

Mgt : If lungs not mature
↓
DOC : Corticosteroid

Worldwide
↓
DOC is Betamethasone

India
↓
DOC is Dexamethasone (cheap)

MCQ

Dose of Betamethasone *

* 2 injections, 24 hr apart, 12 mg each

12 mg each $\xrightarrow{24\ hrs}$ 12 mg

MCQ

Dose of Dexamethasone

* 4 injections, 6 mg each, 12 hr apart

6 $\xrightarrow{12\ hr}$ 6 $\xrightarrow{12\ hr}$ 6 $\xrightarrow{12\ hr}$ 6

⇒ Route of both : i.m

⇒ For lungs to mature, ideally should wait for 24 hr after last injection

⇒ Only C/I : Chorioamnionitis

Benefits of Corticosteroids

* Prevents RDS
* Prevents Necrotising Enterocolitis
* Prevents intraventricular hemorrhage

⇒ DOC for preventing neurological damage in preterm is $MgSO_4$.

Complete Mgt of PTL < 34 wks

1) Corticosteroid (need atleast 24 hr) for lungs to mature
+
2) Short term tocolytics (prevent labor before 24 hrs)
+
3) $MgSO_4$

~~$MgSO_4$~~

⇒ No role of
1) Progesterone
2) Antibiotics (unless the membrane are ruptured)

⇒ Nifedipine is a tocolytic

Tocolytics

* Best / most effective
Nifedipine
* Best tocolytic in heart disease pt
↓ ATOSIBAN > $MgSO_4$
(Oxytocin antagonist)
* Tocolytic with max. maternal s/e
β-agonist.
* β-agonists which are used as tocolytics
1) Isoxsuprine
2) ~~Retrodr~~ Retrodrine
3) Salbutamol
4) Terbutaline
* MC s/e of β-agonist:
Tremors
* Other s/e of β-agonist
- Hyperglycemia
- Pulmonary edema

* So they are C/I in diabetic ♀ → tocolytic of choice in them
Nifedipine

* Tocolytic with max. s/e on fetal ~~cardiac~~.
Indomethacin
(closure of ductus arteriosus)

* $MgSO_4$ acts as a tocolytic at 9-10 meq/L

Test for lung maturation

* MC done → Lecithin / Sphingomyelin ratio in amniotic fluid
MCQ
↓
If L/S ≥ 2 → Lungs mature
L/S < 2 → Lungs not mature

* Best test done → To check the presence / absence of phosphatidyl glycerol in amniotic fluid.
MCQ
↓
if it present → Lungs mature
if not present → Lungs not mature.

(Ques will be like best test done in diabetic ♀ → NO matter which ♀, best test - Phosphatidyl G +nt/-nt)

Antepartum Hemorrhage

* Any bleeding which occurs from the female genital tract beyond the period of viability and uptill delivery.

Causes

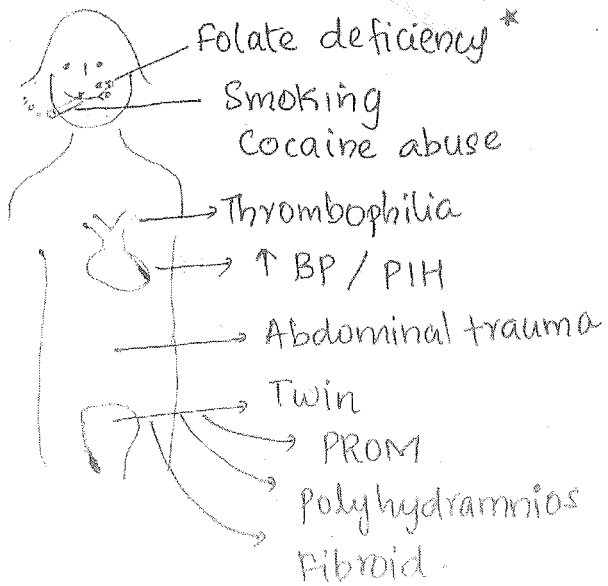
Placental causes	Fetal cause
1) Placenta previa	Vasa previa
2) Abruptio placenta	
3) Circumvallate placenta	

Placenta previa	Abruptio placenta ~~previa~~
* Placenta is located in the lower uterine segment. Best time for USG = 3rd trimester	* Placenta is located normally in the upper segment ~~of~~ but it seperates prematurely.
* Incidence is 1 in 300 pregnancies	* Incidence is 1 in 200 (more common than P. previa)
* Recurrent rate in next pregnancy 5%	* Recurrence rate 12%
* Risk factors: 1) MC is previous H/O p. previa 2) Highest risk is previous H/O C-section (3 times)	* MC risk factor previous H/O abruptio placenta

* Common risk factor for both:
  1) Smoking
  2) Twin pregnancy
  3) ↑ maternal age
  4) ↑ maternal parity

* Other causes of placenta previa
  (i) Due to endometrial damage
    → Endometritis
    → Smoking
    → Dilatation & curretage
    → Myomectomy scar.
  (ii) Due to large size of placenta
    → Twin pregnancy
    → Succenturate lobe
      Placenta bilobata
      Placenta spuria.

* Other causes of abruptio placenta



* PIH also K/A PET (Pre Eclampsic Toximia)

* Classification of placenta previa.

1) Type 1:
Placenta is in the lower uterine segment but it does not reach uptil internal os

Lateral P.P

2) Type 2:
Placenta reaches the margin of internal os.

Marginal P.P

3) Type 3:
Placenta covers the internal os incompletely

Incomplete PP

4) Type 4:
Placenta covers the internal os completely

Complete PP

* In type I & II the placenta can be attached to either anterior wall of uterus / the posterior wall of uterus

Type 1 < Ant. / Post } Minor degrees (can try vaginal delivery)

Type 2 < Ant

Post → Dangerous variety of PP (as it provides less space for fetus to come out)

↓

Do cessarean section

↑

Type 3 } Major degrees
Type 4 }

* Note:

In posterior varieties of placenta previa (ie, type I post. & type II post.)

↓

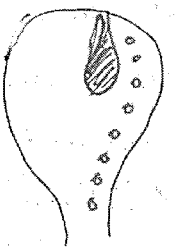
Stallworthy sign is +ve

Abruptio placenta

* Classification k/a Page classification — MCQ

* Varietes of Abruptio P

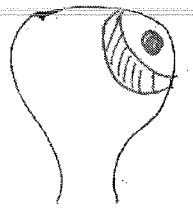
1) Revealed variety



(Blood comes out) (visible)

2) Concealed variety

Bleeding is not getting space to come out



Here blood collects behind the placenta → enters uterine myometrium → uterus starts appearing wine / bluish in colour.

↓

Such a uterus is k/a Couvelaire uterus

↓

It is not an indication for doing hysterectomy

- Complain / History in placenta previa
  - Causeless
  - Painless
  - Recurrent bleeding
  - Initially there is less bleeding k/a warning hemorrhage.
    ↓
    followed by excessive bleeding.
  - Bleeding is bright red in colour.

- complain / History of Abruptio P
  - H/O trauma or H/O PIH
  - Abdominal pain present
  - Not recurrent in same pregnancy (once starts, deliver the baby)
  - No warning hemorrhage
  - Bleeding is dark red in colour.

* In general examination of placenta previa → condition of patient & amount of blood loss (less bleeding - No shock, more bleeding - shock)

* In concealed variety of Abruptio placenta → The condition of patient & amount of blood lost do not correspond with each other.

* In placenta previa, on per-abdominal examination
  ↓
  Uterus is soft, relaxed and non-tender
  - fetal heart sound ↓ easily heard
  - fetal parts ↓ easily palpable

* In case of concealed A.P
  Uterus is rigid, tensed and tender
  ↓
  So fetal heart sound is not easily heard
  &
  Fetal parts not easily palpable.

* In placenta previa:
  Mostly fetus is alive

In Abruptio P
  Mostly fetus is dead

* In placenta previa:
  Height of uterus = Period of gestation.
* In Abruptio placenta
  Because blood collects in uterus → so height of Uterus more than period of gestation.
* Pervaginal examination is C/I in p. previa.
* Pervaginal examination is C/I (also in)
  - PROM
  - Virgin females
* In placenta previa
  IOC → Transvaginal US (TVS)
  (because probe in vagina only, not going to damage it by enter more deep in to it)
* In Abruptio placenta:
  IOC: Clinical diagnosis >> TVS
* In P. previa:
  - IUGR is not present
  - Fetal death is less
  - NO DIC

* In Abruptio P
  - Because PIH is risk
  ↓
  In PIH, maternal BP ↑ (P∝V)
  ↓
  V of blood to fetus ↓
  ↓
  fetal growth ↓
  ↓
  IUGR seen.
  - Fetal death is common
  - Can lead to DIC

Management of Abruptio placenta

* Principles:
  1) Never wait & watch
  2) Never give tocolytics
  ↓
  Always: Termination of pregnancy (because DIC risks mother life)

Prefered mode of delivery
↓
Vaginal

Indication of C-section
1) Fetal distress
2) Pregnancy <32wk

* In Abruptio P → DIC → first correct DIC then vaginal delivery

Management of Placenta previa

Terminate the pregnancy immediately

↓

k/a Active Management.

↓

In any condition where mother's life is at risk.

Continue the pregnancy

↓

k/a Expectant management

↓

Purpose: To let the fetus attain lung maturity (37 wks)

↓

Condition: Never putting mother's life at risk.

↓

* If on USG, found congenital anomaly in fetus which is incompatible with life (eg: Anencephaly)

↓

* If on USG, (N) fetus or congenital anomaly compatible with life (eg: Polydactyly)

Steps of expectant management

Macaffee & Johnson regime

1) Hospitalize
2) Arrange for blood
3) Rh-ve → Anti D
4) inj. corticosteriods → hasten lung maturation of fetus
5) Any contraction +nt → short term tocolytics (Nifedipine)

↓

Carry upto 37 wks

↓

Deliver / Terminate her pregnancy.

Active management	Expectant management
* When patient is hemodynamically unstable	* Hemodynamically stable patient.
* If there is continous bleeding present	* If the bleeding has stopped.
* If gestational age ≥ 37 wks or if patient is in labor	* Gestational age ≤ 36 wks & hemodynamically stable patient
* If fetal distress is present (heart sound not well heard)	* NO fetal distress
↓	↓

Mode of delivery in P. previa

* Type of p. previa identified
  - by USG
  - Double setup examination (pervaginal examination in the OT)

Type of P. previa

- Type I → Ant / Post
- Type II – Post
  - ↓
  - k/a minor degrees of P.P
  - ↓
  - Vaginal delivery
- Type II – post (k/a dangerous variety)
- Type III
- Type IV
  - ↓
  - Major degrees of P.P
  - ↓
  - Cesarean section

DIC:

* It is k/a consumptive coagulopathy.
* Because all clotting factors are consumed.

* Obstetrical causes of DIC
  1) Abruptio placenta
  2) Intrauterine death of fetus
  3) Amniotic fluid embolism
  4) Septic abortion
  5) Severe pre-eclampsia / Eclampsia / HELLP syndrome

* ~~Clotting factors~~ Diagnosis:
  - Serum fibrinogen (CF-I)
    - ↓↓
  - Fibrin degradation products ↑↑

* Drug in management of DIC
  - Blood transfusion
  - Fresh frozen plasma
  - Cryoprecipitate
  - Platelet transfusion
  - Heparin +/-

MCQ
* EACA (Epsilon Amino Caproic Acid) is not used in the management of DIC

MCQ
* Whenever DIC occurs → first correct DIC → then vaginal delivery (not C-section)

Post Partum Hemorrhage (PPH)

* MCC of maternal mortality
* Prevention of PPH is must
* Manage the 3rd stage of labor actively.
* 3rd stage of labor → Time interval in delivery of baby & placenta.
* Main action in 3rd stage
  Delivery of Placenta
* Human placenta is deciduate
  ↓
  shed off after delivery
* In the 3rd stage of Labor
  ↓
  Management
  - Passive
    ↓
    Waiting for placenta to deliver spontaneously
  - Active
    ↓
    Active mangament of 3rd stage of labor
    ↓
    AMTSL

* Human placenta is deciduate
  ↓
  shed off after delivery
* In the 3rd stage of labor
  ↓
  Management
  - Passively
    ↓
    (Doing nothing)
    Waiting for placenta to deliver spontaneously.
  - Actively
    ↓
    Active management of 3rd stage of labor (AMTSL)

* Time taken in passive mgt
  15-20 minutes
  - Bleeding ↑
  - PPH ↑
  - Chances of maternal mortality ↑
* Time taken in AMTSL = 5-10 min
  - Bleeding ↓
  - PPH ↓
  - Chances of maternal mortality ↓
* AMTSL → It is the preffered management.

* Prolonged 3rd stage of labor
↓
> 30 min
↓
Retained placenta
(also means if placenta has not delivered ≥30 mins after the delivery of baby)

* AMTSL has all advantages
* Only advantage → can lead to retained placenta.

Components of AMTSL

1) Injection uterotonic after the delivery of baby (to mother)
2) Delayed cord clamping
3) Delivery of placenta by controlled cord traction/ modified BRANDT ANDREWS technique.
4) Intermittent uterine tone assessment
(earlier: Uterine massage)

Injection uterotonic (All MCQs)

* Uterotonic recommended by WHO → Oxytocin
* Oxytocin can be natural (post pituitary) or synthetic
* t½ of Oxytocin: 3-4 min
* Can be i.m / i.v infusion
* Never be given iv bolus (leads to ↓BP, cardiac arrest)
* Leads to ↑ Uterine contraction (physiologically)
↓
used to augment Labor
* Milk secretion: Prolactin
* Milk ejection: Oxytocin

⇒ If oxytocin is not available

Alternatives

- Methyl ergometrine / ergometrine
↓
Methergin
- PGE_1 → Misoprost
- PGF-2α → Carboprost
- Syntometrine
↘ Oxytocin + Methylergometrine
- Carbetocin - Synthetic oxytocin

Methylergometrine

* It leads to increase in BP (never used in PIH patient)
* It leads to tetanic contractions in uterus
(never be used during pregnancy, before fetus is born)
↓
so never used for augmenting labor. (accelerating)

C/I for Methyl ergometrine/ Methergine

T - After the delivery of 1st twin
O - Organic heart disease
P - Pre-eclampsia } ↑ BP
E - Eclampsia }
R - Rh incompatibility (Rh-ve mother, Rh+ve fetus)

⇒ Here in all cases can use oxytocin
⇒ After delivery → uterus contracts → more blood to heart → ↑ CO → worsen heart disease in mother.

Delayed cord clamping

* Clamp the cord within 1 min of delivery → Early cord clamping
* Clamp cord b/w 1-3 min of delivery → delayed cord clamping → extra 80 ml blood in cord goes to baby = 50 mg of Fe → prevent neonatal anemia
* Normally we prefer delayed cord clamping
* Early cord clamping preferred in
  1) Baby needs resuscitation
  2) Mother is Rh-ve & baby Rh+ve
  3) If diagnosed heart disease in baby
* In preterm babies and HIV +ve mother → now recommended that delayed cord clamping should be done not early cord clamping (it was used earlier)

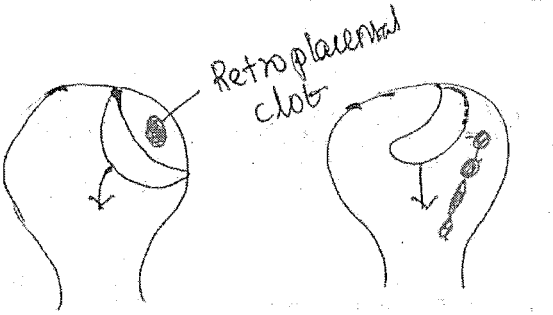
Delivery of placenta by Controlled cord traction

* If the placenta has not seperated from the uterus
  ↓
  cord is pulled
  ↓
  leads to uterine ~~contraction~~ inversion

* MCC of * uterine inversion is Mismanaged 3rd stage of ~~labour~~ labor.
* There is neurogenic shock + hemorrhagic shock → MCC of death in uterine inversion
* A woman after delivery goes in to severe shock, most probable cause → PPH
* A woman after delivery immediately goes in to shock, most probable cause → uterine inversion

Management of uterine inversion

Reposition of uterus normally (with hand)
↓
Johnson method
↓
if fails
↓
OT & ↓ GA (General Anesthesia) ⇒ 4th step of AMTSL
↓
HAULTAIN surgery



Schultz Duncan

Signs of Placental seperation

1) Gush of bleeding
2) Suprapubic bulge
3) Height of uterus ↑ slightly
4) Permanent lengthening of cord.

