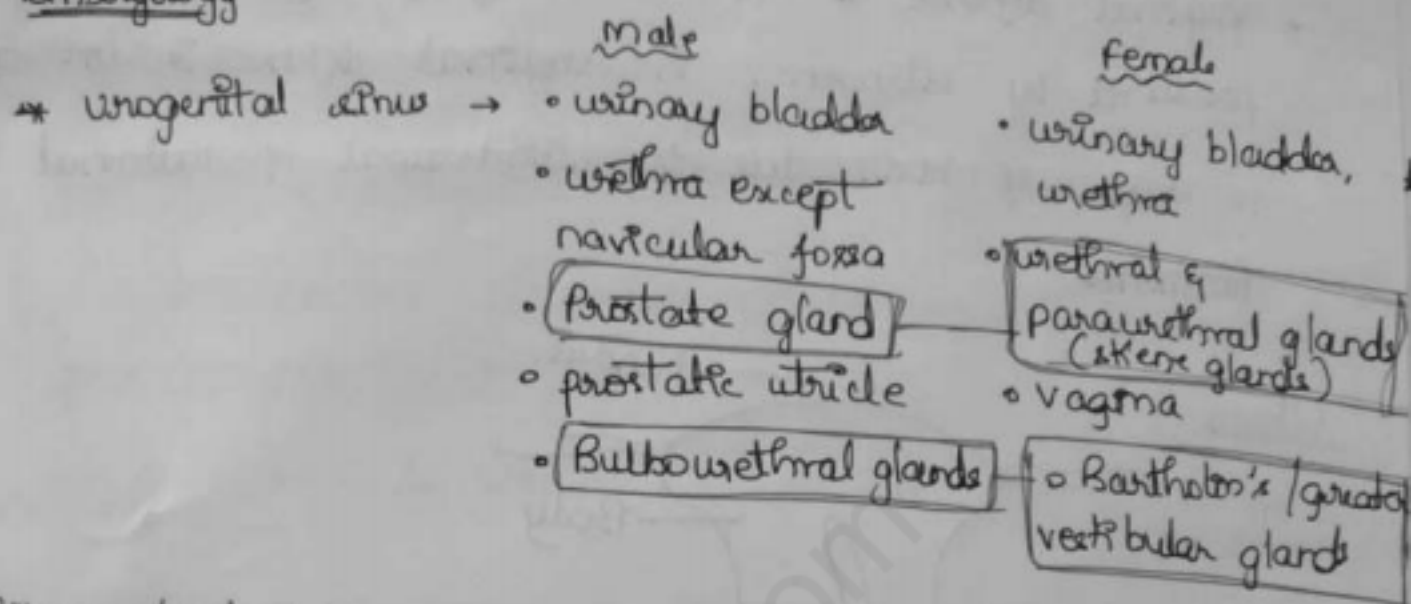


Anatomy of female genital tracts

Embryology



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

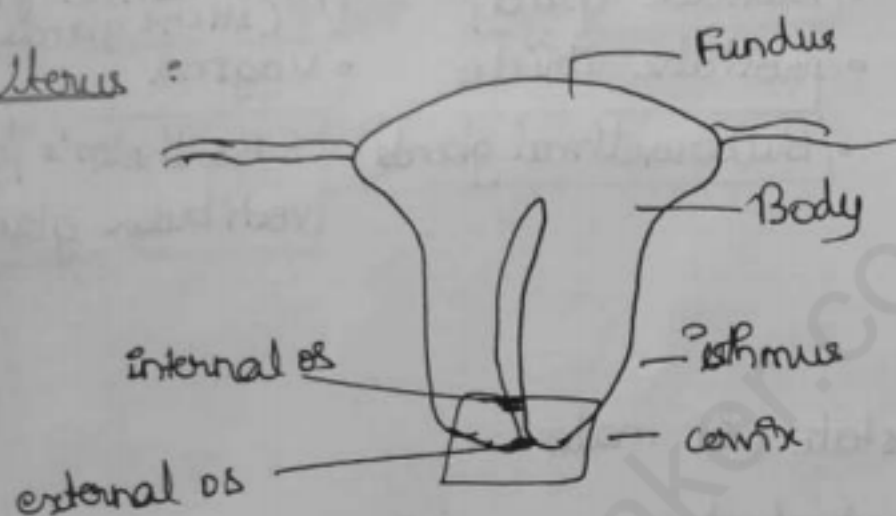
Bartholin's gland:

- opens b/w hymen & labia minora in inner aspect of labia minora (10 o'clock & 5 o'clock positions)
- Bartholin's cyst → TOC: marsupialization
- Bartholin's abscess TOC: IVD

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 ciliated 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 $\therefore$ 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 $\therefore$ 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

* Support 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

Physiological changes in pregnancy

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

Hematological change $\rightarrow$ begins by 6 weeks, max changes are in T2
 $\uparrow$ b/w 28-32 wks

- Blood volume $\rightarrow \uparrow$
- plasma volume $\rightarrow$ 40 to 50% $\uparrow$
- RBC mass $\rightarrow$ 18 - 20% $\uparrow$
- Hb mass $\rightarrow \uparrow$
- Hb concentration $\rightarrow \downarrow$
- Hematocrit $\downarrow$
- viscosity $\downarrow$

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

- ~~Se Iron~~ $\rightarrow$ ~~B~~
- Total requirement of iron during pregnancy $\rightarrow$ 1000 mg
 in T₁ $\rightarrow$ 3-4 mg/day
 T₂ & T₃ $\rightarrow$ 6-7 mg/day
- $\therefore$ • 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 $\uparrow$ in factor II (Fibrinogen)
- Fibrinolytic activity is $\downarrow$ [d/t $\downarrow$ TPA & $\uparrow$ TPAI]
- protein C & S are $\downarrow$
- Antithrombin levels are $\downarrow$ (N) • BT & CT are (N)

- ESR ↑
- leucocyte ALP, C3, C4, CRP, procalcitonin → ↑
- 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 $\rightarrow \uparrow$ / $\downarrow$
- DBP $\rightarrow \downarrow$
- MAP $\rightarrow \downarrow$

- Femoral venous pressure $\rightarrow \uparrow\uparrow$ [N $\rightarrow$ 8mm Hg
pregnancy $\rightarrow$ 24mm Hg]

Other changes

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

• H_2O reabsorption may be seen

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• 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 $\textcircled{N}$ - 25 mg/ml
pregnancy - 150 mg/ml

Thyroid gland

- Total $T_3, T_4 \rightarrow \uparrow$
- Free $T_3, T_4 \rightarrow \textcircled{N}$
- 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 labor pain & ends τ full dilatation of cervix

True labour pain	False pain
◦ $\uparrow$ intensity	◦ —
◦ regular	◦ Irregular
◦ Interval shortens	◦ —
◦ not relieved by enema, sedation	◦ relieved
◦ also $\times$ 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 Cx 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 Cx
- 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

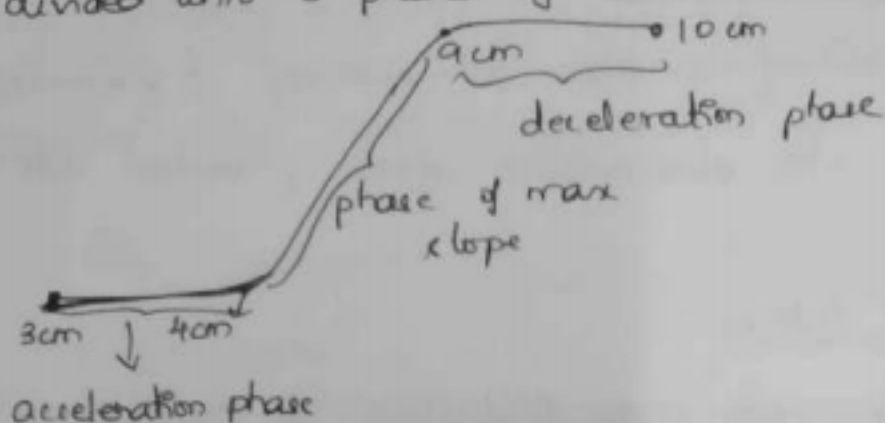
2nd stage

- 2 hrs in primi & 1 hr in multigravida

3rd stage

- 5 mins (due to active management)

- divided into 3 phases by Friedman's curve



Partogram

- New partogram doesn't include latent phase

- Components :
 - * fetal heart rate $\left[\begin{array}{l} \text{N} \Rightarrow 110-160 \\ < 100 \rightarrow \text{brady} \\ > 180 \rightarrow \text{tachy} \end{array} \right]$
 - assessed every 15 min
 - in 1st stage & every 5 min in 2nd stage

- * liquor $\rightarrow$
 - I - intact
 - C - clear
 - M - meconium
 - B - blood stained liquor

- * moulding $\rightarrow$ indicates severity of cephalopelvic disproportion

- 0+ $\rightarrow$ obliteration of parietal bones, but no overriding
- 2+ $\rightarrow$ reducible overriding
- 3+ $\rightarrow$ 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 Critchton method (by FF)

