Anatomy of female genital tracts

Embryology

	Male	Female
* Urogenital sinus →	• urinary bladder	• urinary bladder, urethra
	• urethra except navicular fossa	• urethral & paraurethral glands (skene glands)
	• Prostate gland	
	• prostatic utricle	• vagina
	• Bulbourethral glands	• Bartholin's / greater vestibular glands

Skene glands:

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

Fourchette:

- It is the area where both labia minora posteriorly meet

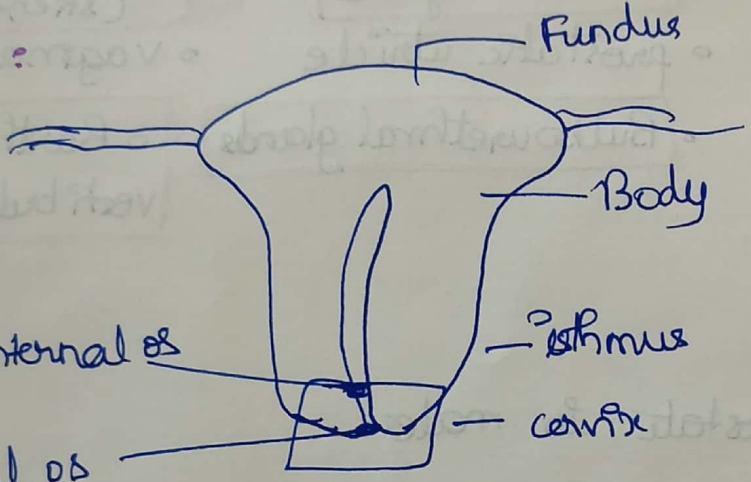
Bartholins gland:

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

e sample for hormonal study is taken from lateral wall of vagina

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

Uterus :



- The lining of cervix is ectated columnar (below anatomi internal os)
- The area bounded by anatomical internal os above & histolog 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

- m/c lymph nodes involved in cancer cervix is obturator lymph nodes

Fallopian tube :

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

Blood supply of uterus :

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

} can be ligated in uncontrollable PPH

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

Perineal anatomy:

- Muscles attached to perineal body are
  - external anal sphincter
  - superficial transverse perinei
  - deep " "
  - bulbospongiosus muscle
  - levator ani

* Ovary is connected to lateral pelvic wall by infundibulopelvic ligament

* Round ligament runs from fundus of uterus to labia majora

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

Vagina

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

* supplied by uterine artery

* lined by stratified sq. epithelium

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

Nabothian follicles → d/t erosion of cervix

Physiological changes in pregnancy

- Non pregnant uterus → 70g, 15 ml, pear/pyriform shaped
- pregnant uterus → 1-1.1Kg, globular - spherical shaped
- Enlargement of uterus is d/t hypertrophy > hyperplasia

Cervix

- cervix becomes soft due to estrogen
- eversion of cervix
- ↑ cervical secretion ~ IgA

- Corpus luteum function maximally by 6-7 weeks of pregnancy
- (N) pH of vagina ⇒ 4.5 to 5.5
  during pregnancy ⇒ 3.5 to 4.5

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

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

Cell count

- Reticulocyte count ↑
- RBC count ↓
- WBC count ↑ (upto 15,000)
- Platelet count ↓ (upto 1 lakh)
- Neutrophils ↑ (neutrophilic leukocytosis)

Iron parameters

- ~~Iron~~ ⇒ ~~Fe~~
- Total requirement of iron during pregnancy → 1000 mg
  in T_1 → 3–4 mg / day
  T_2 & T_3 → 6–7 mg / day

∴
- S. Iron & S. ferritin ↓ in T_2
- S. transferrin ↑
- TIBC ↑
- PSAT ↓

Clotting parameters

- all clotting factors except [11 & 13] increase in pregnancy
  ↓
  N/↓
- maximal ↑ in ~~t~~ factor I (Fibrinogen)
- Fibrinolytic activity is ↓ [d/t ↓TPA & ↑TPA I]
- protein C & S are ↓
- Antithrombin levels are (N).
- BT & CT are (N)

Inflammatory markers

- 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) → ↑↑
- ~~postpartum~~
  - → CO starts ↑ing by 5 weeks & reaches max by 32 weeks
  - → CO during postpartum period (immediate) (70% ↑) > labour (50% ↑) > pregnancy (40% ↑)
  - → CO returns to ~~prepreg~~ prelabour levels by 48 hours & prepregnant levels after peurperium
- Both peripheral & pulmonary vascular resistance ↓↓ during pregnancy (d/t relaxin)
  - ↓
  - ↓↓ diastolic pressure

Pressure changes

- SBP → (N) / ↓
- DBP → ↓↓
- MAP → ↓
- Femoral venous pressure → ↑↑ [(N) → 8mm Hg, pregnancy → 24mm Hg]

Other changes

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

} → water retention & ↑ plasma volume

Respiratory system

- Transverse diameter of chest → ↑ by 2cm
  circumference → 6cm ↑
  } → ↑ TV (tidal volume)
- Diaphragm → moves up by 4cm
  ↓
  → it obliterates RV & ERV space ⇒ ↓ RV & ERV
  → IRV unchanged
  → FRC = RV + ERV → ↓ FRC
  VC (IRV + TV + ERV) → N (unchanged)
  − ↑ ↓
  IC → ↑
  TLC → slightly ↓ or N
- Respiratory rate → unchanged
- minute ventilation → ↑
- max breathing capacity → N
- Respiratory alkalosis ∴ (d/t ↑ min ventilation)

