

Anatomy of female genital tract

Embryology

- * urogenital sinus →
- | <u>male</u> | <u>female</u> |
|----------------------------------|---|
| • urinary bladder | • urinary bladder, urethra |
| • urethra except navicular fossa | • urethral & paraurethral glands (Skene glands) |
| • Prostate gland | • vagina |
| • prostatic utricle | • Bartholin's / greater vestibular glands |
| • Bulbourethral glands | |

Skene glands:

- Homologous to prostate in males
- largest paraurethral glands
- one pair of ducts open on either side of the external urinary meatus

Fourchette:

- It is the area where both labia minora posteriorly meet

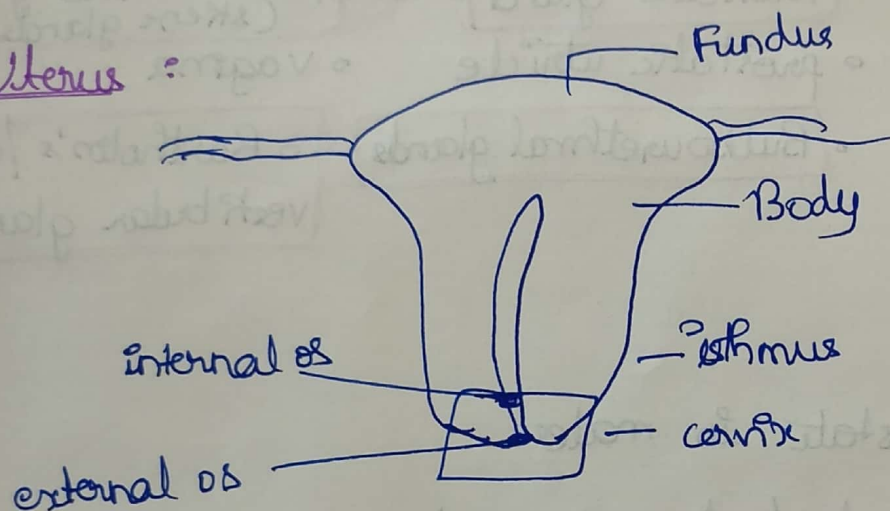
Bartholin's gland:

- opens b/w hymen & labia minora in inner aspect of labia minora (7 o'clock & 5 o'clock positions)
- Bartholin's cyst → TOC: marsupialization
- Bartholin's abscess TOC: IND

wall of vagina

- Vaginal defence is d/t to acidity of vagina $\leq$ is produced by estrogen. The vaginal defence is lost after 10 days of birth due to withdrawal of maternal hormones

Uterus :



- The lining of cervix is ectated columnar (below anatomical internal os)
- The area bounded by anatomical internal os above & histological internal os below is called the isthmus of uterus

• Corpus - cervix ratio before puberty is 1:2,
at puberty is 2:1
adults - 3:1

After menopause uterus & cervix atrophy

obturator lymph nodes

Fallopian tube :

- is lined partially by ciliated & partially by nonciliated columnar epithelium
- surrounded by peritoneum on all sides except along line of attachment of mesosalpinx
- longest part is ampulla = is m/c site of ectopic pregnancy
- site for tubectomy is isthmus
- total length is 10-12 cm
- interstitium is the narrowest part & forms anatomic sphincter whereas physiological sphincter is formed by isthmus

Blood supply of uterus :

- ovarian artery
- uterine artery
- anterior division of internal iliac arteries

can be ligated
in uncontrollable
PPH

* m/c organ = is susceptible to unintentional damage by
hysterectomy is ureter

Perineal anatomy:

• Muscles attached to perineal body are

- external anal sphincter
- superficial transverse perineal
- deep " "
- bulbospongiosus muscle
- levator ani

* ~~Ovary is connected to lateral pelvic wall by infundibulopelvic ligament~~

* Round ligament runs from fundus of uterus to labia majora

* Supports of vagina → perineal body, pelvic diaphragm, levator ani muscle

Vagina

* The main source of physiological secretion found in vagina is cervix

* ~~supplied by uterine artery~~

* ~~lined by stratified sq. epithelium~~

* Its posterior wall is covered by peritoneum in the form of pouch of Douglas (recto uterine pouch)

Nabothian follicles → d/t erosion of cervix

- Non pregnant uterus → 70g, 15 ml, pear/pyriform shaped
- pregnant uterus → 1-1.1kg, globular-spherical shaped
- Enlargement of uterus is d/t hypertrophy > hyperplasia
- Cervix
 - cervix becomes soft due to estrogen
 - **eversion of cervix**
 - **↑ cervical secretion & IgG**
- Corpus luteum functions maximally by 6-7 weeks of pregnancy
- pH of vagina $\Rightarrow$ 4.5 to 5.5
during pregnancy $\Rightarrow$ 3.5 to 4.5

Hematological changes → begins by 6 weeks, max changes is in T2 btw 28-32 wks

- Blood volume → ↑
- plasma volume → 40 to 50% ↑
- RBC mass → 18 - 20% ↑
- Hb mass → ↑
- Hb concentration → ↓
- Hematocrit ↓
- viscosity ↓

Cell count

- Reticulocyte count $\uparrow$
- RBC count $\downarrow$
- WBC count $\uparrow$ (upto 15,000)
- Platelet count $\downarrow$ (upto 1 lakh)
- Neutrophils $\uparrow$ (neutrophilic leukocytosis)

Iron parameters

- ~~Iron~~ $\rightarrow$ $\rightarrow$
- Total requirement of iron during pregnancy $\rightarrow$ 1000 mg
in T₁ $\rightarrow$ 3-4 mg/day
T₂ & T₃ $\rightarrow$ 6-7 mg/day
- S. Iron & S. ferritin $\downarrow$ in T₂
- S. transferrin $\uparrow$
- TIBC $\uparrow$
- PSAT $\downarrow$

Clotting parameters

- all clotting factors except 11 & 13 increase in pregnancy
 $\downarrow$
N/ $\downarrow$
- maximal $\uparrow$ is τ factor I (Fibrinogen)
- Fibrinolytic activity is $\downarrow$ [dlt $\downarrow$ TPA & $\uparrow$ TPAI]
- protein C & S are $\downarrow$
- Antithrombin levels are $\textcircled{N}$,
- BT & CT are $\textcircled{N}$

◦ ESR ↑

◦ leucocyte ALP, C3, C4, CRP, paracalcitonin → ↑

◦ cell mediated immunity is predominantly suppressed during pregnancy

CVS

◦ HR ↑ by 10 bpm

◦ stroke volume ↑

◦ Cardiac output → ↑↑
(CO)

◦ ~~peripher~~

→ CO starts ↑ing by 5 weeks & reaches max by 32 weeks

→ CO during postpartum period > labour > pregnancy
(immediate) (50% ↑) (40% ↑)
(70% ↑)

→ CO returns to ~~prepreg~~ prelabour levels by 48 hours & prepregnant levels after puerperium

◦ Both peripheral & pulmonary vascular resistance ↓↓ during pregnancy (d/t relaxin)



↓ diastolic pressure

Pressure changes

- SBP → $\uparrow$ / $\downarrow$
- DBP → $\downarrow$
- MAP → $\downarrow$
- $\textcircled{X}$ Femoral venous pressure → $\uparrow\uparrow$ $\left[\textcircled{N} \rightarrow 8\text{mmHg} \right.$
pregnancy → 24mmHg $\left. \right]$

Other changes

- apex beat moves up & out → 4th ICS, lateral to mid clavicular line
- All chambers of heart $\uparrow$ in size & pericardial effusion $\pm$
 $\therefore$ cardiac silhouette $\uparrow$
- ECG → $\textcircled{C}$ axis deviation
- S3 may be heard
- S1 → loud & split
- S2 → no changes
- Systolic murmur (low grade) → intensified during inspiration
- diastolic " → transient soft in 20%
- continuous murmur from breast → mammary souffle
- uterine blood flow → $\uparrow$ - from 50 ml to 750 ml at term
- Renin → increased
- Angiotensin → $\uparrow\uparrow$ } $\rightarrow$ water retention & $\uparrow$ plasma volume

◦ Transverse diameter of chest $\rightarrow \uparrow$ by 2 cm
circumference $\rightarrow$ 6 cm $\uparrow$ } $\rightarrow \uparrow$ TV (tidal volume)

◦ Diaphragm $\rightarrow$ moves up by 4 cm

$\downarrow$
 $\rightarrow$ it obliterates RV & ERV space $\Rightarrow \downarrow$ RV & ERV

$\rightarrow$ IRV unchanged

$\rightarrow$ FRC = RV + ERV $\rightarrow \downarrow$ FRC

VC (IRV + TV + ERV) $\rightarrow$ $\textcircled{N}$ (unchanged)
 $\quad \quad \quad \uparrow \quad \quad \downarrow$

IC $\rightarrow \uparrow$

TLC $\rightarrow$ slightly $\downarrow$ $\textcircled{N}$

◦ Respiratory rate $\rightarrow$ unchanged

◦ minute ventilation $\rightarrow \uparrow$

◦ max breathing capacity $\rightarrow \textcircled{N}$

◦ Respiratory alkalosis $\rightarrow$ (d/t $\uparrow$ min ventilation)

Renal system

◦ Kidney $\uparrow$ by 1 cm

◦ RPF $\uparrow$

◦ GFR $\uparrow$ & tubular reabsorption $\uparrow$

◦ urea clearance } $\uparrow \Rightarrow \downarrow$ serum levels
urea " }
creatinine "

• (R) hyponatraemia may be seen

www.FirstRanker.com

www.FirstRanker.com

• all solutes $\downarrow$ $\rightarrow$ plasma osmolality $\downarrow$

Endocrine $\rightarrow$ Pituitary

- Ted $\rightarrow$ GH, prolactin, ACTH, CRH, size of gland
- $\downarrow$ ed $\rightarrow$ FSH, LH
- unchanged $\rightarrow$ TSH
- prolactin (N) - 25 mg/ml
pregnancy - 150 mg/ml

Thyroid gland

- Total $T_3, T_4 \rightarrow \uparrow$
- Free $T_3, T_4 \rightarrow$ (N)
- TBG - $\uparrow$
- fetal thyroid gland starts taking iodine by 11 weeks
- fetus starts releasing thyroxine by 20 weeks
- I_2 req $\rightarrow \uparrow$
- size of thyroid gland $\rightarrow \uparrow$
- TSH $\rightarrow$ slight fall (trimester dependant)
- HCG $\begin{matrix} \alpha \\ \beta \end{matrix} \rightarrow$ resembles TSH, FSH, LH
- BMR $\uparrow$ by 25%

Adrenal

- DHEAS is the only adrenal hormone ↓ during pregnancy
- all other adrenal hormones ↑
- Insulin sensitivity ↓ by 70% during pregnancy
→ ∴ ↑ insulin production

1st stage of labour:

◦ Begins τ true labour pain & ends τ full dilatation of cervix

<u>True labour pain</u>	<u>False pain</u>
◦ $\uparrow$ intensity	◦ —
◦ regular	◦ Irregular
◦ Interval shortens	◦ —
◦ not relieved by enema, sedation	◦ relieved
◦ a/w ex effacement, show & bag of membr.	◦ not associated

* Full cervical dilatation $\rightarrow$ 10 cm

* m/c presentation $\rightarrow$ cephalic

" attitude $\rightarrow$ flexion

" part $\rightarrow$ vertex

m/c diameter $\rightarrow$ suboccipito bregmatic in AP direction
& biparietal is transverse

(both are 9.5 cm)

$\therefore$ req 10 cm dilatation

• full dilatation of C_x to delivery of fetus

3rd stage → delivery of fetus to delivery of placenta

4th stage → 1hr observation after placental delivery

1st stage of labour

latent → upto 4cm dilatation of C_x
active → after 4cm dilatation

• latent phase → 8 hrs in primi (max) & 6 hrs in multigravida

• active phase → proceeds at 1 cm/hr

• ARM is done when dilatation reaches 4cm

• Total duration → 12 hrs in primi & 6 in multigravida

2nd stage

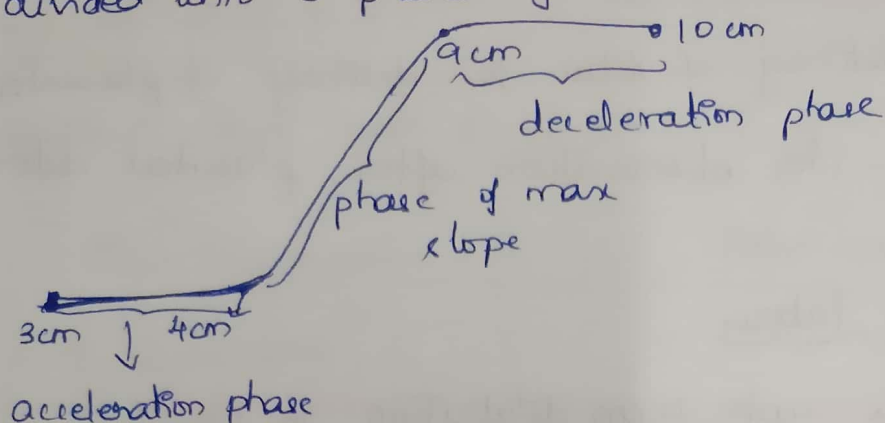
- 2 hrs in primi & 1 hr in multigravida

3rd stage

• 5 mins (due to active management)

Active phase of 1st stage

- divided into 3 phases by Friedman's curve



Partogram

- New partogram doesn't include latent phase

- Components :
 - * fetal heart rate
 - Ⓝ ⇒ 110-160
 - < 100 → brady
 - > 180 → tachy
 - ↓
 - assessed every 15 min
 - in 1st stage & every 5 min in 2nd stage

- * liquor →
 - I - intact
 - C - clear
 - M - meconium
 - B - blood stained liquor

- * moulding → indicates severity of cephalopelvic disproportion

- 0+ → obliteration of parietal bones, but no overriding
- 2+ → reducible overriding
- 3+ → irreducible overriding

• dilatation of Cx

- time in X axis
- dilatation in Y axis
- dilatation is marked by the alphabet 'X'

• descent of head

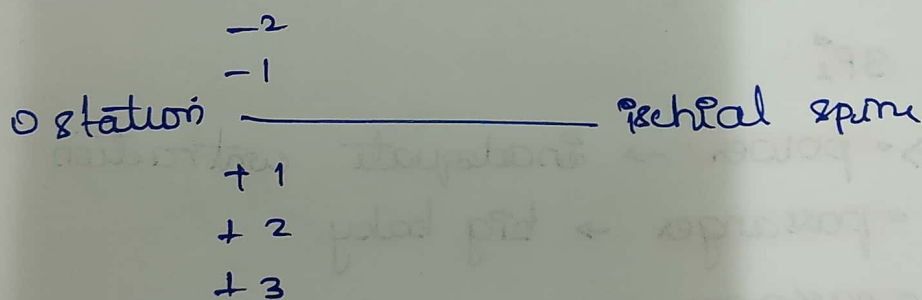
- marked by '0'
- assessed by crichton method (by #)
- scores 5/5 to 0/5

* PV is done only at 4hrs interval unless otherwise needed.

→ action line is 4hrs after alert done

* Crichton scores : $\frac{1}{5}$ & $\frac{0}{5}$ → engaged head &
 $\frac{5}{5}$ → mobile head

* Ischeal spine → 0 station → head engaged



station of head & AFI are not present in partogram

• Contractions

ideal → atleast 3 contractions in 10 mins each
 for 40 seconds

 → 1 contraction < 20s

 → 2 contractions each < 20s

 → 1 contraction 20-40s

 → 1 contraction > 40s

o oxytocin infusion

→ can be fed upto 32 IU/min

o drugs administered

o maternal pulse BP

o temp, urine vol & urine acetone

Abnormalities in partogram:

o prolonged latent phase

o 1° dysfunctional labour (m/c)

→ slow progression from the very beginning

o 2° arrest of c_x dilatation

→ causes - 3P_s

(m/c) → o power → inadequate contraction

o passenger → big baby

o passage

Definite signs of separation of placenta:

o Kustner sign → cord doesn't reved on pushing the uterus upward in abdomen

o Schroeder sign → uterus rises in abdomen as placenta pass down into vagina (filling of placenta in vagina)

o gushing of blood

o lengthening of cord

- uterotonic within 1 min of delivery
[$-m$ is oxytocin 10 U i.m.]
- ~~general~~ controlled cord traction
- uterine massage

Pressure of uterine contraction

1st stage $\rightarrow$ 50 mm Hg

2nd stage $\rightarrow$ 100 mm Hg

Non pregnant & early pregnancy $\rightarrow$ 2 to 3 mm Hg

- Best test for adequacy of pelvis $\rightarrow$ trial of labour
- safest method for pelvimetry $\rightarrow$ MRT

Diabetes complicating pregnancy

Overt diabetes : $F \geq 126 \text{ mg/dl}$

$PP / RBS \geq 200 \text{ mg/dl}$

$HbA_{1c} \geq 6.5 \%$

Gestational DM :

◦ American guidelines :

- selective screening
- 24-28 wks
- 2 step approach

low risk $\rightarrow$ (N) BMI, no family history, past history

intermediate

high $\rightarrow$ BMI ≥ 30 , family history, past history

High risk $\rightarrow$ BMI > 30 , family H/O DM, past H/O DM, previous macrosomic baby $> 4.5 \text{ kg}$, Bad obst. history, recurrent abortions, polyhydramnios / IUD

Sullivan test : 50g glucose $\xrightarrow{1 \text{ hr}}$ $< 140 \text{ mg/dl}$

if $\geq 140 \text{ mg/dl}$

(N)

100g GTT

- done $\bar{c}$ 8 to 14 hrs fasting
- 3 day prior to test - (N) meals
- unrestricted physical activity
- 100g glucose gn & 4 samples taken

- i) Fasting - 95 mg/dl
 ii) 1 hr - 180 mg/dl
 iii) 2 hr - 155 mg/dl
 iv) 3 hr - 140 mg/dl

→ Carpenter & Coustan criteria

2) WHO - no screening test, directly diagnostic test

• after fasting → 75 g glucose

F - 95 mg/dl

1 hr - 180 mg/dl

2 hr - 155 "

3) IADPSG (International association of diabetes pregnancy study) → no screening test

- 75 g glucose after fasting

F - 92 mg/dl

1 hr - 180 "

2 hr - 153 "

4) DIPSI (diabetes in pregnancy study group in India)

* 100% screening at booking visit

* no fasting.