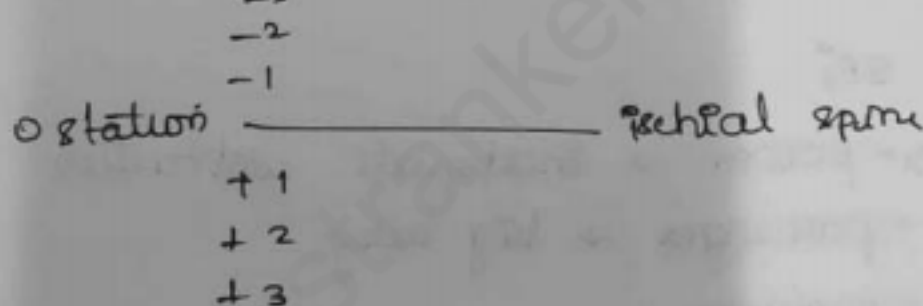
→ scores 5/5 to 0/5

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

* action line is 4hrs after alert done

* Critchton 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

→ can be fed upto 32 IU/min

- drugs administered
- maternal pulse BP
- temp, urine vol & urine acetone

Abnormalities in partogram:

- prolonged latent phase
- 1° dysfunctional labour (m/c)
 - slow progression from the very beginning
- 2° arrest of c_x dilatation

→ causes - 3P₂

- (m/c) →
- power → inadequate contraction
 - passenger → big baby
 - passage

Definite signs of separation of placenta:

- Kurtner sign → cord doesn't reavel on pushing the uterus upward in abdomen
- Schroeder sign → uterus rises in abdomen as placenta pass down into vagina (filling of placenta in vagina)
- gushing of blood
- lengthening of cord

Active management of 3rd stage of labour

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

Pressure of uterine contraction

1st stage $\rightarrow$ 50 mmHg

2nd stage $\rightarrow$ 100 mmHg

Non pregnant & early pregnancy $\rightarrow$ 2 to 3 mmHg

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

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) meal

• unrestricted physical activity

• 100g glucose gm & 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) DIAPSI (diabetes in pregnancy study group in India)

* 100% screening at booking visit

* no fasting.

- 75 g of glucose & sample after 2 hours

< 120

(N)

120 - 140

• Impaired glucose tolerance

140 - 200

• gestational DM

> 200

• 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 $\rightarrow 2.5\%$
- anomaly ^{rate} in a person $\geq$ HbA_{1c} 6-6.9% $\rightarrow 5\%$
7-7.9% $\rightarrow 11\%$
 $\geq 8\% \rightarrow 15\%$
 $\geq 10\% \rightarrow 20\%$

- Human placental Lactogen (HPL)
cortisol
estrogen
progesterone
- } all contribute to $\uparrow$
insulin resistance
 $\downarrow$
GDM

- HPL acts like GH

- ↓
- synthesised from syncytiotrophoblast
- lipolytic $\rightarrow \uparrow$ FFA

$\therefore$ in pregnancy $\rightarrow$ fasting hypoglycemia &
post prandial hyperglycemia

1st trimester

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

2nd Trimester

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

During labour :-

- prolonged labour
- shoulder dystocia
- PPH $\left\{ \begin{array}{l} \text{traumatic} \\ \text{atonic} \end{array} \right.$
- instrumental delivery
- LSCS
- still birth
- ketoacidosis

Postpartum

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

Neonatal complications

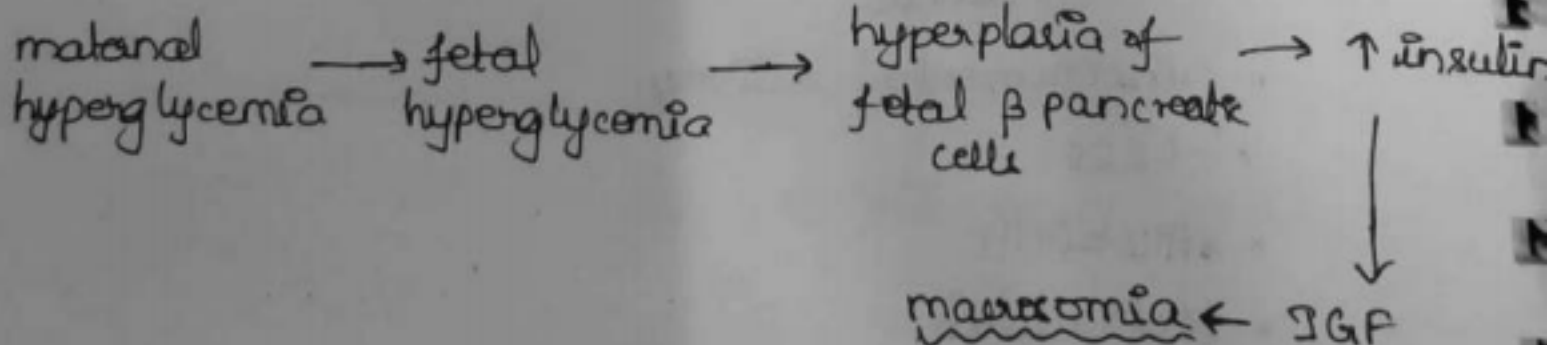
- hypoglycemia
- hypocalcemia
- hypokalemia
- polycythemia → hyperbilirubinemia
- DM
- Low IQ
- cognition & memory disorder
- cardiomyopathy

• 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 $\rightarrow$ only 60-70% sensitivity

* triple test $\rightarrow$ β HCG, unconjugated E₃ (estradiol),
(16-18 weeks)

* maternal serum AFP

or

* quadruple test $\rightarrow$ triple test + inhibin

• Targeted anomaly scan

* 18 to 20 weeks

• fetal echo - at 24 weeks

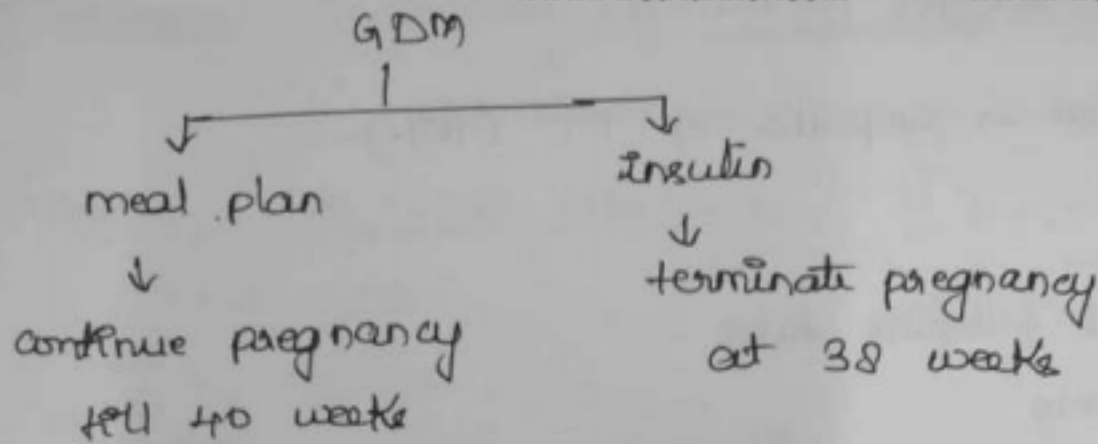
* In case of overt diabetes, admit the pt at 34 weeks &

do i) fetal KKK 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 → ampulla of FT (70%)

Risk factors

- abnormal fallopian tube
- Tubectomy
- prior ectopic pregnancy
- salpingitis & other infections (PID) (m/c) ← N. gonorrhoea
chlamydia
- salpingitis isthmica nodosa
- ART (artificial reproductive technique)
- smoking
- Copper & progestin releasing IUDs
- progestin only contraceptives (POP or minipills)

× No risk in combined OCPs

CF:

- pain abdomen (acute)
- amenorrhoea
- vaginal bleeding
- tenderness on PA ex.

o Passage of decidua

cast mirrors the shape of endometrial cavity

Outcomes in ectopic pregnancy

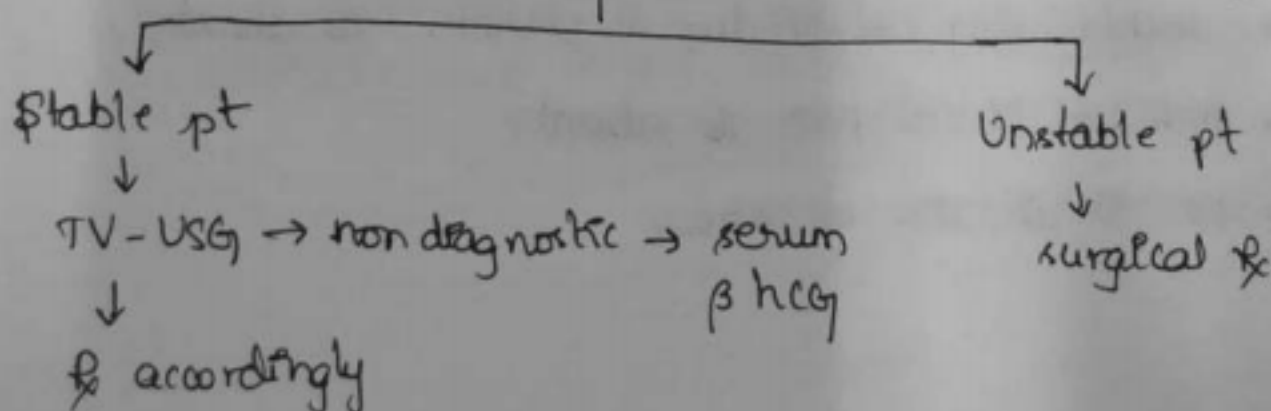
- o Tubal rupture
- o " abortion
- o pregnancy failure = resolution