Renal system

- Kidney ↑ by 1cm
- RPF ↑
- GFR ↑ & tubular reabsorption ↑
- urea clearance
  uric acid "
  creatinine "
  } ↑ ⇒ ↓ serum levels

- (R) hydronephrosis may be seen
- all solutes ↓ → plasma osmolality ↓

Endocrine → Pituitary

- ↑ed → GH, prolactin, ACTH, CRH, size of gland
- ↓ed → FSH, LH
- unchanged → TSH

- prolactin (N) – 25 mg/ml
  pregnancy – 150 mg/ml

Thyroid gland

- Total T_3, T_4 → ↑
- Free T_3, T_4 → (N)
- TBG – ↑
- fetal thyroid gland starts taking iodine by 11 weeks
- fetus starts releasing thyroxine by 20 weeks
- I_2 req → ↑
- size of thyroid gland → ↑
- TSH → slight fall (trimester dependant)
- HCG < α → resembles TSH, FSH, LH
  β
- BMR ↑ by 25%

Adrenal

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

Stages of labour ...

1st stage of labour :

- Begins ~ true labor pain & ends ~ full dilatation of cervix

True labour pain	False pain
↑ intensity	—
regular	Irregular
Interval shortens	—
not relieved by enema, sedation	relieved
↓ cx effacement, show & bag of membr.	not associated

* Full cervical dilatation → 10 cm
* m/c presentation → cephalic
  " attitude → flexion
  " part → vertex

m/c diameter → suboccipito bregmatic in AP direction & biparietal is transverse (both are 9.5 cm)

∴ req 10 cm dilatation

2 stage of labour

- full dilatation of Cx to delivery of fetus

3rd stage → delivery of fetus to delivery of placenta

4th stage → 1 hr observation after placental delivery

1st stage of labour

latent → upto 4 cm dilatation of Cx

active → after 4 cm 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 4 cm
- Total duration → 12 hrs in primi & 6 in multi

2nd stage

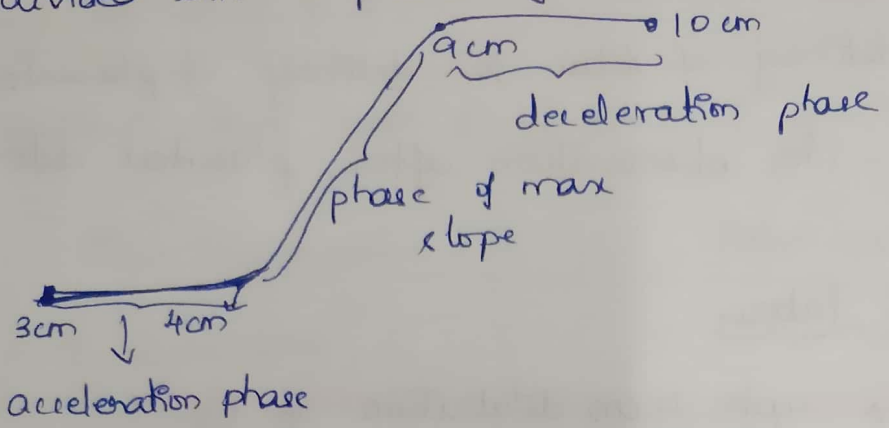
- 2 hrs in primi & 1 hr in multi

3rd stage

- 5 mins (due to active management)

Active phase of 1st stage

- divided into 3 phases by Friedman's curve



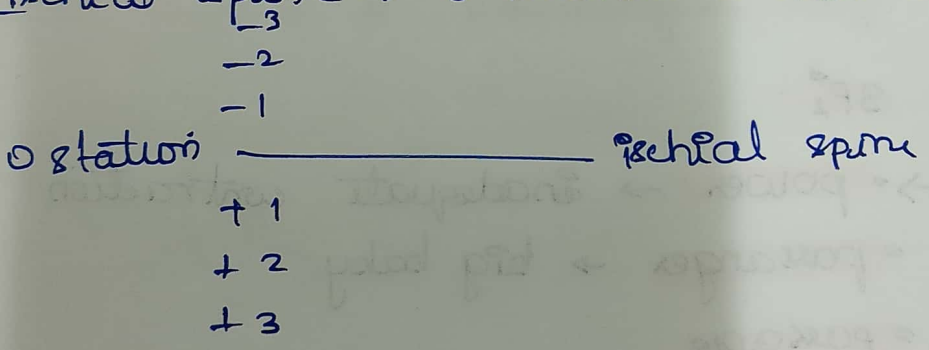
Partogram

- New partogram doesn't include latent phase
- **Components :**
  - * **fetal heart rate** → assessed every 15 min in 1st stage & every 5 min in 2nd stage
    - (N) → 110–160
    - < 100 → brady
    - > 180 → tachy
  - * **liquor** →
    - I – Intact
    - C – clear
    - M – meconium
    - B – blood stained liquor
  - * **moulding** → indicates severity of cephalopelvic disproportion
    - 1+ → obliteration of parietal bones, but no overriding
    - 2+ → reducible overriding
    - 3+ → irreducible overriding