- 75 g of glucose & sample after 2 hours

<u><120</u>	<u>120-140</u>	<u>140-200</u>	<u>>200</u>
Ⓝ	• Impaired glucose tolerance	• gestational DM	• overt DM

↓
do HbA_{1c} to see if it
is overt

The complications are more in overt diabetes than GDM

- Anomaly rate in (N) person → 2.5%
- anomaly ^{rate} in a person w HbA_{1c} 6-6.9% → 5%
7-7.9% → 11%
≥ 8% → 15%
≥ 10% → 20%

Human placental lactogen (HPL)
cortisol
estrogen
progesterone

} all contribute to ↑
insulin resistance
↓
GDM

◦ HPL acts like GH

↓

- synthesised from syncytiotrophoblast
- lipolytic → ↑ FFA

∴ in pregnancy → fasting hypoglycemia &
post prandial hyperglycemia

1st trimester

- abortions - d/t chromosomal abnormalities
- hyperemesis → DKA

2nd Trimester

- anomalies
- macrosomia → CPD
- polyhydramnios → overdistension → abruptio placenta
- malpresentation
- cord presentation & cord prolapse
- still births
- IUD
- IUGR
- preterm labour

During labour :

- prolonged labour
- shoulder dystocia
- PPH $\begin{matrix} \text{traumatic} \\ \text{atonic} \end{matrix}$
- instrumental delivery
- LSCS
- still birth
- ketoacidosis

Postpartum

- subinvolution
- infection → endometritis, puerperal sepsis
- venous thromboembolism
- future DM

Neonatal complications

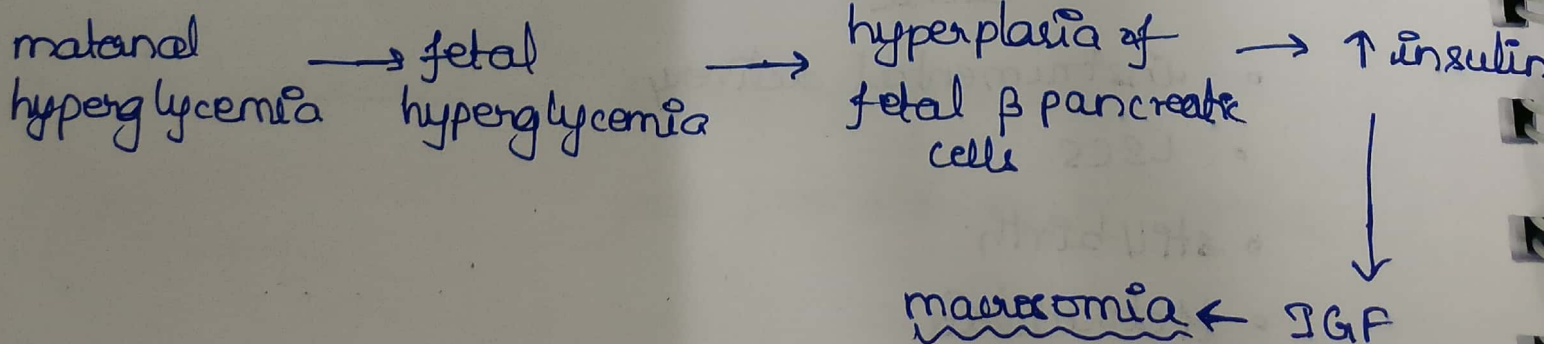
- hypoglycemia
- hypocalcemia
- hypokalemia
- polycythemia → hyperbilirubinemia
- DM
- Low IQ
- cognition & memory disorder
- cardiomyopathy

• macrosomia is the m/c (45%)

• pre eclampsia (28%)

→ delayed lung maturity

Peterson hypothesis



- Overt DM → ◦ insulin
+
◦ Diet → 55% carb, 20% protein,
25% fat & <10% saturated fat

anomalies in overt diabetes

- VSD, ASD, TGA, cardiomyopathy
↓
(most specific)
- neural tube defects
- renal agenesis, duplication of ureters
- imperforate anus, fistulas, atresias
- caudal regression syndrome (rare but specific)
- single umbilical artery

◦ Rx of GDM :

* meal plan → 40% carb, 20% protein, 40% fat
for 2 wks & GTT

↓
F < 95 mg/dl

1 hr < 140 mg/dl

2 hr < 120 mg/dl

} if achieved
then
continue meal
plan till end.

If not, then start

* insulin → regular + intermediate
0.7-1.0 U/kg/d in divided doses

◦ 1st trimester screening — at 11-14 weeks

i) maternal age

ii) nuchal translucency

iii) β HCG

iv) PAPP-A

If +ve, then do chorionic villous screening

If not done, then do

◦ 2nd trimester screening → only 60-70% sensitivity

* triple test → β HCG, unconjugated E₃ (estriol),
(16-18 weeks) * maternal serum AFP
or

* quadruple test → triple test + inhibin

◦ Targeted anomaly scan

* 18 to 20 weeks

◦ fetal echo — at 24 weeks

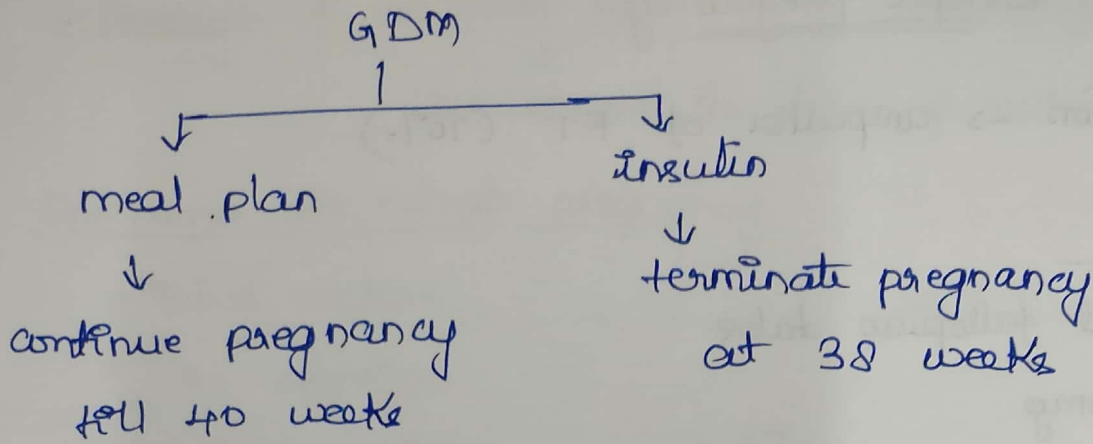
* In case of overt diabetes, admit the pt at 34 weeks &

do i) fetal K₂ count

ii) NST / biophysical profile

iii) o/c scan — AFI & estimated fetal weight

& terminate pregnancy at 38 weeks



Intrapartum

- stop morning dose of insulin
 - maintain blood sugar at 70-100mg/dl
 - if $< 70 \rightarrow$ dextrose
 - $> 100 \rightarrow$ infusion regular insulin
 - hourly CBG
- * target calorie req/day $\rightarrow 30-35$ Kcal/day
- if BMI $> 30 \rightarrow 25$ Kcal/day

Ectopic pregnancy

- m/c location $\rightarrow$ ampulla of FT (70%)

○ Risk factors

- abnormal fallopian tube

- Tubectomy

- prior ectopic pregnancy

- salpingitis & other infections (PID) $\leftarrow$ N-gonorrhoea
(m/c) chlamydia

- vulvovaginitis isthmica nodosa

- ART (artificial reproductive technique)

- smoking

- Copper & progestin releasing IUDs

- progestin only contraceptives (POP or minipills)

* No work in combined OCF

CF =

- pain abdomen (acute)

- amenorrhoea

- vaginal bleeding

- tenderness on PA ex.

- Passage of decidual cast

cast mirrors the shape of endometrial cavity

Outcomes in ectopic pregnancy

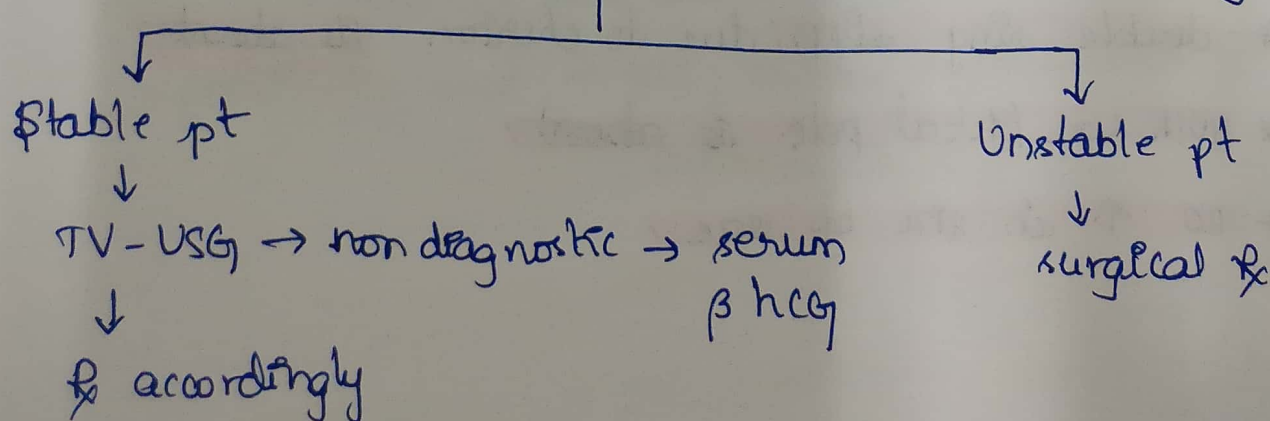
- Tubal rupture
- " abortion
- pregnancy failure & resolution

- Rupture in 1st few weeks → isthmus portion
- ampulla is more distensible
- rupture occurs later in interstitium
- pregnancy survives longer in ampulla
- Tubal abortion → fimbrial & ampullary pregnancies

Diagnosis

- β hCG < Qualitative (~~time~~ UPT)
- Quantitative
- Transvaginal USG (tv USG)
- Laparoscopy
- Laparotomy

+ UPT & abd. pain / vaginal bleeding



- In (N) pregnancies, doubling of βhCG occurs in 48 hours
- ~~discriminatory~~ discriminatory zone is the level of βhCG above $\leq$ failure to visualise an intrauterine pregnancy indicates that either pregnancy is not alive or is an ectopic pregnancy.
- usually around 1500 mIU/ml
- serial follow up & TVS of βhCG is necessary

Serum progesterone levels

- (N) pregnancy $\rightarrow \geq 25 \text{ ng/ml}$
- values $< 5 \text{ ng/ml} \rightarrow$ nonliving uterine / ectopic pregnancy

TVS

- Endometrial findings $\rightarrow$ pseudogestational sac
 - $\downarrow$
 - * anechoic fluid collection within endometrial cavity.
 - * centrally located within the uterus
 - * irregular in outline
 - * double ring sign due to chorion is absent
 - * yolk sac / fetal pole is absent
 - * no $\uparrow$ in size of sac

True gestation sac → • eccentric location in uterus

- round & regular outline
- double ring sign i.e. chorion +
- yolk sac & fetal pole +
- sac ↑ in size 1mm/day

• Adnexal findings:

- colour doppler tvs → ring of fire / interstitial ring
reflects placental blood flow around periphery of

Other modalities

• Laparoscopy

• D&C → chorionic villi float in (N) saline as lacy folds, decidua without villi structure is present

• Laparotomy

• culdacentesis → * used to identify hemoperitoneum

* 18G needle inserted into retroperitoneal

pouch → non clotting blood + in ectopic pregnancy

Management - Medical

• Methotrexate ± or without folic acid rescue

↳ only one used now

- previously used → KCl, PGF_{2α}, hyperosmolar glucose, actinomycin D

Case selection for medical m/m

- hemodynamically stable pt
- serum $\beta hCG < 3000$ IU/L (not absolute req) [upto 10,000 is OK]
- tubal diameter $< 4cm$ without fetal cardiac activity
- no acute intra abdominal bleeding
- level of βhCG is single best predictor of Rx success

c/I to m/x → breastfeeding,
immunodeficiency,
evidence of tubal rupture
intra uterine pregnancy
hepatic / renal / hematological dysfn.
PUD
active pulmonary d/o

• Failure rate is 5-10%

Surgical m/m

- * Done in
 - heterotopic pregnancy
 - unstable pts
- * c/I in pts who can be treated medically

① Conservative surgery

- linear salpingostomy (done now)
- salpingotomy (not done now)
- segmental resection ± or without anastomosis

② Radical surgery

- total salpingectomy

* linear salpingectomy can be done only in patients with unruptured tube. In case of ruptured tube, conservative surgery is resection & end to end anastomosis

* dose of anti D in ectopic pregnancy is 50 mcg (T1 abortion)
(gn for all Rh -ve women & ectopic pregnancy)

Dose of anti-D }
◦ T1 abortion / MTP / ectopic } → 50 mcg

- 2nd trimester onwards → 300 mcg
- within 72 hrs after delivery → 300 mcg

Rare types of ectopic pregnancy

- 1^o abdominal pregnancy → Studiford's criteria
- ovarian pregnancy → Spiegelberg's criteria
- Cervical pregnancy → Rubin's criteria

Ovarian pregnancy

Criteria for diagnosis

- tube on affected site must be intact
- gestational sac in ovary
- ovarian tissue found in gestational sac
- gestational sac is connected to uterus by ovarian ligament

surgery:

- unruptured ectopic → ovarian wedge resection or cystectomy
if diagnosed early
- systemic / local injection of Mtx /
in Mtx
- ruptured → salpingo-oophorectomy

Abdominal pregnancy

* Studdiford criteria: - for 1st abdominal pregnancy

- Both tubes & ovaries are (N)
- absence of uteroplacental fistula
- pregnancy exclusively related to peritoneal surface

* 2nd abdominal pregnancy

- more common
- 1st site being ovary, tube or uterus

M/m:

- surgery is mainstay
- removal of placenta is not done if life threatening maternal hemorrhage → leave placenta in situ → Mtx / embolization
- sometimes both fetus & placenta are left in situ to avoid surgery &
- long term complications are due to infection of placental tissue left in situ

- ~~one~~ multifetal pregnancy = one conceptus at ⑧ site & one at ectopic site
- 1 per 30,000 pregnancies (natural incidence)
- incidence ↑ to 1 in 1000 d/t ART
- ♂ → always surgery

- Spontaneous abortion / miscarriage $\rightarrow$ < 20 weeks or $\leq 500g$
- About 50-70% of spontaneous conceptions are lost
- Most clinical pregnancy losses occur before 8 week

Causes

- Numerical chr. ab[Ⓝ] $\rightarrow$ m/c ($> 50\%$)
- Infections
- DM, thyroid disorder
- Obesity, malnutrition
- Uterine anomalies etc.

Types of numerical chromosomal ab[Ⓝ]

① autosomal trisomy

- m/c - trisomy 16
- it is lethal
- all trisomies except 21, 18 & 13 are lethal
- d/t error in maternal meiosis I & are 9/w advanced maternal age

polyploidy

- tri / tetraploidy

sex chromosome polysomy

eg. 47 XXY, 47 XYY

Monosomy X - single m/c chromosomal ab[Ⓝ] causing spontaneous

① Spontaneous

- a) threatened
- b) inevitable
- c) incomplete
- d) complete
- e) missed

② Induced { medical surgical

Threatened abortion:

- Bleeding PV (mild)
- closed cervix on PV
- size of uterus corresponds to period of gestation
- small area of retroplacental hemorrhage on USG ±
- fetal cardiac activity +

Inevitable abortion:

- severe bleeding PV
- loss of liquor
- cervix open ± palpable conceptus
- size of uterus doesn't correspond / corresponds

Incomplete abortion:

- Bleeding PV & passage of POC
- size of uterus doesn't correspond

Missed abortion

- No Sx
- Baby died in utero
- Size of uterus doesn't correspond to POG

Complete abortion

- vaginal bleeding trace or absent
- os closed
- uterus smaller than POG
- uterine cavity empty

Septic abortion:

- vaginal discharge → purulent, foul smelling & features of sepsis
- size of uterus is variable → may be larger
- os open
- USG → POC retained, free fluid in peritoneal cavity

Recurrent abortion:

- Sequence of ≥ 3 consecutive spontaneous abortion before 20 weeks
- * 1^o → no successful pregnancy
- * 2^o → losses following a viable birth

◦ causes - (m/c is) chromosomal abn

- uterine factors
- cervical incompetence
- endocrine problems
- immunological problems
- inherited thrombophilia

◦ systemic disorders
like miliary TB

Parental chromosomal abnormality

- Karyotyping of parents done to diagnose balanced translocations leading to recurrent miscarriages
- m/m → pre implantation genetic testing

Immunological factors

- AlloImmune factors
- AutoImmune factors → APLA syndrome (APS)

APLA syndrome

- miscarriage after 10 weeks
- treatable
- Diagnostic criteria [Revised Sapporo criteria]
 - * clinical criteria
 - vascular thrombosis
 - pregnancy morbidity
 - * Lab criteria (must be +ve atleast on 2 occasions 12 weeks apart)
 - presence of lupus anticoagulant
 - IgG or IgM anticardiolipin Abs
 - anti $\beta 2$ glycoprotein -1 IgG or IgM Ab

- APs cause thrombosis & not hemorrhage

[DRVVT - dilute russell viper venom test]

- anti β_2 GP Abs may cause false +ve VDRL
- m/c seen in pts w recurrent abortions $\rightarrow$ anticardiolipin Ab
- thrombocytopenia $\pm$

OM/m $\approx$

- * women w no prior thrombosis
 - low dose aspirin (81 mg)
 - UFH / LMWH

when pregnancy is detected & continued till 6 weeks post partum

- * women w prior thrombosis $\rightarrow$ maintenance therapy w warfarin & when pregnancy is confirmed, switch over to aspirin

- Indications for cerclage
 - active preterm labour
 - evidence of chorioamnionitis
 - vaginal bleeding
 - PPRM
 - evidence of fetal compromise / death
- funneling is seen in cervical incompetence in USG
- diameter of internal os in cervical incompetence is $> 1\text{ cm}$
- (N) length of cervix $\rightarrow 4\text{ cm}$
in cervical incompetence $\rightarrow < 2.5\text{ cm}$
- cerclage done based on history of ≥ 2 previous abortions / USG / $\downarrow$ Cx length

Diagnosis in pregnancy

- history
- Shirodkar's digital palpation test
- cervical sonography $\rightarrow$ funneling of Cx & change in form of γ, V & U
- Cx length $< 2.5\text{ cm}$
- funneling of Int. os $> 1\text{ cm}$
- speculum ex
- cervical index -
$$\frac{\text{funnel length} + 1}{\text{endocervical length}}$$

Diagnosis in non pregnant state

- passage of a No. 8 Hegar's dilator without resistance & pain
- cervicogram - funnel shaped cx
- intra cervical balloon studies
- traction studies - 100gm or less to pull folley bulb out of os

Other causes

- uterine causes → diagnosed by TVS, 3D US, MRI, hysteroscopy
- Inherited thrombophilias → diagnosed by factor II leiden mutation, prothrombin gene mutation, protein C & S deficiency
 - * Rx by low dose aspirin & heparin in pregnancy
- Infections → screen for bacterial vaginosis in pregnancy, VDRL testing

Classification

① Hydatidiform moles (chorionic villi present)

- i) Benign → complete & partial mole
- ii) Malignant → invasive mole

② Nonmolar trophoblastic neoplasms (chorionic villi absent)

- i) choriocarcinoma
 - ii) placental site trophoblastic tumor
 - iii) epithelioid trophoblastic tumor
- } malignant

Hydatidiform mole :

- ab(N) placenta = partly degenerative & partly proliferative changes in young chorionic villi
- maternal lesions arise from fetal & not maternal tissue
- forms small cysts of varying sizes
- resembles hydatid cyst

Incidence :

- more in asia, hispanics, american indian
- highest in philippine (1 in 80 pregnancies)
- In India → 1 in 400 pregnancies
- low intake of vit A (carotene) also ↑ R/o complete mole

RF for complete moles

- previous GTD
- maternal age upper > lower extremities
- older paternal age
- combined OCPs
- prior to unsuccessful pregnancies

• No known RF available for partial mole

Pathogenesis of complete mole

- complete moles have a diploid Karyotype - $46XX/46XY$
- paternal chromosome duplication leads to $46XX/46XY$
(only paternal origin)
- Bipaternal origin is AR & very rare

Partial mole

- dispermy leads to triple Karyotype $69XXX/69XXY$
[i.e. 2 sperms fertilise a ovum]
- clonality is also a cause where 2 chromosome sets are donated by father
- scalloping of chorionic villi present

Complete mole - gross

- Grape like cluster
- No fetal tissue / embryo / amniotic sac
- areas of hemorrhage present

Partial mole - gross

- affection of chorionic villi is focal
- fetus or atleast amniotic sac present
- baby is growth retarded & multisystem abn

CF :

- Sx → ◦ vaginal bleeding : white currant (vesicles) in red currant juice
- feature of thyrotoxicosis (d/t β hCG)
- pain, vomiting, breathlessness
- signs → ◦ anemia, features of pre-eclampsia,
 - size of uterus more than POG
 - doughy feel of uterus
 - absent fetal parts / heart sounds

USG

- complete mole → snow storm appearance
- partial mole → fetus & multicystic placenta

Serum β hCG

- In complete mole $\rightarrow$ very much elevated
- false -ve UPT possible d/t oversaturation of test assay
- In partial mole $\rightarrow$ $\uparrow$ ed / $\uparrow$

	Complete mole	Partial mole
◦ Trophoblast hyperplasia	diffuse	focal
◦ Karyotype	46xx / 46xy - paternal origin	◦ 69xxx / 69xxx triploid
◦ hydropic degeneration of villi	diffuse	focal
Immunostaining of P57 ^{KIP2}	-ve	+ve
typical Δ	molar pregnancy	missed abortion