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

Diagnosis

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

+ UPT & abd. pain/vaginal bleeding



- In $\odot$ 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 τ TVS $\&$ βhCG is necessary

Serum progesterone levels

- $\odot$ 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 like 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 ⊕ saline as lacy folds,
decidua without villi structure is present
- laparotomy
- culdocentesis → * used to identify hemoperitoneum
* 18G needle inserted into retroperitoneal
pouch → non clotting blood + in ectopic pregnancy

Management - medical

- Methotrexate ± or without folinic 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 < 4 cm without fetal cardiac activity
- no acute intra abdominal bleeding
- level of βhCG is single best predictor of Δ 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 salpingotomy (done now)
- salpingotomy (not done now)
- segmental resection $\pm$ or without anastomosis

2) Radical surgery

- total salpingectomy

* lesser 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 (1st abortion)
(gn for all Rh-ve women & ectopic pregnancy)

Dose of anti-D }
anti-D } • 1st abortion / MTP / ectopic → 50 mcg

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

Rare types of ectopic pregnancy

- 1st 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 / Im 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 d/t 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

- multifetal pregnancy = one conceptus at $\textcircled{R}$ site & one at ectopic site
- 1 per 30,000 pregnancies (natural incidence)
- incidence $\uparrow$ to 1 in 7000 d/t ART
- $\rightarrow$ always surgery

- Spontaneous abortion / miscarriage $\rightarrow$ < 20 weeks $\&$ $\leq 500g$
- 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 $\pm$
- fetal cardiac activity +

Inevitable abortion:

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

Incomplete abortion:

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

Mixed 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 disorder
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 → APS syndrome (APS)

APS 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 Abx may cause false +ve VDRL
- m/c seen in pts w recurrent abortions $\rightarrow$ anticardiolipin Ab
- thrombocytopenia $\pm$

OM/m :

- * 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

- 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
- Sherodkar'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 ex
- 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 V 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 :

- abn^o placenta = partly degenerative & partly proliferative changes in young chorionic villi
- maternal lesions arise from fetal & not maternal tissue
- forms small cysts of varying size
- resembles hydatid cyst

Incidence :

- more in asia, hispanics, american indians
- 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 40 unsuccessful pregnancies

No known RF available for partial mole

Pathogenesis of complete mole

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

Partial mole

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

Complete mole - gross

- Grape like clots
- 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
- features 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 away
- In partial mole $\rightarrow$ $\uparrow$ / $\uparrow$

	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 20% in complete & 5% in partial mole
- age > 40 | < 20 yrs
- parity > 3
- serum hCG $> 1,00,000$ mIU/ml
- uterine size > 20 weeks
- previous 4th molar pregnancy
- theca lutein cysts > 6 cm size

m/m :: suction evacuation

• anti D in Rh -ve

• follow up

Suction evacuation ::

- preferred in women who wish to retain^m fertility
- under anaesthesia
- curette done after suction evacuation

- β -hCG every week till -ve



every month for 6 months

- usually becomes -ve by 4-8 weeks

- 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

- $\therefore$ TDC $\rightarrow$ hysterectomy

- invasive mole
- choriocarcinoma
- PSTT
- epitheloid trophoblastic tumor

→ diagnosed by clinical / β HCG / pathological / usg

→ Δ^{th} 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 (eg: 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

Rx

• 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 level > 1,00,000
- hCG fails to become ① 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-mole is choriadenoma destruens (invasive mole)

or choriocarcinoma

* GTD $\underline{\text{E}}$ following a full term pregnancy is always a choriocarcinoma

Invasive mole

- all invasive moles arise from partial or complete moles
- 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 / 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

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

◦ Monozygotic ratio is always constant - 1:250

Disygotic ^{incidence} ~~occure~~ variation is due to :

- ① ↑ FSH d/t → ↑ maternal age, ↑ maternal obesity & multiparity
- ② Family h/o twine in maternal family
- ③ Past h/o twins
- ④ pregnancy d/t ovulation induction by clomiphene citrate or gonadotrophins
- 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 →	◦ ⊕	◦ ⊖

	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 are not effective in twins

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

Interventions are useful:

- limited physical activity
- ↑ ANC
- TV & monitoring of Cx length

Vascular anastomosis

◦ ^{seen} ~~not~~ in monochorionic

◦ 2 types → 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 anaemia polycythemia sequence (TAPS)

• Deep anastomosis :

- present in substance of placenta
- always artery to vein anastomosis
- always unidirectional flow
- extend 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 → oliguric, anemic
- Recipient twin → polycythemia

Donor twin	Recipient twin
• oliguric, 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

⊗ 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 the recipient via umbilical artery to recipient; recipient uses this minimal oxygen in its lower ~~limb~~ 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)
- ~~the~~ donor twin is more prone for CCF in TRAP

occur only in TRAP & not TTT

* 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%)
- I/c → transverse-transverse (in conjoint)

- If 1st twin (t1) is non cephalic → 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 → neg 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
ischio-phagus
cranio-phagus
pyrophagus (back to back)

Management of anomalous ~~birth~~ 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 $\geq 15\%$

Rh isoinmunisationProtocols of Rh-isoimmunisation:

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



- 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



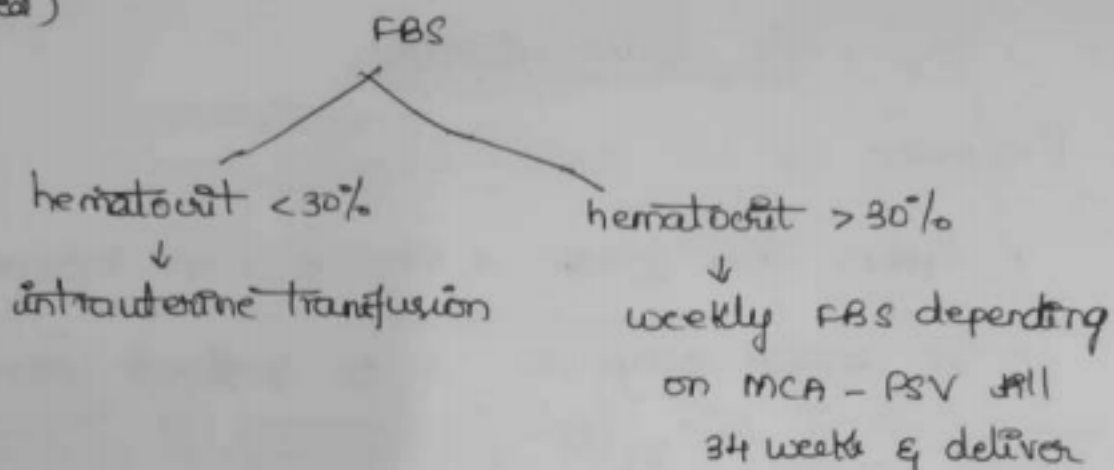
deliver at 37 weeks

> 1.5 mom

- if > 34 wks - deliver immediately
- if < 34 weeks - do fetal blood sampling (FBS)

- no isoinmunisation present
- prevention of isoinmunisation by adm Anti D within 72 hrs of birth of baby or anti D at 28 wks of pregnancy

(continued)



• ~~8~~.

Rh incompetence

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

Anti D Ig = $\begin{cases} \text{monoclonal} \\ \text{polyclonal} \end{cases}$

① polyclonal → • n/c used

- 300 mcg neutralise 15 ml of fetal RBCs
- 50 mcg " 2.5 ml " " "
- t_{1/2} ⇒ 16 to 24 days
- 90% i.m. in deltoid / anterolateral aspect of thigh

◦ foetal anemia

- hydrops (big fetus)
- bilirubinemia

◦ Signs of foetal anemia →

- * polyhydramnios
- * hyperplacentosis
- * hepatosplenomegaly
- * ↑ RA size
- * foetal hydrops

◦ Signs of foetal hydrops →

- * severe anemia,
- * CCF → ascites, PE, pericardial effusion
- * HSM
- * hyperplacentosis
- * still birth

USG → subcutaneous/scalp edema in foetal 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 / Ballantyne syndrome

Intrauterine transfusion:

- when hematocrit $< 35\%$
- O-ve blood, cross matched w mother's blood & tightly packed is transfused to achieve hematocrit of 75%

Routes → • Intra peritoneal → transfused blood absorbed thro' diaphragmatic lymphatics
• Intra vascular
• combined

- Intra vascular route → • 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
- (1) 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 → ^{mf} ASD, VSD, pulm stenosis, COA
 - * cyanotic → ^{mlc} TOF, Eisenmenger's
- RHD - MS (ie m/c), MR, AS, AR
- cardiomyopathy
- IHD
- arrhythmias, syphilitic
- * Regurgitant lesions are better tolerated than stenosis

NYHA functional classification

Gr I $\rightarrow$ no limitation of physical activity

II $\rightarrow$ mild " ; $S_x \leq$ (N) " "

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

IV $\rightarrow$ severe limitation; S_x even at rest

CLARK'S classification of heart disease during pregnancy according to risk :