- **dilatation of Cx**
  - → time in X axis
  - → dilatation in Y axis
  - → dilatation is marked by the alphabet 'X'

- **descent of head** :→ marked by '0'
  - → assessed by crichton method (by PV)
  - → scores 5/5 to 0/5

* PV is done only at 4hrs interval unless otherwise needed.
* action line is 4hrs after alert line
* crichton scores : 1/5 & 0/5 → engaged head & 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 < 20 s ;  → 2 contractions each < 20 s

 → 1 contraction 20 – 40 s

 → 1 contraction > 40 s

- oxytocin infusion / min
  → can be ↑ed upto 32 ImU / 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 Cx dilatation

→ causes – 3P's

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

Definite signs of separation of placenta:

- Kustner sign → cord doesn't recede 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/c is oxytocin 10 U I.m]
- ~~generic~~ controlled cord traction
- uterine massage

Pressure of uterine contraction

1st stage → 50 mm Hg

2nd stage → 100 mm Hg

Non pregnant & early pregnancy → 2 to 3 mm Hg

- Best test for adequacy of pelvis → trial of labour
  safest method for pelvimetry → MRI

Diabetes complicating pregnancy

Overt diabetes : F ≥ 126 mg/dl

PP/RBS ≥ 200 mg/dl

HbA1C ≥ 6.5 %.

Gestational DM :

- American guidelines :
  - selective screening
    - low risk → (N) BMI, no family/past history
    - intermediate
    - high → ~~BMI > 30, family history, past history,~~
  - 24 - 28 wks
  - 2 step approach

High risk → BMI > 30, family H/o DM, past H/o DM, previous macrosomic baby > 4.5 kg, Bad obst. history, recurrent abortions, polyhydramnios / IUD

O Sullivan test : 50g glucose —1 hr→ < 140 mg/dl (N)

If ≥ 140 mg/dl ↓

100g GTT

- done ⊂ 8 to 14 hrs fasting
- 3 day ~~prior~~ to test — (N) meals
- unrestricted physical activity
- 100g glucose gn & 4 samples taken

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

→ Carpenter & Coustan criteria

2) WHO – no screening test, directly diagnostic test

- after fasting → 75g glucose

F – 95 mg/dl
1 hr – 180 mg/dl
2 hr – 155 "

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

– 75g glucose after fasting

F – 92 mg/dl
1 hr – 180 "
2 hr – 153 "

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

* 100% screening at booking visit
* no fasting.

– 75g of glucose & sample after 2 hours

< 120	120 – 140	140 – 200	> 200
(N)	• Impaired glucose tolerance	• gestational DM	• Overt DM

If glucose is btw → GDM
↓
do HbA1C to see if it is overt

The complications are more in overt diabetes than GDM

- Anomaly rate in (N) person → 2.5%
- anomaly rate in a person ∈ HbA1C 6-6.9% → 5%
  7-7.9% → 11%
  ≥ 8% → 15%
  ≥ 10% → 20%

- Human placental lactogen (HPL)
  cortisol
  estrogen
  progesterone
  } all contribute to ↑ insulin resistance
  ↓
  GDM

- HPL acts like GH
  ↓
  - synthesised from syncytiotrophoblast
  - lipolytic → ↑ FFA

∴ in pregnancy → fasting hypoglycemia & post prandial hyperglycemia

Complications

1st trimester

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

2nd Trimester

- anomalies
- macrosomia → CPD / big placenta → placenta previa
- polyhydramnios ↔ overdistension → apruptio placenta
  → malpresentation
- cord presentation & cord prolapse
- still births
- IUD
- IUGR
- preterm labour

During labour:

- prolonged labour
- shoulder dystocia
- PPH < traumatic / atonic
- instrumental delivery
- LSCS
- still birth
- ketoacidosis

Postpartum

- subinvolution
- infection → endometritis, peurperal 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

maternal hyperglycemia → fetal hyperglycemia → hyperplasia of fetal β pancreatic cells → ↑ insulin

↓

macrosomia ← IGF

Treatment of diabetes in pregnancy:

- 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 ureter

→ 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 – 1U / kg / d in divided doses

Screening for anomalies (Antepartum surveillance)

- 1st trimester screening — at 11-14 wks
  - i) maternal age
  - ii) nuchal translucency
  - iii) β HCG
  - iv) PAPP-A

  If +ve, then do chorionic villous screening

  If not done, then do

- 2nd trimester screening → only 60-70% sensitivity
  - * triple test → β HCG, unconjugated E_3 (estriol), maternal serum AFP (16-18 weeks)
    or
  - * quadruple test → triple test + inhibin

- Targeted anomaly scan
  - * 18 to 20 weeks
- fetal echo — at 24 weeks

* In case of overt diabetes, admit the pt at 34 wks & do i) fetal kick count
  ii) NST / biophysical profile
  iii) obs. scan - AFI & estimated fetal weight

& terminate pregnancy at 38 weeks

GDM
↓
meal plan → continue pregnancy till 40 weeks
insulin → terminate pregnancy at 38 weeks

Intrapartum

- stop morning dose of insulin
- maintain blood sugar at 70–100 mg/dl
  - If <70 → dextrose
  - >100 → infusion regular insulin
- hourly CBG

* target calorie req/day → 30–35 Kcal/day
  - If BMI > 30 → 25 Kcal/day

Ectopic pregnancy

- m/c location → ampulla of FT (70%)
- Risk factors
  - abnormal fallopian tube
  - Tubectomy
  - prior ectopic pregnancy
  - salpingitis & other infection (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.