Theca lutein cysts

- complete mole 1/4 theca lutein cysts in 25-60% of women
- A/L
- d/t overstimulation of lutein elements by $\uparrow$ β hCG
- contained fluid is rich in chorionic gonadotropin, also contains estrogen & progesterone

so expectant m/m

- can be aspirated transabdominally under USG guidance
- Oophorectomy done if undergoes torsion & infarction persists after untwisting

RF for malignant change in GTD

- ~~pt~~ age
- chance is 25% in complete & 5% in partial mole
- age >40 | <20 yrs
- parity >3
- serum hCG > 1,00,000 mIU/ml
- uterine size > 20 weeks
- previous 40 molar pregnancy
- theca lutein cysts > 6cm size

m/m :: suction evacuation

◦ anti D in Rh -ve

◦ follow up

Suction evacuation ::

- preferred in women who wish to retain fertile
- under anaesthesia
- curette done after suction evacuation

- β -hCG every week till -ve



every month for 6 months

- usually becomes -ve by 4-8 wks
- pregnancy c/I till hCG levels become -ve
- contraceptives can be used. i.e. combined OCPs, medroxyprogesterone acetate, barrier methods (less effective)
- IUCDs are c/I in GTD

Placental site trophoblastic tumor (PSTT)

- arises from implantation site intermediate trophoblast
- syncytiotrophoblastic cells are absent
- ↓
- β hCG levels are not much elevated
- composed mainly by cytotrophoblastic cells
- resistant to chemotherapy
- $\therefore$ TOC $\rightarrow$ hysterectomy

- invasive mole
- choriocarcinoma
- PSTT
- epitheloid trophoblastic tumor

→ diagnosed by clinical / β HCG / pathological / USG

→ Δ ^{HC} features :

- continued vaginal bleeding
- hCG titres plateau / rise / remain detectable for ≥ 6 months
- histological features of choriocarcinoma

◦ it can occur after any form of pregnancy

→ molar / term or preterm / miscarriage / tubal pregnancy

◦ Cf → * Irregular vaginal bleeding

* continued amenorrhoea

* vaginal nodules

* cough - d/t lung mets → more common

* epigastric pain (liver mets)

* headache / confusion (cerebral mets)

M/m

◦ mets evaluation → CXR, CT brain, pelvic USG

FIGO anatomic staging for GTN

Stage I → confined to uterus

II → spreads outside uterus but confined to genital organs (e.g. vaginal nodules)

III → lung mets

IV → brain, liver or GIT mets

WHO prognostic scoring

• score < 6 → low risk

≥ 7 → high risk

• GTN following term pregnancy → 2 points

pretreatment hCG $> 1,00,000$ → 4 points

Brain mets → 4 points

Re

• low risk GTN (stage I / stage 2 & score < 6)

* single agent CT → Mtx + folic acid
or
actinomycin D

* If (R) → 2nd line drugs

• high risk GTN or stage 4 (stage 2 & 3 & score ≥ 7)

- age > 35 yrs
- initial hCG levels > 1,00,000
- hCG fails to become $\downarrow$ by 7-9 weeks time or there is re-elevation
- histologically Δ^d as invasive mole
- evidence of mets
- previous H_o molar pregnancy
- ~~who~~ women who is unreliable for follow up

* m/c GTD following H_o mole is choriadenoma destruens (invasive mole)

or choriocarcinoma

* GTD $\nsubseteq$ follows a full term pregnancy is always a choriocarcinoma

Invasive mole

- all invasive moles arise from partial or complete mole
- extensive tissue invasion by trophoblast & whole villi
- locally aggressive, but rarely metastasize

◦ Order of mets of choriocarcinoma is lung (80%) > vagina > pelvis > liver > brain

Multifetal gestations

Hellen's rule:

- Gives incidence of multifetal gestation in India

$$\rightarrow \text{Twins} = 1 : 80$$

$$\text{Triplets} = 1 : 80^2$$

Mono & dizygotic

Dizygotic $\rightarrow$ from fertilisation of 2 ova by 2 sperms

Monozygotic $\rightarrow$ from splitting of single fertilised ovum

Chorionicity:

- Type of placentation

- Dichorionicity / ~~di~~ dizygosity $\rightarrow$ each fetus has its own placenta

$\rightarrow$

- Monozygotic $\rightarrow$ ~~monozygotic~~

◦ splitting by < 3 days $\rightarrow$ dichorionic diamniotic

4-8 days $\rightarrow$ monochorionic diamniotic

> 8 days $\rightarrow$ monochorionic monoamniotic

> 13 days $\rightarrow$ conjoined twins

◦ monozygotic diamniotic is the m/c type of monozygotic twin

◦ Monozygotic ratio is always constant - 1 : 250

Disygotic ^{Indidence} variation is due to :

- ① ↑ FSH d/t → ↑ maternal age, ↑ maternal obesity & multiparity
- ② Family h/o twins in maternal family
- ③ Past h/o twins
- ④ pregnancy d/t ovulation induction by clomiphene citrate or gonadotrophine
- ⑤ IVF

	Monozygotic	Disygotic
◦ phenotype →	◦ Identical twins	◦ non identical
◦ genotype →	◦ identical	◦ non identical
◦ gender →	◦ concordant (same) sex	◦ discordant sex
◦ perinatal outcome →	◦ worst	◦ good
◦ ethnic variation →	◦ ④	◦ ⑤

	Monochorionic	Dichorionic
<u>I trimester</u>		
• no of sac	1	2
• dividing membrane thickness	$< 2 \text{ mm}$	$> 2 \text{ mm}$
<u>II trimester</u>		
• placenta	1	2
• sex	same	dyscordant
• dividing membr. layers	2 (1 - chorion & 1 - amnion)	4 (2 chorion & 2 amnion)

Twin peak (lambda) sign:

- triangular projection of placental tissue between dividing membranes.
- seen in dichorionic

T sign:

- diagnostic of monochorionic

Antepartum complications

T1 → hyperemesis, ↑ R/o miscarriage

T2 → HTN, GDM, anemia

T3 → preterm labour, polyhydramnios, APH (placenta previa or abruption)
pressure symptoms (RS, GIT, edema)

Intrapartum complications

1st stage →

- prolonged labour
- PROM

- malpresentation

- cord prolapse

- may req augmentation of labour

2nd stage →

- may req forceps / assisted breech delivery / internal podalic or external cephalic version /

LSCS

- abruption of placenta

3rd stage →

- atonic PPH

- retained placenta

Postpartum complications:

- 2^o PPH

- thromboembolism

- subinvolution

- lactational failure

Interventions $\leq$ are not effective in twins

- bed rest & hospitalisation (in absence of other complications)
- cervical encerclage
- progestins
- tocolytics (in absence of established preterm labour)

Interventions $\leq$ are useful :

- limited physical activity
- $\uparrow$ ANC
- TV & monitoring of Cx length

Vascular anastomosis

- ~~not~~ ^{seen} in monochorionic
- 2 types $\rightarrow$ superficial & deep
- Superficial anastomosis : (present on surface of chorion)
 - seen only in monochorionic
 - 75% is artery to artery (umbilical artery to umbilical artery)
 - 50% is artery to vein or vein to vein
 - always bidirectional (depending on pressure diff. b/w the twins)

- There will be no discordancy in growth or amniotic fluid or Hb differences

→ disturbance in bidirectional flow leads to discordancy in

growth - twin to twin transfusion syndrome (TTTS)
amniotic fluid - Twin oligo polyhydramnios sequence (TOPS)

Hb diff → Twin anemia polycythemia sequence (TAPS)

• Deep anastomosis :

- present in substance of placenta
- always artery to vein anastomosis
- always unidirectional flow
- extent throughout capillary bed of villous structure leading to 3rd circulation at a common villous compartment
- disturbance leads to discordancy in growth, AF & Hb diff like in superficial anastomosis

Twin to Twin Transfusion Syndrome (TTTS)

- Donor or pump twin & recipient twin
- Donor twin → oligemic, anemic
- Recipient twin → polycythemia

Donor twin	Recipient twin
• oliguria, anemia • oligohydramnios • ↓ Renal blood flow → activation of RAAS • non visible bladder • IUGR	• polyuria, polycythemia • polyhydramnios • activation of ANP • overdistended bladder • circulatory overload — prone for hyperviscosity syndrome, HTN, occlusive thrombosis, hyperbilirubinemia, CHF • hydrops possible

Quintero staging:

- applicable for monochorionic twins
- staging of TTTS
- prognostic staging in TTTS

Stage I → • AF discordancy
• urine visible in donor bladder

Stage II → stage I + urine not visible

⑩ Stage III → discordancy in Doppler flow
(umbilical artery or vein / ductus venosus)

Stage IV → frank ascites or hydrops in either of twins

Stage V → death of either twins

Management of TTTS:

- serial amnioreduction
- laser photocoagulation
- septostomy
- selective foetocide

Twin Reversal Arterial Perfusion sequence (TRAP)

• large superficial artery to artery anastomosis in early trimester followed by large vein to vein anastomosis is involved (4 vessels involved)

[donor artery to recipient artery & later ^{also} recipient vein to donor vein]

- deoxygenated blood from donor passes to through umbilical artery to recipient; recipient uses this minimal oxygen in its lower ~~body~~ part of body & further deoxygenated blood passes to upper part of body leading to poor development of upper body
 - ~~poor~~ ^{failure of} head growth (acardiac acephalus)
 - partially developed head & identifiable limbs (acardiac myelocephalus)
 - no identifiable structure (acardiac amorphus)
- } occurs only in TRAP & not TTTF
- ~~the~~ donor twin is more prone for CCF in TRAP

* TOPs & TAPs are subset of TTTs but TRAP is not

Delivery of twin:

- m/c → cephalocephalic (60%)
- cephalo breech (20%)
- breech cephalic (10%)
- breech-breech (6-10%)
- l/c → transverse-transverse (in conjoint)

- If 1st twin (t1) is noncephalic → always CS
- If t1 is cephalic → go for vaginal delivery
- After delivery of t1, see for lie of t2 by abdominal / vaginal examination & reconfirm w/ USG
- If t2 is in transverse lie, do ECV
 - If turns to cephalic → do NVD
 - If turns to breech → assisted breech
 - If ECV fails, shift pt to OT & do ~~ECV~~ & do PV
 - ↓
 - If membranes absent, do LSCS
 - If membranes present, do IPV & go for NVD or breech extraction
- assisted breech → spontaneous delivery till umbilicus & then intervention; no GA required
- Breech extraction → req GA or minimal tocolytic like mag sulph.

Indications for CS in twins

- 1st non cephalic
- monochorionic mono amniotic → always CS at 32-34w
→ absolute Ix
- TTTS
- conjoined twins at term
- severe IUGR w/ abn Doppler

Conjoint twins:

- Types → thoracophagus → m/c
ischiophagus
craniophagus
pyrophagus (back to back)

Management of anomalous ~~twins~~ fetus in twin pregnancy

◦ If dichorionic

→ inject intrathoracic KCl



anomalous fetus will die due to cardiac arrest

◦ If monochorionic

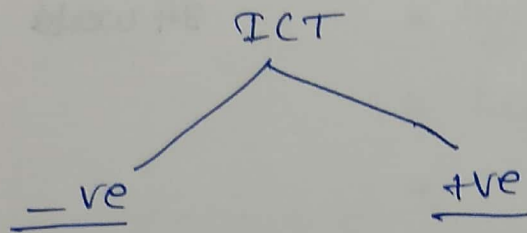
→ bipolar cord coagulation of anomalous twin

Parameters in discordance:

- weight $\geq 25\%$
- BPD $\geq 6\text{mm}$
- head circumference $\geq 5\%$
- abdominal " $\geq 20\text{mm}$
- systolic to diastolic (SD) ratio $\geq 15\%$

Protocols of Rh- isoenimmunisation:

- Spouse blood group $\rightarrow$ Rh -ve $\rightarrow$ no intervention needed
- If spouse group +ve $\rightarrow$ do indirect coombs test (ICT)



- no isoenimmunisation present

- prevention of isoenimmunisation by admin Anti D within 72 hrs of birth of baby.

or

- anti D at 28 wks of pregnancy

- assess titres

$< 1:16$



~~critical titre~~
~~value~~

- do 2 weekly ICT & deliver at 37 weeks

$> 1:16$



- critical titre

- monitor fetal anemia through middle cerebral artery peak systolic velocity (MCA-PSV) expressed in mom (multiple of median)

< 1.5 mom



deliver at 37 weeks

> 1.5 mom

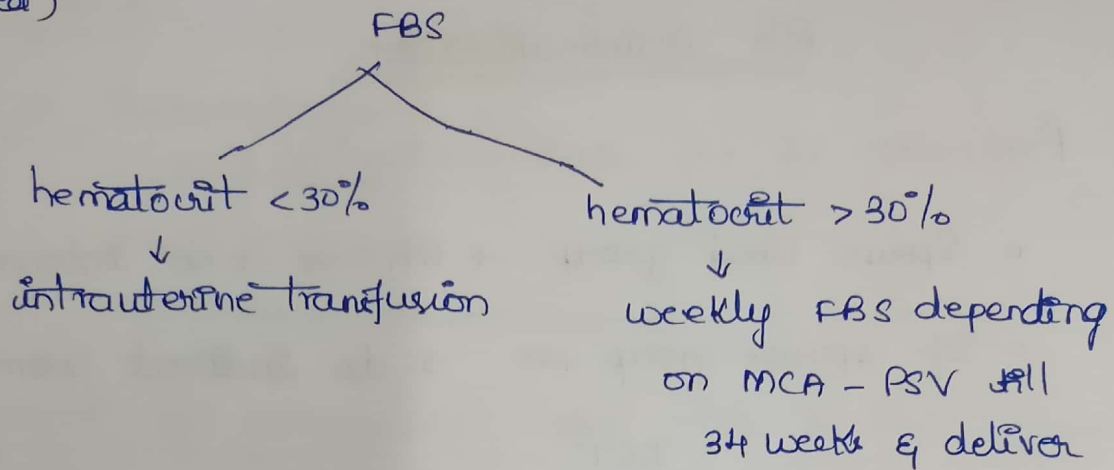
- if > 34 wks

- deliver immediately

- if < 34 weeks

- do fetal blood sampling (FBS)

(continued)



• •

Rh incompetence

- 1st pregnancy → sensitizing pregnancy
response by IgM
weak response
- subsequent pregnancy → sensitized pregnancy
amniotic response → strong response
= IgG
→ fetal anemia, hydrops, bilirubinemia

Anti D Ig = { monoclonal
polyclonal

① polyclonal → m/c used

- 300 mcg neutralise 15 ml of fetal RBCs
- 50 mcg " 2.5 ml " " "
- t_{1/2} ⇒ 16 to 24 days
- 0.9% i.m. in deltoid / anterolateral aspect of thigh

◦ Foetal anemia

- hydrops (big fetus)
- bilirubinemia

◦ Signs of fetal anemia → * polyhydramnios

* hyperplacentosis

* hepatosplenomegaly

* ↑ RA size

* fetal hydrops

◦ Signs of fetal hydrops

→ * severe anemia,

* CCF → ascites, PE, pericardial effusion

* HSM

* hyperplacentosis

* still birth

USG → subcutaneous/scalp edema in fetal hydrops
(Buddha's position) → Halo sign in USG

◦ D/t hyperplacentosis → preeclampsia in mother



edema in mother

∴ both mother & baby are big

→ maternal mirror syndrome / Ballantine syndrome

Intrauterine transfusion:

- when hematocrit $< 30\%$
- O+ve blood, cross matched w mother's blood & tightly packed is transfused to achieve hematocrit of 75%

Routes $\rightarrow$ • intra peritoneal $\rightarrow$ transfused blood absorbed thro diaphragmatic lymphatics
 • intra vascular
 • combined

- Intra vascular route $\rightarrow$ • preferred route
 • intrahepatic portion of umbilical vein
 & umbilical vein at cord insertion into placenta
 are used

Normal physiology

- ↑ in HR, plasma vol, CO, stroke vol, MAP
- ↓ in DBP, systemic vascular (R)

(N) CVS changes

- prominent S1
- S3
- systolic murmur upto grade 3
- (L) axis deviation in ECG
- up & out shift of apex
= seems like slight cardiomegaly in CXR

ab(N)

- diastolic murmur
- systolic murmur > grade 3
- arrhythmias
- marked cardiomegaly
- persistent split S2

Classification of heart disease based on etiology

◦ congenital

* non cyanotic → ^{mk} ASD, VSD, pulm stenosis, COA

* cyanotic → ^{mlc} TOF, Eisenmenger's

◦ RHD - MS (is m/c), MR, AS, AR

◦ cardiomyopathy

◦ IHD

◦ arrhythmias, syphilitic

* Regurgitant lesions are better tolerated than stenosis

NYHA functional classification

Gr I → no limitation of physical activity

II → mild " ; $S_x \leq$ (N) " "

III → marked " ; $S_x \leq$ less than (N) activity, but comfortable at rest

IV → severe limitation; S_x even at rest

CHART's classification of heart disease during pregnancy according to risk :

- Low risk (0-1%) → ASD, VSD, PDA, MS, NYHA-1 & 2, corrected TOF
- Medium risk (5-15%) → MS - NYHA 3, 4 & AF, AS, uncorrected TOF
- High risk (25-50%) → pulm HT, Eisenmenger's, COA & valvular involvement, Marfan & aortic involvement

WHO classification

Risk class I → no ↑ed R/o maternal mortality & no/mild ↑ in morbidity → admission at 36 wk

II → small ↑ risk of mortality or moderate ↑ R/o morbidity → adm at 28 wk

III → severely ↑ed risk → monitor throughout antenatal period

IV → extremely ↑ed risk & needs expert management

◦ pregnancy is C/I in class IV, if possible MTP done
& if can't be done managed like class III

◦ class IV includes → "pulmonary HT,

(pregnancy C/I)

• severe ventricular dysfn

(LVEF $< 30\%$, NYHA 3 & 4)

◦ previous peripartum cardiomyopathy
• residual impairment of LV fn

◦ severe MS, severe symptomatic AS

◦ Marfan syndr. • aorta dilated > 45 mm

◦ aortic dilatation > 50 mm in aortic dis a/w
bicuspid aortic valve

◦ severe COA

Predictors of cardiac events in pregnancy (NOPE)

◦ N - NYHA class > 2

◦ O - obstructive lesion of heart (MS, AS, peak LV outflow tract)

◦ P - prior cardiac event like arrhythmia, cardiac failure

◦ E - EF $< 40\%$

• % complication is 5, 30 & 75% when none, one or more
than one of them is present

M
m

- Admission → WHO class I at 36 wks, II at 28 weeks,
III & IV — preferable to stay in hospital throughout antenatal period
- class II pt not allowed for NVD
- 1st & 2nd stage of labour → bedrest, propped up position, restrict IV fluids
- 1E Px
- adequate analgesia
- cut short 2nd stage
- post partum — watch for signs of cardiac failure & pulm. edema; administer IV furosemide if needed
- oxytocin is uterotonic of choice

Role of anticoagulants

- Tx in mech. prosthetic valves, prosthetic devices, arrhythmias
- DOE → * LMWH for 1st 12 weeks
* warfarin in 13 - 36 weeks
* after 36 wks — LMWH
stop 24 hrs before delivery & start 12 hrs ~~later~~ after delivery
* warfarin resumed 3 days later, it is safe in lactation

◦ In pts on oral anticoagulants, LSCs is preferred to reduce R/o intracranial hemorrhage in fetus.