Methods of placenta seperation

Schultz method
- Placenta seperates from centre
- MC
- Fetal side of placenta comes first (fetal side - shiny)

S < shiny schultz

- Retroplacental clot is formed, so less bleeding

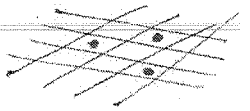
Duncan method
- Placenta seperates from periphery
- Maternal side of placenta comes out first, maternal side - dull

D < dull Duncan

- No clot formed so more bleeding

MCQ
* MCC of PPH ↓ uterine tone

MCQ
* Living ligature → middle layer of myometrium is that



* Out of all 4 components of AMSTL → Most important →
1st step: inj. uterotonic

PPH

* Post partum hemorrhage
* Blood loss ≥ 500 ml after vaginal delivery
≥ 1000 ml after C-section

↓

Till 12 wks after delivery

PPH

1° PPH → Occuring within 24 hr of delivery

2° PPH → after 24 hr - 12wk of delivery

MIA

* Severe PPH is when blood loss ≥ 1000 ml.

* Causes of PPH: (4T)

1) T - ↓ tone of uterus (Atonic uterus - MCC of PPH)
2) T - Trauma (2nd MCC)
3) T - Thrombin (Any bleeding disorder) (HELLPsyn / DIC)
4) T - Tissue (Retained tissue eg: Retained placenta)

MCQs

* MCC of PPH is Atonic uterus
* MCC of 1° PPH → Atonic uterus
* MCC of 2° PPH → Retained placenta
* All placental anomalies can lead to PPH

P. succenturata, P. bilobata, P. spuria.

Risk factors for PPH

1) If uterus is overdistended
   - Multifetal pregnancy
   - Polyhydramnios
   - H. mole
   - Multigravida
2) If Labor is prolonged (>12 hr)
3) Precipitate labor (entire labor occurs within 3 hrs)
4) If labor has not been conducted properly
   - Overused anesthetic agent
   - Oxytocin
5) If female is anemic
6) Forceps delivery → Traumatic PPH.

Management of PPH

* 1st step → General resuscitative measures
  1) i.v lines
  2) Blood arrangement
  3) Catheterization
  4) ABC
* 2nd step → Identify type of PPH: Atonic / Traumatic

Because MCC of PPH are atonicity & trauma → both has different management

Try to palpate the uterus per abdominally

If uterus can be palpated
↓
Traumatic PPH (either vaginal tear or cervical tear)
↓
Take patient to OT & repair tear.

If uterus cannot be palpated
↓
Atonic PPH

Management of Atonic PPH

1) Medical management (drugs)
↓ (if fails)
2) Compression / Mechanical methods
↓ (if fails)
3) Surgical method

Medical management

* Drugs ↑ uterine tone k/a oxytocics or uterotonic drugs
* 1st drug : Oxytocin / Syntocinon
  2 : Methylergometrine / Methergin
  3 : Syntometrine (Oxy. + Methergin)
  4 : Prostaglandins

* Dosages

Drug	To prevent PPH (3rd stage)	To treat PPH
(Best) Oxytocin	10 IU	30-40 IU
Methergin	0.2 mg (i.m)	0.2 mg (i.m) repeat after every 15 min
Carboprost (PGF-2α)	250 mcg (i.m)	250 mcg (im) 8 doses max. at 30 min interval s/e : Diarrhea
Misoprost (PGE_1)	* 600 mcg per oral (MCQ)	* 800 mcg sublingual / per-rectal (MCQ)

Prostaglandins

PGE_2 (analogue)

* k/a Dinoprost
* Main action on Cervix
* Helps to soften cervix during labor k/a ripening of cervix
* Also used in inducing labour.
* PGE_2 does not acts on uterus, so it is not used for PPH management.

PGF-2α

* It is Carboprost
* Also K/a Hembate
* Acts on uterus
* Helps in managing PPH
* Since not acts on cervix, cannot used for inducing labor.

PGE 1

* K/a Misoprost
* Acts both on cervix & uterus
* Used for
  - inducing labor
  - inducing abortion
  - ripening of cervix
  - managing PPH.

MCQs

• PG analogue C/I in bronchial asthma

PGF-2α

(can be use PGE-1 here)

• PG analogue C/I in scarred uterus (previous C-section scar)

PGE_1 (Misoprost)

⇒ If bleeding is not controlled by drugs → go for mechanical method.

→ i.e, compression of uterus by

• Bimanual compression of uterus (one hand through vagina, inside uterus, other hand above abdomen)

• SB trans oesophageal catheter

↓

MCQ 200-500 ml of warm saline

↓

If not available then condom catheter/ BAKRI balloon catheter

⇒ If it not works then go for surgical method.

If ♀ completed her family

↓

• Remove Ute + Cx

TAH / Simple hysterectomy

• Removing only uterus

↓

Subtotal hysterectomy.

If family not completed

↓

B-LYNCH suture

↓

• It is applied on uterus
• Used for atonic PPH

* If B-LYNCH fails → ligate/ embolize
  - Uterine artery
  - Ovarian artery.
  - Ant. division of int. iliac A (uterine A branch of it)

* If still bleeding continues
↓
do hysterectomy.

Shock index

* Shock index = $\frac{\text{Heart rate}}{\text{sys. BP}}$

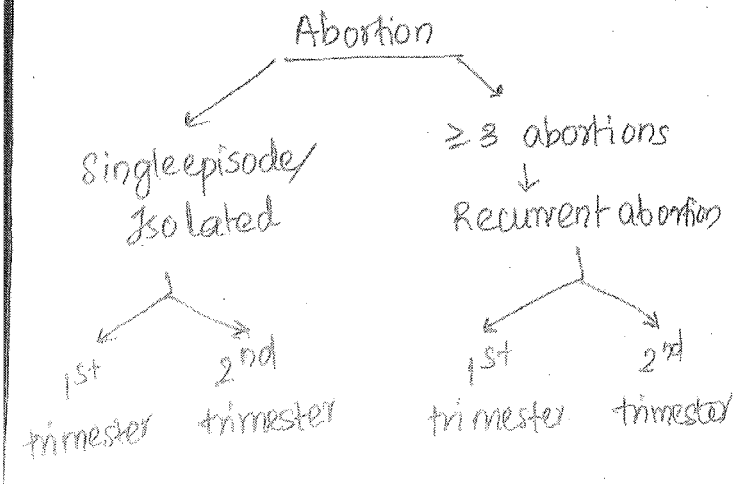
* (N) 0.5 – 0.7

* If shock index becomes
0.9 to 1.1 → severe
hemorrhage → blood is needed.

ABORTION

* It is interruption of pregnancy/ death of fetus before 20 wks of pregnancy (period of viability) or before is 500 gm in wt.

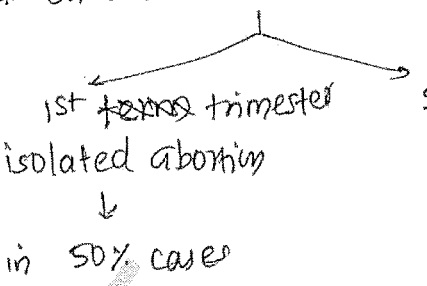
* After period of viability → Rla intra uterine death.



* MC time of abortion ≤ 8 wks

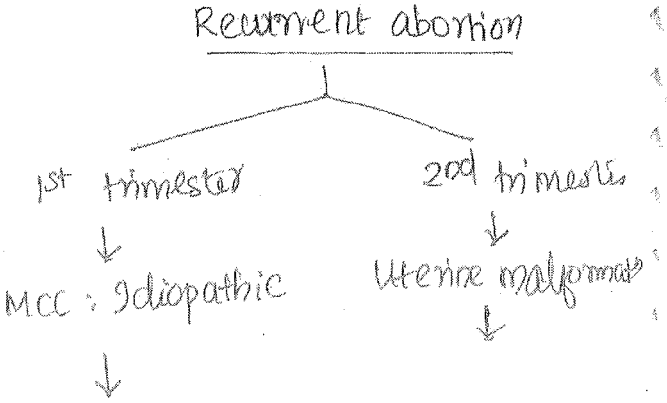
* MCC of isolated abortion
– 1st / 2nd trimester
Chromosomal anomaly / Germplasm defect.

* Chromosomal anomalies can lead to



* MC chromosomal anomaly causing isolated abortion
1st best ans. → Aneuploidy
2nd " → Trisomy
3rd " → Monosomy X (20%)
4th " → Trisomy 16 (16%)

* Infections can lead to a single episode of abortion (not recurrent)



(1st trimester)

↓

Immunological - Antiphospholipid Ab syndrome (APLA syndrome)

↓

Then endocrinal causes
- Diabetes
- Hypothyroidism
- ↑ prolactin level

↓

Chromosomal anomaly -
Balanced translocation of chromosomes.

(2nd trimester)

↓

They can be congenital or acquired

↓

In congenital - Septate / bicornuate uterus

Acquired causes is (MCC) Cervical incompetence

↓

Other cause is Rh-ve pregnancy

⇒ Should karyotyping is done in recurrent abortion
Yes
(of aborted material)

⇒ MCC of 2nd trimester abortion
Cervical incompetence.

Note (MCQs)

* Infections (TORCH) can never lead to recurrent abortions.
(So TORCH test is not done)

* Only one infection leads to recurrent abortion → Syphilis
Test : VDRL

Antiphospholipid Ab syndrome

* 3 types of Ab (any 1 present)
1) Lupus anticoagulant Ab (MC)
2) Anticardiolipin Ab
3) β_2 microglobulin Ab

* Lupus anticoagulant → it is a misnomer → it leads to thrombosis in all blood vessels → placental blood vessels.
~~→ obstruction there~~

↓

Leads to

1) PIH (↑ pressure due to obstruction)
2) IUGR (↓ blood supply to fetus)
3) If blood supply to fetus stops
⇒ At < 20 wks → Abortion
⇒ > 20 wks → IUD
⇒ during delivery → still birth
4) Small placenta (blood supply to placenta ↓)

* Diagnosis:
i) Detect the antibodies
ii) Lupus anticoagulant ↑ APTT (Activated partial thrombin time) but no effect on PT (Prothrombin time)
iii) Prolonged russal vipes venom clotting time

Mgt

* Heparin & Aspirin

Cervical incompetence

* MCC of second trimester reaurrent abortions
* Characterized by sudden painless dilatation of cervix, rupture of membranes & expulsion of fetus
* MC time → 16-24 wks of abortion
* Diagnosis:

= In a pregnant ♀, diagnosis by
- History
- TVS

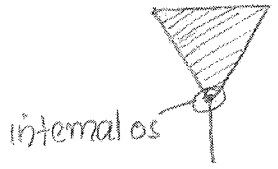
TVs shows:

	Normal ♀	Incompetence of os
Cx length	40 mm	≤ 25 mm
Diameter of int. os	< 8 mm	≥ 8 mm
Shape of Cx		U shaped

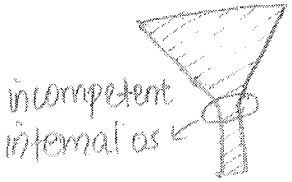
= In non pregnant ♀ → pass Hegar dilator no: 8 without ♀ offering any resistance → indicates cervical incompetence

↓

Next is do a hysterocervicography (dye in to uterus using foley's catheter)



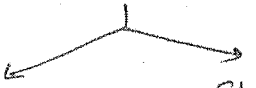
Normal (dye not to Cx & vagina)



Funnel shaped appearance

* Mgt:

circlage surgery (suture at level of int os)



McDonald circlage

Shirodkar circlage

* Time of Sx → 14-18 wks
* Removal of suture → when patient goes in to labor or at 37 wks

Types of Abortion

Type	Definition	Symptom	P/A (Per abdominal examination)	Int os	USG
1. Threatened abortion	Process of abortion has begin, still it is reversible	spotting	Size of Uterus = Period of gestation	Closed	Live fetus
2. Inevitable abortion	Abortion is not reversible	• Bleeding +/- • Pain abd • No H/O product of conception coming out	Size of Uterus = Period of gestation	Open	Dead fetus
3. Incomplete abortion	Process of abortion begins but it is incomplete	• Bleeding • Pain • ~~Product of~~ H/O passage of product of conception	Ht of Uterus will be less than period of gestation	Open & can see product of conception are coming out through it	Dead incomplete fetus
4. Complete abortion	Process of abortion is complete	Initially bleeding + product of conception comes out & now bleeding has stopped	Ht of Uterus less than the period of gestation	Closed	Empty uterus
5. Missed abortion	Fetus is dead but patient is unaware	Dirty brown discharge	Ht of uterus less than the period of gestation	Closed	Dead macerated fetus

⇒ In all Rh-ve females, after abortion, Anti-D should be given.

⇒ If abortion → ≥ 12 wks
↓
Dose of Anti-D is
300 mcg (i.m)
(in all types of abortion)

⇒ If abortion → ≤ 12 wks
↓
Dose of Anti-D is
50 mcg (i.m)
↓
Except
- Threatened abortion
- Complete abortion
(don't give Anti-D)

MTP

* Medical termination of pregnancy
* MTP Act given in 1971
* But came in to action in 1972
* In India, MTP is legal upto 20 wks of pregnancy
* MTP ≥ 12 wks → opinion of 2 doctors is essential (sex of baby identified)

* MTP → In 1st trimester
↓
- Best upto 7 wks: Medical abortion
- 7-15 wks: Suction evacuation
- Others are
  - Menstrual regulation
  - Manual vaccum aspiration
  - Dilatation & Curretage (D & C)

* MTP in 2nd trimester:
- Best ≥ 15 wks is Use of prostaglandins (PGE_1 / PGE_2)
- Others are:
  - Oxytocin
  - Extra amniotic ethacridine
  - Intra amniotic saline
  - Suction using ovum forceps
  - ~~Hysterectomy~~ Hysterotomy (opening uterus & taking out product of conception)

Medical Abortion

* According to WHO/ACOG → medical abortion can be done uptill 9 wks
* In India → Uptill 7 wks.

< 7 wks

* On Day 1 give 200 mg of oral mifepristone (1 tab) (send her back home)
* On day 3 = 400 mcg of Misoprost (oral / vaginal) (400 mcg = 2 tabs)
* Observe for 2-3 hrs & send her home ~~back~~.
* Day 15 → Do a clinical exam? to ensure complete abortion
* In < 1% cases → curettage will be required to complete the process.