- Passage of decidual cast
  - ↓
  - cast mirrors the shape of endometrial cavity

Outcomes in ectopic pregnancy

- Tubal rupture
- " abortion
- pregnancy failure i.e. resolution

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

Diagnosis

- β hCG < Qualitative (~~urine~~ UPT)
  - Quantitative
- Transvaginal USG (TV USG)
- laparoscopy
- laparotomy

+ UPT & abd. pain / vaginal bleeding

Stable pt
↓
TV-USG → non diagnostic → serum β hCG
↓
℞ accordingly

Unstable pt
↓
surgical ℞

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

Serum progesterone levels

- (N) pregnancy → ≥ 25 ng/ml
- values < 5 ng/ml → nonliving uterine / ectopic pregnancy

TVS

- Endometrial findings → pseudogestational sac
  - ↓
  - * 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 ↑ in size of sac

True gestation sac → ◦ eccentric location in uterus
◦ round & regular outline
◦ double ring sign d/t chorion +
◦ yolk sac & fetal pole +
◦ sac ↑ in size 1mm/day

◦ Adneral findings:
- colour doppler TVS → ring of fire / interstitial ring
reflects placental blood flow around periphery of pregnancy

Other modalities
◦ Laparoscopy
◦ D&C → chorionic villi float in (N) saline as lacy fonds, decidua without villi structure is present
◦ laparotomy
◦ culdocentesis → * used to identify hemoperitoneum
* 18G needle inserted into retrouterine pouch → non clotting blood + in ectopic pregnancy

Management - Medical
◦ Methotrexate ± or without folinic acid rescue
↳ only one used now
- previously used → KCl, PGF2α, hyperosmolar glucose, actinomycin D

Case selection for medical m/m

→ hemodynamically stable pt
→ serum βhCG < 3000 IU/L (not absolute req) [upto 10,000 is OK]
→ tubal diameter < 4cm without fetal cardiac activity
→ no acute intra abdominal bleeding

- level of βhCG is single best predictor of Rx success

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

- Failure rate is 5 – 10%

Surgical m/m

* Done in
  - heterotropic pregnancy
  - unstable pt
* c/I in pt who can be treated medically

① Conservative surgery
- linear salpingostomy (done now)
- salpingotomy (not done now)
- segmental resection ± or without anastomosis

② Radical surgery
- total salpingectomy

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

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

Dose of anti-D
- T1 abortion / MTP / ectopic → 50 mcg
- 2nd trimester onwards → 300 mcg
- within 72 hrs after delivery → 300 mcg

Rare types of ectopic pregnancy

- 1° 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 infection of Mtx / in Mtx
- ruptured → salpingo-oophorectomy

Abdominal pregnancy

* Studiford criteria: - for 1° abdominal pregnancy
  - Both tubes & ovaries are (N)
  - absence of uteroplacental fistula
  - pregnancy exclusively related to peritoneal surface
* 2° abdominal pregnancy
  - more common
  - 1° 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

Heterotopic pregnancy

- multifetal pregnancy ~ one conceptus at (N) site & one at ectopic site
- 1 per 30,000 pregnancies (natural incidence)
- incidence ↑ed to 1 in 7000 d/t ART
- Rx → always surgery

ABORTION

- Spontaneous abortion / miscarriage → < 20 weeks or ≤ 500g
- About 50-70% of spontaneous conceptions are lost
- Most clinical pregnancy loses occur before 8 week

Causes

- Numerical chr. abⓃ → 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 c/w advanced maternal age

polyploidy

- tri / tetraploidy

sex chromosome polysomy

eg:- 47 XXY, 47 XYY

Monosomy X - single m/c chrosomal abⓃ causing spontaneous

Classification of abortion

① Spontaneous
- a) threatened
- b) inevitable
- c) incomplete
- d) complete
- e) missed

② Induced < medical / surgical

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

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

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

Missed abortion

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

Complete abortion

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

Septic abortion:

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

Recurrent abortion:

- Sequence of ≥3 consecutive spontaneous abortion before 20 weeks
  - * 1° → no successful pregnancy
  - * 2° → loses following a viable birth
- cause – (m/c is) - chromosomal abN
  - uterine factors
  - cervical incompetence
  - endocrine problems
  - immunological problems
  - inherited thrombophilias
  - systemic disorder like military TB

o TORCH infections cause sporadic & not recurrent abortions

Parental chromosomal abnormality

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

Immunological factor

- Alloimmune factor
- Autoimmune factors → APLA syndrome (APS)

APLA syndrome

- miscarriage after 10 weeks
- treatable
- Diagnostic criteria [Revised Sapporo criteria]
  - * clinical criteria
    - → vascular thrombosis
    - → pregnancy morbidity
  - * Lab criteria (must be +ve atleast on 2 occasions 12 weeks apart)
    - → presence of lupus anticoagulant
    - → IgG or IgM anticardiolipin Abs
    - → anti β2 glycoprotein-1 IgG or IgM Ab