◦ adm FFP & vit K to newborn

* warfarin embryopathy includes chondrodysplasia punctata, microcephaly, nasal hypoplasia, optic atrophy

IE

◦ Ix for Px are prosthetic valve, unrepaired cyanotic congenital heart d/c, completely repaired CHD w/ prosthetic devices during 1st 6 months after surgery, cardiac transplant recipients

AB regimen → qn 6-8 hrs before & 6 hrs after delivery

◦ ampic 2g or xone 1g iv + genta 80mg iv

◦ if penicillin allergic, cephalosporins or iv clinda 600mg

◦ if enterococcal inf. is suspected, adm vancomycin

Peripartum cardiomyopathy

◦ A form of dilated CMP w/ LV systolic dysfunction that results in s/s of heart failure

Criteria → development in last month of pregnancy or first 5 months after delivery

◦ absence of heart disease prior to last month of pregnancy

- absence of identifiable cause of heart failure
- LV systolic dysfunction
- Unknown etiology
- Theories : genetic predisposition, autoimmunity, viral infection
- associated RF → age > 35, twin pregnancy, PIH, multiparity, use of tocolytic therapy, african-american race
- mortality rate : 25-50%

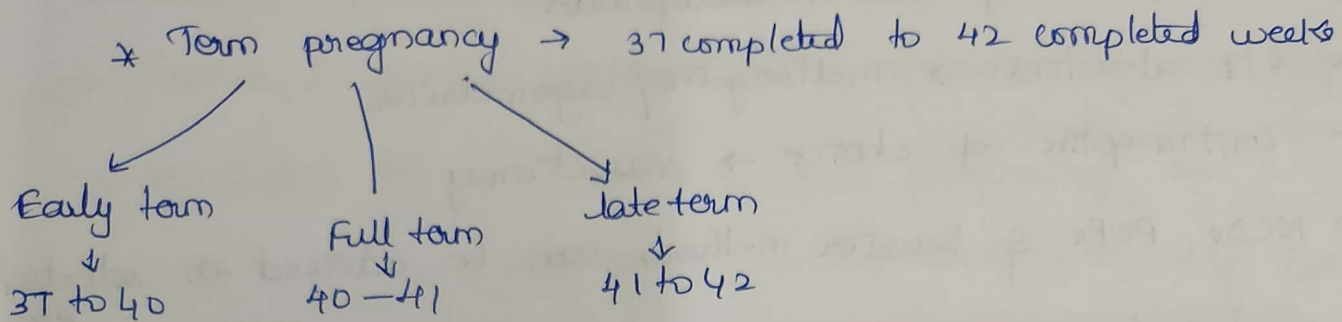
⊗ pointer

- R/o cardiac failure → immediate > labour > 28-32 wks postpartum
- Main R/o maternal mortality → Eisenmenger's syndrome
- m/c of maternal mortality → MS
- mitral balloon valvotomy done best in T2 → 14-18 wks
- Induction of labour → mech methods like Foley's balloon are preferred
 - FGE 2 gel can be used
- RF for cardiac failure → anaemia, infections, preeclampsia, hyperthyroidism

- Anaesthesia of choice in cardiac C/I → general
- Tocolytic of choice → atosiban (oxytocin receptor ~~agonist~~ ^{antagonist})
- **DOE for arrhythmias → metoprolol**
- C/I uterotonic → methergine / ergometrine
- Contraceptive of choice → vasectomy
- IUCDs, POPS & barrier methods can be advised in selected _{pts}
- **combined OCPs are C/I**

Preterm Labour

- EDD is calculated using Naegele's rule



- * Preterm → ~~before~~ 24-37 weeks [India → 24 to 26]
WHO → 22

- Late preterm → 34 to 37 weeks
- early/moderate preterm → 32 to 34 weeks
- very early preterm → 28 to 32 weeks
- extreme " → < 28 weeks

Preterm labour

- cervical dilatation 1-3 cm → early preterm labour
- cervical " > 3 cm → advanced preterm labour
- c_x length < 2.5 cm in USG & no contractions
- cervical incompetence
- few contractions & no cervical dilatation → threatened preterm

- D - distended uterus (multiple pregnancy, polyhydramnios)
- I - infections (bacterial vaginosis, GBS, PROM, UTI)
- M - maternal (preeclampsia, anaemia, heart disease, APH, low BMI, stress, depression)
- I - iatrogenic (wrong dates, surgeries in uterus or CX)
- N - nicotine (smoking)
- I - interval b/w pregnancies
- S - short CX (< 20.5 cm, funneling of os)
- H - H/O preterm labour.

Predictors (FLUSH)

- F - fetal fibronectin ($N < 50$ ng/ml)
- L - length of CX ($N > 20.5$ cm)
- U - urine culture & high vaginal swab
- S - salivary estriol (more than 1.8 - 2 cm suggests PTL)
- H - HUAM (Home uterine activity monitoring)

Shapes of CX in USG

① shape - T

ab ① $\rightarrow$ $\gamma, \vee, \cup$ $\bar{c}$ bulging membranes

Immunological factors

IL-1 β , TNF α , IL-6, IL-8, MCP-1, PG ϵ , MMB, etc

Management (SMART)

- S - Steroids - betamethasone is preferred
 - c/t in chorioamnionitis
- M - magnesium sulphate - reduces contractility & neuroprotective if GA < 32 weeks
- A - antibiotics - GBS, asymptomatic bacteriuria or associated PROM; preferred - penicillin/clindamycin
- R - refer to a center w high level intensive neonatal care unit
- T - tocolytics

Classification of tocolytics

- β_2 adrenergic agonists - terbutaline, ritodrine, isoxsuprine
- MgSO₄
- ~~CCB~~ CCBs - nifedipine, nicardipine
- oxytocin receptor antagonist - atosiban (preferred in heart & complicating pregnancy)
- PG synthetase or - indomethacin, aspirin, ibuprofen, sulindac
- NO donors - nitroglycerine
- Halothane

* Nifedipine is the most preferred tocolytic as it has the least adverse effects

- may be given in a pt $\geq$ previous $\geq$ preterm labour, not useful in active preterm labour

Role of cerclage

- previous $\geq$ preterm births / 2nd trimester loss
- USG $\rightarrow$ CX < 2.5 cm
- Done in ~~1st~~ < 28 weeks of pregnancy
- Not done in PPRM, chorioamnionitis, twins, APT
- preferred is McDonald method
- others $\rightarrow$ transabdominal cerclage, Shirodkar method

PROM

- mostly dit infections
- sterile speculum examination to demonstrate leak in the 1st step
- other tests $\rightarrow$
 - nitrazine test (yellow strip turns blue)
 - fern test
 - fetal fibronectin levels
- deliver if > 34 weeks

M/M < 34 wks

- a 48 hr course of i.v ampicillin & erythromycin foll by 5 days of amox & erythro for expectant m/m
- single course of antenatal corticosteroids gn to women & PROM at 24 to 31 weeks

Post dated pregnancy

- pregnancy that extends to 42 weeks of gestation or beyond
- 'post maturity' is used to describe features of a post term neonate. Described by Clifford & consists of 3 stages

I - wrinkled, peeling skin, & thin body

II - stage I + fetal distress, meconium present

III - stage I & 2 & meconium stained skin or nails, loss of subcutaneous fat

Post term

Placental maturation



macrosomia



hypoglycemia, hypothermia, shoulder dystocia

Placental insufficiency



oligohydramnios, fetal distress, meconium aspiration syndrome, cord compression

Causes:

- wrong dates
- previous too prolonged pregnancy
- irregular ovulation
- ↓ fetal estrogen (CRH production → d/t placental sulfatase deficiency, anencephaly, fetal adrenal hypoplasia)

Physiological changes in post-term gestation

- placental changes - senescence (ageing, infarcts, calcifications)
- amniotic fluid changes - oligohydramnios, cloudy (flakes of vernix)
 - L/S ratio = $> 4:1$
 - presence of meconium

Complications

- Fetal →
 - Fetal distress
 - MAS
 - fetal trauma (d/t macrosomia) like brachial plexus injuries, clavicle fx
 - ↑ perinatal mortality
 - dysmaturity syndrome

Management

- confirmation of gestational age
- 1st trimester USG (CRL $\pm$ 7d)
- 2nd trimester BPD ($\pm$ 14d)
- quickening 16-18 wks

Termination of pregnancy

40 weeks



Do stripping, perform non stress test
& AFI

1

If AFI adequate,
NST reactive



Repeat mod. BPP
twice weekly

1
induce @ 42 weeks

If oligo or non reactive
NST - induce labour

Induction of labour

Modified Bishop scoring system

1	0	1	2	3
---	---	---	---	---

Methods of induction

- Mechanical dilators →
 - Foley's balloon,
 - laminaria tent
 - osmotic dilators
- stripping of membranes
- amniotomy
- Pharmacological —
 - PGE_2 gel
 - oxytocin infusion
 - misoprostol (PGE_1 analogue)

Medical disorders of pregnancy

Hepatic disorders

Classification

- ① specific to pregnancy, resolves spontaneously or following delivery
 - hepatic dysfunction from hyperemesis
 - intrahepatic cholestasis
 - acute fatty liver
 - hepatocellular damage $\rightarrow$ preeclampsia $\rightarrow$ HELLP syndrome.
- ② acute liver disorder coincidental to pregnancy
- ③ chronic " " predating pregnancy

④ Liver disease findings in pregnancy

- elevated serum ALP
- palmar erythema
- spider angioma

Intrahepatic cholestasis of pregnancy

- aka recurrent pruritus of pregnancy. cholestatic hepatosis

o CF:

- pruritis: late in pregnancy, no constitutional pr, predilection for soles & palms, skin changes d/t excoriations from scratching, pruritis precedes lab findings by several weeks (mean of 3)
- chronic hepatitis C is a/w significantly ↑ed ALP cholestasis
- m/c in multifetal pregnancy
- (N) liver enzymes at presentation
- slight jaundice ($< 5 \text{ mg/dl}$)
- No BP change / proteinuria
- recurs in subsequent pregnancies & with estrogen containing contraceptives
- ∴ OCPs are c/I in such pts
- Rise in serum bile acids is the earliest & most consistent change / best marker; there is 10-100 fold ↑ in serum cholic acid foll by serum chenodeoxycholic acid
- always only direct bilirubin ↑ed
- serum ALP ↑ed
- ↑ed chance of PPH & CS
- good prognosis

Pathogenesis

- ? gene defect
- ↓ clearance of bile acids
- ALT / AST elevated (never > 250)
- hyperbilirubinemia (never > 4-5)

Liver Bx

- mild cholestasis & bile plugs in hepatocytes & ~~canal~~ canaliculi of centrilobular regions but without inflammation or necrosis
- disappear after delivery, but often recur in subsequent pregnancies or with estrogen containing contraceptives

dx:

- pruritus → antihistamines, topical emollients
- cholestyramine may ↓ absorption of fat soluble vitamins → leads to vit K deficiency & fetal coagulopathy
- UDCA → DOC, relieves pruritus, lowers bile acid & serum enzyme concentrations & may ↓ certain neonatal complications

- m/c of acute liver failure during pregnancy
- 1 in 10,000
- doesn't recur in subsequent pregnancies
- characterised by accumulation of microvesicular fat that "crowds out" (N) hepatocytic function
- Grossly the liver is small, soft, yellow & greasy
- a/w defect in fatty acid oxidation, mainly LCHAD
- manifest late in pregnancy
- a/w male fetus, multiple fetal gestation
- CF - persistent nausea, vomiting, epigastric pain, progressive jaundice (bilirubin 4-10 mg/dl), HTN, proteinuria, edema
- Mod to severe cases manifest by hypofibrinogenemia, hypoalbuminemia, hypocholesterolemia, prolonged clotting time
- serum bilirubin levels usually $< 10 \text{ mg/dl}$ &
- serum aminotransferases are usually $< 1000 \text{ U/L}$
- hypoglycemia, hepatic encephalopathy, severe coagulopathy (d/t hypofibrinogenemia) & some degree of renal failure

• M/m

- Induction of labour
- Blood & blood products (PPH & coagulopathy)
- hepatic dysfunction resolves within a week post partum

* m/a of pyelonephritis in pregnancy → E. coli

* ABX are safe in pregnancy

- cephalosporins
- penicillin
- erythromycin
- metronidazole
- nitrofurantoin

Infectious diseases in pregnancy

HSV

- HSV-1 → non genital & HSV-2 → genital
- sexually transmitted
- 1° inf → papules in genital area → painful vesicles
→ multiple vulval & perineal ulcers
- inguinal adenopathy may be present
- reactivations are less severe

- Perinatal transmission is m/c
- Neonatal disease →
 - localized skin, mouth, or eye disease
 - CNS d/c = encephalitis
 - disseminated d/c

◦ fb:

- maternal acyclovir
- CS if there are active lesions during labor
- invasive monitoring should be avoided in labour

Toxoplasmosis

- Toxoplasma gondii
- Perinatal problems
 - 1° infection causes fetal abn d/c transplacental transmission
 - max severity of infection is in 1st trimester
 - max incidence of transmission is in later pregnancy (but ↓ abn teratogenicity)
- more babies are asymptomatic at birth
- severe disease → triad of hydrocephalus or
intracranial calcifications, chorioretinitis & cerebral

- $\Delta \rightarrow$ acute infection : IgG & IgM Abs or 4 fold $\uparrow$ in paired specimens 4 weeks apart
- M/m $\rightarrow$ - spiramycin - if 1st infection is confirmed
 - pyrimethamine - sulfonamide to be alternated
 - spiramycin if fetal infection is confirmed

Varicella zoster

- HZV doesn't cause congenital malformations
- perinatal effects :
 - fetal varicella syndr - highest risk b/w 13 - 20 wks. occurs in 2% of fetus infected at this time
 - Neonatal varicella - maternal infection at term if the infant is born within 7 days of onset of rash

Fetal varicella syndrome

- skin scarring in geographical distribution
- hypoplastic limbs
- eye defects
- neurological abn
- m/m $\rightarrow$ oral acyclovir 800mg 5 times daily for 7 days - help to ameliorate severity of disease in mother

◦ prevention - VZIG within 72hrs of exposure

Causative

Teratogenic sequelae

- 1) Toxoplasma - Tetrad of hydrocephalus or microcephaly, convulsions, chorioretinitis, cerebral calcification
- 2) Rubella - Tetrad of Gregg's PDA, cataract, sensorineural hearing loss
- 3) Parvovirus - nonimmune hydrops
- 4) Syphilis - Bone defects, hydrops

Syphilis

◦ Transplacental transmission (m/c route)

- occurs only after 20 weeks

◦ through contact & lesions during delivery

Effects of syphilis on pregnancy & labour

- preterm labour, fetal death, fetal growth restriction, neonatal infection
- fetal hepatic abscess → anaemia & thrombocytopenia
→ ascites & hydrops
- neonatal faunus & petechiae or purpuric skin lesions

- Placental effects →
 - large & pale placenta
 - Blood vessels ↓ & dis appear d/t endarteritis & stromal cell proliferation

• tx:

- penicillin (preferred)
- erythro & azithro → curative to mother but not fetus
- early syphilis → benzathine penicillin single dose
- >1yr duration → " " ~~3 doses~~ weekly → 3 doses
- Neurosyphilis? aqueous crystalline penicillin iv
- X ray shows → moth eaten appearance of syphilitic femur

Epilepsy

- seizure frequency is unchanged in 50%, ↑ed in 30% & ↓ed in 20%.
- women who were seizure free for 9 months atleast before conception will likely remain so during pregnancy
- Folate 0.4mg/day is to be given pre-conceptionally & ↑ed to 4mg/day after conception
- seizure frequency is ↑ed in pregnancy d/t episodes of subtherapeutic levels of anticonvulsants

- subtherapeutic levels and d/t nausea, vomiting, and GI motility, antacid use, pregnancy hypervolemia, placental enzymes & induction of hepatic enzymes & red GFR

Pregnancy complications

- red Rlo CS & seizure disorders in baby
- red Rlo pre eclampsia & PPH
- untreated epilepsy is not also ↑ fetal malformation rate
- monotherapy is preferred & lowest possible dose
- valproate, phenytoin & phenobarbitone → also ↑ Rlo GPT defects
- newer anticonvulsants are better
- M/M → targeted USG for women on anticonvulsant drs
- Contraception & breastfeeding
 - Breastfeeding is not contraindicated
 - red failure rates & combined OCPs & POPs & enzyme inducing anticonvulsant agents

Abnormal fetal presentation

- incidence of malpresentation at term $\rightarrow$ 3 to 5%

Breech presentation:

- podalic pole presents at pelvic brim
- m/c malpresentation
- m/c is prematurity

Complete breech: ◦ more common in multigravida

- pv $\rightarrow$ feel buttock & 2 feet of baby
- fully flexed posture
- legs flexed at both hips & knees

Incomplete breech:

- various degrees of deflexion present
- foot is presenting part (footling)

Frank breech:

- hips flexed & knees extended

- Presenting part is a ...

Breech presentation

Causes of breech presentation

Factors

- ↓ed vertical polarity of uterus
- ↑ed / ↓ed fetal mobility
- obstructed pelvic inlet
- fetal malformations
- prematurity

causes

- multiparity
- placentation high in fundus or low in pelvis
- uterine myomata (fibroids)
- intrauterine synechiae
- ~~an~~ müllerian duct fusion abn
- prematurity & polyhydramnios
- ↓ed fetal muscle tone / activity
- CPD
- hydrocephalus
- oligohydramnios

Δ:

- clinical
- USG (gold std)
- position - sacrum is denominator of breech.

* 1st position → ① sacro anterior (LSA) - m/c

Mechanism of labour in breech

- Principal movement occurs at - buttocks, shoulders & head
- Buttocks - diameter of engagement of buttock is one of the oblique diameters of inlet. The engaging diameter is ischiochantic - sacrum toward isopubic eminence
- when the diameter passes thro' pelvic brim, the breech is engaged
- shoulders - engaging diameter is biacromial diameter
- head - engaging diameter is suboccipitofrontal diameter

Pinard maneuver / front breech decomposition:

- used to deliver the legs in front breech

Lowet maneuver:

- used to deliver the shoulder / arm in breech

Burne Marshall maneuver:

- delivers head in breech
- baby taken in an arc & put on mother's abdomen

Mauriceau maneuver:

- delivers head in breech
- operator places his hand on baby's face & assistant gives

Delivery of breech with forceps:

◦ to deliver head

* The aftercoming head of breech, chin to pubis is delivered by

- manual rotation & extraction by Pipers forceps
- modified prague maneuver

* cause of death in breech delivery is

- intracranial hemorrhage
- atlantoaxial dislocation
- asphyxia

◦ Breech presentation & hydrocephalus is managed by

- CS
- transabdominal decompression
- PV ~~can~~ decompression
- craniotomy of aftercoming head (not done down)

Occipitoposterior position & deep transverse arrest :

◦ 90% corrects itself

◦ cause ⇒ ◦ android or anthropoid pelvis (wide occiput in

- anterior attachment of placenta (wider post. segment)
- high pelvic inclination
- abn(Ⓢ) uterine contraction

- long rotation ($3/8^\circ$)
- short anterior rotation ($1/8^\circ$)
 - nonrotation
 - short posterior rotation ($1/8^\circ$)
- } unfavourable
↓
face to pubis

Deep transverse arrest :

- sagittal suture is placed in transverse ischiospinous diameter & there is no progress in descent of head even after $1/2 - 1$ hour following full dilatation of Cx

Management of occipitoposterior position

- forceps delivery

M/m of deep transverse arrest

- CS
- vacuum extraction
- Kielland forceps (not $\text{\textcircled{A}}$ forceps)
- Manual rotation & forceps delivery

• baby at 90° to long axis of uterus $\rightarrow$ transverse lie
[45° to long axis of uterus is oblique lie]

- Presentation in transverse lie is shoulder presentation
- If labour progresses $\rightarrow$ hand prolapse, cord prolapse

Face presentation:

- head is hyperextended
- presenting part is chin / mentum (denominator)
- it may be mentoposterior / mentoanterior
 - $\downarrow$
 - no mechanism of labour
 - only LSCS possible

causes

- • preterm

• enlargement of neck

• coil of cord around neck

• hydramnios, contracted pelvis

• fetal malformations, anencephaly

• high parity - pendulous abdomen permits back

to sag anteriorly in same direction of occiput

• diameter of engagement is submentobregmatic

Engagement diameters

- Brow presentation - mentovertical (14 cm)
- (N) flexed vertex presentation - submento bregmatic (9.5 cm)
suboccipito bregmatic (9.5 cm)
- Breech (partially deflexed head) - suboccipito frontal (10 cm)
- Fully deflexed head - occipito frontal (11.5 cm)
- Face presentation - submento bregmatic (9.5 cm)

Brow presentation

- On pv examination, anterior fontanelle & supraorbital ridge is felt
- In (N) cephalic presentation, only posterior fontanelle is felt

- begins as soon as placenta is expelled & lasts for 6 weeks
- Immediate → within 24 hours
- early → upto 7 days
- remote → upto 6 weeks

Lochia

- vaginal discharge for first 15 days during puerperium
- offensive fishy smell
- Colour
 - lochia rubra (red) - 1 to 4 days
 - " serosa (yellowish/pink/pale brown) - 5 to 9 days
 - " ~~alb~~ alba (pale white) - 10 to 15 days
- amount : for first 5-6 days - 250 ml
- (N) duration - 3 weeks
- Immediately after delivery, wt of uterus - 1000 gm
after 6 hrs - 60 gm
- Carunculae myrtiformes → nodular tags of hymen after childbirth

- Hormones involved in lactation are
 - oxytocin, prolactin, human placental lactogen (HPL), thyroid hormone, progesterone
- The m/c urinary problem during post partum period is stress incontinence

Contraceptive effect of breast feeding:

- for women who are doing EBF $\rightarrow$ 98% contraceptive effect upto 6 months
- Non lactating mother contraception $\rightarrow$ 3rd week postpartum
- Lactating mother $\rightarrow$ 3rd month postpartum

Postpartum mastitis:

- Re: • analgesic, ABs, gentle hand expression, hot & cold compress
- do not stop BF
 - m/cc & staph aureus especially MRSA
 - source of infection $\rightarrow$ infant's nose & throat
 - m/m $\rightarrow$ milk from affected breast for C&S
 - dicloxacillin / another penicillin

- Breast abscess:
 - suspected when a mass & fluctuation is palpable
 - no defervescence within 48-72hrs of Rx
 - USG finding
 - Rx: ~~1st~~ I&D
- Decreased lactation is seen in maternal anxiety, cracked nipple, breast abscess, bromocriptine therapy
- C/I for BF
 - sputum +ve TB
 - breast cancer

Colostrum

- 1st 2 to 4 days
- 50-100ml/day
- more protein, secretory IgA, more lactose, less fat
- * Contraception is preferred in lactating mother is POP.