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

WHO classification

Risk class I $\rightarrow$ no $\uparrow$ ed R/o maternal mortality & no/mild $\uparrow$ in morbidity $\rightarrow$ admission at 36 wks

II $\rightarrow$ small $\uparrow$ risk of mortality or moderate $\uparrow$ R/o morbidity $\rightarrow$ adm at 28 wks

III $\rightarrow$ severely $\uparrow$ ed risk $\rightarrow$ monitor throughout antenatal period

IV $\rightarrow$ extremely $\uparrow$ 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 LVfn
 - 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%
- R/o complications 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
- IE 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
- DOAC → LMWH for 1st 12 weeks
 - * warfarin in 13 - 36 weeks
 - * after 36 wks — LMWH
- stop 24 hrs before delivery & start 12 hrs ~~late~~ after delivery
 - * warfarin resumed 3 days later, it is safe in lactation

- In pts on oral anticoagulants, LMWH 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 & 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 & 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%

⑧ pointer

- R/o cardiac failure → immediate → labour → 28-32 wk postpartum
- Max 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 is preferred
 - FGE 2 gel can be used
- RF for cardiac failure → anaemia, infections, preeclampsia, hypothyroidism

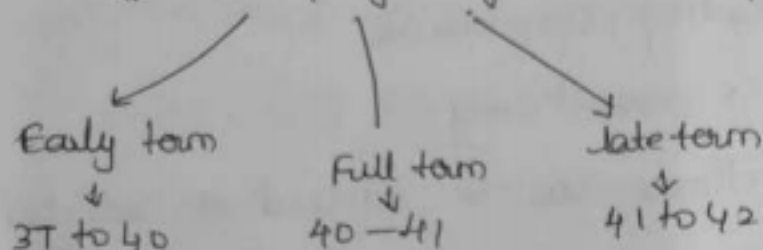


- Anaesthesia of choice $\rightarrow$ general anaesthesia
- Tocolytic of choice $\rightarrow$ atosiban (oxytocin receptor ~~agonist~~ ^{antagonist})
- DCC for arrhythmias $\rightarrow$ metoprolol
- C/I uterotonic $\rightarrow$ methergine / ergometrine
- Contraceptive of choice $\rightarrow$ vasectomy
- IUCDs, POPs & barrier methods can be advised in selected pt
- combined OCPs are C/I

Preterm Labour

- EDD is calculated using Naegele's rule

* Term pregnancy → 37 completed to 42 completed weeks



* 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
- cx length < 2.5 cm in USG & no contractions
- cervical incompetence
- few contractions & no cervical dilatation → threatened preterm

Causes

(PTL)

DINH WILCH PREGNANCY

- D - distended uterus (multiple pregnancy, polyhydramnios)
- I - infections (bacterial vaginosis, GB, PROM, UTI)
- M - maternal (preeclampsia, anemia, 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 (< 2.5 cm, funneling of OA)
- H - H/O preterm labour.

Predictors (FLUSH)

- F - fetal fibronectin ($N < 50$ ng/ml)
- L - length of CX ($N > 2.5$ 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 $\rightarrow$ Y, V, U $\bar{=}$ bulging membranes

Immunological factors

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

Management (SMART)

- S - Steroids - betamethasone is preferred
 - c/e 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 & high level intensive neonatal care unit
- T - tocolytics

Classification of tocolytics

- β_2 adrenergic agonists - terbutaline, metoprolol, isoxsuprine
- MgSO₄
- 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 $\pm$ previous $\pm$ l preterm labour, not useful in active preterm labour

Role of cerclage

- previous $\pm$ l 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 $\pm$ l infections
- sterile speculum examination to demonstrate leak is 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 amoxi & erythro for expectant m/m
- single course of antenatal corticosteroids go to women $\geq$ 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, $\pm$ thin body

II - stage I + fetal distress, meconium present

III - stage I & II $\pm$ 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 placenta)
sulfatase deficiency, anencephaly, fetal adrenal hypoplasia

Physiological change = post term gestation

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

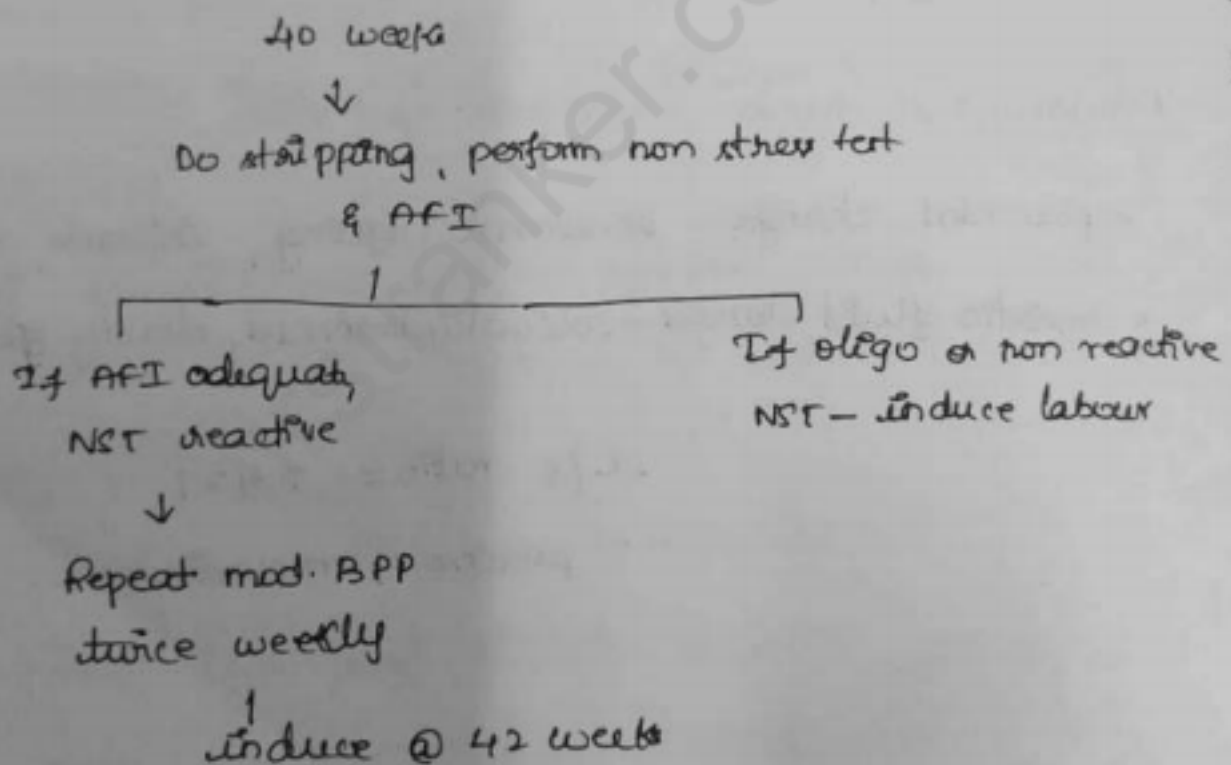
Complications

- 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



Induction of labour

Modified Bishop scoring system

1	0	1	2	3
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- Methods of induction
- o Mechanical dilators →
 - Foley's balloon,
 - laminaria tent
 - osmotic dilators
 - o stripping of membranes
 - amniotomy
 - Pharmacological —
 - PGE₂ gel
 - oxytocin infusion
 - misoprostol (PGE₁ 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

- after recurrent pruritus of pregnancy, cholestatic hepatoma

o CF:

- pruritis: late in pregnancy, no constitutional sx, predilection for soles of palms, skin changes & 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
- (2) liver enzymes at presentation
- slight jaundice ($< 5 \text{ mg/dl}$)
- No BP change / proteinuria
- recurs in subsequent pregnancies & with estrogen containing contraceptives
- $\therefore$ OCPs are c/I in such pt
- o 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 & ~~canaliculi~~ canaliculi of centrilobular regions but without inflammation or necrosis
- disappears after delivery, but often recurs 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 occur in subsequent pregnancies
- characterised by accumulation of microvesicular fat that "crowds out" the 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
- LF - persistent nausea, vomiting, epigastric pain, progressive jaundice (bilirubin 4-10 mg/dl), HTN, proteinuria, edema
- Mod to severe cases manifest by hypofibrinogenemia, hypoalbuminemia, hypcholesterolemia, 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 due to coagulopathy)
- hepatic dysfunction resolves within a week postpartum

→ 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 - ex m/c
- Neonatal disease →
 - localised 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
- Perinatal problems
 - 1st infection causes fetal abn^o d/c transplacental transmission
 - max severity of infection is in 1st trimester
 - max incidence of transmission is in late pregnancy (but ↓ teratogenicity)
- many babies are asymptomatic at birth
- severe disease → triad of hydrocephalus or
microcephaly, 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 syndrome - 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 calcifications
- 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 → anemia & thrombocytopenia
 - ascites & hydrops
- neonatal pneumonia & petechiae or purpuric skin lesions

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

• fb:

- penicillin (preferred)
 - erythra & azithro → curative to mother but not fetus
 - early syphilis → benzathine penicillin single dose
 - >1yr duration → " " ~~given~~ weekly → 3 doses
 - Neurosyphilis? aqueous crystalline penicillin iv
- Xray 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, altered 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 $\uparrow$ fetal malformation rate
- monotherapy is preferred & lowest possible dose
- valproate, phenytoin & phenobarbitone $\rightarrow$ also $\uparrow$ Rlo birth defects
- newer anticonvulsants are better
- M/M $\rightarrow$ targeted USG for women on anticonvulsant dr
- 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 as 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