- APS cause thrombosis & not hemorrhage

[DRVVT – dilute russell viper venom test]

- anti β2 GP Abs may cause false +ve VDRL
- m/c seen in pts ~ recurrent abortion → anticardiolipin Ab
- thrombocytopenia ±
- M/m :
  - * women ~ no prior thrombosis
    - low dose aspirin (81 mg)
    - UFH / LMWH

when pregnancy is detected & continued till 6 wks post partum

- * women ~ prior thrombosis → maintanence therapy ~ warfarin

& when pregnancy is confirmed, switch over to aspirin

- CI to cerclage
  - active preterm labour
  - evidence of chorioamnionitis
  - vaginal bleeding
  - PPROM
  - evidence of fetal compromise / death
- funneling is seen in cervical incompetence in USG
- diameter of internal os in cervical incompetence is >1cm
- (N) length of cervix → 4 cm
  in cervical incompetence → <2.5 cm
- cerclage done based on history of ≥2 previous abortions / USG / ↓ Cx length

Diagnosis in pregnancy

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

Diagnosis in non pregnant state

- passage of a No. 8 Hegar's dilator without resistance & pain
- cervicogram – funnel shaped cx
- intracervical balloon studies
- traction studies – 600gm 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

  * tx by low dose aspirin & heparin in pregnancy
- Infections → screen for bacterial vaginosis in pregnancy, VDRL testing

Gestational Trophoblastic diseases

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 } malignant
iii) epithelioid trophoblastic tumor

Hydatidiform mole :

- abN placenta ī partly degenerative & partly proliferative changes in young chorionic villi
- maternal lesions arise from fetal & not maternal tissue
- forms small cysts of varying sizes
- resembles hydatid cyst

Incidence :

- more in asians, hispanics, american Indians
- highest in philippines (1 in 80 pregnancies)
- In India → 1 in 400 pregnancies
- low intake of vit A (carotene) a/w ↑ R/o complete mole

RF for complete moles

- previous GTD
- maternal age upper > lower extremities
- older paternal age
- combined OCPs
- prior #o 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 cluster
- No fetal tissue / embryo / amniotic sac
- areas of hemorrhage present

partial mole – gross

- affection of chorionic ville 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 βHCG)
  - 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 βhCG

- In complete mole → very much elevated
- False -ve UPT possible d/t overcaturation of test away
- In partial mole → Tod / N

	Complete mole	Partial mole
• Trophoblast hyperplasia	diffuse	focal
• Karyotype	46 XX / 46 XY — paternal origin	• 69 XXY / 69 XXX triploid
• hydropic degeneration of villi	diffuse	focal
Immunostaining of $P57^{KIP2}$	-ve	+ve
typical Δ	molar pregnancy	missed abortion

Theca lutein cysts

- complete mole d/t theca lutein cysts in 25-60% of women
  - B/L
- d/t overstimulation of lutein elements by ↑ βhCG
- contained fluid is rich in chorionic gonadotropin, also contains estrogen & progesterone

- regress within 2 months (spontaneously)
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

- ~~old 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 H/o molar pregnancy
- theca lutein cysts > 6cm size

m/m : - suction evacuation
- anti D in Rh -ve
- follow up

Suction evacuation :

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

Follow up

- β-hCG every week till -ve
  ↓
  every month for 6 months
- usually becomes -ve by 4-8 wks
- pregnancy c/I till hCG levels become -ve
- contraceptives ∠ can be used. ie 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
  ↓
  ∴ βhCG levels are not much elevated
- composed mainly by cytotrophoblastic cells
- resistant to chemotherapy
- ∴ TOC → hysterectomy

GTNs :-

- invasive mole
- choriocarcinoma
- PSTT
- epithelioid trophoblastic tumor

→ diagnosed by clinical / βHCG / pathological / USG

→ Dx features :

- continued vaginal bleeding
- hCG titres plateau / rise / remain detectable for ≥6 months - hs
- 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 + folinic acid or actinomycin D
  - If (R) → 2nd line drugs
- **high risk GTN or stage 4** (stage 2 & 3 & score ≥7)

- Regular chemotherapy

- age > 35 yrs
- initial hCG level > 1,00,000
- hCG fails to become (N) by 7-9 weeks time or there is re-elevation
- histologically ~~s~~ as invasive mole
- evidence of mets
- previous Ho molar pregnancy
- ~~woman~~ women who is unreliable for follow up

* m/c GTD following H. mole is choriadenoma destruens (invasive mole)
or choriocarcinoma

* GTD ~~is~~ follows a full term pregnancy is always a choriocarcinoma

Invasive mole

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

- Orders of mets of choriocarcinoma is lung (80%) > vagina > pelvis > liver > brain

Multifetal gestations

Hellen's rule:

- Gives incidence of multifetal gestation in India
  - → Twins = 1 : 80
  - Triplets = $1 : 80^2$

Mono & dizygotic

Dizygotic → from fertilisation of 2 ova by 2 sperms

Monozygotic → from splitting of single fertilised ovum

Chorionicity:

- Type of placentation
- Dichorionicity / dizygosity → each fetus has its own placenta
  →
- Monozygotic → [illegible]
  - splitting by < 3 days → dichorionic diamnionic
    4–8 days → monochorionic diamnionic
    > 8 days → monochorionic monoamnionic
    > 13 days → conjoint twins

- monochorionic diamnionic is the m/c type of monozygotic twin
- Monozygotic ratio is always constant – 1:250

Dizygotic ~~incidence~~ variation is due to :

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

	Monozygotic	Dizygotic
• phenotype →	• Identical twins	• non identical
• genotype →	• Identical	• non identical
• gender →	• concordant (same) sex	• dyscordant sex
• perinatal outcome →	• worst	• good
• ethnic variation →	• ④	• ⑤

	Monochorionic	Dichorionic
I trimester		
◦ no of sac	1	2
◦ dividing membrane thickness	< 2 mm	> 2mm
II trimester		
◦ 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° PPH
- thromboembolism
- subinvolution
- ~~lactational failure~~

Interventions ↓ are not effective in twins

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

Interventions ↓ are useful:

- limited physical activity
- ↑ ANC
- TVS monitoring of Cx length

Vascular anastomosis

- seen 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 btw the twins)

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

→ disturbance in bidirectional flow leads to discordancy in

growth - twin to twin transfusion syndrome (TTTS)

amniotic fluid - Twin oligo polyhydramnios sequence (TOPS)

Hb diff → Twin anemia polycythemia sequence (TAPS)

- Deep anastomosis :

- present in substance of placenta
- always artery to vein anastomosis
- always unidirectional flow
- extent throughout capillary bed of villous structu

leading to 3rd circulation at 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 & recepient twin
- Donor twin → oligemic, anemic
- Recepient twin → polycythemia

Donor twin	Recepient twin
◦ oliguria, anemia	◦ polyuria, polycythemia
◦ oligohydramnios	◦ polyhydramnios
◦ ↓ Renal blood flow → activation of RAAS	◦ activation of ANP
◦ non visible bladder	◦ overdistended bladder
◦ IUGR	◦ circulatory overload – prone for hyperviscosity syndrome, HTN, occlusive thrombosis, hyperbilirubinemia, CCF
	◦ 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 foeticide

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 recepient artery & later also recepient vein to donor vein]

- deoxygenated blood from donor passes through umbilical artery to recipient; recipient uses this minimal oxygen in its lower part of body & further deoxygenated blood passes to upper part of body leading to poor development of upper body
  - → failure of head growth (acardiac acephalus)
  - → partially developed head & identifiable limbs (acardiac myelocephalus)
  - → no identifiable structure (acardiac amorphus)

  } occurs only in TRAP & not TTTF
- donor twin is more prone for CCF in TRAP

* TOPS & TAPS are subset of TTTS but TRAP is not

Delivery of twin:

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

- If 1st twin (t1) is non cephalic → always LSCS
- If t1 is cephalic → go for vaginal delivery
- After delivery of t1, see for lie of t2 by abdominal / vaginal examination & reconfirm ± 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 PV
    - If membranes absent, do LSCS
    - If membranes present, do IPV & go for NVD or breech extraction

assisted breech → spontaneous delivery till umbilicus & then intervention; no GA required

Breech extraction → req GA or minimal tocolytic like mag sulph.

Indications for LSCS in twins

- 1st non cephalic
- monochorionic monoamniotic → always CS at 32-34 wks
  - → absolute Ix
- TTTS
- conjoint twins at term
- severe IUGR ± ab (N) doppler

Conjoint twins :

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

Management of anomalous fetus in twin pregnancy

- If dichorionic
  → inject intrathoracic KCl
  ↓
  anomalous fetus will die due to cardiac asystole

- If monochorionic
  → bipolar cord coagulation of anomalous twin

Parameters in discordance :

- weight ≥ 25%
- BPD ≥ 6mm
- head circumference ≥ 5%
- abdominal " ≥ 20mm
- systolic to diastolic (SD) ratio ≥ 15%

Rh isoimmunisation

Protocols of Rh- isoImmunisation :

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

ICT

-ve

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

+ve

- assess titres

< 1:16

- 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 (multiples of median)

< 1.5 mom

↓

deliver at 37 weeks

> 1.5 mom

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

(Continued)

FBS

hematocrit < 30% → intrauterine transfusion

hematocrit > 30% → weekly FBS depending on MCA – PSV till 34 weeks & deliver

Rh incompetence

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

Anti D Ig: < monoclonal / polyclonal

① polyclonal → . m/c used
- 300 mcg neutralise 15 ml of fetal RBCs
- 50 mcg " 2.5 ml " " "
- $t_{1/2}$ ⇒ 18 to 24 days
- gn i.m in deltoid / anterolateral aspect of thigh

Effects of isoimmunisation

- Foetal anemia
  - hydrops (big fetus)
  - bilirubinemia

- Signs of fetal anemia → * polyhydramnios
  * hyperplacentosis
  * hepatosplenomegaly
  * ↑ RA size
  * fetal hydrops
- Signs of fetal hydrops → * severe anemia,
  * CCF → ascites, PE, pericardial effusion
  * HSM
  * hyperplacentosis
  * still birth

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

- D/t hyperplacentosis → preeclampsia in mother
  ↓
  edema in mother
  ∴ both mother & baby are big
  → Maternal mirror syndrome / Ballantine syndrome