Thromboembolism in pregnancy:

Virchow triad → stasis, local trauma, hypercoagulability

RF

- previous Hb thromboses
- twins
- anemia
- APH / PPH
- CS foll by prolonged immobility
- hyperemesis
- obesity
- multiparity
- HTN
- stillbirth
- peripartum hysterectomy

DVT

- iliofemoral > iliac > calf veins
- (L) side > (R) side d/t compression of (L) iliac vein by (R) iliac & ovarian artery

S/S :

- abrupt pain & edema of leg & thigh
- 'white limb' - pale, cool extremity & diminished pulsations d/t arterial spasm
- Homan's sign

- $\Delta \rightarrow$ proximal compression USG (initial diagnostic test of choice)
 - $\rightarrow$ doppler imaging of iliac vein / CT or MR venography
 - $\rightarrow$ D-dimer testing
- M/m : - LMWH (preferred) / UFH
 - postpartum : warfarin

Pulmonary TE :

- 1 in 1000 pregnancies
- CF : dyspnoea, pleuritic chest pain, cough
- Δ :
 - leg Sx $\rightarrow$ compression USG
 - CXR
 - CT pulm. angiography
 - V/Q scintigraphy
- M/m :
 - anticoagulants
 - vena caval filters in case CS needed
 - thrombolysis
 - embolectomy

Obstetric neuropathies :

- m/c $\textcircled{N}$ injured during NVD $\rightarrow$ lateral femoral cutaneous $\textcircled{N}$ of thigh
- RF $\rightarrow$ nulliparity, prolonged 2nd stage, prolonged pushing in semi-Fowler position

- Common (N) injured in LS → ilioinguinal & iliofemoral (N)

(especially in herniated disc)

- Common (N) injured in NVD → common peroneal nerve (CL + foot drop)

Anemia in pregnancy

Physiological anemia of pregnancy:

- plasma vol ↑ by 40-50%.
- RBC mass ↑ by 30%.
- ∴ Hb conc. ↓ by 2g/dl d/t hemodilution

Iron requirement in pregnancy

- It is 2x higher
- ∴ 1000mg / pregnancy
 - 500mg for erythropoiesis
 - 300mg for fetus & placenta
 - 200mg shed thru skin, urine & stool

WHO grading

mild 10-11 g/dl

moderate 7-10 "

severe <7 "

ICMR grading

- mild → 10-10.9
- mod → 7-9.9
- severe → <7
- very severe → <4

CDC

- Hct 33% & Hb 11g/dl - 1st & 2nd trimester
- Hct 32% & Hb 10.5 g/dl - T2

Causes:

- Deficiency ^(mle) → iron, folate, vit B12
- hemoglobinopathies
- aplastic anemia
- AIHA
- leukemia

Development of IDA: - 3 stages

① depletion of iron stores → ↓ ferritin

② Iron deficient erythropoiesis

→ ↓ ferritin, ↓ sr. Fe, ↓ PSAT, ↓ Hct

→ no clinical Sx / morphology change

→ (N) Hb

③ Frank IDA

→ change in RBC morphology

→ clinical Sx

→ ↓ Hb

Indicators :

- earliest indicator → serum ferritin
- most sensitive indicator → MCHC (usually unchanged in 1st pregnancy)
- most accurate method to estimate Hb → cyanmethemoglobin
- all parameters ↓ but TIBC & transferrin increases
- Hb should be estimated at 1st AN visit, 30th - 32nd week & 36 weeks

Complications

In mother

During pregnancy

- pre eclampsia
- infection
- heart failure
- preterm labour

Labor

- uterine inertia
- PPH
- cardiac failure
- shock

Puerperium

- puerperal sepsis
- subinvolution
- lactation failure
- venous thrombosis
- pulm. thromboembolism (PTE)

In fetus

- prematurity
- LBW
- IUD
- fetal anemia
- developmental delay

Management:

1) Oral Iron / parenteral iron

2) Blood transfusion

Choice of method

Depends on - severity of anemia, GI, additional RF

Px:

• National Nutritional Anemia Control Programme of India

- 100 mg Iron + 500 mcg folic acid x 100 days

• 12 by 12 initiative

- aim at 12 g/dl Hb by 12 yr of age

WHO Px

• 60mg of elemental Iron & 250 mcg folic acid for

6 months & continue 3 months postpartum

Rx:

• Iron should be given 2 hrs before or 4 hrs after ingestion of antacids

• Iron is absorbed as ferrous salt in acidic medium

- Rise in Hb after oral iron is seen after 3 weeks
- Can be taken $\pm$ food if adverse effects are present
- Parenteral therapy is indicated in dialysis pt,
& moderate to severe anemia in 2nd trimester

Preparations

- Iron dextran 50-100mg i.m / i.v - 1g after test dose as anaphylaxis is possible
- Ferric gluconate } iv only
- Iron sucrose }
↳ safer

- Dose calculation

Iron needed = Hb deficit $\times$ wt in Kg $\times$ 2.21 + 1000 mg
 (to replenish the stores)

- Role of Ferric carboxymaltose (FCM) → used in postpartum anaemia

Blood transfusion

- marked / severe anemia in T3
- APH
- Packed cells in @ anemia
- exchange transfusion in cur

M/m during labour

1st stage

- propped up position
- analgesia
- D₂
- reserve blood
- avoid fluid overload

2nd stage

- cut short
- avoid ergometrine
- Px ABx
- iron & folate - 3 months postpartum
- appropriate contraceptive advice

• Pt who is severely anaemic develops Sx by end of T2

• ↑ R/o CCF

Other types of anaemias

SCA:

- m/c hemoglobinopathy seen in pregnancy
- AR inheritance
- In pregnancy maintain target Hct above 25%
- ↑ R/o thrombosis → m/m by thrombolytic Px, analgesics
- NVD is preferred
- POP & IUDs are safe contraceptives

Thalassemia

- α → deletion of 1 or 4 genes on chr 16
- β → deletion / point mutation of 1 or 2 genes on chr 11
- ↓ MCH, MCV & MCHC
- serum iron elevated
- NESTROF Test - screening test
- Hb electrophoresis - definitive test
- Blood transfusion is the m/m
- iron is C/I (parenteral iron)
- chelation $\pm$ desferrioxamine after TI is advisable
- concurrent folic acid supplementation

PPH

• Blood loss > 500 ml in NVD & > 1000 ml in LSCS

• massive PPH > 2000 ml

• a drop in Hct > 10%

• obstetric shock index - HR / systolic BP

* ② value is 0.5 - 0.7

* values > 1 → massive hemorrhage

Types

• 1° → within 24 hrs, m/c → atony

• 2° → 24 hrs to 12 weeks, m/c → retained placenta

Causes: 4 T's of PPH

• Tone 70% (over ~~distension~~ ^{dilatation} of uterus)

• Trauma - 20% (hematoma, inversion, rupture, laceration)

• Tissue (retained placenta → 10%
Invasive ")

• Thrombin (coagulopathies) - 1%

Atonic PPH

o m/c

o causes → o over distended uterus

o maternal anaemia, malnutrition, multigravida

o labour complications → prolonged 2nd stage, precipitate labour, APH

Degree of shock

	Degree 1/ compensated	Degree 2/ mild	3 moderate	4 severe
o Blood loss	15% (500ml)	15-30% (1500)	30-40% (2L)	>40% (>2L)
o HR	mild tachy	>100	>120	>140, weak pulse
o RR	Ⓝ	↑	>30	>40
o BP	Ⓝ	DBPT SBP Ⓝ	SBP < 100 mmHg	SBP < 70 mmHg
o capillary refill	Ⓝ	delayed	delayed	absent
o mental status	Ⓝ	mild anxious	altered	altered conscious, coma
o urine output	Ⓝ	20-30ml/hr	<20 ml/hr	nil

Px :

◦ WHO recommendations for AMTSL, 2012 :

① use of uterotonic to prevent PPH during 3rd stage of labour for all birth — oxytocin
10 IU, iv/im

② sustained uterine massage is not recommended

③ controlled cord traction (cct) → recommended in
NVD

only if skilled attendants available

④ postpartum abdominal uterine tone assessment for early id of uterine atony is recommended for all women

Medical m/m :

◦ DOc → oxytocin → 10 IU im / iv

→ 3-5 min for onset of action &
DOA is 2-3 hrs by im

→ ↑ contraction by ↑ing Ca^{2+}

- methergene → 0.2 mg ^{im} / PV ~~mg~~ every 2-4 hours - 5 doses
→ c/I in HTN, scleroderma & Raynaud's
- **Carbo prest** (PG F_{2α}) → 0.25 mg p.m.
→ c/I in **asthma**
→ can be repeated upto 8 doses
- **Misoprostol** (PG E₁) → 800-1000 mcg
→ oral / SL / PR / vaginal ^{preferred}
→ A/E → fever, vomiting

Blood product utilisation

- Whole blood → 500 ml → ↑ Hct 3%
- PRBCs → 300 ml → ↑ Hct 3% less fever
- Platelets → 50 ml → ↑ PLT 10,000
- FFP → fibrinogen, AT III, clotting → 250 ml → ↑ fibrinogen factors, plasma 5-10 mg/dl
- cryo → fibrinogen, factor 8, 13, → 40 ml → ¹¹
VWF

Other methods

- Bimanual compression
- NASG - anti shock garments

Surgical m/m :1) Condom tamponade

Balloons used $\rightarrow$ obstetrical silicone balloons (Bakri balloon - 500 ml)

$\rightarrow$ Foley's catheter balloons

$\rightarrow$ sangster - Blackmore tube

$\rightarrow$ Ruch catheter

$\rightarrow$ condom "

2) Compression sutures

commonly used $\rightarrow$ ~~also~~ B Lynch sutures (m/c)

o hayman "

o cho "

3) stepwise devascularisation

o uterine A ligation

o int. iliac A "

o arterial embolisation

if all fails $\rightarrow$ hysterectomy - subtotal

APH

- Bleeding PV after viability of baby - 22 weeks (WHO)
24 to 26 (India)

Causes:

- placenta previa (30-35%)
- abruptio placenta (35-40%)
- Others - placenta accreta, vasa previa, etc.

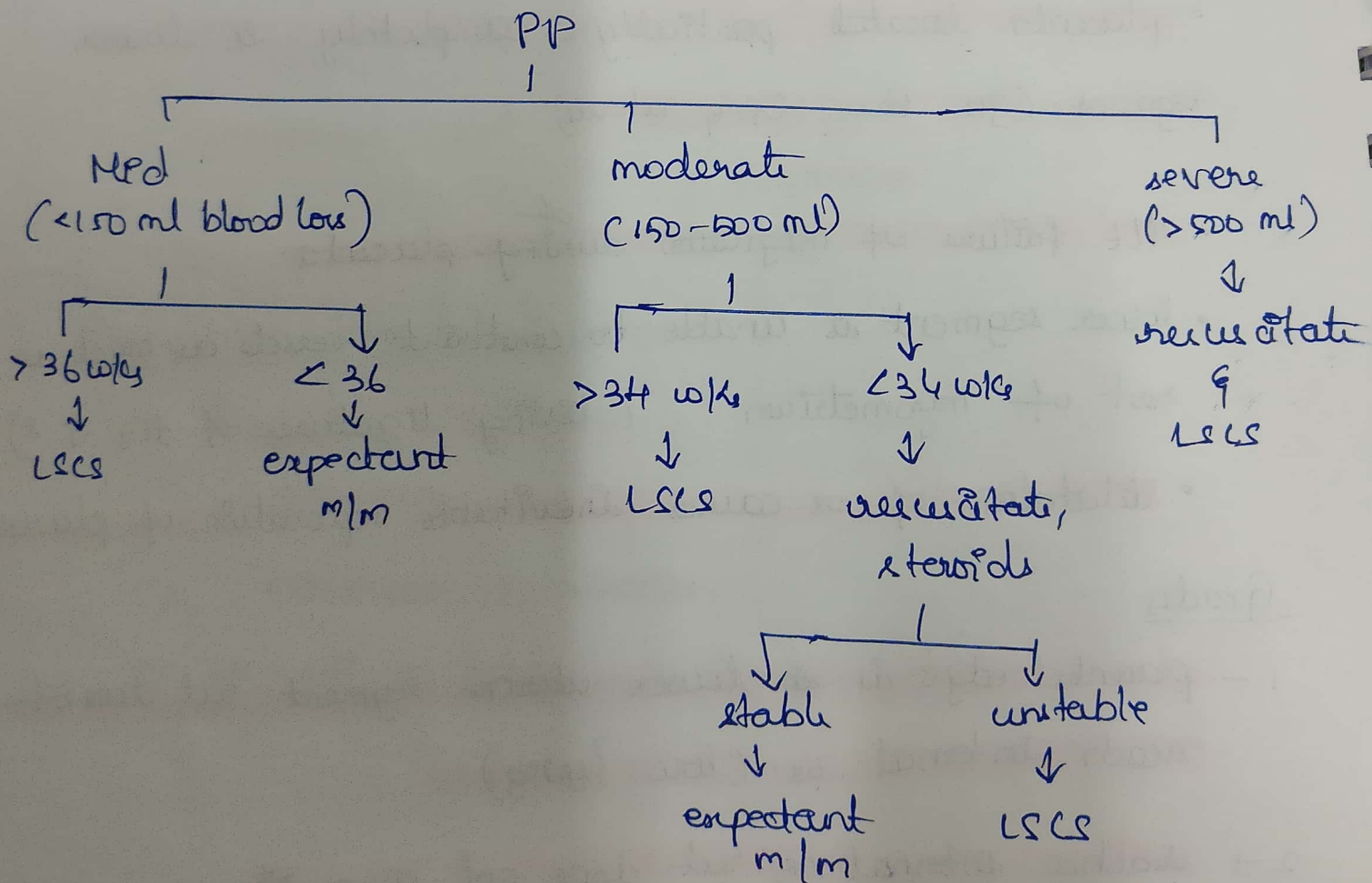
Placenta previa

- placenta located partially or completely in lower segment (in btw Cx & uterus)
- d/t failure of migration ^{of} during placenta
- lower segment is unable to constrict vessels as much as rest of myometrium (living ligatures of fig of 8)
- dilatation of os causes inevitable separation of placenta

Causes:

- 3 previous histories → 1) placenta previa
ii) uterine surgeries
iii) smoking
- 3 Thing factors → ↑ maternal age
↑ parity
↑ no. of fetus (multiple pregnancies)

Management



McAfee & Johnson Regimen

- complete bed rest, tocolysis & observation
- steroids
- criteria for this regimen
 - mother hemodynamically stable
 - no fetal distress
 - pregnancy < 36 weeks
- there is 25% ↑ R/o hysterectomy in women undergoing repeat CS for previa compared to 6% in 1st CS for previa
- Transabdominal USG → simplest, safest
- PV is C/I in previa
- painless bleeding diff previa from abortion
- Type 2b → dangerous → posterior placenta previa
 - ↓
 - might not be detected in routine USG
- Stallworthy's sign → slowing of fetal HR on pressing the head down into pelvis is seen in posterior placenta previa
- CS is indicated in all previa

- severe & settles by itself

Abruptio placenta

- hemorrhage d/t accidental separation of a normally situated placenta

classifications

① Shen's grading

Gr I (retrospective) → not recognized before delivery,
retroplacental hematoma discovered
on maternal surface of placenta,
no APH

II → mild bleeding PV, uterine tenderness & tetany,
no fetal distress / maternal shock

It → severe bleeding, tenderness, tetany, fetal distress & death, maternal shock

III $a \rightarrow$ without DIC

Ex b $\rightarrow$ with DIC

classification

ages

agnosie purely on pathology, no sx

uicent form & a live baby, mild bleeding

ld form & onset of clotting problems & fetal

distress

severe form & coagulation defects & fetal death

in utero

- same as previa +

- previous abruption, smoking, cocaine abuse

- 3Ps → polyhydramnios, PROM, preeclampsia

- 2Ts → trauma, thrombophilias

- Ted AFP in T2

of delivery

Abruption

- **also uterine apoplexy**
- **rare complication in severe abruption**
- **distal vascular damage within placenta = causes hemorrhage that progresses to & infiltrates the wall of uterus. can be diagnosed only by direct visualization or biopsy (seepage of blood into myometrium)**
- **self limiting.**

DIC

- **most dreaded complication of abruption**
- **Over hypofibrinogenemia ($< 150 \text{ mg/dl}$ of plasma)**
- **$\tau \uparrow$ FDP, D dimer & variable $\downarrow$ in other coagulation factors $\rightarrow$ seen in 30% τ abruption placenta severe enough to kill fetus**
- **Release of thromboplastin in abruption $\uparrow$ DIC**
- **M/M $\rightarrow$ FFP, cryoprecipitate**
- **PLT transfusion if plt $< 50,000$**

Placenta accreta

- morbidly adherent placenta
- d/t absence of decidua basalis & Nitabuch's layer
- $\left\{ \begin{array}{l} \text{inacreta} \rightarrow \text{invade upto serosa, but doesn't breach it} \\ \text{periacreta} \rightarrow \text{breach the serosa \& may involve bloodles etc.} \end{array} \right.$
- placental localisation using MRI & plan for hysterectomy & LSCS

Vasa previa

- d/t velamentous insertion of cord (ie cord inserted into fetal membrane)
- Blood loss is of fetal origin $\rightarrow$ $\uparrow$ ed fetal mortality, - maternal mortality is not $\uparrow$ ed
- can be diagnosed antenatally by doppler usg
- when bleeding occurs $\rightarrow$ sinusoidal fetal HR pattern seen
- Δ at time of bleeding by - Singers alkali denaturation test / Apt test

OH/m - emergency CS

- on staining blood $\pm$ Wright stain, if RBC's appear nucleated the blood is of fetal origin

IUD

- WHO → death of fetus that has reached **wt of $\geq 500g$**
or GA 22 weeks

India → 28 wks

- Antepartum death → delivery of a macerated fetus
- Intrapartum → " " " " fresh stillbirth

Causes

- Maternal causes → HTN, DM, infections - malaria, hepatitis, influenza, toxoplasma, syphilis
 - hyperpyrexia
 - APLA
 - Thrombophilia - factor V Leiden, protein C & S def.
 - SLE
 - post term
 - intrapartum - ab @ labor, rupture uterus
- Fetal causes - (m/c)
 - chr. ab @, structural anomalies, chorioamnionitis, IUGR, Rh-incompatibility, non immune hydrops fetalis, Intrapartum asphyxia

placenta →

1) ~~an~~ malposition →

www.FirstRanker.com

2) cord prolapse, true knot, cord around neck

3) TTTs

4) placental insufficiency

• Iatrogenic → ECV, drugs

• Idiopathic

Pathology :

• dead fetus undergoes aseptic degenerative process termed maceration

• epidermis is 1st structure to undergo maceration that appears 12-24 hrs after death

• Blistering & peeling of skin occurs. Fetus becomes swollen & looks dusky red

• gradually aseptic autolysis of ligamentous structures & liquefaction of brain matter & other viscera occur, producing characteristic radiological signs

Δ :

• S₀ - loss of fetal movements

• Signs → regression of breast changes of pregnancy

• PA → ~~red~~ fundal height smaller than POG

• FHS absent



• Egg shell crackling feel of fetal head - late feature

- USG - earlier diagnosis
- evidence of IUD → lack of fetal movement & cardiac activity for 10 mins
- Robert's sign → appearance of gas shadow in chambers of heart & great vessels
 - earliest sign
 - seen 12 hrs after IUD
 - seen in X ray
- Spalding sign → irregular overlapping of cranial bones & hyperextension liquefaction of brain matter
 - appear after 7 days after IUD
- Other X ray features → hyperflexion of spine, hyperextension of neck, crowding of ribs & loss of $\textcircled{N}$ parallelism

Complications

- Infections → m/c - *C. Welchii*
- DIC if fetus retained for > 4 weeks & thromboplastin released from dead tissue
- ↑ chance of uterine inertia, retained placenta, PPH during labour.