7-9 wks

* Day 1 = 200 mcg of oral Mifepristone (1 tab)
* Day 3 = 800 mcg of Misoprost (vaginally) (4 tabs)
* Day 15 → Do clinical exam? to ensure complete abortion

(If not → do D & C)

* Misoprost is teratogenic → leads to congenital malformation

MCQ

↓

Mobius syndrome

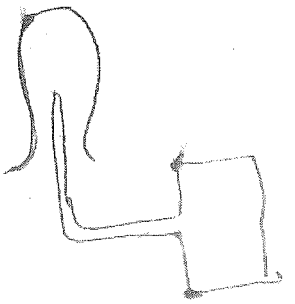
Suction Evacuation

MCQ

* Plastic canula = Karmans canula (size = period of gestation / 1 less than it)

MCQ

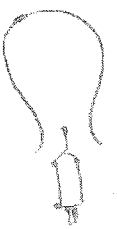
* Pressure = 600 mm Hg



* If in a rural setting → No electricity → then

7-12 wks : Manual vaccum aspiration

↓



60 ml syringe to suck out all product of conception

↓

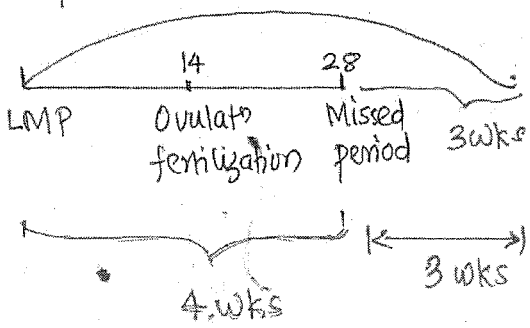
Pressure : 600 mmHg

↓

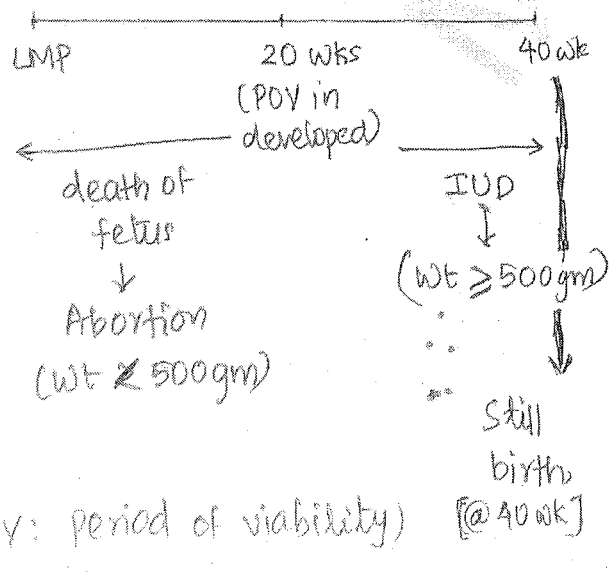
done b/w 7-12 wks

Upto 7 wks

* Best method : Medical abortion
* Alternative / Not best method :
  Menstrual regulation
* Here Karman canula is used → but no suction machine.
* Using canula, product of conception is taken out



* Here 7 wks of pregnancy is = 3 wks after the missed period



(POV: Period of viability)

IUP

* Death of fetus > 20 wks
* wt of fetus ≥ 500 gm
* Signs of IUP:

Clinical → Fetal movement (- nt)
Clinical → Fetal heart sound (-nt)

* Best investigation to detect IUD → USG
  (Fetal heart sound → Steth / Doppler)
* On USG → Cardiac activity of
  - fetus will be absent
  - Amniotic fluid ↓ (color - tobacco juice)
  - Overlapping of skull bone k/a Spalding sign

* On x-ray, some signs are seen

	Description	Seen at
• Spalding sign	Overlapping of skull bones of fetus	Within 1 wk of fetal death
• Robert sign (1st sign)	Presence of gas in great vessels of fetus	Within 12 hrs of fetal death
• Ball sign	Hyperextension/ hyperflexed of fetal spine	After 3-4 wks of fetal death

Management of fetal death

* Induce labor
* Best mode of delivery is
  Vaginal
  ↓
  Except if the pelvis is contracted or dead fetus in transverse lie
  ↓
  Here mgt is C-section.
* Milk secretion hormone: Prolactin
  In this case give ♀
  Bromocriptine / Cabergoline
  ↓
  to stop milk secretion
* After fetal death, it takes 4 wks to DIC happens.

Diagnosis of Pregnancy

Symptoms of first trimester: (1-12 wks)

U - Urinary symptoms (uterus is pelvic organ → irritate bladder → ↑ frequency of urine.

Are - Amenorrhea
My - Morning sickness (HCG)
Best - Breast discomfort
Friend - Fatigue

Signs of early pregnancy

1) Goodell sign: Softening of cervix.
   It is the earliest sign to become +ve → at 6 wks of pregnancy.

2) Hegar sign:
   Because isthmus is empty and soft during pregnancy
   ↓
   Do a bimanual examination (one hand in vagina & other behind uterus perabdominally
   ↓
   fingers of both hand can touch each other
   ↓
   at 6-10 wks of pregnancy

⇒ Rest all signs +ve at 8 wks

- Jacquiemer sign / Chadwick sign
  Bluish discolouration of the vagina.
- Osiander sign:
  Pulsations can be felt in the lateral fornix of vagina.
- Palmer's sign:
  Regular rhythmic contractions of uterus in 1st trimester

Size of uterus in 1st trimester

* @ 6 wks → = to size of hens egg
* @ 8 wks → " cricket ball
* @ 12 wks → " fetal head

Position of uterus

* Uterus is inside pelvis in 1st trimester
* It cannot be palpated perabdominaly
* At 12 wks → uterus lies at the level of pubic symphysis

2nd trimester (13-28 wks)

* Urinary symptoms are relieved as uterus becomes an abdominal organ
* Quickening → Perception of fetal movement by mother
  - Primigravida - is felt @ 18 wks
  - Multigravida → felt @ 16 wks

Signs of 2nd trimester

① * Colostrum can be expressed by 16 wks
* K+, Fat, Carbohydrates - KFC
* Colostrum is rich in all ingredients
* in comparison to breast milk, except KFC (K+, fat, carbohydrate) (less in colostrum)

② Braxtons hicks contractions are present

They are painless contractions which are irregular → throughout pregnancy from 2nd trimester onwards.

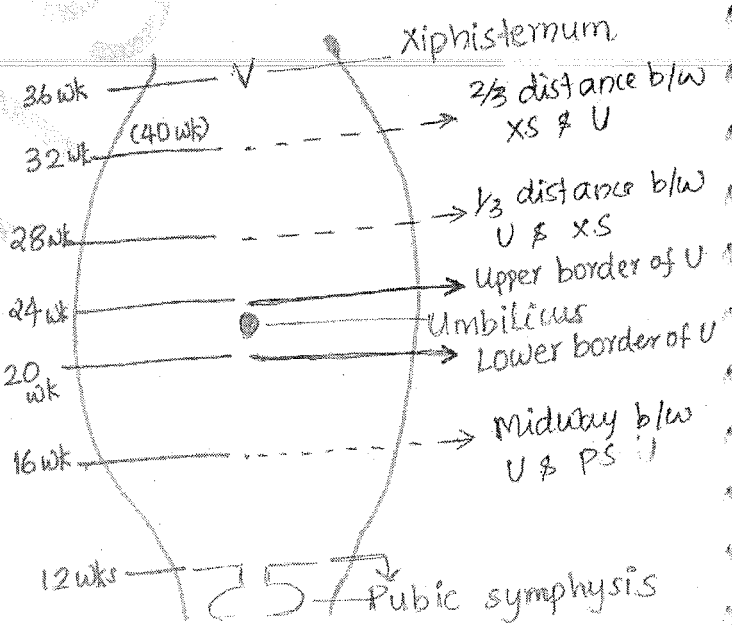
↓

Towards the end of the pregnancy → become painful → false labor pains.

③ Fetal parts can be palpated at 20 wks.

④ Fetal heart sound can be heard by
- Doppler : 10-12 wks
- Stethescope : 18-20 wks

Height of the uterus



* If fetal head is inside abdomen → 32 wks
* If head of fetus goes inside pelvis → 40 wks
* Both 32 wks & 40 wks same level

* At 36 wks, height of uterus is at the level of xiphistemum → so a pregnant ♀ experiences lot of respiratory discomfort

↓

After this head of fetus goes down in to pelvis → k/a Engagement

↓

So height of uterus ~~comes~~ falls & come down to 32 wks height

↓

Because of this ♀ have relief from resp. discomfort

↓

k/a Lightening

↓

Symptom of 3rd trimester

Symptoms of 3rd trimester

1) Lightening
2) Because head of fetus goes down in to pelvis → it irritates bladder → ♀ will ↑ urinary frequency.

So ↑ urinary frequency is seen in 1st & 3rd trimester

Definitive signs of pregnancy

1) Presense of fetal movement
2) Presence of fetal heart sound by steth / doppler
3) On USG → see fetus, cardiac activity etc..
4) X-ray → see fetal skeleton by 16 wks

↓

C/I in pregnancy

⇒ Pseudocyesis: False pregnancy (she starts assuming as pregnant)

↓

Here definitive ~~sound~~ signs of pregnancy will never seen.

Antenatal visits

Ideally

- Every month uptill 28 wks
- Every 2 wks uptil 28 - 36 wks
- After 36 wks → delivery → visit every week

Total 12-15 visits

* WHO says, minimum 4 visits

1st wk → 16 wk (most congenital malformation detected)

2nd → 24 - 28 wks (gestational (heart disease also) diabetes)

3rd → 32 wks (localize placenta)

4th → 36 wks (pelvis assessment)

* In India → rural → minimum is 3 visits → can skip 2nd visit as heart disease & diabetes not common

* Booking visit → First antenatal visit.

* Booked case → If the ♀ visited the hospital for atleast 3 times (3 antenatal visits) (in Govt. hospitals)

MLA

Que: All of the following vaccines can be given during pregnancy except

a) Hepatitis
b) Tetanus
✓c) Cholera
d) Rabies

a) Hepatitis
✓b) BCG
c) Cholera
d) Rabies

Vaccination during pregnancy:

* All killed vaccines can be given during pregnancy
* All live vaccines are C/I

Absolutely safe vaccines	Given only during epidemics
H = Hepatitis I = Influenza T = Tetanus Rabies = Rabies	Tab - Typhoid P - Pneumococcus C - Cholera M - Meningococcus

Given only when ♀ travelling to an endemic area.	Absolutely C/I vaccines
• Yellow fever • Japanese encephalitis • Polio	• M - Mumps • M - Measles • R - Rubella • Small pox • Chicken pox • BCG

Obstetrical services in India

Basic services

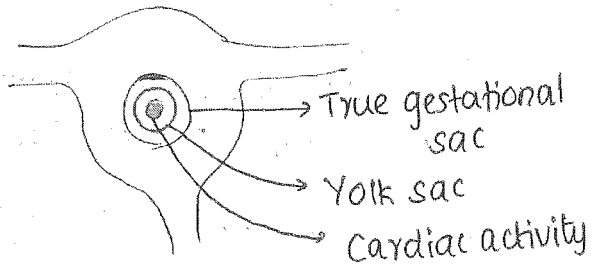
* Facilities to give antibiotics, anticonvulsants
* Facility to give them oxytocin/oxytocics
* Facility for assisted delivery
* Facility for manual removal of placenta.
* Facility for retained products.

Comprehensive services

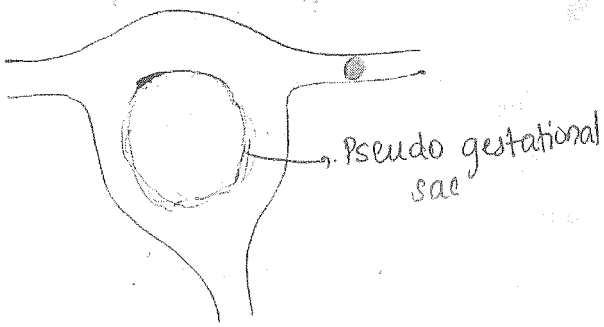
* 2 additional
  1) Facility for blood transfusion
  +
  2) Facility for C-section

USG in pregnancy

* First evidence of pregnancy on USG is appearance of gestational sac



Intrauterine pregnancy



Ectopic pregnancy

* In ectopic P → decidua appears like a gestational sac k/a Pseudo gestational sac
* Here corpus luteum → progesterone → converts endom to decidua → appears like gestational sac → ie, pseudogestational sac.

True gestational sac

* Seen in intra-uterine pregnancy
* Grow by 1-2 mm/day
* On one side of uterus → Eccentric
* Later on have a yolk sac ↓ Double ring sign +ve

Pseudo G.S

* Seen in ectopic pregnancy
* Never grow
* At centre of uterus
* No yolk sac so no double ring sign

⇒ So first evidence of
- pregnancy on USG is Gestational sac
- intrauterine pregnancy Yolk sac.

Critical titre of hcg

	TVS	TAS
Gestational sac (hcg)	4^{+1} – 4^{+3} wks (1500 IU)	5 wks (5000 – 6000 IU)
Yolk sac	5 wks	5–6 wks
Cardiac activity	5–6 wks	6–7 wks

⇒ If nothing mentioned in que, take it as TAS (transabdominal USG)

⇒ Critical titre of hcg → that value of hcg at ≤ ges-sac should be visible on USG → TVS: 1500 IU, for TAS 5000–6000 IU

(Every 48 hrs, hcg levels doubles) →

Que: On USG, cardiac activity seenby

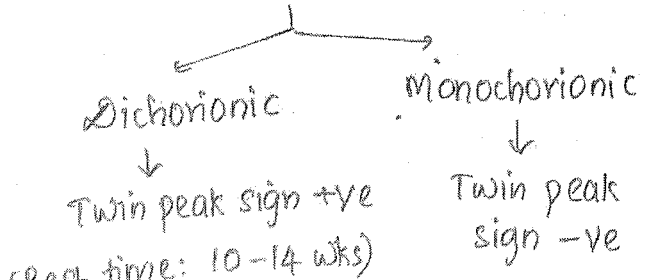
a) 5 wks (6-7wk) (TAS)
✓ b) 6 wks
c) 7 wks
d) 8 wks

Que: On USG, cardiac activity can be seen earliest by

✓ a) 5 wks (Earliest) ↓ (TVS)
b) 6 wks
c) 7 wks
d) 8 wks

Uses of USG

1) To detect pregnancy +/not (GS, YS, cardiac activity)
2) Detect whether pregnancy is intra uterine / ectopic
3) Twin pregnancy → detect chorionicity

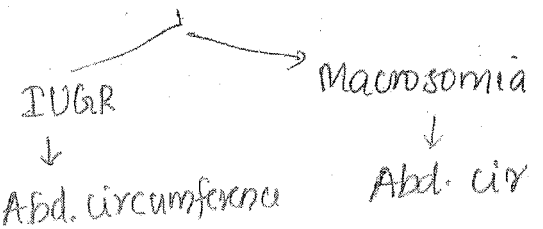


4) Determine the gestational age.
5) Assess the growth of fetus
   - Detect IUGR
   - Detect macrosomia
6) Detect congenital malformations
7) Detect everything about placenta ~~previa~~ / amniotic fluid

Parameters for estimation of fetal age on USG

* In the 1st trimester – uptill 14 wks
  Crown Rump Length (CRL)
  (uterus not palpable abdominally)
* 2nd trimester (14-28 wks)
  Biparietal diameter (BPD)/ Head circumference
* 3rd trimester
  Femur length
* Overall best:
  Crown Rump length
* Overall best time to do USG
  1st trimester

⇒ The only USG parameter ⊆ tells abt fetal growth is Abdominal circumference
ie, Best parameter for



To detect congenital anomalies

* Best time : 16-20 wks
  : (Organogenesis is completed)
  - Also in India MTP is legal uptill 20 wks
* Best USG to detect anomalies
  Level 2 USG → k/a TIFA
  (Targeted Imaging for fetal Anomalies)
* If ♀ → only 1 USG < TIFA / 16-20 wks

mcq
* Earliest congenital anomaly detect on USG → Anencephaly

↓

mcq 1) Earliest detected at 10 wks
mcq 2) Best time : 14 wks
mcq 3) Signs of anencephaly
  - Mickey mouse sign (△le)
  - Frog eye sign (Bulging eyes)

* Signs of spina bifida on USG
  - Lemon sign (frontal bone)
  - Banana sign (cerebellum protrudes)

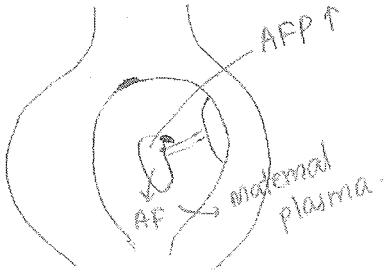
Anencephaly

* It is a neural tube defect in which cranial vault (skull bone) is absent.
* So not compatible with life
* Whenever detected → even if beyond 20 wks (≥ 20 wks)

↓

do MTP

* Adrenal glands are also absent / hypoplastic
* Face is normal → so MC presentation in anencephaly → Face presentation
* Also face presentation is MC in ♀ anomaly
  Anencephaly.
* In anencephaly, levels of Alpha feto proteins (AFP) are raised in the fetus