Intrauterine transfusion:

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

Routes → ∘ intraperitoneal → transfused blood absorbed thro diaphragmatic lymphatics
∘ intra vascular
∘ combined

- Intravascular route → ∘ preferred route
∘ intrahepatic portion of umbilical vein & umbilical vein at cord insertion into placenta are used

Cardiac disease in pregnancy

Normal physiology

- ↑ in HR, plasma vol, CO, stroke vol, MAP
- ↓ in DBP, systemic vascular ®

(N) CVS changes

- prominent S1
- S3
- systolic murmurs upto grade 3
- (L) axis deviation in ECG
- up & out shift of apex

∴ seems like slight cardiomegaly in CXR

ab(N)

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

Classification of heart disease based on etiology

- congenital
  - * non cyanotic → ASD (mc), VSD, pulm stenosis, COA
  - * cyanotic → TOF (mc), eisenmenger's
- RHD – MS (is mc), MR, AS, AR
- cardiomyopathy
- IHD
- arrythmias, syphilitic

* Regurgitant lesions are better tolerated than stenosis

NYHA functional classification

Gr I → no limitation of physical activity

II → mild " ; Sx ∓ Ⓝ " "

III → marked " ; Sx ∓ less than Ⓝ activity, but comfortable at rest

IV → severe limitation; Sx even at rest

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

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

WHO classification

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

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

III → severely ↑ed risk → monitor throughout antenatal period

IV → extremely ↑ed risk & needs expert management

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

- Class IV includes → pulmonary HT, (pregnancy C/I)
  - severe ventricular dysfn (LVEF < 30%, NYHA 3 & 4)
  - previous peripartum cardiomyopathy ± residual impairment of LV fn
  - severe MS, severe symptomatic AS
  - Marfan syndr. ± aorta dilated > 45 mm
  - aortic dilatation > 50mm in aortic d/s 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 arrythmia, cardiac failure
- E – EF < 40%

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

M | m

- Admission → WHO class I at 36 wks, II at 28 weeks,
  III & IV – preferable to stay in hospital throughout antenatal period
- class IV pt not allowed for NVD
- 1st & 2nd stage of labour → bed rest, 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

- Ix in mech. prosthetic valve, prosthetic devices, arrythmias
- DOC → * LMWH for 1st 12 weeks
  * warfarin in 13 – 36 weeks
  * after 36 wks – LMWH

stop 24 hrs before delivery & start 12 hrs ~~hrs~~ after delivery

* warfarin resumed 3 days later, it is safe in lactation

- In pts on oral anticoagulants, LSCS is preferred to reduce R/o intracranial hemorrhage in fetus.
- adm FFP & vit K to newborn

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

IE

- Rx for Px are prosthetic valve, unrepaired cyanotic congenital heart d/s, completely repaired CHD ± prosthetic devices during 1st 6 months after surgery, cardiac transplant recepients

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

- ampi 2g or xone 1g iv + genta 80 mg 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 dysfn 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%

(X) points

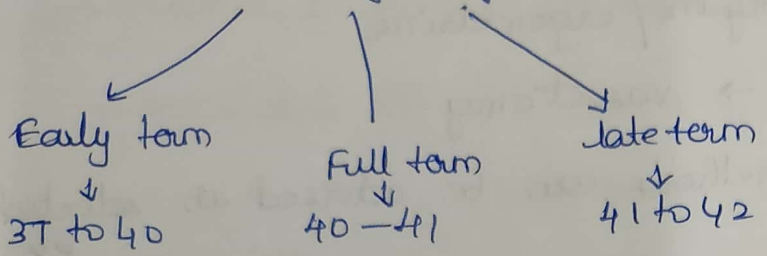
- R/o cardiac failure → immediate postpartum > labour > 28-32 wks
- Most R/o maternal mortality → eisenmenger's syndrome
- m/cc of maternal mortality → MS
- mitral balloon valvotomy done best in T2 → 14-18 wks
- Induction of labour → mech methods like foley's balloon is preferred
  - PGE2 gel can be used
- RF for cardiac failure → anemia, infections, preeclampsia, hyperthyroidism

- Anaesthesia of choice in cardiac c/s → ~~spinal~~
- Tocolytic of choice → atosiban (oxytocin receptor antagonist)
- DOC for arrhythmias → metoprolol
- c/I uterotonic → methergine / ergometrine
- contraceptive of choice → vasectomy
- IUCDs, POPs & barrier methods can be advised in selected pts
- combined OCPs are c/I

Preterm labour

- EDD is calculated using Naeglese 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 DIMINISH PREGNANCY)

- D - distended uterus (multiple pregnancy, polyhydramnios)
- I - infections (bacterial vaginosis, GBS, PROM, UTI)
- M - maternal (preeclampsia, anemia, heart disease, APH, low BHI, stress, depression)
- I - iatrogenic (wrong dates, surgeries in uterus or Cx)
- N - nicotine (smoking)
- I - interval btw pregnancies
- S - short Cx (< 2.5 cm, funneling of OS)
- 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

(N) shape - ☤ T

ab(N) → Y, V, U ± bulging membranes

Immunological factors

IL 1β, TNFα, IL-6, IL-8, MCP-1, PGs, MMPs, etc

Management (SMART)