- ABO & Rh
- Blood sugar
- Urine ex
- VDRL
- RFT, TFT
- TORCH screening
- lupus anticoagulant, anticardiolipin Abs

M/m

◦ Expectant → in 80% spontaneous expulsion occurs in 2 weeks of death

→ sr. fibrinogen estimation - done weekly

current → terminate pregnancy once IUD is ^{ed} Δ

Mode of delivery → always by medical induction

◦ oxytocin infusion - if Cx favourable

◦ PGs - vaginal (PGE₂) gel, if Cx unfavourable

◦ Misoprostol (PGE₁) 25-50 μg vaginal or oral every 4 hr

④ ARM in C/I d/t R/o infections

◦ exclude 2° abdominal pregnancy if repeated attempts at induction fails to start labour

Examination of fetus for

- malformations
- umbilical cord entanglement
- no. of vessels in cord
- placenta for meconium staining

Ix for autopsy & chromosomal studies

- H/o recurrent IUDs
- fetus has anomalies or dysmorphic features
- either parent is a carrier for balanced translocation

Ix for CS in IUD ?

- major degree placenta previa
- previous ≥ 2 LSCs
- transverse lie

- Contracted pelvis is one in which one or more diameters in one or more planes are shorter than N
- m/c type is small gynecoid pelvis foll by small platypelloid pelvis

Rachitic pelvis

- vit D deficiency
- aka star-shaped pelvis
- kidney / reniform shaped pelvic inlet

Naegle's pelvis:

arrested development of one ala of sacrum

Robert's pelvis:

arrested development of both ala of sacrum

CPD:

- There is disproportion in size of pelvis & the fetal head can be minor / major
- Minor $\rightarrow$ trial labour
- major $\rightarrow$ elective LSCs
- minor + obstetric complications $\rightarrow$ elective LSCs

Trial of labour

- in minor CPD
- during labour there occurs an adjustment b/w fetal head & maternal pelvis d/t
 - moulding of fetal head
 - giving way of pelvis
 - good uterine contractions

Criteria to allow trial of labour

- vertex presentation
- must not be postmature
- ideally spontaneous onset
- monitoring = partogram
- FHS monitored & continuous CTG
- Platypelloid pelvis → is favourable as pelvis is contracted at one diameter & only one level (cavity/brim/outlet)

Obstructed labour:

in spite of good uterine contractions, progressive descent of fetal head is arrested d/t mech obstruction

- causes →
 - i) fault in passage - CPD, contracted pelvis, cervical fibroid, impacted ovarian tumour
 - ii) fault in passenger → malpresentation, hydrocephalus, macrosomia, fetal ascle

Features :

- maternal exhaustion
- Cx fully dilated
- membranes are absent

- upper uterine segment retracts & lower segment dilates & thins out → Bandl's ring (pathological retraction ring)
- Bandl's ring can be palpated in b/w upper & lower uterine segment
- Bandl's ring moves closer to & closer to umbilicus

[Gold std for diagnosis for CPD → CT pelvimetry
◦ Ht < 140 cm is RF for NVD]

Bandl's ring - features

- patient in agony
- upper segment is hard & tender > lower segment is distended & tender

fb: ◦ rupture of uterus to be excluded
◦ CS is done in majority of cases

Maternal pelvis & fetal skull

Maternal pelvis:

- 4 bones → sacrum, coccyx, two innominate bones
↓
ilium, ischium & pubis
- pelvic jt →
 - symphysis pubis - fibrocartilaginous
 - sacrospinous jt - synovial
 - sacrococcygeal jt - synovial hinge joint

True pelvis

- Below brim of pelvis
- divided into
 - inlet
 - cavity
 - outlet

False pelvis

- above brim of pelvis

Pelvic inlet

- almost round (in gynecoid) = AP diameter being shortest
- in erect posture, tilted forward, making an angle of inclination of about 55°
- ↑ in angle of inclination
 - d/t sacralization of L5 vertebra
 - delay in engagement because uterine axis fails to coincide with inlet
 - favour occipito posterior
 - difficulty in descent of head & long birth canal & flat sacrum interfering with internal rotation

AP diameters of pelvic inlet

- True/anatomical conjugate → from sacral promontory to upper border of pubic symphysis → 11 cm
- Obstetric conjugate → from midpoint of sacral promontory to prominent point of posterior pubic symphysis → 10 to 10.5 cm
- Diagonal conjugate → from tip of sacral promontory to lower border of pubic symphysis → 12.5 cm
(only measurable conjugate)
↳ measured by doing PV

Obstetric conjugate

◦ smallest AP diameter

- pelvic inlet is considered to be contracted, if it is < 10 cm
- obstetric conjugate = diagonal conjugate - 1.5 cm
- 1.5 cm for inclination, height & thickness of pubic symphysis

Pelvic cavity

◦ Round in shape

◦ Boundaries

above - pelvic brim

below - plane of least pelvic dimensions

anterior - symphysis pubis

posterior - sacrum

Mid pelvis

- At the level of ischial spine
- Bipinnous diameter $\rightarrow$ 10cm
- aka plane of least pelvic dimensions
- (F) in labour
 - internal rotation occurs at this level
 - beginning of forward curve of pelvic axis
 - deep transverse arrest occurs here
 - station of head
 - external os lies at this level
 - origin of levator ani muscles
 - landmark for pudendal block

Pelvic outlet

- isolated outlet contraction is very rare
- transverse diameter - 11cm
- AP " $\rightarrow$ 13cm
- subpubic angle $\rightarrow$ 85 degrees
- $\downarrow$
- if < 75 degrees $\rightarrow$ lead to dystocia

Caldwell Moley classification of pelvis

Gynaecoid pelvis

- m/c female pelvis
- transverse diameter of inlet is slightly more than AP
- posterior segment is round, wide & roomy
- ant & post. segment are equally spacious
- is well curved

- ischial spines are not prominent
- sacrospinous notch is wide
- subpubic angle is 90 degrees
- in small gynecoid pelvis, all diameters are proportionately reduced, but shape is (N)

Anthropoid / ape like pelvis :

- 2nd m/c
- shape is long anteroposteriorly oval
- only pelvis τ AP diameter $>$ transverse
- side walls convergent, spines are likely prominent
- sacrum has 6 segments usually

Android pelvis

- male pelvis
- inlet heart shaped
- cavity funnel " "
- anterior segment is narrow, sharply angulated & post " " shallow
- causes deep transverse arrest
- sacral promontory indents the inlet & reduces its capacity
- subpubic angle is acute ($< 90^\circ$)

Platypelloid pelvis ◦ least common type

- flat female pelvis
- inlet is transversely oval
- subpubic angle is wide

Out comes

- Gynaecoid → ◦ m/c & ② type
 - diameter of engagement : transverse / oval
 - no difficulty in engagement
 - ② occipitoanterior (LOA) / ① occipito transverse (LOT) are m/c positions
- Anthropoid → ◦ direct OP m/c in this type
 - face to pubis delivery is m/c in this type
- Android → ◦ OP is more common (occipito posterior)
 - persistent OP is "
 - Deep transverse arrest (DTA) is m/c
- Platypelloid → ◦ head engages in transverse diameter & marked asynclitism
 - engaging diameter in asynclitism is super ~~and~~ parieto - subparietal diameter (8.5 cm)

Dystocia dystrophica syndrome

- oft is stockily built & bull neck, broad shoulders & short thighs
- male pattern hair distribution
 - android pelvis
 - subfertile
 - ↑ incidence of preeclampsia, postmaturity, uterine inertia
 - failure of lactation

• asynclitism → sagittal suture deflected from transverse diameter of pelvis

• anterior asynclitism → Nagele's obliquity
posterior " → Letzmann "

Fetal skull

• Sinciput → btw glabella & bregma
↓ ↓
(root of nose) (ant. fontanelle)

• BPD → most ~~(X)~~ for engagement → 9.5 cm

• Bimastoid diameter → 7.5 cm
↓
cannot be reduced

• Bitemporal diameter → 8.5 cm

• Engagement → BPD of fetal head is through pelvic inlet

Maternal serum AFP (MSAFP)

- AFP is synthesized by fetal yolk sac & later by fetal GIT & liver
- analogous to albumin
- Defects in integument such as neural tube & ventral wall defects permit AFP to leak into amniotic fluid, resulting in ↑ed MSAFP
- production peaks around 13 weeks in fetal plasma (mg/ml) amniotic fluid (μg/ml)
- seen in maternal serum in ng/ml
- Ratio of fetal plasma : maternal serum AFP is 50,000 : 1
- MSAFP screening done b/w 15 - 20 weeks
- Measured in ng/ml & ~~expressed~~ reported in MoM (multiples of median)
- upper limit of (N) → 2 to 2.5 MoM
- Factors affecting AFP:
 - maternal wt → (falsely ↓ed in obese women)
 - gestational age
 - race / ethnicity
 - diabetes → false low
 - multifetal gestation

◦ Elevated levels

- multifetal gestation
- fetal death
- neural tube defect
- gastroschisis/omphalocele
- cystic hygroma
- maternal hepatoma/teratoma
- underestimated GA
- gastroschisis > omphalocele/spina bifida occulta

Low levels

- Obesity
- DM
- Trisomies 21 or 18
- GTD
- fetal death
- overestimated gestational age

Screening tests

- T1 → CVS
- T2 → amniocentesis
- ~~T3~~ → cordocentesis → 18 wks
- In Down's syndr → ↓ AFP & ↑ hCG
- β-hCG is ↑ in Rh incompatibility
- Acetyl cholinesterase in amniotic fluid is present in open neural tube defect

Procedures for prenatal genetic diagnosis:

- amniocentesis or CVS to obtain fetal cells or placental tissue
- Cordocentesis/PUBS (percutaneous umbilical blood sampling)
- is performed to obtain fetal blood

Amniocentesis

- It is transabdominal withdrawal of amniotic fluid
- performed b/w 15 to 20 wks, but may be performed later as well
- If pt is RhD-negative, anti D Ig given after procedure
- C/c → midtrimester loss (1 in 500)
 - amniotic fluid leakage
 - chorioamnionitis
- amniocentesis
 - Early ~~amniocentesis~~ - b/w 11 to 14 weeks
 - also more fluid leakage, fetal loss & talipes equinovarus

CVS :

- Bx of chorionic villi (placenta) performed b/w 10-13 wks gestation - trans cervical or transabdominal
- Results available earlier in pregnancy allowing safe abortion
- If done < 7 wks → limb reduction defects & oromandibular limb hypogenesis
- C/c → chromosomal mosaicism → do amniocentesis vaginal spotting
- anti D Ig gn in Rh -ve pts

PUBS / cordocentesis / funipuncture

- performed after 18 wks
- m/c Ix → fetal malformation detected during T2 & T3
→ Rh sensitization to detect fetal Hct.
- Differentiate b/w fetal blood & maternal blood:
 - fetal blood cells (140+L) are larger than maternal cells (80+L)
 - The MCV of a sample of fetal blood should be higher than 100
 - Apt test, or Hb alkaline denaturation test, especially before 28 wks of gestation

MCA

- CVS doesn't detect neural tube defects
- (R) pregnancy can be earliest detected by TVS at a β hCG level of around 1000 IU/ml

Best marker of GA

- in T1 → CRL
- in T2 → BPD
- T3 → femur length
- Doppler USG detect IUGR
- X ray pelvimetry is indicated in outlet obstruction, breech presentation in vaginal delivery, osteomalacia

Neural tube defects:

- Incomplete closure of neural tube by embryonic age of 26-28 days
- 2nd m/c malformations after cardiac anomalies
- spina bifida & anencephaly make up 95% of NTDs

Anencephaly:

- Absent cranium & telencephalic structures
- skull base & orbits are covered only by angiomatous stroma
- pituitary is hypoplastic or absent
- ↓ in size of adrenal gland
- structures not affected → cerebellum, base of skull, facial bones
- Risk of recurrence:
 - 1 affected child → 5%
 - 2 " " → 10 to 13%
- 70% of anencephalic fetuses are female
- Δ:
 - T2 → ↑ AFP in amniotic fluid
 - often hydramnios develops
 - USG at 10 wks → absence of cranial vault
 - angiomatous brain tissue

- acrania → shower cap appearance in USG
- 1st fetal anomaly detected in utero by USG
- Δ made as early as 8-10 wks
- USG → Mickey mouse shaped head
- Best time for Δ → early T2 (12-14 wks)
- C → - hydramnios
 - malpresentation → face or breech
 - preterm labour → d/t hydramnios
 - post maturity
 - shoulder dystocia
- M/M → if Δ^{ed} before 20 wks, pregnancy terminated

Spina bifida

- defect in vertebrae in dorsal arch = exposure of meninges & spinal cord
- open spina bifida → defect includes skin & soft tissues

Types

occulta → skin/soft tissue not involved, small defect covered by tuft of hair (lipoma)

open → ~~occulta~~ ~~open~~

aperta → covered by thin membr, giving appearance of cystic tumor

- affects lumbosacral region in 63%.

◦ ventral defects of spine are extremely rare

◦ Barana sign → compressed cerebellum

◦ lemon sign → indentation of frontal bone

Anterior abdominal wall defects

① Omphalocele :

◦ Sac covering the content is very thin consists of 3 layers → outer amniotic membr, middle Wharton's jelly & inner peritoneal layer. Sac may get ruptured during birth

◦ often a/w cardiac & genitourinary anomalies
-70%

② Gastroschisis :

◦ small defect in anto abd-wall just lateral to umbilicus, common in premature babies

◦ defect in involution of 2nd umbilical vein

◦ common in mothers < 20yrs who take aspirin, ibuprofen, pseudoephedrine during TI & who regularly smoke & take alcohol

◦ not a/w other anomalies

Etiology

- 95% → trisomy 21 / non disjunction during meiosis
- 3-4% → robertsonian translocation
- 1-2% → isochromosome or mosaicism
- Risk ↑ w ↑ maternal age, > 35 yrs
- But 70% of Down syndr are seen in women < 35 yrs
- m/c nonlethal trisomy
- seen in 1 in 500 recognised pregnancies, & 1 in 740 live births
- adult women w Down syndr are fertile & a third of their offspring have Down syndr.

Risk factors

- Age : ^{or} advanced maternal age
- prior pregnancy w autosomal trisomy (triploidy)
- woman or partner w balanced chromosomal translocation

T1 screening

- b/w 11-14 wks
- components →
 - fetal nuchal translucency (NT scan)
 - PAPP-A
 - Free β hCG

NT scan

- as an isolated marker, NT detects 65-70% of fetus = Down

NT + PAPP-A + hCG

- detection rate 80 to 90%
- NT $\rightarrow$ $\uparrow$ ed
- PAPP-A $\rightarrow$ $\downarrow$ ed
- hCG $\rightarrow$ $\uparrow$ ed

T2 screening

- lower maternal serum AFP, $\uparrow$ hCG, $\downarrow$ unconjugated estradiol levels (uE3)
- dimeric inhibin $\alpha \rightarrow$ $\uparrow$ in Down

Triple test $\rightarrow$ MSAFP + β hCG + uE3

Quad test $\rightarrow$ MSAFP + β hCG + uE3 + dimeric inhibin α

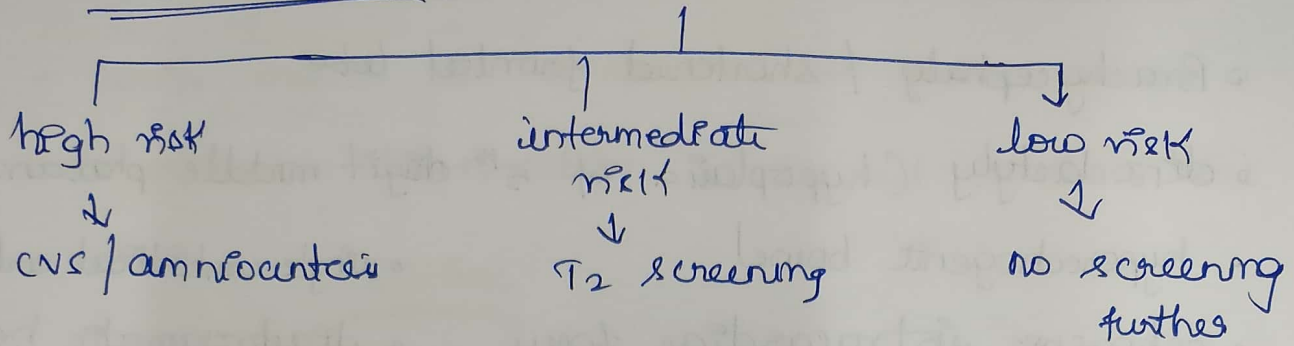
Abn low uE3 levels

- seen in Smith Lemli Opitz syndrome & X linked ichthyosis

Combined T1 & T2 screening

- Integrated screening $\rightarrow$ all pts undergo T1 & T2 screening
- Sequential screening
 - $\rightarrow$ stepwise sequential screening
 - $\rightarrow$ contingent " "

contingent screening → T1 screening



• This method of screening is ideal & cost effective

cell free fetal DNA testing:

- fetal DNA is released into maternal blood by apoptotic trophoblast, the DNA is used for screening
- 98% detection rates for trisomies 21, 18 & 13
- false +ve rates of $\leq 0.5\%$
- uses the tech of massively parallel sequencing or chromosome selective sequencing
- Recommendations:
 - women > 35 yrs
 - USG finding of 1 R/o fetal anomaly
 - risk factor for Down

Sonographic markers (soft markers) for Down synd in T2

- Brachycephaly / shortened frontal lobe
- clinodactyly (hypoplasia of 5th digit middle phalanx)
- hyperechogenic bowel
- echogenic intracardiac focus
- nasal bone absence / hypoplasia
- nuchal fold thickening
- short femur / humerus
- single umbilical artery
- diaphragmatic hernia
- duodenal atresia

→ presence of ≥ 2 of above suggest down synd.