* ↑ AFP in fetus → to AF → spread to maternal plasma
* So screening test for anencephaly → maternal serum AFP levels → done : 15-20 wks

If AFP ↑↑

Diagnostic test these days → Level 2 USG / TIFA

Earlier Diagnostic test → Amniocentesis → In Amniotic fluid (AF) check level of AFP

If AFP ↑↑ → Problem in fetus → Fetal conditions → ↑AFP

1) Neural tube defect
2) Abdominal wall defects
   - Omphalocele
   - Gastrochisis
3) Pilonidal sinus

→ So check the levels of Acetyl cholinesterase levels in AF → if raised ⇒ Neural tube defect

⇒ Best biochemical marker of NTD
Acetyl cholinesterase

⇒ Conditions in which AFP ↓↓

G → Gestational Trophoblast disease like H. mole

O → Maternal obesity
O → Overestimated gestational age

A → Abortion

T → Trisomy 21 (Down's syndrome)

⇒ Prevention of NTD:
Folic acid

⇒ Dose of folic acid

In all ♀ → 400 µg/days → Ideally should be given 1 months before conception till 3 months after conception → Prophylactic dose of folic acid

In a ♀ H/O
1) Previous NTD baby
2) Gestational diabetes
3) On valproic acid

→ 4 mg/day → Therapeutic dose of folic acid

⇒ Govt of India supplies free Fe + folic acid tabs

↓

Dose : 500 µg (or mcg)

⇒ So routine daily administration of folic acid to ♀

500 µg (or mcg)

⇒ RDA of iodine to ♀

250 mcg

⇒ RDA of calcium to ♀

1000 mg

(RDA : Routine daily administration)

Physiological changes during pregnancy

General changes

1) Basal metabolic rate ↑ (10-20%)

2) Water retension during pregnancy

MCQ Que: How much → 6.5 L

↓

Because of H_2O retension

↓

Edema	Plasma osmolarity	Levels of serum Na^+ & S. K^+
↓	↓↓	↓↓
Physiological		

3) Weight gained during pregnancy

- Total weight gain : 11-12 kg

1st trimester → 1 kg

2nd → 5 kg

3rd → 5 kg

- Net weight gained : 5-6 kg

(after delivery → wt of baby, placenta, fluid gone)

- Factors affecting wt gain during pregnancy

1) Socio economic status
2) Ethnicity (American ♀ > African ♀)
3) Weight before pregnancy
4) Parity (in 1st is more)

- Weight gain is not dependent on Smoking

(Smoking → IUGR → so can affect fetal weight ~~gain~~ → not maternal weight gain)

Changes of uterus

* Wt of non pregnant U : 70 gm
* Wt pregnant U : 1000 gm
* Length of
  non pregnant → 7.5 cm
  pregnant → 35 cm
* Capacity of
  non pregnant → 10 ml
  pregnant → 5000 ml

Changes in vagina

1) Bluish discolouration - Chadwick sign
2) Cells predominate
   Intermediate cells
3) Doderlein bacteria ↑ during P
   ↓
   More of glycogen in to lactic acid
   ↓
   Acidity of vagina ↑
   ↓
   pH of vagina ↓ (pH = 3-5)
   ↓
   • MC vaginitis during pregnancy
   Candidiasis

Changes in hematological system during pregnancy

Increases

1) Blood volume (30-40%)
2) Plasma volume (40-50%)
3) RBC volume (20-30%)

ie, liquid component is ↑↑ more than cellular component
↓
k/a Hemodilution of pregnancy
↓
So ↓ in viscosity/osmolality

* Hemodilution leads to physiological anemia of pregnancy
↓
But Hb never < 11 gm/dl

Decreases

1) Viscosity/osmolality
2) Physiological anemia (↓ Hb)
3) Packed cell volume ↓
   $\left(\frac{RBC}{Plasma} \times 100\right)$
4) ~~RBC~~
~~5) Hb~~

Increases

1) Blood volume
2) Plasma volume
3) RBC volume
4) Hb mass
5) WBC count
6) Total amt of plasma protein (gm) ↑
7) Globulin ↑
   (Sex hormone binding globulin)
   (Thyroid hormone binding G)

Decreases

1) Viscosity/osmolality
2) ↓ Hb
3) ↓ PCV
4) RBC
5) Hb concentration
6) Platelet count
   ↓
   ↓ is k/a benign gestational thrombocytopenia
7) Plasma protein conc (g/dL) ↓
8) Albumin ↓

⇒ Normally Alb: G = 1.7:1

MCQ During pregnancy, Alb: G = 1:1

Increases	Decreases
* All clotting factor ↑ ie, pregnancy is a hypercoagulable state	* Except factor 11, 13 (↓↓)
* Clotting factor 1 → serum fibrinogen → ↑↑ MCQ during pregnancy. ↓ ESR & C-reactive protein ↑↑	

⇒ Parameters remained unaffected in pregnancy:
- Bleeding time.
- Clotting time.

Anemia during pregnancy

- **Physiological A**
  - ↓
  - All ♀ due to hemodilution
  - ↓
  - Hb ≥ 11 gm%
- **Pathological A**
  - ↓
  - Seen only in few ♀
  - ↓
  - Hb < 11 gm%
  - Anemia of pregnancy
    - Severe A: Hb < 7 gm%
    - Very severe: Hb < 4 gm%

Physiological Anemia

MCQ * Total Fe needed during pregnancy = 1000 mg

MCQ * Amt of Fe needed by fetus during pregnancy = 300 mg

MCQ * Daily requirement of Fe during pregnancy = 4-6 mg/day.

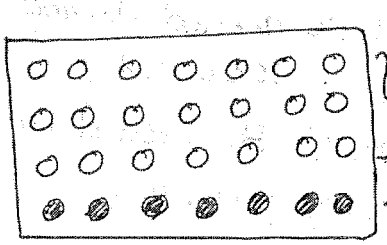
* Only 10% of dietary Fe is absorbed → so to fulfill the requirement of 4-6 mg/d → Fe intake in diet: 40-60 mg/day
  - ↓
  - Not possible, so iron supplementation is mandatory to all ♀.

* Govt. of India supplies Fe + folic acid tab free of cost

MCQ Fe + Folic acid
- ↓ 100 mg
- ↓ 500 ~~mg~~ mcg
- ↓
- Salt: Ferrous sulphate

* These tablets should be taken 1 tab/day × 100 days by all ♀ do not have pathological A
  - ↓
  - K/a prophylatic dose of Fe (1 tab/d → 100 mg Fe)

* Mala-D / Mala-N → 28 tab



OCPs

→ Fe tab

Salt of Fe → Ferrous fumarate

Pathological Anemia during pregnancy

* Hb < 11 gm%
* MCC of anemia during pregnancy
  Fe deficiency anemia

* MCC of anemia during pregnancy in developing countries
  Fe + Folic acid def. A
  ↓
  k/a Dimorphic anemia

* 1st parameter to ↓ in Fe def A / earliest indicator of IDA / Most sensitive indication is
  Serum ferritin
  (storage form of iron)

Proplylactic Fe : 1 tab/d
Therapeutic Fe : 2-3 tab/d

(Therapeutic dose of Fe)

* If ♀ with anemia → dose of Fe
  2-3 tab/day till her blood parameters become normal

* First parameter to ↑ after giving oral Fe
  Reticulocyte count (7-10 days)
* Hb ↑ after 3 wks of oral / Parental iron. at a rate of 0.7 gm – 1 gm/week

↓

Then give maintainence dose
1 tab/day.

↓

continue throughout pregnancy and for 100 days / 3 months after delivery

MCQ

↓

to replenish the iron stores

MCQ

⇒ If a ♀ at 36 wks of pregnancy, Hb = 7 gm%.

↓

Best management:
Blood transfusion

* Indications of blood transfusion:

1) In a pregnant ♀ c severe anemia beyond 36 wks of pregnancy.
2) Anemia due to blood loss like placenta previa.
3) Associated infection
   - Refractory anemia.

Changes in CVS during pregnancy

Increases

1) Cardiac output (CO = SV × HR)
   - ↑ CO, ↑ SV, ↑ HR
   - Max. ↑ CO → immediately after labor > 2nd stage of labor > late 1st stage > 28-32 wks of pregnancy.

MCQ

↓

Or que: as when are ~~changes~~ chances of heart failure maximum during pregnancy.

Decreases

1) Progesterone is a smooth muscle relaxant → ↓ peripheral vascular resistance during pregnancy → ↓ BP

CVS factors unaffected during pregnancy

1) JVP / central venous pressure
2) Pulmonary capillary wedge pressure.

Supine Hypotension syndrome

* During late third trimester if a ♀ lies supine → gravid uterus press against IVC
↓
↓ venous return in mother
↓
↓ CO in mother

- HR - ↑
- BP - ↓
- ↓
- Hypotension

- Blood flow to fetus ↓↓
- ↓
- fetal distress

⇒ Best position to lie: Left lateral

Changes in clinical indicators of heart during pregnancy

1) ↑ HR → ↑ pulse rate
2) ↓ BP
3) Heart is rotated ⊕ upward & outward (to left) during pregnancy (diaphragm pushing it)

- Apex beat can be heard in the 4th ICS, 2.5 cm lateral to mid. clavicular line
- On ECG, ↓ Left axis deviation
- Patient experience palpitation

4) Heart sounds:
- S_1 is loud & has exagerated splitting.
- S_2 is normal during pregnancy
- S_3 is easily heard in pregnancy.

5) Murmurs: 2 murmurs are normal in pregnancy
- Ejection systolic murmur < grade 3
- Continous murmur (mammary murmur)

6) On x-ray → mild cardiomegally may be present & straightening of left heart border.

Heart disease in pregnancy

■ Indicators of heart disease during pregnancy.
- Cyanosis
- Clubbing
- Pulmonary edema
- Any arrhythmia
- JVP ↑
- Heart sounds → S_2 loud with a prominent split / S_4 heard.
- Murmurs: Diastolic murmur
- Marked cardiomegally on x-ray.

MC heart disease during pregnancy

* MCC of heart disease in developing countries.
  Rheumatic HD
  ⇓
  MC : Mitral Stenosis

* MCC of heart disease (HD) in developed countries
  Congenital HD
  ⇓
  MC: Atrial Septal Defect.

* MC heart disease in pregnancy
  Mitral stenosis

* MC congenital heart disease during pregnancy
  ASD

* MC cyanotic HD during pregnancy
  TOF

* MC congenital valvular HD during pregnancy
  Mitral valve prolapse.

* Like in medicine → NYHA classification
* In obs, : Clarke's classification.
  Here HD is divided based on the maternal mortality.

Clarke's classification

- **Class I**
  - All congenital HD
  - Maternal mortality < 1%.
- **Class II**
  - Maternal mortality (15-25%)
- **Class III**
  - Maternal mortality (25-50%)

Includes
1) Pulmonary HTN < 1° / 2° (Eisenmenger synd)
2) Marfan syndrome with aortic involvement
3) Coarctation of Aorta

* Due to pulmonary HTN in Class III
  ⇓
  MCQ HD in which pregnancy is C/I & MTP is advised is Class III

* HD in which max. risk of maternal mortality
  Eisenmenger syndrome

* In HD patients, MTP should be done at ≤ 12 wks & not after that
  ↓
  The risk of doing MTP is same as pregnancy

Management of labor

Time of admission

* Clarke's Class I → 2 wks before EDD (expected date of delivery)
* Clarke's Class II → at 28 wks of pregnancy.
* Clarke's Class III → Throughout pregnancy (Pregnancy is C/I & MTP is done usually)

Intrapartum management

* Let her go in to Spontaneous labor
  - if spontaneous labor pain does not occur
    - MCQ Induction of Labor can be done (before not)
* Best method is vaginal delivery within 30 min.
  - if not then vaccum >> forceps
* Labor analgesia for painless labor can be given
  - Give Epidural analgesia

HD in which C-section has to be done

1) HD involving Aorta
   - Coarctation of Aorta
   - Aortic stenosis
   - Aortic aneurysm
   - Marfan syndrome with involvement of aorta
2) Recent MI
3) If patient is on Warfarin.

⇒ Give O_2 to patient

⇒ Position in which delivery is conducted.
Left lateral position / semi recumbent position.
(or otherwise venous return ↑)

⇒ Fluid @ 75 ml/hr (not more)

Immediately after delivery

1) Donot give methyl ergometrin (Methergin) (Can use oxytocin)
(C/I → TOPER ; O - organic heart disease).
2) MC time of heart failure is immediately after delivery
Just aftr deli. > @ time of labor > 28-32 wks of preg
Give diuretics

Earlier

3) In all HD patients antibiotics are given to prevent infective endocarditis → But now it is not recommended

Contraception in HD

* Till now best temporary method is barrier method → ie, condom
* OCP's are C/I → leads to thromboembolism
* IUCD C/I → leads to infection
* Recently IUCD's are approved in HD patient

∴ Now best is IUCD not condom

IUCD > Condom (Barrier method)

Permanent

* Best is husband → Vasectomy
  ↓ if refuses
  ♀ = Tubectomy
* Best method of Tubectomy is Mini laprotomy
* Laparoscopy is C/I
* Best anesthesia → Local anesthesia
* Best time → At the end of first week after delivery.

Clinical case 1

Suppose ♀ ∈ HD, in her valve replacement is done → then mechanical valves are put & she is advised to use Warfarin throughout life → she conceives

↓

should replace Warfarin

↓

Anticoagulant of choice during pregnancy : ~~Heparin~~ different at diff. period

2 Anti coagulants

	Warfarin	Heparin
	↓	↓
	• Strong anticoagulant (advantage) (Pregnancy is hyper coagulated state)	• It does not cross placenta & harm fetus (adv)
	• But it can cross placenta & leads to chondro-dysplasia in fetus	• But it is a weak anticoagulant
Period of gestation		Anticoagulant of choice.
• Uptill 12 wks		Heparin
• 12 - 36 wks (organogenesis already completed)		Warfarin
• After 36 wks uptill delivery		Heparin

(Because ↑↑ chance of PPH & antidote is available for H, not for W)

* 6 hrs after vaginal delivery/ 24 hrs after C-section
↓
Restart anticoagulant
↓
Either Heparin or Warfarin
↓
MCQ Because Warfarin is not C/I during breast feeding.

MCQ
* If patient is on Warfarin and goes in to labor → do C-section

Clinical case 2

* If pt has MS during pregnancy
↓
MCQ Surgery for MS

- Surgery of choice → Balloon valvuloplasty
- Best time → 2nd trimester (14-18 wks)
- Sx ≤ is C/I → Valve replacement

MCQ
* Any Sx in pregnancy, best time is 2nd trimester.

Changes in renal system during pregnancy

* Anatomical changes

1) Size of kidney ↑ by 1 cm
2) Due to smooth muscle relaxing effect of progesterone
↓
Hydroureter
&
Hydronephrosis
3) Due to bladder congestion, bladder pressure ↑↑ & intraurethral pressure ↑↑

* Physiological changes:

MCQ Blood volume ↑
↓
blood to mother's kidney ↑
↓
Renal blood flow & ↑
↓
GFR ↑↑
(filtering capacity of the kidney ↑)

- ↑ clearance of urea, uric acid & creatinine
- MCQ ↓↓ in
  - serum urea
  - serum uricacid
  - serum creatinine

Asymptomatic Bacteuria

* On urine examination (midstream clean catch sample)
↓
if no: of bacteria are $\geq 10^5$/ml without any symptoms of UTI
↓
k/a asymptomatic bacteuria

* Risk factors are
  - Diabetes
  - Sickle cell trait

* MC organism → E-coli
* Mgt: Ampicillin / Amoxicillin
* Recurrent infection, DOC → Nitrofurantoin

MCQ
⇒ Effect of PIH on GFR is
↓ GFR ($P \propto 1/V$, RBF↓, GFR↓)

Changes in carbohydrate metabolism during pregnancy

1) Insulin secretion ↑ during pregnancy
2) But insulin resistance during pregnancy
↓
hormones which leads to IR
↓
- Human Placental Lactogen
- Estrogen (main)
- Progesterone
- Cortisol

3) Insulin resistance is max. b/w 24 - 28 wks
4) Because of IR, pregnancy is a diabetogenic state.