Knee presentation

Causes of breech presentation

Factors

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

causes

- • multiparity
- placenta high in fundus or low in pelvis
- uterine myomata (fibroids)
- intrauterine synechiae
- ~~and~~ 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 → 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 ischiochancele & sacrum toward iliopectineal eminence
- when the diameter passes thro' pelvic brim, the breech is engaged
- shoulders - engaging diameter is biacromial diameter
- head - engaging diameter is suboccipitofrontal diameter

Pinnard maneuver / frank breech decomposition:

- used to deliver the legs in frank breech

Lovect maneuver:

- used to deliver the shoulder / arm in breech

Burns Marshall maneuver:

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

Mauriceau maneuver:

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

Delivery of breech by forceps:

◦ to deliver head

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

- manual rotation & extraction by P. P. forceps
- modified prague maneuver

* cause of death in breech delivery is

- intracranial hemorrhage
- atlantoaxial dislocation
- asphyxia

◦ Breech presentation & hydrocephalus is managed by

- CSF
- 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

◦ ab(6) uterine contraction

- 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 ischial 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/o 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

- 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
 - " ~~alt~~ 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 postpartum 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 48-72 hrs 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
 - 50-100 ml/day
 - more protein, secretory IgA, more lactose, less fat
- * Contraception E is preferred in lactating mother is POP.

Thromboembolism in pregnancy:

Virchow triad → stasis, local trauma, hypercoagulability

RF

- previous Hb thromboses
- trauma
- anemia
- APH / PPH
- CS foll by prolonged immobility
- hyperemesis
- obesity
- multiparity
- HTN
- stillbirth
- postpartum 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 ① injured during NVD $\rightarrow$ lateral femoral cutaneous ② of thigh
- RF $\rightarrow$ nulliparity, prolonged 2nd stage, prolonged pushing in semi-Fowler position

- Common (N) injured in CS → ilioinguinal & iliofemoral (N)
(especially in femoral hernia)
- Common (N) injured in NVD → common peroneal nerve
(+ foot drop)

Anemia in pregnancy

Physiological anemia of pregnancy:

- plasma vol $\uparrow$ by 40-50%.
- RBC mass $\uparrow$ by 30%.
- $\therefore$ Hb conc. $\downarrow$ by a g/dl d/t hemodilution

Iron requirement in pregnancy

- It is a $\times$ higher

$\therefore$ 1000 mg / pregnancy

- 500 mg for erythropoiesis
- 300 mg for fetus & placenta
- 200 mg shed thru skin, urine & stool

WHO grading

mild 10-11 g/dl

moderate 7-10 "

severe < 7 "

ICMR grading

• mild $\rightarrow$ 10-10.9

• mod $\rightarrow$ 7-9.9

• severe $\rightarrow$ < 7

• very severe $\rightarrow$ < 4

CDC

- Hct 33% & Hb 11g/dl - 1st & 3rd trimester
- Hct 32% & Hb 10.5 g/dl - T2

Causes:

- ^(mle) Deficiency → 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, ↓ ASAT, ↓ 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 2nd 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

ComplicationsIn motherDuring 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, GA, additional RF

Px:

- National Nutritional Anemia Control Programme of India
 - 100 mg iron + 500 mcg folic acid x 100 days
- 12 by 12 initiative
 - aims at 12 g/dl Hb by 12 yr of age

WHO Px

- 60 mg 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

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

Preparation

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

Dose calculation

$$\text{Iron needed} = \text{Hb deficit} \times \text{wt in Kg} \times 2.21 + 1000 \text{ mg}$$

(to replenish the stores)

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

Blood transfusion

- marked / severe anemia in T3
- APTT
- Packed cells in 2nd anemia
- exchange transfusion in cef

M/m during labour1st 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 anaemiasSCA:

- m/c hemoglobinopathy seen in pregnancy
- AR inheritance
- In pregnancy maintain target Hct above 25%
- ↑ R/O thromboses → m/m by thromboprophylaxis, analgesia
- 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 & (N) 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 = deferoxamine after TI is advisable
- concurrent folic acid supplementation

PPH

- Blood loss > 500 ml in NVD & > 1000 ml in ces
- massive PPH > 2000 ml
- a drop in Hct > 10%
- octet shock index - HR / systolic BP
 - * $\odot$ value is 0.5 - 0.7
 - * values > 1 $\rightarrow$ massive hemorrhage

Types

- 1 $^{\circ}$ $\rightarrow$ within 24 hrs, m/c $\rightarrow$ atony
- 2 $^{\circ}$ $\rightarrow$ 24 hrs to 12 weeks, m/c $\rightarrow$ retained placenta

Causes: 4 Ts of PPH

- Tone $\rightarrow$ 70% (over ~~contraction~~ ^{dilatation} of uterus)
- Trauma - 20% (hematoma, inversion, rupture, laceration)
- Tissue (retained placenta $\rightarrow$ 10%
Invasive ")
- Thrombin (coagulopathies) - 1%

Atonic PPH

om/c

• causes → • over distended uterus

• maternal anaemia, malnutrition, multigravida

• labour complication → prolonged 2nd stage, precipitate labour, PPH

Degree of shock

	Degree 1/ compensated	Degree 2/ mild	3 moderate	4 severe
• Blood loss	15% (500ml)	15-30% (1500)	30-40% (2L)	>40% (>2L)
• HR	mild tachy	>100	>120	>140, weak pulse
• RR	Ⓝ	↑	>30	>40
• BP	Ⓝ	DBPT, SBP Ⓝ	SBP < 100 mmHg	SBP < 70 mmHg
• capillary refill	Ⓝ	delayed	delayed	absent
• mental status	Ⓝ	mild anxious	altered	altered conscious, coma
• 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 1st 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} / ^{IV} ~~any~~ every 2-4 hours - 5 doses
→ c/I in HTN, ^{dermo}dermo & Raynaud's
- Carbo prest (PGF_{2α}) → 0.25 mg i.m.
→ c/I in asthma
→ can be repeated upto 8 doses
- Misoprostol (PG E₁) → 800-1000 mcg
→ oral / SL / ^{performed}PR / vaginal
→ A/E → fever, vomiting

Blood product utilization

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

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 in 1st half of pregnancy - 22 weeks (WHO)
24 to 26 (India)

Causes:

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

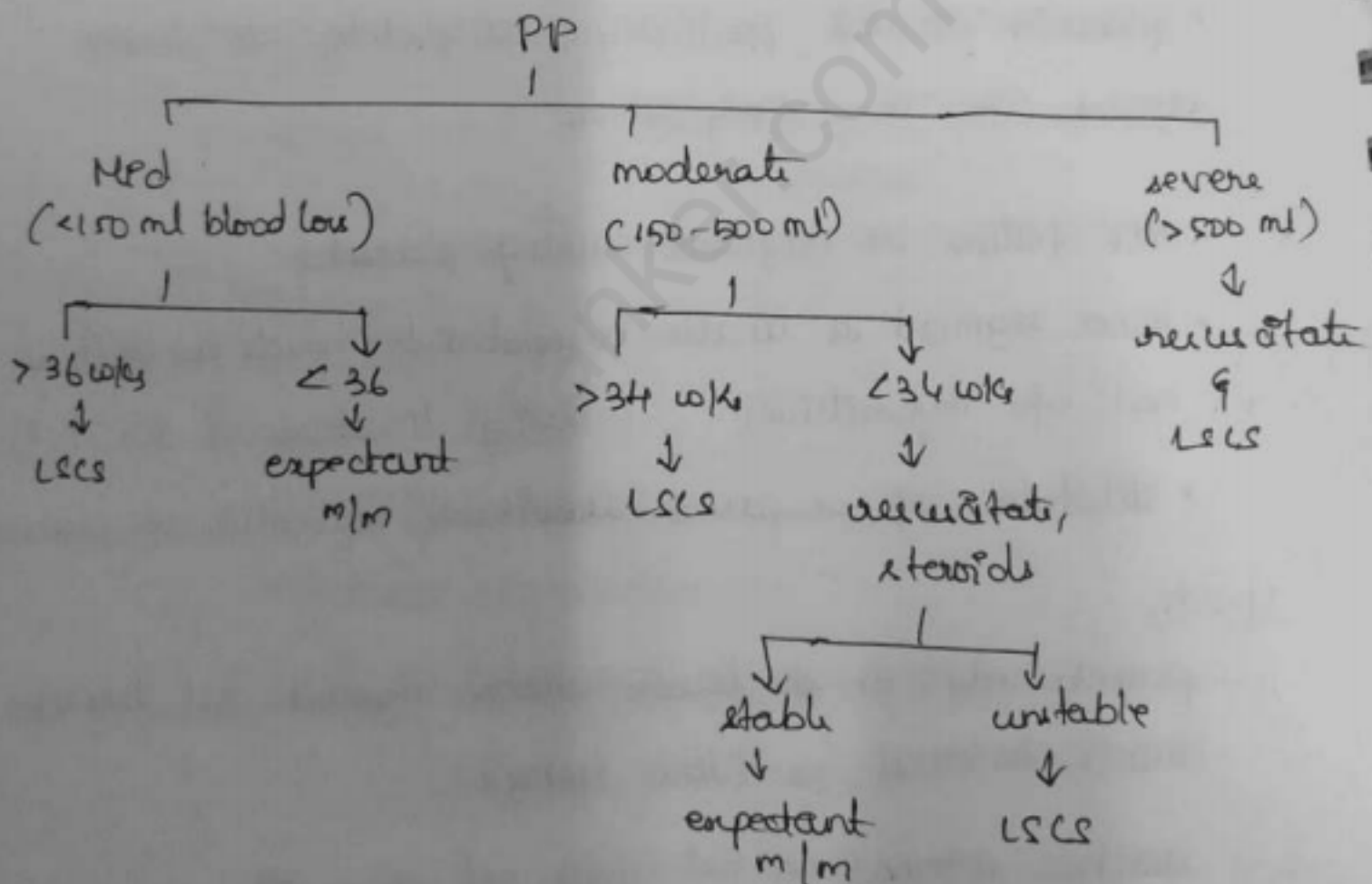
Placenta previa

- placenta located partially or completely in lower segment (in b/w cervix & uterus)
- due to failure of migration ^{of} placenta
- lower segment is unable to construct vessels as much as rest of myometrium (living ligatures of fig of 8)
- dilatation of os causes inevitable separation of placenta