Confirmatory test:

- only 100% confirmatory test in karyotyping

Anomalies also high risk of aneuploidy

- cystic hygroma (50% - 70%)
- holoprosencephaly (30-40%)
- Dandy walker (40%)
- duodenal atresia (30%)
- omphalocele (30-50%)
- gastroschisis → no risk

- 100% chance of recurrence in ~~both~~ parents & Robertsonian translocation (balanced translocation)

IUGR

- Birth wt below 10th percentile of average for GA

Symmetrical

- uniformly small
- ponderal index (Birth wt / crown heel length) — $\textcircled{R}$
- HG : AC & FL : AC ratios $\textcircled{R}$
- etiology $\rightarrow$ genetic disease or infection (intrinsic to fetus)
- Total cell no. — less
cell size — $\textcircled{R}$
- Neonatal course $\rightarrow$ complicated
 $\hat{=}$ poor prognosis
- less common

Asymmetrical

- head larger than abdomen
- low
- elevated
- chronic placental insufficiency (extrinsic to fetus)
- $\textcircled{R}$
smaller
- uncomplicated $\hat{=}$ good prognosis
- more common

* amniotic fluid volume is low in IUGR

* smoking, alcohol, cocaine cause IUGR

* IUGR babies are prone for hypoglycemia

MTP

mothers in
↑ sick
anomalous baby
↑
MTP act 1971 → MTP is in base of therapeutic, eugenic, humanitarian, social (feature of contraception in married)

• who can terminate

* one who has assisted in atleast 25 MTP in authorized centre & having a certificate

* one who has 6 months house surgeon training in OBG

* one ~~has~~ with diploma or degree in OBG

• can be done only in government established or approved hospitals

• consent → consent of woman is sufficient

written consent of guardian in minor or mentally ill

• pregnancy < 12 wks → opinion of single doctor

12-20 wks → 2 doctors

• ~~tertiary~~ MTP 41 in > 20 wks

• T1 abortion

Medical

• mifepristone

• mifepristone & misoprostol (Pg E1)

• M+x & misoprostol

• Tamoxifen & u

Surgical

• menstrual regulation

• vacuum aspiration

• suction evacuation &/or curettage

• Dilatation & evacuation

- rapid method

- slow method

- PGs → PGE₁ (misoprostol), 15 methyl PGF_{2α} (carboprost), PGE₂ (dinoprostone) & their analogs
- Dilatation & evacuation (13-14 wks)
- Intra uterine instillation of hyperosmotic solution
 - * extra amniotic → ethacridine lactate, PGs (PGE₂, PGF_{2α})
 - * extra amniotic saline infusion (isotonic) + a transcervical catheter balloon
 - * intra amniotic hypertonic urea, saline (not used now)
- oxytocin infusion — high dose + either of above 2 methods
- hysterotomy (abdominal) — less commonly done

T1 MTP

① mifepristone (RU-486) & misoprostol

↓
progesterone antagonist

→ effective upto 63 days & highly successful within 49 days of gestation

② oral mifepristone 200mg (1 tab) + vaginal misoprostol 800µg

(4 tab, 200 µg each) differs 6-48 hrs is equally effective

→ approved upto 63 days of pregnancy

* Mifepristone can be used in fibroids, molar pregnancy, ectopic pregnancy

→ not used in threatened abortion

Operative obstetrics

ECV :

- conversion of non cephalic to cephalic presentation
- done ideally at 37 weeks
- I_x → non cephalic presentations
- C/I → conditions where NVD is C/I as in placenta previa / abruption (APH)
- → twin pregnancy (can be done in 2nd twin if intact membranes)
- → oligohydramnios
- previous LSC is not a C/I.
- if bradycardia of baby occurs, wait & watch for 20 mins

Episiotomy :

- Median episiotomy is painless & heals easily (not done now)

IPV :

- delivery of 2nd twin is the only I_x of IPV in modern OB/G.
- done under GA

Ventouse :

- C/I in prematurity, face presentation, breech presentation

Forceps: delivery

- used in mento anterior position
- maternal heart disease

Duhrssen's incision :

- incision of ex in 2 & 10 o'clock positions
- used in trapped fetal head, cervical dystocia d/t fibrosed ex or uterovaginal prolapse

Drugs in pregnancy

Oxytocin

- each ampule contains 5U/ml; stored at 4-8°C
- Ix → early pregnancy
 - augment expulsion of products in abortion
 - induction & augmentation of labour
 - prevention & control of PPH
- Routes → iv (mlc), im, buccal tablets, nasal sprays
- Ad/E →
 - water intoxication & anti diuretic at high doses
 - hypot, formation of constriction ring
 - fetal distress

Methergin:

- Syntometrin → oxytocin 5U + ergometrine 0.5U
- Ix → control bleeding foll. abortion & evacuation of molar pregnancy
 - PPH
- C/I → pre eclampsia & eclampsia, heart disease, twin pregnancy following delivery of 1st twin, Rh incompatibility, labour induction/augmentation
- causes tetanic uterine contractions
- Ad/E → ↑ in BP, N & V
- Pv (fastest) / im / oral
- Misoprostol → PG E1 analogue
 - 25, 50, 100, 200, 400 mcg tablets
- uses → abortion, induction of labour, PPH
- oral / SL / vaginal / rectal

• Dosage : 1) abortion - 800 - 1000 mg

2) induction - 25-50 mcg @ 4-6 hrs

3) PPH - 800-1000 mcg PR

• advantages → selective for uterus & has no effect on bronchus (not CI in asthma)

• Ad/E → N&V, diarrhoea, chills, rigors, fever, meconium staining of amniotic fluid

Dinoprostone (PG E₂)

• gel contains 0.5 mg of PG E₂

• Tx → cervical ripening, induction of labour

• Dosage → intracervical application - 0.5 mcg

• Ad/E → fetal distress, rupture of uterus

Carboprost (PG F_{2α})

• each ampule - 250 mcg / ml

• Tx → PPH

• dose → 250 mcg as i.m inj only

• max of 8 doses can be repeated at 15 min interval

• Ad/E → exaggeration of asthma

choice of drugs

• MTP → misoprostol = mifepristone

• control of bleeding in abortion → misoprostol, oxytocin, methergin

• induction $\left\{ \begin{array}{l} \text{C}_x \text{ unfavourable} \rightarrow \text{PG E}_2, \text{PG E}_1 \text{ (Bishop score } < 6) \\ \text{C}_x \text{ favourable (Bishop score } > 6) \rightarrow \text{oxytocin \& PG E}_2 \end{array} \right.$

• augmentation → oxytocin is DOc

AMTSL → oxytocin 10 U IV or IM

- control of PPH → oxytocin, methylergometrine, misoprostol, carboprost

Antihypertensives in pregnancy

- 1st line → methyl dopa or labetalol
- 2nd line → nifedipine
- hypertensive emergencies → labetalol, hydralazine, sodium nitroprusside
- ACE Inh are C/I

α methyl dopa

- MOA → central & peripheral acting anti adrenergic
- dose → 250mg bd or tds orally, ↑ed upto max 2g/day in divided doses
- Ad/E → postural hypot, sedation, depression, hemolytic anemia
fetal → intestinal ileus

Labetalol :

- combined α & β adrenergic blocker → ↓ peripheral vascular (R)
- dose → 100mg tds oral.
- Ad/E → postural hypotension, headache, tremor

Nifedipine :

- used for HTN & as tocolysis
- MOA → direct arteriolar vasodilatation (CCB)
- Ad/E → flushing, hypot, headache, tachycardia, ⊕n of labour

Anti convulsant

- $MgSO_4$ is Doc for control & prevention of eclampsia
- others → phenytoin & diazepam

$MgSO_4$

- anticonvulsant in eclampsia
- fr in severe pre-eclampsia & "
- Tocolysis - in preterm labour
- MOA → suppress motor and plate sensitivity to ACh & reduce release of ACh from N endings
- competitive $\ominus$ of Ca^{2+}
- direct depressive action on uterine muscle
- reduces cerebral edema

Dosage → PRITCHARD Regimen

- loading dose → 4g slow iv (20% soln) over 3-4 min, foll by 10g (50% soln) deep im gn as 5g in each buttock
- maintenance dose → 5g (50% soln) deep im on alternate buttock every 4 hrs only on monitoring
- Knee jerk, urine output (100 ml in 4 hrs / 30ml in 1 hr), RR > 14/min

- C/I → myasthenia gravis, oliguria
- Ad/E → respiratory depression, muscle weakness
- Therapeutic level of $MgSO_4$ → 4 to 7 mEq per litre
- antidote → calcium gluconate (10ml of 10% slow iv)

- CCB - nifedipine
- $MgSO_4$
- β mimetics $\rightarrow$ terbutaline, nitroglycerine, doxapram

- NSAIDs - indomethacin, zolindac
- atazanavir - oxytocin antagonist
- Glyceryl trinitrate - NO donor

Anticoagulants:

- $I_x \rightarrow$ venous TE, APLA, AF, prosthetic valves, cardiomyopathy

Heparin

- MOA $\rightarrow$ $\ominus$ action of thrombin
enhances action of AT III
- dose $\rightarrow$ 5000 - 10,000 units SC every 12 hrs (i.e. aPTT monitoring)
- A/E $\rightarrow$ maternal - hemorrhage, thrombocytopenia, osteoporosis
fetal $\rightarrow$ no effects as it doesn't cross placenta
- antidote $\rightarrow$ protamine sulphate

LMWH:

- longer DOA than UFH (single daily SC inj)
- no need of monitoring & $\downarrow$ A/E
- enoxaparin, dalteparin
- dose: 2000 to 4000 units

Warfarin:

- more effective than heparin in preventing TE
- A/E $\rightarrow$ warfarin in T1 causes warfarin embryopathy
 $\rightarrow$ $\uparrow$ R/O abortion, still birth & fetal bleeding
- BF is not C/I $\pm$ warfarin therapy
- vit K & FFP are given in overdose

Protocol for anticoagulation:

- 6 to 12 wks → heparin is given
 - 12 - 36 wks → warfarin or continue heparin
 - > 36 wks → heparin - stopped at onset of labour
restarted then following NVD & 24 hrs full CS
 - warfarin is started in perinatal period overlapping with heparin
full desired INR is achieved & heparin is stopped
- heparin therapy : aPTT is kept 2 times the control
warfarin " : INR btw 2.5 - 3.5

Teratogens

- 2-3% of birth defects are d/t drug exposure
- 4 to 12 wks → maximum teratogenicity
- before 31 ~~days~~ days → all or none effects

FDA risk categories:

- A → safe drugs, studies available
- B → no risk in human, risk in animals +
- C → risk cannot be ruled out, well controlled human studies lacking
- D → +ve evidence of risk, given if benefit outweighs risk in life threatening conditions
- X → CI

X drugs → alcohol, ALEs, Li, Mtx, misoprostol, danazol, isotretinoin, radioactive iodine

Drugs & fetal effects

Analgesics

- para - safe
- indomethacin - premature closure of DA
- aspirin → premature closure of DA, persistent PHT & Kernicterus in newborn

Anticoagulants

- warfarin → ~~causes~~ condrati syndrome / chondrodysplasia punctata

Antihypertensives

- ACE In → renal tubular dysplasia, lung hypoplasia, oligohydramnios, limb constriction defects

Others

- carbimazole → fetal aplasia cutis, choanal atresia, esophageal atresia
- Li → Ebstein anomaly, fetal goitre / hypothyroidism, fetal DI
- DES → vaginal clear cell Ca, T-shaped uterus, vaginal adenosis
- Isotretinoin → 10% microtia, hypertelorism, outflow tract defects, hydrocephalus
- chloramphenicol → gray baby syndrome (peripheral vascular collapse)
- Sulphonamides → neonatal hemolysis, jaundice
- corticosteroids → high doses - fetal & neonatal adrenal suppression
prolonged systemic use → IUGR
- androgenic steroids → masculinization of female offspring
- phenytoin, valproate → • benefits outweigh risk, R to NTD, neonatal bleeding
• avoid polytherapy
- phenobarbitone → safe
- zidovudine, acyclovir → safe
- all live viral vaccines → C/I in pregnancy

Substance abuse

- Alcohol → IUGR, cranio facial defects - microcephaly, microphthalmia, flat nasal bridge, hypoplastic philtrum, behavioural anomalies
- Smoking → IUGR, LBW
- Cocaine → microcephaly, cerebral infarcts, abortion, LBW

Abnormal uterine action

Ⓐ uterine contractions

- Braxton Hicks contractions → painless, irregular, throughout pregnancy, no Cx changes
- Effective uterine contractions -
 - * start from perimetrium at cervix (R stronger than L), propagates downwards, depolarises whole uterus within 15 sec
 - * contractions predominant over fundus
 - * upper segment contracts, LUS relaxes

Stages of labour

- preparatory division → latent phase + acceleration phase
- dilatation " → phase of maximum dilatation
- pelvic delivery → deceleration phase + 2nd stage of labour

Assessment of uterine contractions

- Basal tone, peak pressure, duration & frequency
- assessed by - clinical method (inaccurate)
 - tocodynamometer - external transducer
 - intrauterine pressure catheter (most accurate)
- measuring unit $\rightarrow$ monte video unit

$$1MV = \text{intensity of contraction (mmHg)} \times \text{no. of contraction in 10 min}$$

Parameters

- (N) basal tone - 5 to 20 mmHg
- peak pressure - 60 mmHg
- adequate uterine contractions $\rightarrow$ 3 in 10 mins lasting for 45 sec
 - $\rightarrow$ causing intrauterine pressure of 65-75 mmHg or 220 MV units
- Tachysystole - >5 contractions in 10 mins
- Hypertension - tachysystole causing fetal distress

Dysfunctional labour:

- more common in primi
- (N) $\times$ dilatation in primi - 1 cm/hr
 - multi gravida - 1.5 cm/hr
- 1° dysfunctional labour - $\times$ dilates <1 cm/hr following (N) latent phase of labour; mostly gets corrected by amniotomy/ oxytocin augmentation

• 2nd aspect of dilatation - (x dilatation stops or slows after start of ④ active phase of labour

Abnormal uterine ~~cont~~ action

< coordinated uterine action → polarity is maintained
inco-ordinate uterine action → polarity lost

Co-ordinated

- ① Hypotonic
- uterine inertia
- ② Hypertonic
 - ① no obstruction - precipitate labour
 - ② obstruction - generalised tonic contraction & retraction
pathological retraction ring

In co-ordinate

- ① spastic lower uterine segment
- ② constriction ring
- ③ Hypertonic uterine contractions (tetany)
- ④ colicky uterine
- ⑤ Asymmetrical uterine contractions

Hypotonic uterine contractions

- m/c disorders
- < 3 contractions in 10 min, each < 45 sec / < 180 MV units
- occur at any stage of labour
- polarity maintained, FHS good
- 4/m → rule out CPD, malpresentation, malpositions
→ labour augmentation & ARM + oxytocin

precipitate labour:

- rapid expulsion of baby d/t hyperactive uterine contractions
- combined duration of stage I & II < 2 hrs
- Cx dilatation ≥ 5 cm/hr in multiparous & 10 cm/hr in multi
- common in multipara, recurrence is common
- C/C $\rightarrow$ injury to Cx, vagina, perineum
 - PPH, uterine rupture / inversion, infections
- fetal $\rightarrow$ ICH

Bandl ring / pathological contraction ring / Tonic uterine contraction & retraction

- D/t obstruction
- formed at jn of upper & lower US
- Keeps moving towards umbilicus

End result

- primi $\rightarrow$ further retraction stops - uterine exhaustion
- multi $\rightarrow$ progressive thinning of LUS $\rightarrow$ uterine rupture

CF

- dehydration & Ketacidosis
- pt exhausted
- upper segment - tender, LUS - distended
- Palpable ring

M/m $\rightarrow$ analgesics, correct dehydration & acidosis
rule out uterine rupture

CS done

Prevention $\rightarrow$ partogram

Spastic lower segment

- Reversed polarity, lack of fundal dominance
- LUS contractions stronger
- Basal tone ↑
- pt in unbearable pain
- tender uterus, difficulty in palpating fetal parts
- fetal distress
- M/m → CS

Constriction ring / Schroeder's ring:

- localised myometrial contraction forming ring at junction of upper & LUS, usually around neck of fetus in cephalic presentation; abnormal polarity (⊗)
- usually reversible
- causes → injudicious oxytocin
→ premature attempt at instrumental delivery
- Δ → ring not felt by PA. It is revealed during CS in I stage
forceps application in II stage
manual removal of placenta in III stage (hour glass contractions)
→ PV = no features of obstruction
- maternal condition not much affected
- fetus may be in distress, usually FHR good
- uterus never ruptures
- No alteration in position of fetus

M/m - o delivery by CS

o ring usually passes off by deepening the plane of anaesthesia or cut vertically to deliver the baby

o difficulties in II or III stage → deep anaesthesia releases the ring

Generalised tonic contraction (tetany)

o contractions involve whole of uterus upto internal os

o no diff b/w upper & LUS

o tonic muscular spasm of uterus & fetus inside

o no uterine rupture

o cause → CPD, methergine, injudicious oxytocin

o CF → severe continuous pain, tender & tense uterus, fetal distress ±, fetal parts not well defined

o M/m → *in hypercontractility induced by oxytocin, oxytocin stopped. Terbutaline given

* in CPD & fetal distress → CS

Cervical dystocia

o Failure of cervical dilatation

o cause → inefficient ut. contractions, malpresentation, malposition

o Cx spasm < 1° - primie, Cx rigid & fails to dilate

2° → scarring of Cx from previous surgery or d/s

o M/m ⇒ o malpresentation / position → CS

o Thin rim of Cx → rim pushed up manually before NVD

o Dubresne's incision at 2 & 10 o'clock position

Amniotic fluid

- The amniotic fluid is replaced every 3 hours
- Origin
 - * early pregnancy - ultrafiltrate of maternal plasma across placenta
 - * T2 - transudation across fetal skin
 - * > 20 wks - fetal urine & fetal lungs - main source
- Amniotic fluid is kept in balance by
 - fetal swallowing
 - intramembraneous flow across skin, cord & placental surface
- Composition & volume
 - * water - 98%, solids 1-2%
 - * osmolality - 250 mosm/l, pH - 7 to 7.5, Sp gravity - 1.010
 - * rate of turn over → 550 cc/hr
 - * replaced every 3 hrs
 - * volume of AF
 - 12 wks - 50 ml
 - 20 wks - 400 ml
 - 36-38 wks - 1000 ml
 - 40 wks - 800 ml
 - 42 wks - 500 ml
- Colour
 - * near term - straw colour (N)
 - * * greenish yellow - post maturity
 - * green - meconium stained, breech

* golden yellow - Rh incompatibility (bilirubin) www.FirstRanker.com www.FirstRanker.com

* dark coloured - absorption

* dark brown - IUD

o Functions

* NOT MUCH NUTRITIVE VALUE

* shock absorber

* temp. maintenance

* movement of fetus - prevents adhesion

* growth of fetus

* during labour → bag of membrane helps in dilatation of cx

o Assessment

* USG → antepartum fetal surveillance (APFS)

* **AFI** → sum of 4 quadrant measurement of largest vertical pocket

→ **(N): 5 - 25**

* Single deepest pocket (**SDP**) • measurement : **(N) → 2 - 8 cms**

Polyhydramnios

or
(SVP)

o **liquor > 2000 ml**

o **AFI > 25** / **SDP ≥ 8 cm**

Grades

SVP (cm)

o mild

8-11 (mlc)

o moderate

12-15

o severe

>16

◦ Fetal causes → * **anencephaly** - transudation from exposed meninges
- absent fetal swallowing reflex
- suppression of fetal ADH

* **open spina bifida**

* **cleft lip / palate, neck mass**

* **esophageal / duodenal atresia**

* **fetal aneuploidy**

◦ **Multiple pregnancy** - **TTTS**, monzygotic twins

◦ Maternal → **diabetes, Rh incompatibility**

◦ Placental → **chorioangioma of placenta**

◦ **Idiopathic** (m/c cause) - 60%.