* Maternal insulin cannot cross the placenta

MCQ
* Fetus starts producing insulin @ 12 wks

* Main source of fetal energy is maternal glucose
↓
transfered to fetus
* Main hormone leading to fetal growth is Insulin
* If maternal glucose levels are increased (hyperglycemia)
↓
MCQ
fetal hyperglycemia
↓
Fetal pancreas → β cells produce more insulin (β-cell hypertrophy)
↓
more insulin in the fetus causes
↓

↑ Insulin causes

↓

1) ↑ Growth of fetus → Macrosomia

2) ↓ surfactant → ↑ chances of respiratory distress syndrome

3) * Fluctuating glucose → ↑ chances of IUD of fetus → MC time is last 2 wks of pregnancy.

Diabetes in Pregnancy

* Diabetic patient becomes pregnant

↓

Overt diabetes

↓

Blood glucose - abnormal from day 1 of pregnancy.

* Patient was normoglycemic before pregnancy, during pregnancy because of insulin resistance becomes diabetic

↓

Gestational diabetes

↓

IR is max: 24 - 28 wks

↓

Blood glucose is abnormal b/w 24 - 28 wks

* In diabetes → hyperglycemia

↓

Free radicals are formed

↓

Congenital malformations in the fetus

* Gestational diabetes → free radical will be formed b/w 24 - 28 wks → Organogenesis is completed → no congenital malformations

* Overt diabetes → free radicals + nt from day 1 → so congenital malformation in fetus

MCQ

* Congenital malformations are seen in pregnant ♀ with overt diabetes not in gestational diabetes.

Congenital malformation in Overt diabetes

* Best time to predict the chances of congenital malformation in a diabetic ♀

↓

HbA1C

< 6.5	6.5 - 9.5	≥ 9.5
↓	↓	↓
No chances	5%	20%

* Best test to detect congenital malformation in a diabetic ♀

↓

TIFFA / Level 2 USG

↓

@ 16-20 wks.

* MC system involved in congenital malformation in diabetic (baby)

↓

CVS > CNS

* MC congenital anomaly in babies of diabetic ♀

VSD > NTD.

* Most specific congenital anomaly

Sacral agenesis / Caudal regression syn.

(rare)

* Most specific CVS anomaly:

Transposition of great arteries (TGA)

* Best method to prevent congenital anomalies in babies of overt diabetes:

1) Tight controll of blood sugar level

↓OC : Insulin

(does not cross placenta)

2) Folic acid : dose - 4 mg

(therapeutic dose)

Gestational Diabetes

Diagnosis of GD

* Screening Test : Glucose Challenge Test

Time → 24-28 wks

* Procedure : No need of fasting

Pt comes → 50 gm oral glucose is given

↓

After 1 hr, check her blood sugar levels.

↓

Result

<140 mg/dL	140-200 mg/dL	≥200
↓	↓	↓
Not diabetic	May be diabetic	Confirmed diabetic (No need of confirm. test)
	↓	
	Do confirmatory test - Glucose Tolerance Test (GTT)	

* Asian population is a high risk population for diabetes

(India → diabetic capital of world)

↓

So in India → Universal screening for all ♀ do GTT @ 24-28 wk

Glucose Tolerance Test

* Recommended by ACOG is
  3 hr, 100 gm GTT
* Procedure → Need overnight fasting (8 hr)
  ↓
  Sample 1 : Fasting blood sugar (FBS) sample
  ↓
  100 gm of Glucose
  ↓
  Check her blood glucose levels after 1 hr, 2hr, 3hr → 3 samples
  ∴ Total 4 samples (FBS + 3)
* Out of total 4 samples, if 2 or > 2 values are abnormal
  ↓
  then patient is confirmed diabetes

Values:

	Upperlimit of (N)
Fasting	95
1hr	180
2 hr	155
3 hr	140

* Recommended by WHO is
  2 hr, 75 gm GTT
* Procedure → Overnight fasting
  ↓
  FBS sample
  ↓
  75 gm glucose given
  ↓
  Check blood glucose @ 1 hr, 2 hr
  ∴ Total samples 3 (FBS + 2)
  Out of total 3, if 1 or more than 1 is abnormal
  ↓
  then patient is said to be diabetic

⇒ During pregnancy, GTT done in India
  3hr, 100 gm GTT

⇒ For non pregnant ♀, male, children
  2 hr, 75 gm GTT

Mgt of GDM

* 1st : Diet modification and exercise
  ↓
  should aim @ following goals

Fasting → 70-95

1 hr PP → < 140

2 hr PP → < 120

& HbAIC → < 6.5

(PP : Post prandial)

* Diet & exercise should be done for 3 weeks
* After 3 wks, check

Metabolic goals are achieved → continue diet & exercise

If metabolic goals not achieved → DOC : Insulin

- Insulin cannot cross placenta
- Diabetogenic state is pregnancy → so strong drug : Insulin

* Dose of insulin ↑ during pregnancy
* Oral hypoglycemic drugs C/I during pregnancy
* which drugs can be used:
  - Metformin
  - Glyburide

Obstetric Mgt of Diabetes

* In case of gestational diabetes managed on diet → Terminate pregnancy @ 40 wks (macrosomia risk)
* In case of gestational DM managed on insulin → Terminate @ 38 wks
* Overt DM → @ 38 wks

↓

Because IUD is max during last 2 wks

* Mode of delivery :-
  Vaginal delivery.
* Only if weight of baby ≥ 4.5 kg is DM pt → then go for C-section.

Complications of DM in pregnancy

Maternal Complications :

1) ↑ infection (like asymptomatic bacteuria, UTI)
2) Mother glucose ↑ → fetal glucose ↑ → polyuria → Polyhydramnios
3) Polyhydramnios can lead to
   - Preterm labour
   - PROM
   - PPH

4) In future can have DM → 6 wks after delivery → do 75 gm GTT → repeat ∴ every 3 years

* Neonates of diabetic mother do not have
  - Mental retardation
  - Anemia

* Which is the best test in diabetic mothers to access the fetal lung maturity
  Phosphatidyl Glycerol

Fetal complications:

1) Fetal hyperglycemia
2) Prematurity
3) ↑ insulin → Macrosomia ∴ IUD
4) Shoulder dystocia
5) ↑ chances of abortion ∴ still birth

Neonatal complications

1) Respiratory ~~dystre~~ distress syndrome (premature)
2) Fetus hyperinsulin → when baby born → connection b/w mother ∴ baby is lost → so source of ↑ glucose goes → remains is ↑ insulin → Neonatal hypoglycemia
3) Hypocalcemia ($\downarrow Ca^{2+}$)
4) Hypomagnesemia ($\downarrow Mg^{2+}$)
5) Hypokalemia ($\downarrow K^{+}$)
6) Hyperbilirubinemia + Polycythemia

k/a hyperviscosity syndrome.

Macrosomia

* Wt of fetus ≥ 4.5 kg
* In India, ≥ 4 kg
* Risk factors:
  - Post dated pregnancy
  - DM
  - Male fetus.
* Best USG parameter to detect macrosomia:
  Abdominal circumference (of fetus)
* Best mode of delivery is Vaginal delivery.
* Only if the wt of fetus

≥ 4.5 kg in diabetic ♀ | ≥ 5 kg in nondiabetic ♀

→ Do C-section

* Complication during vaginal delivery of macrosomic fetus: Shoulder dystocia

Shoulder dystocia

* ℞ is difficulty in delivery of shoulder after delivery of fetal head (≥ 1 min delay)

* Risk factors for shoulder dystocia
  D - Maternal DM
  O - Mat. obesity, fetal obesity = Macrosomia
  P - Post dated pregnancy
  A - Anencephaly.

* Mgt
  - Give a big episiotomy
    - Stop giving fundal pressure
    - Give suprapubic pressure
  - Best maneuver / most effective / 1st maneuver done
    ↓
    Mc Roberts maneuver
    ↓
    Flex the thigh of ~~fetus~~ patient and abduct it
  - Last maneuver done is
    ↓
    Zavanelli maneuver
    ↓
    Push delivered head back to uterus & do C-section

Before try Zavanelli maneuver
- Wood cork screw maneuver
- # clavicle of baby → Cleidotomy
- Divide pubic symphysis of mother: Symphisiotomy

↓ (if fails)

Zavanelli maneuver

* MC fetal complication during shoulder dystocia
Brachial plexus injury

* MC maternal complication is in-sid PPH.

Ectopic Pregnancy

* Condition in which ~~implantation~~ fertilization is normal but implantation occurs outside the uterus.
(fertilization → ampulla)

* Zygote formed, corpus luteum of pregnancy formed → progesterone will be present but it will be less than the normal pregnancy
↓
Endometrium is converted to decidua, (so decidual reaction +nt)
↓
Arias stella reaction +nt (+ve)
(due to progesterone)

* In case of ectopic pregnancy, β-hCG levels are higher than non-pregnant female → but they are lower than (N) intra-uterine pregnancy → also do not double in 48 hrs like in (N) pregnancy

* Because β-hCG levels are higher than non pregnant ♀ → urine pregnancy test will be +ve (99% cases)
In 1% case → -ve.

MCQ
⇒ UPT is ✱ +ve in all cases of ectopic pregnancy
False (1% → -ve)

MCQs

✱ MC site of ectopic pregnancy
Fallopian tube

✱ In the fallopian tube
Ampulla is in Interstitium
Ampulla > Isthmus > Infundibulum > Interstitium.

✱ MC site in FT is Ampulla because fertilisation occurs in the ampulla.

✱ In FT, least common site is Interstitium

✱ Overall, the least common site is
1) Scar of C-section > cervical ectopic

Risk factors for ectopic pregnancy (EP)

- Associated c genital tract
  - MCQ • Highest risk of ectopic pregnancy → if previous H/O ectopic pregnancy.
  - MCQ • MC risk factor for EP
    - Salphingitis or PID (Inf. FT) (Tubes + Ovaries + Uterus)
  - Previous H/O tubal Sx
  - Endometriosis (adhesions are formed)
- Not associated with genital tract
  - Smoking
  - Early age of intercourse
  - Multiple partners
  - In utero exposure to Diethyl stilbesterol (DES)

Role of contraceptives in EP

* As such the chances of EP are ↓ by all contraceptives. Because contraceptives protects against all kinds of pregnancies.
* But if contraceptive failure occurs, it leads to EP
* Among contraceptives, highest chances are with
  Tubectomy > IUCD > Progesterone only pills
* Least chances of EP are with MCQ oral contraceptive pills.

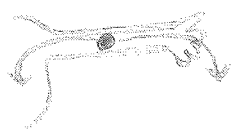
* Symptoms of ectopic pregnancy

(P/V - per vaginally)

- Bleeding P/V
- Amenorrhea 6-10 wks
- Pain in abdomen ↓ MCQ Most consistent symptom
  - ruptured ectopic ↓ pain is due to hemoperitoneum ↓ irritates the diaphragm ↓ leads to shoulder tip pain. (k/a Danforth sign)
  - unruptured ectopic ↓ pain is due to stretching of FT

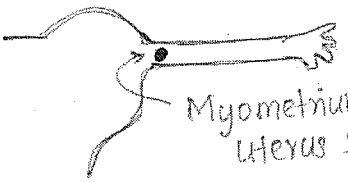
Important one liners

* 2 fates of EP:
  - EP aborts ↓ k/a tubal abortion ↓ MCQ MC site of tubal abortion Ampulla
  - EP continues to grow ↓ Tubal rupture ↓ MC site of tubal rupture is Isthmus



* In which site, EP survives for longer time

- Part it survives longest in overall → Abdominal ectopic (it can go uptill term)
- In FT, it lasts longest in Interstitium. (as myometrium supports EP)



Myometrium of uterus supports it

* where is EP survives for least/ shortest time?

Isthmus

MLR

* What is dangerous EP?

EP @ interstitium (because here EP lasts the longest)

Heterotopic pregnancy

* It is twin pregnancy in which one pregnancy is intrauterine & other is ectopic pregnancy.

Ruptured Ectopic Pregnancy

MLR

* Diagnosis → ♀ with UPT +ve, amenorrhea 6-10 wks, pain in abdomen & bleeding P/V & shock (P/V: Pervaginal)

(UPT: Urine Pregnancy Test)

* Always think of EP, no other diagnosis

* Mgt:

- 1st step: General resuscitation + Open her abdomen ↓ Laparotomy (preferred method)

(Here vitals can be stable or unstable)

Laparotomy → opening the abd & wide incision

- Some surgeons, these days perform Laparoscopy for ruptured ectopic (2/3 small incisions) ↓

Laproscopy can be done only if vitals are stable

- 2nd step: Remove the ruptured tube ⟹ Salpingectomy

* Whether patient is nulliparous or multiparous, in ruptured ectopic, in all cases, salpingectomy is done.

Other imp. points (MCQs)

* Ectopic pregnancy occurs in young females. ∴ when tubes are removed, ovaries are not removed
  ie, salpingo-opherectomy is not done in EP.

* In ruptured ectopic, there is no role of ~~conservative Mgt~~
  No role of:
  - Conservative Mgt
  - Medical Mgt
  - Any other Sx except salpingectomy.

Unruptured Ectopic:

* Diagnosis →
  IOC: Transvaginal USG (TVS)

- Gestational sac +nt inside uterus → Intrauterine pregnancy (Ectopic P cannot be ruled out → pseudo gestational sac)
- Empty uterus (1st sign → raise suspicion of ectopic pregnancy) → Differential diagnosis → ⊘/⊘ with UPT +ve
  - Ectopic pregnancy
  - Abortion
  - Early intrauterine pregnancy

After ⊘/⊘ →

2nd step → Check the FT & ovaries

- if gestational sac/cardiac activity is +nt in FT → indicates EP → further confirm by doing Doppler → On Doppler, in EP → Ring of Fire appearance.
- If nothing is seen in FT → Do 3rd step: Check the levels of β-hCG

  - If β-hCG ≥ 1500 IU (critical titre of β-hCG when gest. sac should be visible in case of IU pregn.) → still if no gest. sac is visible → Ectopic pregnancy
  - When β-hCG < 1500 → Do 4th step → Repeat β-hCG after 48 hrs
    - If β-hCG levels doubled → IU pregnancy
    - If levels ↑ not double → EP
    - Levels ↓↓ after 48 hr → Abortion

Mgt of unruptured ectopic

(3 options)

- → Conservative / Expectant Mgt
- → Medical Mgt
- → Surgical Mgt

Conservative Mgt	Medical Mgt
• Do nothing - wait for EP to resolve spontaneously.	• Doc is Methotrexate (single dose regime)
• Only in unruptured EP	• Only in unruptured EP
• Requirement - Vitals stable - β-hCG < 2000 IU - Size of EP < 3 cms - Cardiac activity should be absent	• Requirement - Vitals stable - β-hCG < 5000 IU - Size of EP < 3.5 - 4 cm - Cardiac activity should preferably be absent
• Not preferred as it carries a risk of rupture.	MCQ • Preferred Mgt of unruptured ectopic pregnancy

Surgical Mgt:

* 1st step → Open the abdomen

- ← Laparoscopy (Preffered in unruptured EP) (Vitals stable)
- → Laprotomy (Ruptured EP)

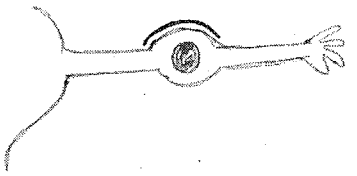
* 2nd step → Surgery:

It depends on parity of ♀ (whether she has completed her family or not)

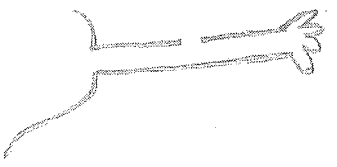
- ← Multiparous [Completed her family] → Salpingectomy (remove tubes)
- → If ♀ is nulliparous → Surgery of choice is **Salpingostomy**
  - Alternative is Salpingotomy

Details of Sx:

Salpingostomy



* Incision is given on the tube over the ectopic
  ↓
  by hydro dissection, remove the ectopic pregnancy
  ↓
  leave the tube as such & out suturing incision site. (to heal by 2° intention)



Salpingotomy

* Everything is same like salpingostomy.