Cause:

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

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
- Stillworthy'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

- warning hemorrhage → seen in T2 or early T3, severe & settles by itself

Abruptio placenta

- hemorrhage d/t accidental separation of a normally situated placenta
- 2 types —
 - concealed hemorrhage (35%)
 - revealed hemorrhage (60%)

Classification

① Shenk'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)

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

III a → without DIC

III b → with DIC

Classification

ages

agnosia purely on pathology, no sy

quiescent form is a live baby, mild bleeding

id form is onset of clotting problems & fetal

distress

severe form is coagulation defects & fetal death

in utero

- same as previa 4

- previous abruption, smoking, cocaine abuse

- 3Ps → polyhydramnios, PROM, preeclampsia

- 2Ts → trauma, thrombophilias

- Ted AFP in T2

of delivery

Abruption

- uterine apoplexy
- rare complication in severe abruption
- d/t vascular damage within placenta $\pm$ causes haemorrhage that progress to & infiltrates the wall of uterus. can be diagnosed only by direct visualisation or biopsy (seepage of blood into myometrium)
- self limiting.

DIC

- most dreaded complication of abruption
- Overt 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 d/t 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
 - $\left\{ \begin{array}{l} \text{inacreta} \rightarrow \text{invade upto cervix, but doesn't breach it} \\ \text{periacreta} \rightarrow \text{breach the cervix \& may involve bladder etc.} \end{array} \right.$
 - placental localization using MRI & plan for hysterectomy
- E Lcs

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 - Senger's alkali denaturation test / Apt test

OH/m - emergency cs

- on staining blood $\bar{=}$ 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
- Thrombophilias - 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) www.FirstRanker.com

www.FirstRanker.com

2) cord prolapse, true knot, cord around neck

3) TTTs

4) placental insufficiency

• Pathogenic → 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 movement
- 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 - earliest diagnosis
- evidence of IUD → lack of fetal movement & cardiac activity for 10 min
- 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 d/t 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 $\odot$ parallelism

Complications

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

• ABO & Rh

• Blood sugar

• Urine ex

• VDRL

• RFT, TFT

• TORCH screening

• Lupus anticoagulant, antinuclear Abs

M/m

• sr. fibrinogen level

• aPTT, when fetus is retained for > 2 wks

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

→ sr. fibrinogen estimation - done weekly

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

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

⊕ ARM in C/I d/t R/o infection

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

Examination of fetus for

- malformation
- 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

- 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 Irradiate 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 passanger → 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 basin of pelvis
- divided into
 - inlet
 - cavity
 - outlet

False pelvis

- above bottom 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 w/ inlet
 - favour occipito posterior
 - difficulty in descent of head d/t long birth canal & flat sacrum interfering w/ 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
- ④ 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 pelvisGynaecoid 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

- 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

Outcomes

- Gynaecoid → ◦ m/c & (N) type
 - diameter of engagement = transverse / oval
 - no difficulty in engagement
 - (C) occipitoanterior (LOA) / (C) 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 ~~sub~~ parieto - subparietal diameter (8.5 cm)

Dystocia dystrophica syndrome

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

- Fetal skull

- Scanned by CamScanner

Diagnosis in obstetricsMaternal 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 ↑ 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 ↓ 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
- Truomis 21 or 18
- GTD
- fetal death
- overestimated gestational age

Screening tests

- T1 → CVS
- T2 → amniocentesis
- ~~T3~~ → cordocentesis → 18 weeks

• In Down's syndr → ↓ AFP & ↑ hCG

• β-hCG is ↑ in Rh incompatibility

• Acetyl cholinesterase in amniotic fluid is present in open neural tube defect

Procedure 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 $\rightarrow$ fetal malformation detected during T2 & T3
 - $\rightarrow$ Rh sensitisation to detect fetal Hct.
- Differentiate b/w fetal blood & maternal blood:
 - fetal blood cells (140 fL) are larger than maternal cells (80 fL)
 - 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 $\rightarrow$ CRL
- In T2 $\rightarrow$ BPD
- T3 $\rightarrow$ 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

- cranial → 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^d 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

• Banana sign $\rightarrow$ compressed cerebellum

• Lemon sign $\rightarrow$ indentation of frontal bone

Anterior abdominal wall defects

① Omphalocele:

• Sac covering the content is very thin consists of 3 layers $\rightarrow$ 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

Down syndromeEtiology

- 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 age
- prior pregnancy w autosomal trisomy (triploidy)
- women 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 α $\rightarrow$ $\uparrow$ in Down

Triple test $\rightarrow$ MSAFP + β hCG + UE3

Quad test $\rightarrow$ MSAFP + β hCG + UE3 + dimeric inhibin α

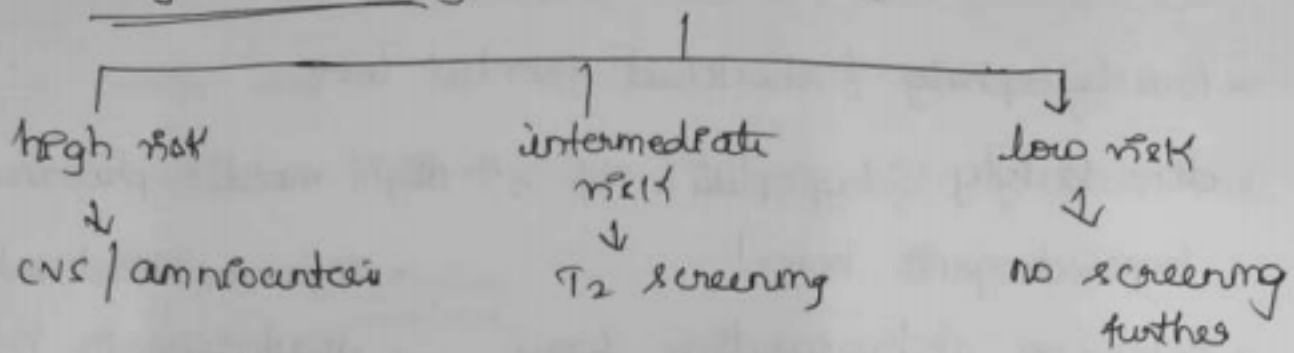
Ab α low UE3 levels

- 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, this 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 or R/o fetal anomaly
 - risk factor for Down

Sonographic markers (soft markers) for Down synd in 12

- 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 syndr.

Confirmatory test:

- only 100% confirmatory test is 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 with ~~parent~~ 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) - $\downarrow$
- HC : AC & FL : AC ratio $\downarrow$
- etiology $\rightarrow$ genetic disease or infection (concurrent to fetus)
- Total cell no. - less
cell size - $\downarrow$
- Neonatal course $\rightarrow$ complicated
 $\rightarrow$ poor prognosis
- less common

Asymmetrical

- head larger than abdomen
- low
- elevated
- chronic placental insufficiency (concurrent to fetus)
- $\downarrow$ smaller
- uncomplicated $\rightarrow$ good prognosis
- more common

* amniotic fluid volume is low in IUGR

* smoking, alcohol, cocaine cause IUGR

* IUGR babies are prone for hypoglycemia

MTP

- o MTP act 1971 → MTP Ex in base of therapeutic, eugenic, humanitarian, social (feature of contraception in married)
- o 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
- o can be done only in government established or approved hospitals
- o consent → consent of woman is sufficient
written consent of guardian in minor or mentally ill
- o pregnancy < 12 wks → opinion of single doctor
12-20 wks → 2 doctors
- o ~~term~~ MTP 41 in > 20 wks

T1 abortion

Medical

- o mifepristone
- o mifepristone & misoprostol (Pg E₁)
- o M+2 & misoprostol
- o Tamoxifen & u

Surgical

- o menstrual regulation
- o vacuum aspiration
- o suction evacuation &/or curettage
- o 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

↓
progestrone antagonist

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

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

(4 tab, 200µg each) after 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 obstetricsECV :

- conversion of non cephalic to cephalic presentation
- done ideally at 37 weeks
- Ix → non cephalic presentation
- 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 LSCS 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 Ix of IPV in modern OBG.
- 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 Cx in 2 & 10 o'clock positions