Clinical types

① **Acute type** → usually **before 20 wks**

◦ **very rare**

◦ **alw TTTS, chorioangioma of placenta**

② **chronic type** → m/c type

Δ:

◦ **FH (fundal ht) > PAG**, **↑ed abdominal girth**

◦ **difficulty in palpating fetal parts, presentation, position, FHS**

Investigations → **USG**, **blood grouping & typing**, **GTT**, **MSAFP**

Complications

◦ Maternal

Antepartum

- pre eclampsia
- malpresentations
- PROM
- preterm labour
- abruption

Intrapartum

- early ROM
- cord prolapse
- uterine inertia
- operative interferences
- PPH

post partum

- subinvolution

◦ Fetal → ↑ morbidity & mortality d/t congenital anomalies & prematurity

Management

◦ mild → no Rx req

◦ **Severe** → Rule out anomalies

* **indomethacin therapy** (105-3 mg / kg / d)

- **stopped at 32 wks**

* **if pt doesn't respond / pt in respiratory distress**

- **serial amniocentesis**

- **500 ml/hr, max upto 1-1.5 L/day**

◦ Intrapartum → * stabilising oxytocin foll by low ROM, allow slow escape of liquor

* Plv following spontaneous ROM to rule out cord prolapse

* **AMTSL**

* examine newborn for anomalies

Oligohydramnios

- liquor < 200 ml at term / AFI < 5 / SVP < 2 cm /
- absent amniotic fluid pocket

causes

- fetal $\rightarrow$ * renal agenesis (m/c)
* fetal anomalies, obstructive uropathy (post-urethral valve),
* IUGR, post maturity
- maternal $\rightarrow$ * hypertensive disorder (uteroplacental insufficiency)
* PROM
* dehydration
- placental $\rightarrow$ amnion nodosum, AFE etc.
- Idiopathic $\rightarrow$ m/c cause of mild oligohydramnios

Δ

- FH $<$ POG, $\downarrow$ ed abd. girth
- uterine feels 'full of fetus'
- USG

Complications

- Maternal - $\uparrow$ ed operative interferences
- Fetal $\rightarrow$ * fetal pulm. hypoplasia
* fetal deformities d/t intra amniotic adhesions - alteration in shape of skull, CTEV, amputation of limb
* cord compression
* meconium aspiration

M/m $\rightarrow$ rule out anomalies

$\rightarrow$ isolated oligohydramnios in T3 - conservative m/m $\pm$ APFS upto term $\rightarrow$ induction of labour

Intrapartum

- * fetal monitoring → CTG / intermittent auscultation (every 15 min in stage I, stage II - every 5 min)
- * amnio infusion → warm saline infusion into amniotic cavity of 250 ml in 30 mins

Indications of amnio infusion

- * Prophylactic / therapeutic
 - prevents cord compression in oligohydramnios
 - of variable deceleration
 - dilutes or washes out thick meconium
- * Diagnostic amnio infusion
 - in T2 for better USG visualization of fetal anomalies in severe oligohydramnios

Variable deceleration

- not in relation to uterine contractions
- seen in cord prolapse

Early deceleration

- seen in head compression
- starts with uterine contraction & passes off before contraction passes

Late deceleration:

- peak of acceleration after peak of contractions, lasts even after contraction has passed off
- seen in uteroplacental insufficiency.

PIH

- HT in pregnancy $\Rightarrow$ SBP $\geq$ 140 mm Hg or DBP $\geq$ 90 mm Hg on 2 occasions 4 hrs apart

Measurement of BP :- sitting / lateral lying down position

- manometer at level of heart
- Korotkoff's ~~2nd~~ sound IV is taken as DBP

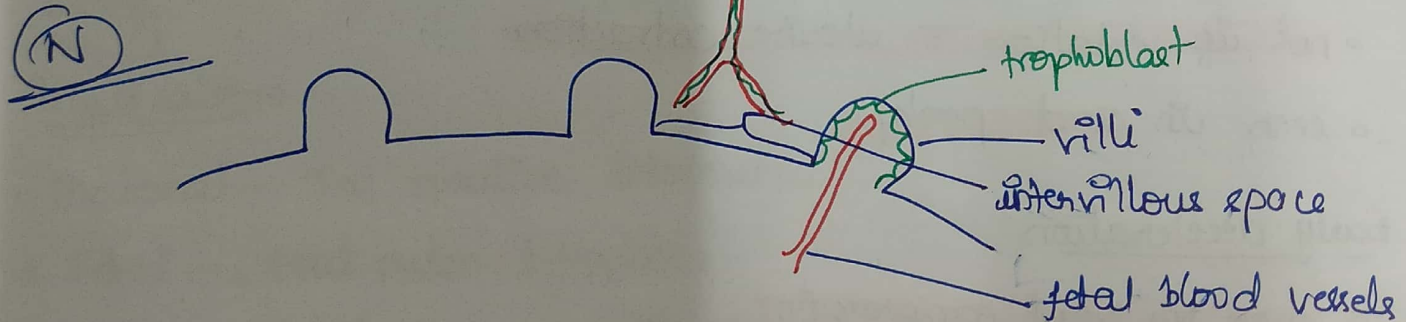
Chronic HT during pregnancy

- $\uparrow$ in BP before 20 wks (H/o chr. HT)
- BP doesn't come back to $\textcircled{N}$ after 12 wks of delivery

PIH :-

- $\uparrow$ in BP after 20 wks of pregnancy
- BP comes back to $\textcircled{N}$ before 12 wks of pregnancy

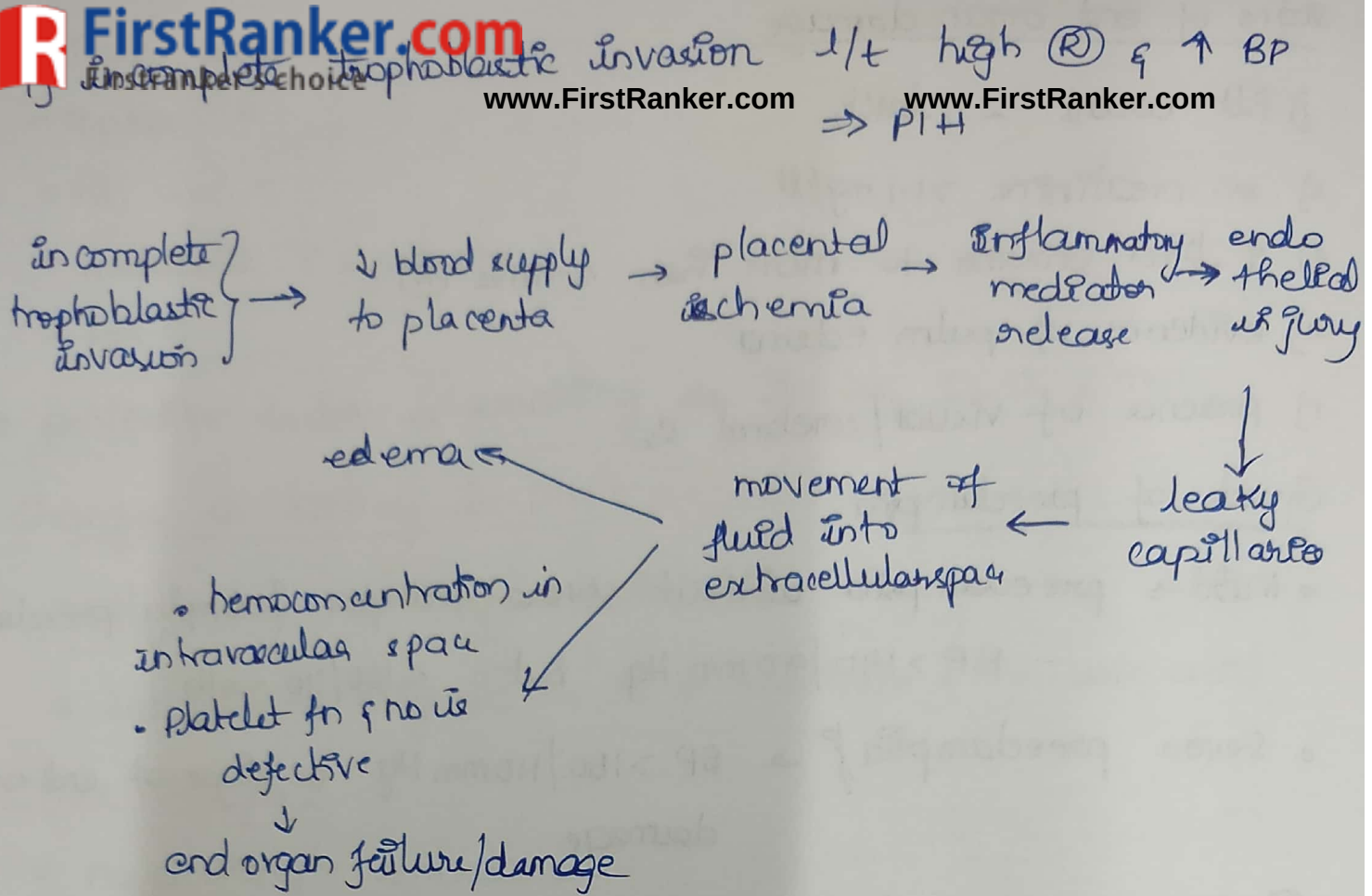
Pathogenesis



- Trophoblast replaces maternal lining, $\therefore$ spiral arteries become low $\textcircled{R}$ vessels

ie trophoblastic invasion

- step I $\rightarrow$ 10 to 12 wks
 - trophoblast replaces lining of arteries upto decidua
- step II $\rightarrow$ 16 to 18 weeks
 - replaces upto myometrium



∴ main culprit in PIH is placenta → removal of placenta is the

- the size of placenta is ↓ed in PIH
- ↑ed in DM, Rh incompatibility, twin
- m/c hematological finding in PIH → ↓ed platelet count

Types of PIH

- Gestational HTN → all 3 characters of PIH seen
- Preeclampsia → proteinuria or signs of end organ failure + other 3 characters of PIH

Proteinuria

- excretion of proteins > 300mg in 24hr urine sample or > 30mg/dl of urine

Signs of end organ damage

- 1) Plt count $< 1 \text{ lakh}$
- 2) sr. creatinine $> 1.01 \text{ mg/dl}$
- 3) $\uparrow$ Liver enzymes to more than 2 times $\textcircled{N}$
- 4) evidence of pulm edema
- 5) presence of visual/cerebral sx

Grade of preeclampsia

- Mild $\rightarrow$ preeclampsia without severe features / simply preeclampsia
 $\text{— BP } > 140/90 \text{ mm Hg but } < 160/110 \text{ mmHg}$
- Severe preeclampsia $\rightarrow$ BP $> 160/110 \text{ mmHg}$ or signs of end organ damage

criteria is removed to classify b/w mild & severe

- $\rightarrow$ • amount of proteinuria
- oliguria
- IUGR

• In PIH, as $P \propto \frac{1}{V} \Rightarrow$ fetal blood flow is led

$\rightarrow$ IUGR (brain sparing effect)
 $\rightarrow$ fetal oliguria
 $\downarrow$
 oligohydramnios

Chronic HTN + superimposed preeclampsia =

• a HT female +

- i) BP suddenly becomes uncontrollable after 20 wks or
- ii) new onset proteinuria after 20 wks or
- iii) signs of end organ damage after 20 wks

RF : * previous H/o preeclampsia + obesity
 * primi
 * chr. HT
 * diabetes
 * chr. renal dis
 * extremes of maternal age
 $< 18 / > 40$

- Molar "
- Rh -ve "
- metabolic syndrome
- APLA

* protective factor → smoking as it ↓ IL-2 & TNF

changes in kidney in PIH:

* glomerular capillary endotheliosis

* ↓ed GFR → oliguria → ↑ sr. creatinine & ↑ sr. uric acid

Other changes:

- * no electrolyte imbalance
- * levels of renin, aldosterone & angiotensin II are ↓ed in PIH, but sensitivity of vessels is ↑ed to them
- * In brain → post. hemisphere changes → visual disturbances
→ scotomas

Eclampsia

- It is severe preeclampsia & GTCS or coma & cannot be attributed to any other cause
- maternal mortality is 4-6% & perinatal mortality is 45% in India
- incidence of eclampsia < globally → 1 in 1500-2000
India → 1-5%

Signs of impending eclampsia:

- epigastric pain, N & V
- headache & dizziness (dit cerebral hypoxia)
- visual sr like blurring / diplopia / scotomas / blindness

- eclampsia is irreversible after delivery
- It can be d/t occipital lobe edema or retinal detachment
- convulsions is d/t cerebral hypoxia
- eclampsia
 - ↳ antepartum
 - ↳ intrapartum
 - ↳ postpartum (within 48 hrs after delivery)
- m/c is antepartum eclampsia
- antepartum eclampsia has worst prognosis
- m/c MRI finding → subcortical white matter edema
- m/c of death → intracranial bleeding

HELLP syndrome

- feature of severe preeclampsia
- Not a form of DIC → here PT, aPTT, fibrinogen all are $\uparrow$
- 15% pts have $\uparrow$ BP
- m/c in 3rd trimester
- maternal mortality rate - 1%
- Sx → malaise, nausea, vomiting, epigastric pain
- H → hemolysis → LDH > 600 , PS → signs of hemolysis, low / absent haptoglobin → most specific
- EL → elevated liver enzymes → AST ≥ 70 IU/L
- LP → low platelet < 1 lakh

Criteria for Δ of HELLP ⇒ Tennessee criteria

- platelet < 1 lakh
- AST > 70 IU/L
- LDH > 600 IU/L

Mississippi classification of HELLP syndrome :

- class 1 (most severe) - plt count $< 50,000$
- class 2 $\rightarrow$ plt count btw 50000 - 1 lakh
- class 3 $\rightarrow$ 1 lakh - 1.5 lakhs, but AST & LDH $\uparrow$

M/m :

- Immediate termination of pregnancy & MgSO₄ > 34 ^{wks}
- If btw 27 - 34 wks, give corticosteroid & deliver after 24 hrs of last dose of corticosteroid

Corticosteroid

- DOC $\rightarrow$ betamethasone - 2 doses of 12mg 24 hrly i.m worldwide
- In India $\rightarrow$ dexamethasone 6mg, 12 hrly 4 doses i.m

D/D of HELLP :

- Acute fatty liver of pregnancy (AFLP)
 - AFLP has additional features of hypoglycemia, deranged coagulation profile, hepatorenal syndrome, pancreatitis
- ↓
think of AFLP & not HELLP

Predictors of pre-eclampsia :

- Uterine artery doppler $\rightarrow$
 - $\rightarrow$ shows diastolic notch
- It disappears by 24 weeks in $\textcircled{N}$ pregnancy
- persists after 24 weeks in PIH

Predictors no longer used

- hemoconcentration
- uric acid levels
- hypocalcaemia
- Ted homocysteine levels
- giant roll over test

Basis of giant roll over test → supine hypotension syndrome
or
ZVC syndrome

But if instead of ↓ BP, BP ↑ by 20 mmHg → +ve test
↓
future PIH +ve

done btw 28-32 wks

Drugs to prevent pre eclampsia:

- Best - aspirin - 50 - 150 mg / day, continued throughout pregnancy & stopped 7 days before labour
- Calcium supplementation — only in hypocalcaemia

No roles in PIH → salt restriction, fish oil, antioxidants

M/m of PIH:

- Termination of pregnancy
- Antihypertensives → as per ACOG guidelines → only in $>160/110$ mmHg
as per NICE " → $>150/100$ mmHg
→ labetalol, hydralazine, a methyldopa,
nifedipine, nitroglycerine, sodium nitroprusside

- Anti HT of choice in PHT / pre-eclampsia / chronic HTN / acute HT / hypertensive crisis → **labetalol**
- Target BP in pre-eclampsia - SBP ⇒ 130-140 mmHg
DBP ⇒ 80-90 mmHg
- in chr HT during pregnancy → systolic 140-150 mmHg
DBP 90-100 mmHg

Labetalol

- oral dose → 100mg bid or tds
- iv → 20mg iv bolus → if not effective within 10 mins
↓
80mg every 10 mins ← if not effective ← 40mg
- max dose → 220mg
- 2nd line drug in acute HTN - iv hydralazine - 5mg initial dose
followed by 5-10mg at 15-20 mins. Max - 30mg

Anti hypertensive CI in pregnancy

- ACE OR
- losartan
- diltiazem
- diuretics → CI only as antihypertensive, but if pt has CHF, diuretic can be given

M/m of mild pre-eclampsia / gestational HT

- Antihypertensives ±
- Definitive M/m → termination pregnancy at 37 weeks
- NVD is preferred

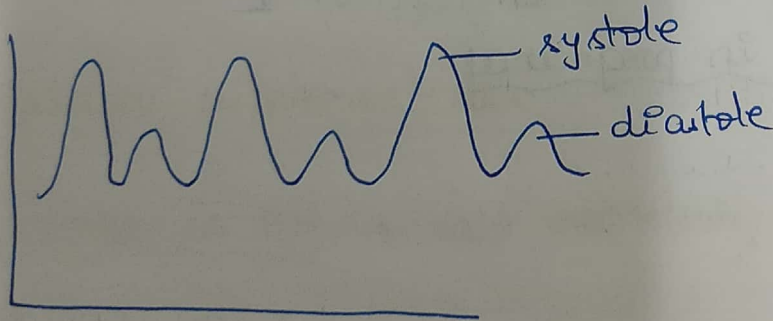
M/m of severe pre eclampsia

- $MgSO_4$ + antihypertensive
- Definitive m/m $\rightarrow$ termination of pregnancy ≥ 34 weeks
- mode of delivery $\rightarrow$ vaginal delivery

IX for immediate termination irrespective of GA :

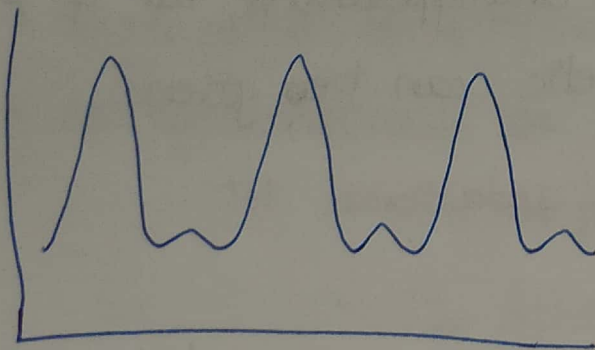
- impending eclampsia
- eclampsia
- fetal distress / abruption placenta
- uncontrolled BP or rising S creatinine levels
- HELLP syndrome
- reversal of end diastolic flow in umbilical artery doppler

Umbilical artery doppler



in (N) pregnancy

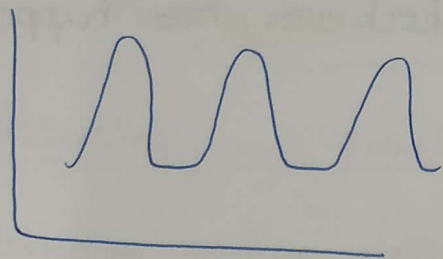
Ⓟ of blood vessels $\downarrow$
 $\downarrow$
 Diastolic flow $\uparrow$
 $\therefore$ S/D ratio $= \downarrow$



in PIH

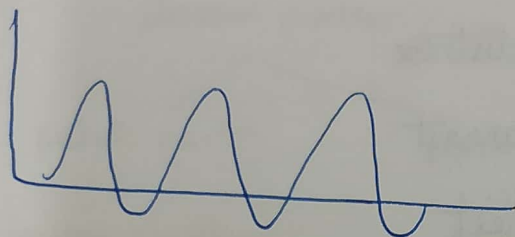
diastolic flow $\downarrow$ d/t $\uparrow$ (R)
 $\therefore$ S/D ratio $= \uparrow$

Severe preeclampsia



absent end diastolic flow
↓

terminate pregnancy ≥ 34 wks



reversal of end diastolic flow
↓

terminate pregnancy irrespective of GA

m/m of eclampsia :

- 1st line $\rightarrow$ airway management
 - then to convulsion $\rightarrow$ $MgSO_4$ & antihypertensive $\rightarrow$ i.v labetalol
 - Definitive m/m $\rightarrow$ terminate pregnancy irrespective of GA
 - Mode of delivery $\rightarrow$ try vaginal - if it occurs in 24 hrs of last convulsion, otherwise cesarean section
- * DOC for status epilepticus in pregnancy $\rightarrow$ thiopentone sodium
> phenobarbitone

$MgSO_4$:

- MOA $\rightarrow$ blocks NMDA receptors in brain, causes cerebrovasodilation, peripherally it blocks calcium channels (use cautiously $\pm$ CCBs)

- $MgSO_4$ should be continued till 24hrs after delivery / 24hrs after last convulsion whichever has happened later

$MgSO_4$ toxicity

- Therapeutic range $\Rightarrow$ 4 to 7 mEq/L
- @ 10 mEq/L $\Rightarrow$ DTR lost
- @ 12 mEq/L $\Rightarrow$ respiratory distress
- @ 15 mEq/L $\Rightarrow$ respiratory arrest
- > 30 mEq/L $\Rightarrow$ cardiac arrest
- antidote for $MgSO_4 \rightarrow$ 10% calcium gluconate i.v
- Currently ACOG recommends $\rightarrow$ iv $MgSO_4$ d/t R/O gluteal abscess in i.m dose

It is like Sibal regime

- $\rightarrow$ loading dose - 6mg iv
- $\rightarrow$ then 2g/hr iv infusion
- $\rightarrow$ if convulsion occurs - 2g iv is given again

Uses of $MgSO_4$:

- severe pre-eclampsia
- HELLP syndr.
- eclampsia
- neuroprotective function to prevent CP in neonate, so used in preterm infants
- short term tocolytic