↓

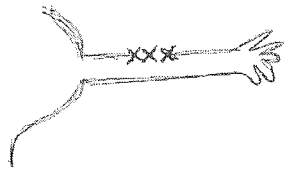
Incision

⇓

Remove EP by hydrodissection

↓

(But) Put sutures on the incision site



Other sites of Ectopic Pregnancy

Other sites	Names of diagnostic criteria
• Cervical ectopic	Rubin criteria
• Abdominal ectopic	Studdiform C
• Ovarian ectopic	Spigelberg C

[C : Criteria]

Down's Syndrome

* Trisomy 21 (47, 1 extra chromose in 21)

* It can be due :
  - Non dysjunction of chromosome (MCC)
  - Translocation of chromosome → b/w 14 & 21 or 21 & 21 → k/a balanced translocation

* Clinical case 1 :

♀ with baby with Down syndrome → chances of Down syn. in her next baby.

↓

Check her present baby

- if Down syn. due to nondysjunction of chromosome → ↓ → 1–2% chances
- if Down syn due to balanced translocation (b/w 21 & 21) → ↓ → 100% chances

* Chances of Down syndrome ↑ as maternal age ≥ ~~30 yrs~~ 35 yrs

* Screening of Down syndrome is Universal screening irrespective of maternal age.

* Screening methods in first trimester : (11–13 wks)
  1) Biochemical markers
  2) USG

* In biochemical marker → in 1st trimester → has 2 markers (Dual test)

a) PAPP-A (Pregnancy associated plasma protein A) (↓↓)

+

b) β-hCG (↑↑)

* In Down syndrome, all markers are ↓↓ except

h → hCG → high

* In USG, will get
  - ↑ nuchal translucency (area under neck)

  ≥ 3mm → indicates Down's syndrome

Causes of ↑ nuchal translucency mcq
- Downs syndrome
- Turners syndrome
- Congenital heart disease

* Screening in 1st trimester is done : 11-13 wks

Screening in 2nd trimester (15-20 wks)

- Biochemical markers
  - ↓
  - i) Triple Test
    - i) β-hCG (↑)
    - ii) α-Feto protein (AFP) (↓)
    - iii) Unconjugated estrogen (↓) (E_3)
  - 2) Quadraple test (QUAD Test)
    - ↓
    - Triple test + Inhibin (↑↑)
      - I → Inhibin Increase
- USG
  - ↓
  - Thickness of the neck area (Nuchal fold thickness)

USG
↓
Nuchal fold thickness
↓
≥ 5mm indicates Down syndrome

* Diagnostic test : (confirmatory test)

Karyotyping
↓
Need viable fetal cells (obtained by)

- Chorionic villi sampling in 1st trimester mcq
  - ↓
  - Study material Chorionic villi ie, trophoblast
  - ↓
  - Best time : 11-13 wks should not be done @ < 10 wks
  - ↓
  - Can detect any • chromosomal anomaly of fetus
- Amniocentesis in the 2nd trimester mcq
  - ↓
  - Study material is fetal skin cells or fibroblast
  - ↓
  - Best time is 15-20 wks (16-18 wks)
  - ↓
  - can also detect • Chromosomal anomaly

(CVS)

- Metabolic problems in fetus like Phenylketonuria
- Hemoglobinopathies like sickle cell anemia.

* Cannot detect neural tube defect (NTD)

MCQ

* MC complication
  Fetal loss/Abortion
  ↓
  1%

MCQ

* MC complication if done at 9 wks / <10 wks
  ↓
  - Oromandibular defects in fetus

(CAC)

»

»

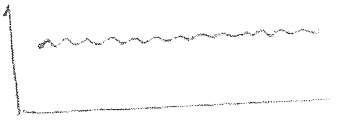
* Can detect NTD (AFP levels ↑↑)

* Rate of abortion
  1 in 300 – 1 in 500

(Safer)

Whenever → ♀ → previous H/O Down syn → do not do screening test → directly do confirmatory test (Karyotyping)

*



If Fetal HR appears fixed → k/a Sinusoidal heart rate pattern.

↓

It is seen in

- Case of fetal anemia
  1) Twin to twin transfusion syndrome (donor twin has anemia)
  2) Vasa previa
  3) Hydrops fetalis (Rb -ve mothers)

Fetal Monitoring

Fetal heart sound

* Fetal heart rate : ① 110 – 150 beats/min
  < 110 → Bradycardia
  ⩾ 150 → Tachycardia

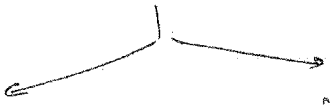
* Fetal heart sound has beat to ② beat variability → so FHR is never fixed → keeps on varying

③ * Fetal heart rate accelerations
↓
↑ in FHR by 15 beats/min for 15 sec
↓
Whenever there is fetal movement, FHR acceleration occurs → denotes that fetus is healthy

④ * ~~Fear~~ Fetal heart rate decelerations → whenever uterine contraction occurs

MCQ

↓
Pressure ↑, so volume of blood going to fetus ↓
↓



if fetus is healthy it can tolerate this ↓ in blood flow ↓ & HR remains normal.

if fetus is compromised, it cannot tolerate ↓ in blood flow ↓ So HR ↓↓

* A decrease in fetal HR by 15 bpm for 15 sec is called as deceleration (15 bpm x 15 sec)
* Deceleration → never (N) → indicates compromised fetus.
* Deceleration → can be of 3 types

Types of deceleration

(1)

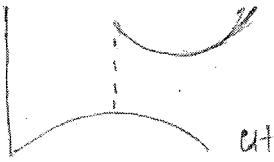
- Dip in fetal HR begins with uterine contraction begin & ends with uterine contraction ends.
- Gradual in onset (≥ 30 sec)

↓

k/a Early deceleration

- It is seen in head compression
- Early decelerations are physiological

(2)



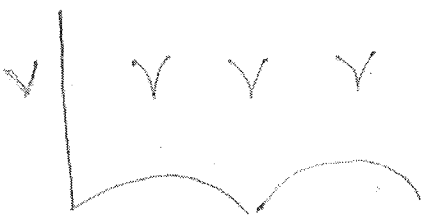
* Dip in fetal HR begins when uterine contraction is at its peak & it persists even after the contraction is over
* Gradual in onset (≥ 30 sec)

↓

k/a Late deceleration

* Seen in uteroplacental insufficiency.
* ie, it is pathological & it is the worst type of deceleration
* Whenever get late deceleration → always terminate the pregnancy immediately.

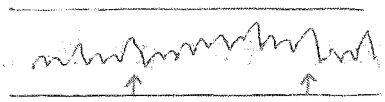
(3)



* There is no fixed relationship b/w dip in FHR & uterine contraction.
* It is sudden in onset (< 30 sec)

* K/a variable deceleration
* seen in cord compression
(Vice Chancelor → Variable Cord Compress)

* Variable deceleration for some time is Ⓝ during labor
* But if it is persistant, then it leads to fetal distress and is an indication for terminating pregnancy immediately.



After 20 mins: Read the strip

If getting ≥2 acceleration in 20 min
↓
fetus healthy

If no acceleration/ single acceleration
↓
Repeat CTG/NST for 20 more minutes
↓
Total = 40 min
↓
In 40 min, if get < 2 acceleration
↓
CTG/NST is non-reactive & fetus may be in distress
↓
Do confirmatory test → Biophysical score

- If get → late deceleration
↓
Immediately terminate pregnancy
or
- Persistent variable deceleration
↓
Delivery

Antepartum fetal monitoring

1) Daily fetal movement count by mother
↓
If any day she experiences less movement
↓
report

⇒ If a ♀ c/o ↓ fetal movement
↓
Do CTG / NST (Non Stress Test)
↓
Screening Test.

NST: Done for 20 minutes
(Tie a belt around mother's abd, lie on left lateral position, when she experience movement, press button → comes as arrow in the strip)

⇔ NST should be done weekly in all ♀ from 32-34 wks onwards & it should be done in 72 hrs in high risk pregnancy like diabetic pregnancy (ie, twice weekly)

Biophysical score

* Diagnostic test for fetal distress
* Also k/a Manning score.
* Parameters

T – Fetal tone

B – Breathing movement of the fetus

Meningitis – Gross body movements

Always – Depth of single amniotic fluid pocket.

Notorious – Non stress test

* To each of these parameter → give a score of 0/2.
* Total score = 10
* If score is 10/10 or 8/10 then → Normal
* The two most important parameters are:
  1) NST → Acute distress
  2) AF pocket → Chronic distress.
* Modified Biophysical score
  1) NST
  2) Amniotic fluid index

Bishop score

* Bischop score is used before inducing labor (initiating labor)
* Parameters are:

Delhi – Dilatation of cervix

Police – Position of cervix

Employed – Effacement of cervix

Special – Station of fetal head

Commandos – Consistency of cervix.

* To each of these parameters → give a score of 0 to 3
* Total = 15
* If score ≥ 9 → induction of labor will be successful
* If score < 4 → first ripen the cervix and then induce labor
* DOC for ripening of Cx is PGE_2 (Dinoprost)
* Which other PG can be used PGE_1 (Misoprost)
* Most imp parameter in Bishop score → Dilatation of cervix
* Modified Bischop score:

Delhi
Police
Left → Cervical length
Special
Commandos

* Here 0–2 score → Max : 10 score
* If score ≥ 6 → induction will be successful.

Intrapartum fetal monitoring

1) Fetal heart sound by stethescope

	Normal ♀	High risk ♀
1st stage of labor	30 mins	15 mins
2nd stage of labor	15 mins	5 mins

2) Continous CTG

3) Fetal scalp pH monitoring
   - → Invasive (so not generally recommended)
   - → If scalp pH b/w 7.25 - 7.35 it is normal.
   - → If pH b/w 7.20 - 7.25, repeat it after 30 minutes
   - → < 7.20 → Cesarean immediately (fetal ~~distress~~ acidosis)

Pelvis

* Female pelvis is making an angle of 55° with the horizontal
  ↓
  k/a angle of inclination

* Pelvic brim is formed ant. → past. by:
  Pubic symphysis → sup. pubic rim → ileo pectineal line → sacroiliac joint → ala of sacrum → sacral promontory.

Pelvic brim divides the pelvis in to two parts

- Part above the brim
  ↓
  False pelvis
  ↓
  - No role in labor
  - It only supports the uterus
- Part below the brim
  ↓
  True pelvis
  ↓
  Further divided in to 3 parts
  ↓
  1) Pelvic inlet: @ level of the brim
  2) Pelvic cavity / midpelvis: lies @ level of ischial spine
  3) Pelvic outlet @ level of ischial tuberosity.

Pelvic Inlet

- AP diameter
- Oblique dia. (12 cm) → right sacroiliac joint to the left iliopectineal eminence
- Transverse diameter (13 cm) → b/w 2 far points in the ileum both side

AP diameter

Conjugate	Description	measurement
1) True conjugate	Distance b/w upper border of pubic symphysis to sacral promontory	11 cm
2) Obstetric conjugate	Middle of PS → SP	10–10.5 cm
3) Diagonal conjugate	Lower PS → SP	12 cm

PS: Pubic symphysis
SP: Sacral promontory

* If diagonal conjugate is 'a' cm in a patient

True C → (a−1) cm
Obst. C → (a − 1.5 to 2) cm

[DC = 12 cm
TC = 11
OC = 10–10.5]

* Shape of pelvic inlet in a female pelvis is transverse oval (as TD > AP diameter)

⇒ **Mid pelvis**
(at the level of ischial spine)

- AP diameter (Line joining lower border of pubic symphysis & the sacrococcigeal joint) → 11.5 cm
- Transverse diameter (distance b/w 2 ischial spines) → k/a bispinous diameter / interischial diameter → 10 cm (Very less)

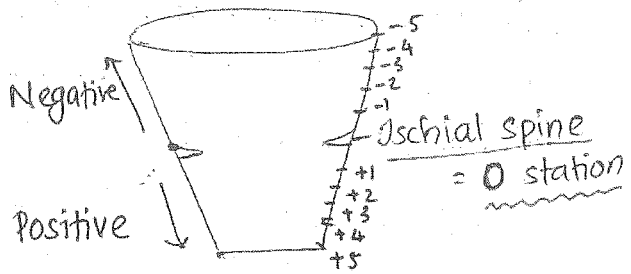
→ Plane of midpelvis is k/a plane of least pelvis diameter

MCQs

* Most important AP diameter of inlet → Obstetrical conjugate
* If obstetrical conjugate < 10 cm, vaginal delivery is not possible → k/a Contracted pelvis
* Critical obstetrical conjugate is 10 cm
* AP diameter of inlet which can be measured clinically is diagonal conjugate. (on P/V examination)

Significance of ischial spine

1) Station of fetal head:



2) Internal rotation of fetal head occurs at this level

3) Deep transverse arrest occurs at this level.

4) It is the site for giving pudental nerve block

5) Levator ani muscle is attached here.

Subpubic angle

It is the angle b/w the pubic rami of pubic bone

↓

females: Obtuse angle

males: Acute angle.

Major diameter of pelvis

	Inlet	Midpelvis	Outlet
AP dia	TC - 11 OC - 10 to 10.5 DC - 12	11.5 cm	11.5 to 13.5
Oblique dia	12 cm	×	×
Transverse dia	13 cm	10 cm	11 cm

* Smallest major diameter of pelvis → bispinous diameter → 10 cm.

Pelvic outlet

- AP diameter
  - ↓
  - distance b/w lower border of pubic sym & tip of coccyx
  - ↓
  - 11.5 — 13.5 cm
  - ↓
  - pt is in labor
- Transverse diameter
  - ↓
  - distance b/w 2 ischial tuberosities
  - ↓
  - 11 cms
  - ↓
  - Bituberous diameter/ Intertuberous dia.

Contracted pelvis

- contracted at inlet
  - ↓
  - when OC < 10 cm
- contracted midpelvis
  - ↓
  - when inter ischial dia (IID) < 8 cm
- contracted outlet
  - ↓
  - when the bituberous dia < 8 cm

Types of contracted pelvis.

1) Naegle's pelvis → in ⊆ one ala of sacrum is absent
(N)ALA

2) Robert's pelvis → in ⊆ both ala of sacral bone are absent.

3) Tri radiate pelvis → it is seen in Vit D deficiency.

4) Rachitic pelvis → it is seen in rickets

⇒ Mode of delivery in contracted pelvis is always Cesarean section

⇒ Recurrent cause of C-section Contracted pelvis

Time of pelvis assessment

* Primigravida → @ 37 wks
* Multigravida → @ time of labor

⇒ Best method of pelvis assessment is MRI.

Cephalopelvic disproportion (CPD)

* Means either the fetus is too big or pelvis is small for this delivery. Does not mean pelvis is contracted.

CPD can occur at level of
> Inlet
> Mid pelvis
> Outlet

* If mild CPD occur @ inlet
→ we can try vaginal delivery
→ K/a trial of labor.

* Trial of Labor is different from trial of ✂ scar.

* Trial of labor → trying vaginal delivery in a female who has mild CPD @ inlet

* Trial of scar → trying vaginal delivery in a ♀ with previous C-section & adequate pelvis

↓

Also K/a VBAC
(Vaginal birth after cessarean)

* Trial of labor is C/I in previous cesarean patient

Clinical case 1

* A G_2P_{1+0} ♀ had a previous normal delivery → this time there is mild CPD @ level of inlet

↓

Mgt: Try vaginal delivery k/a Trial of labor.

Clinical case 2

* G_2P_{1+0} ♀ had previous C-section because of fetal distress → this time pelvis is normal & no fetal distress

↓

Mgt: Try vaginal delivery k/a Trial of scar or VBAC

Clinical case 3

* G_2P_{1+0} ♀ had precious C-section due to fetal distress → this time mild CPD @ level of inlet & no fetal distress.