- used in trapped fetal head, cervical dystocia d/t fibrosed Cx or uterovaginal prolapse

Drugs in pregnancyOxytocin

- 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 diuresis at high dose
 - 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 (fastat) / 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 mg @ 4-6 hrs
- 3) PPH - 800 - 1000 mg PR
- advantages → selective for uterus & has no effect on bronchus (not c/I 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 mg
- 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 →
 - Cx unfavourable → PG E₂, PG E₁ (Bishop score < 6)
 - Cx favourable (Bishop score > 6) → oxytocin & PG E
- augmentation → oxytocin is DOc
- AMTSL → oxytocin 10 U IV or IM

- control of FPH $\rightarrow$ misoprostol, carboprost

Antihypertensives in pregnancy

- 1st line $\rightarrow$ methyl dopa or labetalol
- 2nd line $\rightarrow$ nifedipine
- hypertensive emergencies $\rightarrow$ labetalol, hydralazine, sodium nitroprusside
- ACE are use C/I

α methyl dopa

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

Labetalol:

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

Nifedipine:

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

Anti convulsants

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

 $MgSO_4$

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

Dosage $\rightarrow$ PRITCHARD Regimen

- loading dose $\rightarrow$ 4g slow iv (40% soln) over 3-4 min, foll by 10g (50% soln) deep im gn as 5g in each buttock
- maintenance dose $\rightarrow$ 5g (50% soln) deep im on alternate buttock every 4 hrs only on monitoring
 - $\rightarrow$ Knee jerk, urine output (100 ml in 4 hrs / 30 ml in 1 hr), RR > 14 / min
- C/S $\rightarrow$ myasthenia gravis, oliguria
- Ad/E $\rightarrow$ respiratory depression, muscle weakness
- Therapeutic level of $MgSO_4 \rightarrow$ 4 to 7 mEq per litre
- antidote $\rightarrow$ calcium gluconate (10 ml of 10% slow iv)

• CCB - nifedipine

• $MgSO_4$

• β blockers $\rightarrow$ terbutaline, nitroglycerine, diltiazem

• NSAIDs - indomethacin, sulindac

• 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 (w/ aPTT monitoring)

• Ad/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 & $\downarrow$ Ad/E

• enoxaparin, dalteparin

• dose: 2000 to 4000 units

Warfarin:

• more effective than heparin in preventing TE

• Ad/E $\rightarrow$ warfarin in T1 causes warfarin embryopathy

$\rightarrow$ $\uparrow$ R/O abortion, still birth & fetal bleeding

• BF is not C/I w/ 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 three following NVD & 24 hrs till CS
 - warfarin is started in postnatal period overlapping with heparin
till desired INR is achieved & heparin is stopped
- heparin therapy : aPTT is kept 2 times the control
warfarin " : INR b/w 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 drug, 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, ACE ins, Li, M-tx, misoprostol, danazol, isotretinoin,
radioactive iodine

Drugs & fetal effects

Analgesics

- para - safe
- Indomethacin - premature closure of DA
- Aspirin → premature closure of DA, persistent PFT & Kernicterus in newborn

Anticoagulants

- warfarin → ~~causes~~ conduction 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 → ectopic anomaly, fetal goitre / hypothyroidism, fetal DI
- DES → vaginal clear cell ca, T shaped uterus, vaginal adenosis
- Isotretinoin → B/L 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, abruption, LBW

Abnormal uterine action(A) uterine contractions

- Braxton Hicks contractions → painless, irregular, throughout pregnancy, no Cx changes
- Effective uterine contractions -
 - * start from perimetrium at cervix (i.e. stronger than U), 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 unit
- Tachysystole - >5 contractions in 10 min
- 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 stage 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 locally weak
- 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, FHR good
- 4/m → rule out CPD, malpresentation, malposition
→ labour augmentation ± ARM + oxytocin

Precipitate labour:

- Rapid expulsion of baby d/t hyperactive uterine contraction
- combined duration of stage I & II < 2 hr
- Cx dilatation ≥ 5 cm/hr in multiparous & 1 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 uterine segment
- 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 $\rightarrow$ analgesia, correct dehydration & acidosis
rule out uterine rupture

CS done

Prevention $\rightarrow$ partogram

Spasmic 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 (X)
- usually reversible
- cause → 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 - delivery by CS

• ring usually paves off by deepening the plane of anaesthesia or cut vertically to deliver the baby

• difficulties in II or III stage $\rightarrow$ deep anaesthesia releases the ring

Generalised tonic contraction (tetany)

• contractions involve whole of uterus upto internal os

• no diff b/w upper & LUS

• tonic muscular spasm of uterus & fetus inside

• no uterine rupture

• cause $\rightarrow$ CPD, methergine, injudicious oxytocin

• CF $\rightarrow$ severe continuous pain, tender & tense uterus, fetal distress $\pm$, fetal parts not well defined

• M/m $\rightarrow$ *in hypercontractility induced by oxytocin, oxytocin stopped. Terbutaline gn

* in CPD & fetal distress $\rightarrow$ CS

Cervical dystocia

• Failure of cervical dilatation

• cause $\rightarrow$ inefficient ut. contractions, malpresentation, malposition

• Cr spasm $\begin{cases} 1^\circ - \text{primae, Cr rigid \& fails to dilate} \\ 2^\circ \rightarrow \text{scarring of Cr from previous surgery or d/s} \end{cases}$

• M/m $\Rightarrow$ • malpresentation / position $\rightarrow$ CS

• Thin rim of Cr $\rightarrow$ rim pushed up manually before NVD

• Dehriesenk 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
 - intramembranous 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 $\rightarrow$ 500 cc/hr
 - * replaced every 3hr
 - * volume of AF
 - $\rightarrow$ 12 wk - 50 ml
 - $\rightarrow$ 20 wks - 400 ml
 - $\rightarrow$ 36-38 wks - 1000 ml
 - $\rightarrow$ 40 wks - 800 ml
 - $\rightarrow$ 42 wks - 500 ml
- Colour
 - * near term - straw colour (N)
 - * * greenish yellow - post maturity
 - * green - meconium stained, breech

* golden yellow - RBC compatibility (bilirubin)

* 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 cm**

Polyhydramnios

or
(SVP)

o **liquor > 2000 ml**

o **AFI > 25** / **SDP > 8 cm**

Grades

SVP (cm)

o mild

8-11 (m/c)

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, monozygotic 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
 - a/w TTTS, chorioangioma of placenta
- ② Chronic type → m/c type

Δ:

- FH (fundal ht) > POG, Red 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 interference
- PPH

post partum

- subinvolution

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

Management

◦ Mild → no tx req

◦ Severe → Rule out anomalies

* indomethacin therapy (1.5-3 mg/kg/d)

- stopped at 32 wks

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

- serial amniocentesis

- 500ml/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)
 - * IVUR, 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 $\rightarrow$ need operative interference
- Fetal $\rightarrow$ fetal pulm. hypoplasia
 - * fetal deformities d/t intraamniotic 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
- R 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 contraction
- seen in cord prolapse

Early deceleration

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

Late deceleration:

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

PIH

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

Measurement of BP = sitting / lateral lying down position

- manometer at level of heart
- Korotkoff's sound V is taken as DBP

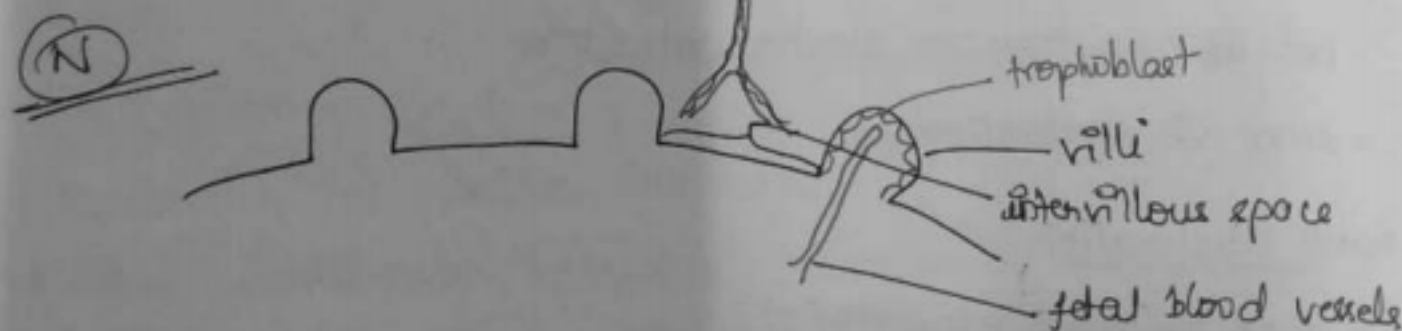
Chronic HT during pregnancy

- $\uparrow$ in BP before 20 wks (info 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