↓

Mgt: Cesarean section

⇒ Trial of labor for mild CPD @ level of inlet should not be done in previous cesarean section

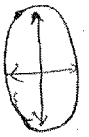
mcq
* Best method for CPD assessment
Trial of labor

mcq
* Best investigation for CPD assess
MRI

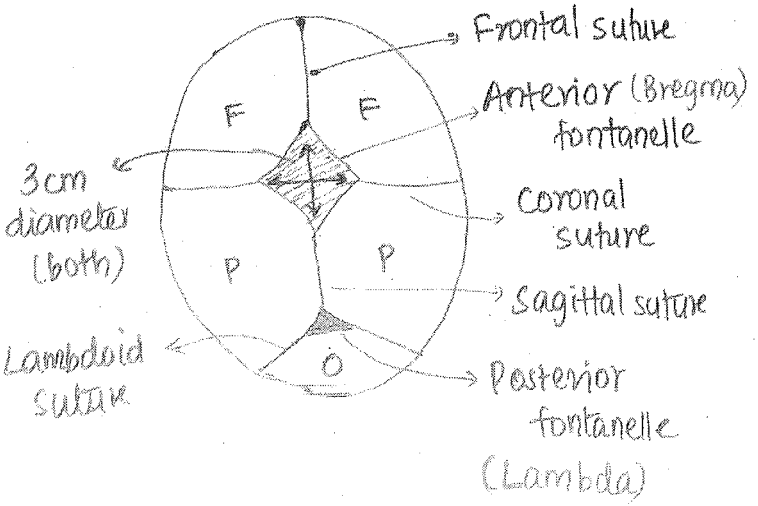
Types of pelvis

* By Caldwell & Mohoy classification
* 4 types:
  1) Gynaecoid pelvis (50%)
  2) Anthrapoid pelvis (25%)
  3) Android pelvis (20%)
  4) Platy pelloid pelvis (5%)

* These are types of normal pelvis

	Gynaecoid	Android	Anthropoid	Platypelloid
	MCQ MC (50%)			MCQ Least common (5%)
	Female like pelvis	Male like pelvis		Flat bowl like pelvis
Shape of inlet	 Transverse oval TD > AP dia.	 Heart shaped TD > AP dia ↓ MCQ Ischial spines are prominent only here	 Vertical oval Only here MCQ AP > TD	 MCQ TD >>> AP
Subpubic angle	Obtuse	Acute		

Fetal skull



* Fontanelles are gap b/w suture lines
* At time of birth → newborn skull → 6 fontanelles
↓
2 are important

- Anterior fontanelle
- Posterior fontanelle

* The part b/w anterior fontanelle & posterior fontanelle is k/a
Vertex

* The part b/w ant. fontanelle on one side & root of nose & the supra orbital ridges on other side is k/a
Brow

* The part b/w root of nose & supra orbital ridges on one side & chin on other side is k/a
Face.

Diameters of fetal head

* AP diameters : Are always longer than transverse dia.

* Longest AP dia is Mentovertical diameter
↓
14 cms
↓
Brow presentation
↓
Always do C-section
(because Cx dilated to 10cm only, can't pass 14 cm)

* 2nd longest AP dia → Submento vertical dia. & Occipitofrontal diameter → Both are 11.5 cms

* Transverse diameters:
Always smaller than AP dia.

Smallest
Miss – Bimastoid dia – 7.5 cm
Tina – Bitemporal dia – 8 cm
SO – Supersubparietal diameter – 8.5 cm
Longest Pretty – Biparietal dia – 9.5 cm

Que: Which is smallest diameter
a) Bitemporal
b) Occipitobregmatic
c) Occipitofrontal
d) Biparietal

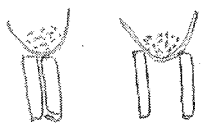
Ans: Bitemporal

LABOR

* Prelabor is characterized by
  1) Lightening
  2) Cx becomes soft → k/a ripening of the Cx
  3) False labor pain felt

* True labor will begin with the onset of true labor pain.

False labor pain	True labor pain
• Irregular or constant	• Regular, rhythmic (on & off)
• Felt in abdomen	• Felt in abd but radiate to back & to thigh
• Remain same	• ↑ in intensity & duration
• Never lead to dilatation of the cervix	• Leads to the dilatation of cervix
• Bag of water absent	• Bag of water present
• Show is absent (mucus mixed with blood)	• Show is present



• False labor pain are relieved by sedation, rest & enema	• True labor pain are never relieved by sedation, rest and enema.

Stages of true labor

1st stage

* From onset of true labor pains till the full dilatation of cervix (10 cm dilated)

↓

Membranes rupture

- if membranes rupture before the onset of labor → ↓ → k/a PROM (premature rupture of membrane)
- if membrane ruptures < 37 wk → ↓ → k/a PPROM (Preterm premature rupture of membranes)

* 1st stage of labor → can be divided in to 2 phases
  - Latent phase
  - Active phase

Latent phase

- Begins ∴ true labor pain

  End = 5 cm dilated cervix

- Main actions

  ⊘ilation of Cx + Cx becomes short (effacement)

- Normal duration

  Nulliparous – 12 hrs

  Multiparous – 8 hr

- Prolonged latent phase

  Nulliparous ≥ 20 hrs

  Multiparous ≥ 14 hrs

- Mgt of prolonged LP

  ↓

  Therapeutic rest (sedation)

  (if false LP → pain subside)

  (if true LP → she will automatically be in the active phase of labor)

Active phase

- ≥ 6cm dilated Cx till the Cx is fully dilated

- Main actions

  ⊘ilatation of Cx + ⊘escend of fetal head

- Normal dilatation

  Nulli → 1.2cm/hr

  Multi → 1.5 cm/hr

- Acc to WHO, min → 1 cm/hr

- Normal descent of fetal head

  Nulli → 1cm/hr

  Multi → 2cm/hr

- Prolonged active phase / Protracted active phase

  ↓

	Nulli	Multi
⊘ilatation	< 1.2cm/hr	< 1.5cm/hr
Descent of head	< 1cm/hr	< 2cm/hr

Active phase (continuation)

Arrest of active phase

* 4 hrs have passed & no dilatation of cervix
* 2 prerequisites before diagnosis of arrest:
  1) Adequate contraction should be present
  2) Membranes should be ruptured

- Mgt of protracted active phase

  - Rule out CPD → ↓ ⊘o C-section (if CPD present)
  - No CPD & pelvis is adequate → ↓ Augment labor (speed up)
    - ~~slow~~ Oxytocin
    - Artifical rupture of membranes

⇒ Mgt. of arrest of active phase is C-section.

~~Important Questions~~

2nd stage of labor

* It begins with full dilatation of cervix & ends with delivery of baby.
* Normal:
  - Nulliparous → 1 hr
  - Multiparous → 30 min
* Prolonged 2nd stage
  - Nulliparous – 2hr
  - Multiparous – 1 hr
* 2nd stage arrest: (+1 hr)
  - Nulliparous – 3 hr
  - Multiparous – 2 hr
* 2nd stage arrest & obstructed labor are the same thing.

Mgt of prolonged 2nd stage

* Cervix should be 10 cm dilated
* See the station of head

≥ +2 (below +2) → Forceps delivery → can be applied only at Station +2 / below it

Above +2 (< +2) → Cesarean section

Mgt of 2nd stage arrest / Obstructed labor

* Condition of mother:
  - Exhausted
  - Tachypnea
  - Acidotic breathing
* Per abdominaly → upper uterine segment is tonically contracted & lower uterine segment is relaxed.
  ↓
  So a ring can be ~~see~~ felt per abdominaly kla
  Bandl ring
* FHS = Fetal distress / fetal death
* Pervaginally (P/V):
  - Foul smelling discharge
  - Hot dry vagina
  - Bleeding P/V
  - Hematuria
  - Caput (swelling on fetal head) present

Mgt of obstructed labor

* 2 principles → Never wait & ~~wait~~ watch
  → Never give oxytocin (uterine rupture)
* Always do immediate C-section
* Complications
  → Rupture of uterus
  → Vesicovaginal fistula

3rd stage of labor

* It begins with delivery of baby & ends with delivery of placenta
* Normal duration
  - Passively → 15-20 min
  - Actively managed → 5-10 min
* Prolonged 3rd stage : ≥ 30 mins

4th stage of labor

* It is 1 hr observation period after the delivery of placenta
* Patient experiences "physiological chills" in this 4th stage (MCQ)

For normal labor

Need 3 things [Passage Push Passenger]

1) Passage → Pelvis → Normal
2) Push → Uterine contractions
   - Pacemaker of contraction is Cornua of uterus (Rt > Lt)
   - @ 2cm/sec
   - Travel down

- Max. contraction is felt at the fundus of uterus
- Uterine contractions ↑↑ intrauterine pressure → measured in 2 units
  1) mmHg
  2) Montevideo unit

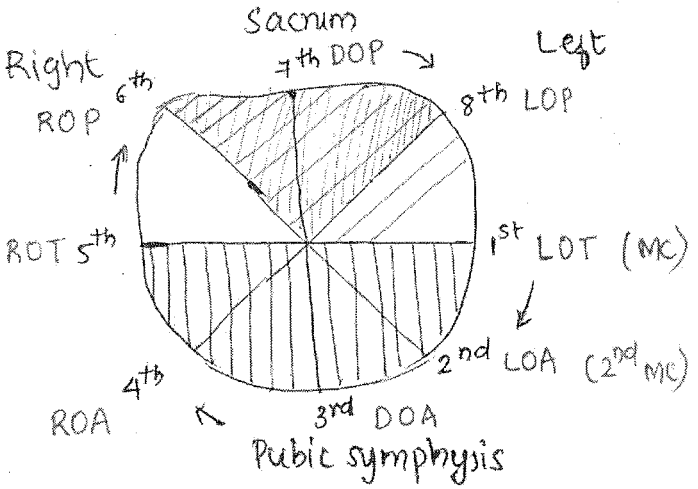
Event	Intrauterine pressure
• Uterine contractions are palpable	10 mm Hg
• " are painful	15 mm Hg
• Early 1st stage of labor	20 mm Hg
• Late 1st stage of labor	50 mm Hg
• 2nd & 3rd stage of labor	100 - 120 mm Hg

Adequate uterine contractions

* It is when ~~to~~ 3 contractions in 10 mins, each lasting for 45 sec with IUP of 60-70 mmHg (220-250 Montevideo units)
* If intrauterine contractions are not adequate → augement labor by → Oxytocin or by artificial rupture of membranes (ARM)
* ARM is C/I in
  - Maternal HIV infection
  - Maternal genital herpes infection
  - IUD of fetus
  - Polyhydramnios

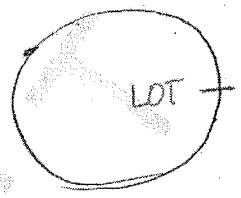
(IUD → can be some reaction with vagina. In poly HA → sudden fluid loss, uterus shrinks, can lead to abruptio placenta)

3) Passenger → Fetus



* MC position in early labor
LOT
* MC position in late labor
DOA
(Direct occipito anterior)
* MC occipito anterior position
LOA
* MC occipito posterior position
ROP

* D → Direct P → Posterior
O → Occipito T → Transverse
A → Anterior



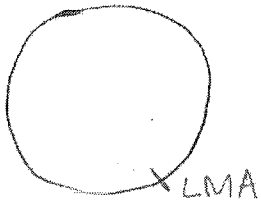
Normal delivery

OP position

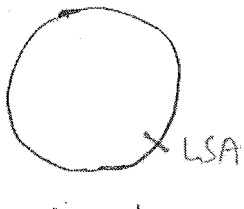
mcq
* MC is LOT (Left occipito transverse)

* Positions from 1 to 5 are positions for normal delivery (normal vaginal delivery)

* Position 6,7,8 together k/a Occipito posterior position
↓
This is malposition



Face



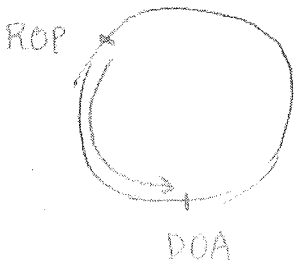
Breech

LMA : Left mento anterior
LSA : Left sacro anterior

* Presentation → Cephalic → Occiput
* MC position in normal delivery is LOT
* 2nd MC → LOA
* Denominator in
Cephalic → Occiput
Face → Mentum
Breech → Sacral

Occipito posterior position

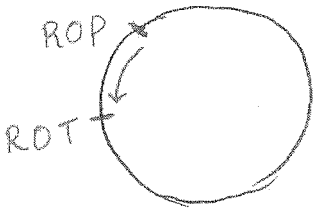
* MC malposition (not malpresentation).
* Lie → Longitudinal
* Presentation → Cephalic
* Presenting part → Vertex
* Position → Occipito posterior (OP)
* OP position is MC in the primigravida females
* All malpresentations are MC in multigravida females.
* MC OP position → ROP
* (MC OA position is LOA)
* In 90% of cases, head will rotate automatically & become occipito ant. & vaginal delivery will occur.
* But this takes a bit longer time
* So best management in OP position → Wait & Watch. MLQ



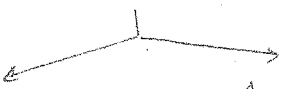
* In 6-8% head of baby starts rotating → it reaches right OT position (ROT) → if it not rotate any further → if head of baby stays in this position for > 30 min inspite of good uterine contractions

↓

Kla Deep transverse arrest



* Deep transverse arrest → occurs @ level of ischial spine
* It is occuring MC in Android pelvis (ischial spine prominent here)
* Mgt of deep transverse arrest is



Android pelvis
↓
Cesarean section

In other pelvis
↓
1) Vaccum delivery
2) Manual rotation followed by forceps delivery
3) Kielland forceps (outdated)
4) C-section (Best)

Note : Vaccum can rotate fetal head but forceps cannot

- Only forceps which can rotate fetal head is

  Kielland forceps

  (which is outdated now)

* Rarely instead of rotating anteriorly, head of fetus rotates posteriorly and becomes

  ↓

  Direct occipito post. position

  ↙ ↘

MC pelvis is

↓

Anthropoid pelvis

(AP dia > T Ø)

↓

Vaginal delivery will occur k/a Face to pubes delivery

If it occurs in any other pelvis

↓

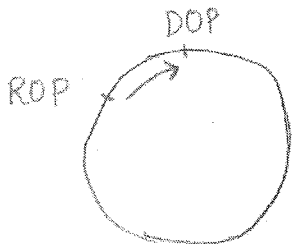
baby will not deliver & remain in that position for ≥ 30 min

↓

k/a Deep sacral arrest

↓

Mgt: C-section



Face presentation

* Here denominator is chin/mentum
* Mentoanterior → Vaginal delivery possible
* Mentoposterior → Always C-section
* In which anomaly face presentation is common

  Anencephaly
* MC ~~presentation~~ position in face

  LMA
* Head in face is delivered by

  Flexion.

Brow presentation

* Always C-section
* Engagement diameter

  Mentovertical (14 cm)

Transverse lie

* Lie is transverse
* Presentation is shoulder
* Denominator → Dorsum

  ↙ ↘

Dorso anterior Dorsoposterior

- Vaginal delivery is not possible
- Always C-section (if baby alive or dead)

MCQ

* Maximum chances of cord prolapse in
  Transverse lie
  (so should do C-section)

* If a female with transverse lie comes during pregnancy/early labor
  ↓
  1 more option
  ↓
  Try external cephalic version
  ↓
  Per abdominally try to rotate the baby & make it cephalic
  ↓
  Its an OPD procedure
  ↓
  Indications
  - Tranverse lie
  - Breech presentation

* Here there is risk of fetal distress
  → so ~~time~~ might go for C-section.