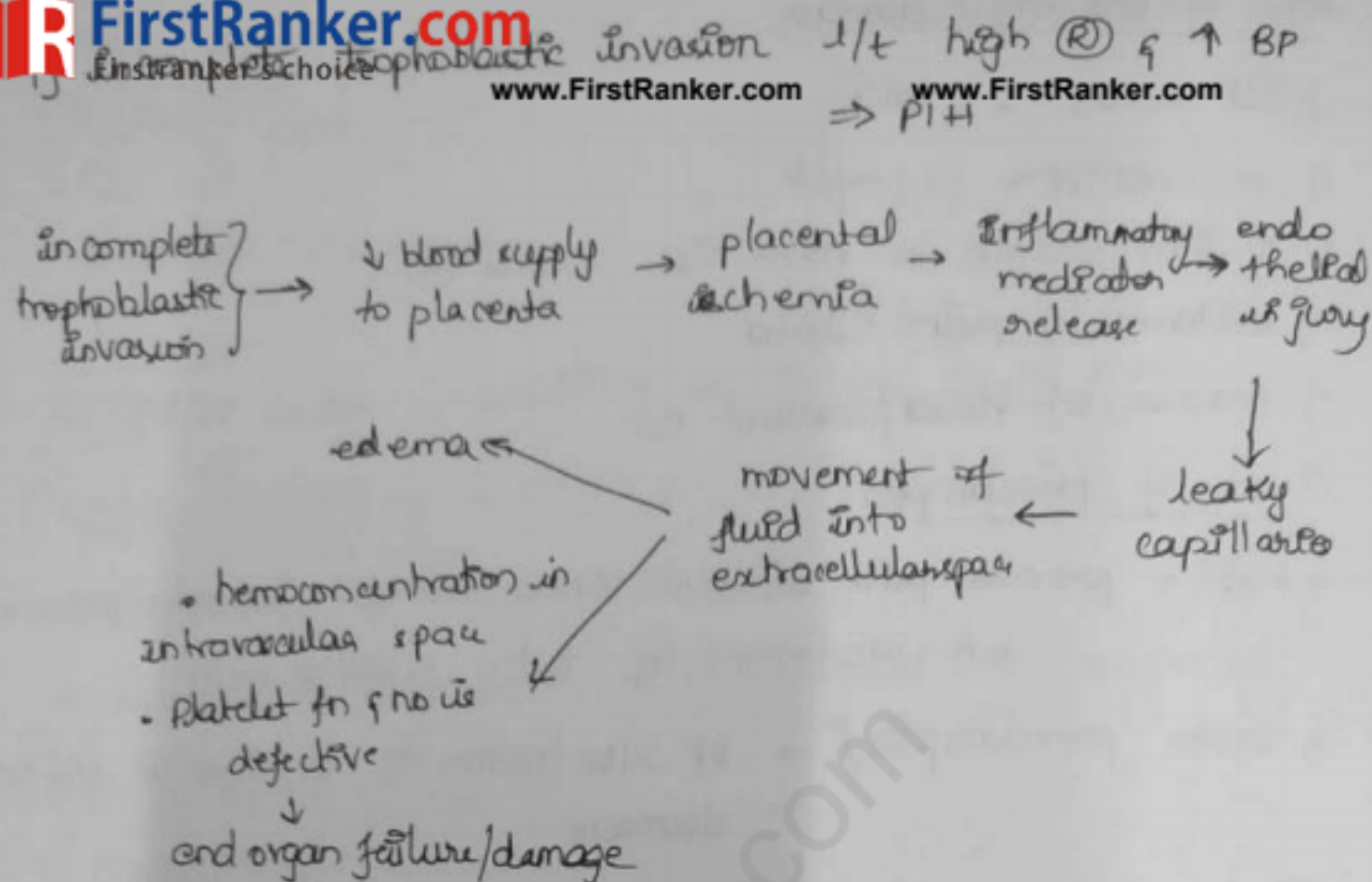
ie trophoblastic invasion

step I $\rightarrow$ 10 to 12 wks

- trophoblast replaces lining of arteries upto decidua

step II $\rightarrow$ 16 to 18 weeks

- replaces up to myometrium



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

- the size of placenta is ↓ in PIH
- ↑ in DM, Rh incompatibility, twin
- m/c hematological finding in PIH → ↓ 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 $> 1.1 \text{ mg/dl}$
- 3) $\uparrow$ liver enzymes to more than 2 times $\text{\textcircled{N}}$
- 4) evidence of pulm edema
- 5) presence of visual/cerebral sx

Grade of preeclampsia

- Mild $\rightarrow$ preeclampsia without severe features / simply preeclampsia
 - 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 into mild & severe

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

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

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

Chronic HTN + superimposed preeclampsia =

• a HT female is

- 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

* primu

* chr. HT

* diabetes

* chr. renal gls

* extremes of maternal age
 $< 18 / > 40$

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

* protective factor $\rightarrow$ smoking as it has IL-2 & TNF

Changes in kidney in PIH:

- * glomerular capillary endotheliosis
- * $\downarrow$ ed GFR $\rightarrow$ oliguria $\rightarrow$ $\uparrow$ sr. creatinine & $\uparrow$ sr. urea acid

Other changes:

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

Eclampsia

- It is severe preeclampsia $\pm$ 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 $\rightarrow$ 1 in 1500-2000
India $\rightarrow$ 1-5%

Signs of impending eclampsia:

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

- It can be d/t occipital lobe edema or retinal detachment
- convulsions is d/t cerebral hypoxia
- eclampsia $\left\{ \begin{array}{l} \text{antepartum} \\ \text{intrapartum} \\ \text{postpartum (within 48 hrs after delivery)} \end{array} \right.$
- m/c is antepartum eclampsia
- antepartum eclampsia has worst prognosis
- m/c MRI finding $\rightarrow$ subcortical white matter edema
- m/c of death $\rightarrow$ intracranial bleeding

HELLP syndrome

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

Criteria for Δ of HELLP $\Rightarrow$ Tennessee criteria

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

Major classification of HELLP syndrome :

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

M/m :

- Immediate termination of pregnancy if mgSO₄ > 34 ^{wks}
- If b/w 27 - 34 wks, give corticosteroid & deliver after 24 hrs of last dose of corticosteroid

Corticosteroid

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

DD 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 $\rightarrow$
 - shows diastolic notch
- It disappears by 24 weeks in $\odot$ pregnancy
- persists after 24 weeks in PIH

Predictors no longer used

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

Basix 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 nitroprusate

- Anti HT of choice in PHT / preeclampsia / chronic HTN / acute HT / hypertensive crisis $\rightarrow$ labetalol
- Target BP in preeclampsia - SBP $\rightarrow$ 130-140 mmHg
DBP $\rightarrow$ 80-90 mmHg
- in chr HT during pregnancy $\rightarrow$ systolic 140-150 mmHg
DBP 90-100 mmHg

Labetalol

- oral dose $\rightarrow$ 100mg bid or tds
- iv $\rightarrow$ 20mg iv bolus $\rightarrow$ if not effective within 10 mins
 $\downarrow$
80mg every 10mins $\leftarrow$ if not effective $\leftarrow$ 40mg
- max dose $\rightarrow$ 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 INH
- losartan
- diltiazem
- diuretics $\rightarrow$ CI only as antihypertensive, but if pt has CHF, diuretic can be given

M/m of mild preeclampsia / gestational HT

- Antihypertensives $\pm$
- Definitive m/m $\rightarrow$ termination pregnancy at 37 weeks
- NVD is preferred

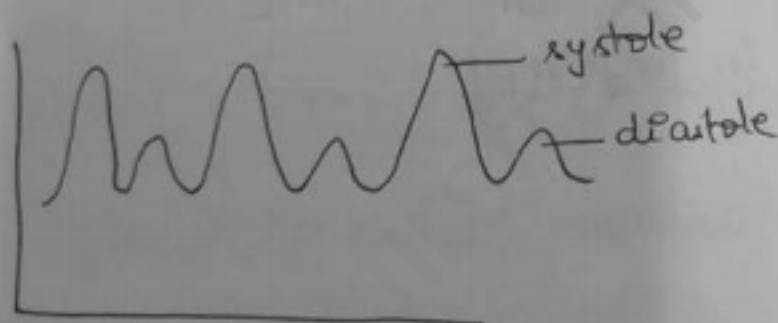
M/m of severe pre eclampsia

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

Ix for immediate termination irrespective of GRA

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

Umbilical artery doppler



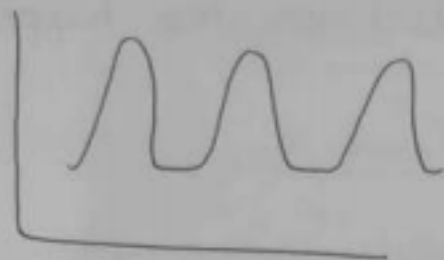
in (N) pregnancy

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

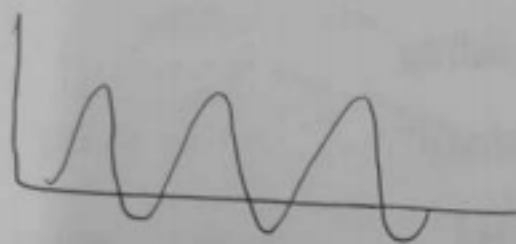


in PIH

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

 $MgSO_4$:

- MDA $\rightarrow$ blocks NMDA receptors in brain, causes cerebral vasodilation, 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 last

$MgSO_4$ 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 K/a Sibal regime

$\rightarrow$ loading dose - 6mg iv

$\rightarrow$ then 2g/hr iv infusion

$\rightarrow$ if convulsion recurs - 2g iv is given again

Uses of $MgSO_4$:

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