* Time: ≥ 36 wks pregnancy upto latent phase of labor
  (Because lungs of fetus should be mature → so ≥ 36 wk)

Pre-requisite

1) Membranes should be intact
   (i.e, adequate amniotic fluid)
2) No C/I to vaginal delivery
   (eg: placenta previa)
3) Patient should not be high risk and pregnancy should not be precious
4) Continous fetal heart rate monitoring should be done during procedure.
5) Not done in previous cesarean section patient

MCQ

* Vaginal delivery is not possible in
  1) Transverse lie
  2) Oblique lie
  3) Brow presentation
  4) Face → Mento posterior

Breech Presentation

* Lie → Longitudinal
* Presentation → Breech
* Breech is MC malpresentation
* Incidence of breech:
  @ 28 wks → 20%
  @ 34 wk → baby spontaneously rotate → 5%
  @ term → 3-4%

* MCC → Prematurity
* MCC of recurrent breech
  Uterine malformation

Types of breech

1) Thigh flexed + Knee flexed
↓
Complete breech
↓
P/V = Feet + Genitalia + Buttocks

* MC in multigravida females
* Chances of cord prolapse : 6 %

2) Thigh flexed + Knee extended
↓
Frank breech
↓
P/V : Buttocks + Genitalia

* MC in primigravida females
* Least chances of cord prolapse

3) Thigh extended + Knee flexed
↓
Knee presentation
↓
Should do C-section

* ~~MCXI~~

4) Thigh extended + Knee extended
↓
Footling presentation

* Maximum chances of cord prolapse.

Cord Prolapse

* Highest chances / risk
  Transverse lie > Footling presentation > Knee presentation
* It is an emergency as the baby can die within minutes of cord prolapse → leads to fetal distress

Cord prolapse / fetal distress
- Dead → Vaginal deli. except if baby in transverse lie
- Alive
  - Cx fully dilated (2nd stage of labor) → (station of fetal head)
    - < +2 (above +2) → C-section
    - ≥ ±2 (below +2) → Forceps
  - Cx not dilated (1st stage) → C-section

* Transverse lie + Cord prolapse

Mgt: C-section (always)

Note:

* At or below +2 station, forceps is much quicker than C-section

So in fetal distress at or below +2 station forceps is prefered.

* In fetal distress, vacuum is C/I.

———— × ————

Breech presentation (continuation)

* In both knee & footling presentation do C-section

* If the head of fetus is extended in breech → Stargazer breech

Indication for C-section.

Mgt of breech

* Depends on

1) Patient coming during pregnancy or early labor
2) Patient coming during late labor

* If patient comes during pregnancy or early labor then

Mgt → External cephalic version

* If patient coming during late labor → 3 options

(1) C-section: done in

- Footling
- Knee presentation
- Stargazer breech
- Breech with previous C-section
- Breech with weight of baby ≥ 3.5 kg
- Preterm baby

Relative

Primi with breech

(2) Assisted breech delivery

Vaginal breech delivery occurs due to mothers effort → whenever needed obstetrician provides assistance.

(3) Breech extraction

Mother under general anesthesia → entire delivery is done by obstetrician ∑ out effort of mother.

Indication: If 2nd twin is transverse lie & a successful IPV has been done.

Maneuvers used in assisted delivery
Breech extraction

1) If buttocks delivered first
   - Engaging diameter is Bitrochanteric dia (10cm)
   - Manoeuver used is
     i) Groin & traction
     ii) If legs are extended → Pinard manoeuver
     P < Pinard / Popliteal fossa

Note: After the delivery of buttocks, baby is wrapped in warm towel so that the cord does not shrink → k/a
~~Salvagena~~
Savage technique.

2) 2nd part → Shoulder
   - Engaging diameter is Bisacromial dia (12 cm)
   - Manoeuver used
     Lovset manoeuver
     (rotate the baby keeping back anterior)

3) Last part → Head → ie, k/a after coming head of Breech
   - Engaging diameter is Sub occipito frontal

Note: In breech, head is delivered by flexion
- Manoeuver used:
  i) Bum Marshall technique
  Let baby hang by its weight → hold legs → take towards mothers abdomen.
  ii) Malar flexion & shoulder traction
  (Mauriceu smillie viet technique)
  iii) Best is Piper's forceps

* Sometimes in delivery of head → face of baby faces towards pubic symphysis → use Prague manoeuver (N occiput to pubic symphysis)

* If head of breech gets entrapped

Preterm breech → Mgt: Dubresen's incision of Cx

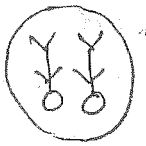
Cx

Term breech → Zavaneilli Manoeuver → Push the delivered parts back in to the uterus and then do C-section

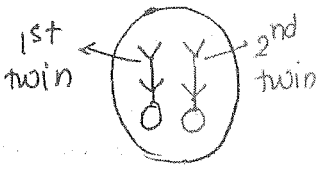
Delivery in Twin Pregnancy

* MC presentation in twins

Both the twins vertex



* Mode of delivery in twins depends on the presentation of first twin.

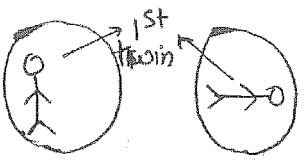


↓

Vertex

↓

Vaginal delivery possible.

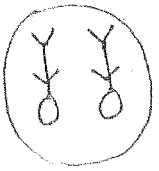


↓ ↓

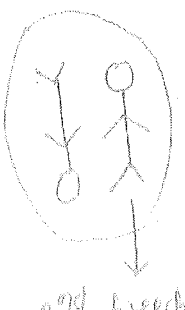
Breech Transverse

C-section

If first twin is Vertex :

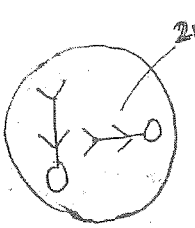


* First & 2nd vertex
* Normal vaginal delivery of first twin
* Do not give inj. methyl ergometrine after delivery of 1st twin
* Deliver 2nd twin vaginally
* Then can give inj. methyl ergometrine



@ 1st twin → normal vaginal

@ 2nd twin → Assisted breech delivery



* First twin delivered vaginally.

↓

After delivery of first twin

↓

Take patient to OT, give general anesthesia so that uterus relax

↓

Hand inside uterus → hold the leg of baby & rotate it so that it becomes breech

↓

K/a internal podalic version

↓

this should be followed by Breech extraction.

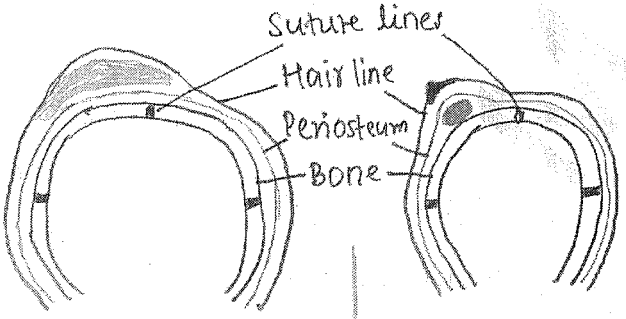
Internal podalic version

* Done under general anesthesia (Ext. Cephalic V → no GA)
* Done in OT (ECV → OPD)
* Only indication is if 2nd twin in transverse lie
* Risk of IPV is Uterine rupture
* C/I in previous C-section patients.

Bandl's ring	Schroeder's ring
* Seen in obstructed labor	* Injudicious use of oxytocin
* Pathological ring	* Physiological ring.
* Retraction ring	* Constriction ring.
* Felt per abd. (P/A) not felt per vaginaly	* Felt P/V not felt P/A.
* Can lead to rupture uterus & VVF (~~vagino~~ vesicular vaginal fistula)	* —
* Mgt: C-section	* Mgt: Relax the ring

* Because it is edema → pits on giving pressure	* Not pit on giving pressure
* Above the periosteum → can cross suture lines	* Below periosteum so cannot cross the suture lines
* Present @ time of birth & disappears within few hours	* Appear within few hrs of birth & disappears automatically in few days
* ~~X~~	* Collection of blood → associa. with jaundice
* Never associated c # of the underlying bone	* Can be asso. with # of underlying bone

Swellings on fetal head



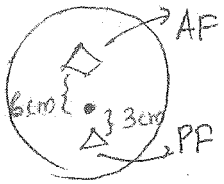
CAPUT SUCCEDENUM	CEPHAL HEMATOMA
• Diffuse edematous swelling above the periosteum	• Localised collection of blood below periosteum
• Formed because head stays in one position for a very long time during labor	• Traumatic or instrumental delivery.

Instrumental delivery:

* It is done only if head of foetus @ +2 station or below that.
* Forceps + Vacuum (Ventouse)
* If forceps is applied at +2 station k/a Low forceps application
* Various forceps can be used for this application

* If forceps is applied below +2 station k/a
Outlet forceps application

* Wrigley's forceps is only used for it.



Pre-requisites for forceps delivery

F - Favourable position & station
O - Os should be fully dilated (i.e, cervix dilated to 10 cm)
R - Membrane should be ruptured and head should be rotated
C - Uterus should be contracted
E - Episiotomy should be given
Bladder should be empty
Head should be engaged
P - Pelvis should be adequate & No CPD

* In case of vacuum, everything is same except
- Can be applied when Cx ≥ 6 cm
∴ Can rotate the head of fetus

* Forceps is applied along the occipitomental diameter

* Vacuum is applied along the flexion point (3cm ant to post fontanelle & 6cm post to ant fontanelle)

Forceps

- Pressure / Traction
Primi – 20 kg
Multi – 13 kg

- Indication:
1) Maternal distress
2) Fetal distress in 2nd stage
3) Prolonged 2nd stage of labor
4) Prophylactically used in CVS patients & in PIH.

- Advantages:
1) Can be used in fetal distress
2) Can be used in preterm deliveries
3) Can be applied in all these presentation where C-section is not mandatory
* Vertex
* Face - Mento ant
* After coming head of breech (Piper's forcep)

Vacuum

- Initial pressure is 0.2 kg/cm^2
- Max: 0.8 kg/cm^2
- During process, a caput (swelling) is created artificially or hydrogenically
↓
k/a Chignon

- Indications are same except
↓
- Not used in fetal distress
- In CVS patients preferred is vacuum.

- Disadvantages
x
x
- Vaccum can be used only in vertex presentation.

Forceps

- Leads to more maternal injuries
- Max. no: of pulls is 3
- If after 3 attempts baby is not delivered → k/a Failed forceps
- 2ts Mgt : C - section

Vacuum

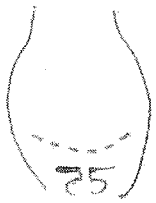
- Leads to more fetal injury (Cephal hematoma) Thus not used in preterm babies
- Max pulls is 3
- If not delivered after 3 attempts k/a Failed vacuum
- Mgt → C-section

Cesarean Section

* It is perabdominal delivery of a viable fetus after giving skin incision and uterine incision

Incisions

Skin incision → MC used is PFANNEN STEIL incision (Cosmetically good)



Uterine incision → Lower segment C-section (LSCS)

MCQ: In LSCS chances of rupture are less in next pregnancy (as LS only passively dilates)

Rupture : 0.2 - 1.5%

Uterine incision → Upper segment C-section (USCS) → k/a Classical C-section

MCQ: Chances of rupture in next pregnancy are high as upper segment actively contracts & relax during labor

4-9% chances

LSCS

* As chances of rupture are less, in next pregnancy if pelvis is adequate & there is no CPD
↓
Vaginal delivery can be tried
↓
k/a Trial of scar or VBAC (Vaginal Birth after C-section)

- Type of incision

MC done → KERR incision → less chance of rupture (0.2 to 1.5%)

DE LEE incision or KRONIG incision
- 1-7% chances of rupture (so not used)

USCS

* As chances of rupture are high, in next pregnancy always repeat C-section is done.
↓
Vaginal delivery is not tried.

- Type of incision

SANGERS incision
Rupture : 4-9%

T-shaped incision (4-9%)

Indications for Classical C-section

* Done only if lower segment cannot be approached.

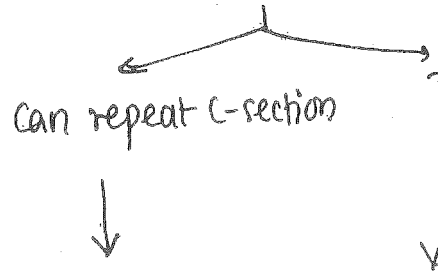
  eg:- Carcinoma cervix
  - Placenta previa with blood vessels running all over lower segment
  - Huge fibroid in lower part of uterus / cervical fibroid
  - If adhesions are present in the lower part of uterus
  - If a preterm C-section is done & lower segment has not yet been formed.
  - Post mortem C-section

Note

* If LSCS has been done twice, next time (ie, 3rd time) LSCS should be done and vaginal delivery should not be tried.

Clinical Case

* If a ♀ c previous C-section is in labor & previous C-section indication was fetal distress.



Repeat C-section

1) If classical C-section done before
2) If previous 2 LSCS have been done
3) Previous H/O uterine rupture
4) If indication of C-section was contracted pelvis

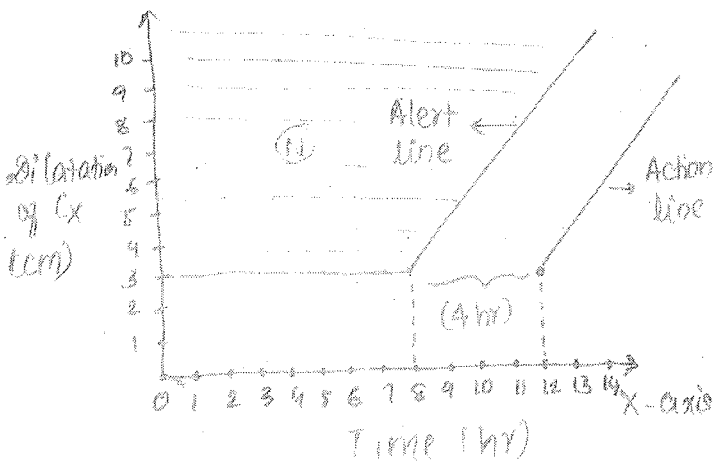
* In same patient → trying vaginal delivery → k/a VBAC
  ↓
  Danger: Uterine rupture
  ↓
  Signs of impending rupture:
  1) Uterine scar will become tender (not visible)
  2) Fetal distress → Fetal tachycardia
  ↓
  Mgt: Immediate C-section

* Signs of Uterine rupture:
  1) Fetal bradycardia
  2) Mother → Shock ← Tachycardia, Hypotension
  3) P/A uterine contractions will stop.
  4) Fetal parts will be felt superficially
  5) P/V - Hematuria, fresh bleeding, loss of fetal station

* Mgt of uterine rupture
  - Immediate laprotomy & repair of uterus.

Partogram

* Suggested by WHO → as a part of safe motherhood programme
* Based on principles:
  - Active phase begins when $C_x \geq 3$ cm
  - In active phase, minimum rate of dilation of C_x should be 1 cm/hr
  - Latent phase was said to be prolonged if it was > 8 hrs
  - Two parallel lines on the partograph
    i) Allert line
    ii) Action line
    Time b/w them is 4 hrs
  - Labor is said to be normal till it remains to the left of alert line.



* (N) → when it lies left to alert line
* If it reaches alert line → there is some problem

* In a partogram, the following are recorded.

Parameter	Time interval at which recorded
1) Pulse rate	30 min
2) Uterine contn	30 min
3) Oxytocin given	30 min
4) Time interval	30 min (each small box in partogram → 30 mins)
5) BP	4 hrs (If PIH → 1 hrs)
6) Temperature	4 hrs
7) Fetal heart rate	1 hr

* FHR is recorded every 1 hr on partograph but measured

	Low risk	High risk
1st stage of labor	30 min	15 min
2nd stage of Labor	15 min	5 min

Per vaginal (P/V) examination during labor

1) At time of admission

Latent phase → 4 hrly

Giving oxytocin → after every 2 hr

If Cx is ≥ 7 cm → 2 hrly

If Cx is ≥ 9 cm → 1 hrly

---

* What is the station of head, when it is engaged?

0 station

* When head is engaged then what is ~~pre~~ per abdominal finding →

≤ 1/5th of head is palpable per abdominally.

Engaging diameter of fetal head

- Transverse dia → Biparietal diameter
- AP diameter

Vertex → Suboccipito bregmatic (9.5 cm)

Brow → Mentovertical (14 cm)

Face → Submento bregmatic (9.5 cm) or submentovertical (13.5 cm)