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

Classification of tocolytics

- β2 adrenergic agonists - terbutaline, retordine, isoxsuprine
- $MgSO_4$
- CCBs - nifedipine, nicardipine
- oxytocin receptor antagonist - atosiban (preferred in heart d/s complicating pregnancy)
- PG synthetase Gr - indomethacin, aspirin, ibuprofen, sulindac
- NO donors - nitroglycerine
- Halothane

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

Role of progesterones

- may be given in a pt of previous hx preterm labour, not useful in active preterm labour

Role of encirclage

- previous hx preterm birth / 2nd trimester loss
- USG → Cx < 2.5 cm
- Done in < 28 weeks of pregnancy
- Not done in PPROM, chorioamnionitis, twins, APH
- preferred is McDonald method
- others → transabdominal encirclage, shirodkar method

PROM

- mostly UTI infections
- sterile speculum examination to demonstrate leak is the 1st step
- other tests → • 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 gn to women ε 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, ε thin body

II - stage 1 + fetal distress, meconium present

III - stage 1 & 2 ε 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 h/o prolonged pregnancy
- Irregular ovulation
- ↓ed fetal estrogen (CRH production → d/t placental sulfatase deficiency, anencephaly, fetal adrenal hypoplasia

Physiological change in postterm gestation

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

Complications

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

Management

- confirmation of gestational age
- 1st trimester USG (CRL +/- 7d)
- 2nd trimester BPD (± 14d)
- quickening 16-18 wks

Termination of pregnancy

40 weeks
↓
Do stripping, perform non stress test & AFI
↓

If AFI adequate, NST reactive
↓
Repeat mod. BPP twice weekly
↓
induce @ 42 weeks

If oligo or non reactive NST – induce labour

Induction of labour

Modified Bishop scoring system

	0	1	2	3

Methods of induction

- Mechanical dilators → • Foley's balloon,
  - laminaria tent
  - osmotic dilators
- stripping of membranes
- amniotomy
- Pharmacological — • PGE_2 gel
  - oxytocin infusion
  - misoprostol (PGE1 analogue)

Medical disorders of pregnancy

Hepatic disorders

Classification

① specific to pregnancy, resolves spontaneously or following delivery
- hepatic dysfn from hyperemesis
- intra hepatic cholestasis
- acute fatty liver
- hepatocellular damage ~ preeclampsia → HELLP syndr.

② acute liver disorder coincidental to pregnancy

③ chronic " " predating pregnancy

④ liver disease findings in pregnancy

- elevated serum ALP
- palmar erythema
- spider angioma

Intrahepatic cholestasis of pregnancy

- aka recurrent jaundice of pregnancy, cholestatic hepatosis

- Cf :

→ pruritis : late in pregnancy, no constitutional Sx, predilection for soles & palms, skin changes d/t excoriations from scratching, pruritis precedes lab findings by several weeks (mean of 3)

- chronic hepatitis C is also significantly ↑ed Rlo cholestasis
- m/c in multifetal pregnancy
- Ⓝ liver enzymes at presentation
- slight jaundice (<5 mg/dl)
- No BP change/ proteinuria
- recurs in subsequent pregnancies & with estrogen containing contraceptives
- ∴ OCPs are C/I in such pts

- Rise in serum bile acids is the earliest & most consistent change / best marker; there is 10-100 fold ↑ in serum cholic acid foll by serum chenodeoxycholic acid
- always only direct bilirubin ↑ed
- serum ALP ↑ed
- ↑ed chances 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 of centrilobular regions but without inflammation or necrosis
- disappears after delivery, but often recurs in subsequent pregnancies or ± estrogen containing contraceptives

Rx:

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

Acute Fatty liver of pregnancy

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

- M/m

- Induction of labour
- Blood & blood products (PPH d/t coagulopathy)
- hepatic dysfn resolves within a week post partum

* m/c of pyelonephritis in pregnancy → E. coli
* ABs c 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 m/c
- Neonatal disease → • localised skin, mouth or eye disease
  - CNS d/s ~ encephalitis
  - disseminated d/s

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

Toxoplasmosis

- Togondii
- Perinatal problems
  → 1° infection causes fetal abn d/t transplacental transmission
  → max severity of infection is in 1st trimester
  → max incidence of transmission is in later pregnancy (but ↓ w/o teratogenicity)
- most babies are asymptomatic at birth
- severe disease → triad of hydrocephalus or [illegible] chorioretinitis & cerebral

- Δ → acute infection : IgG & IgM ABs or 4 fold ↑ in paired specimens 4 weeks apart
- M/m → - spiramycin - if 1° infection is confirmed
  - pyrimethamine - sulfonamide to be alternated = spiramycin if fetal infection is confirmed

Varizella zoster

- HZV doesn't cause congenital malformations
- perinatal effects :

i) fetal varicella syndr - highest risk btw 13 - 20 wks. occurs in 2% of fetus infected at this time

ii) 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 abⓃ

- m/m → oral acyclovir 800 mg 5 times daily for 7 days - help to ameliorate severity of disease in mothers

- prevention - VZIG within 72hrs of exposure

Causative	Teratogenic sequale
1) Toxoplasma	- Tetrad of hydrocephalus or microcephaly, convulsions, chorioretinitis, cerebral calcification
2) Rubella	- Tetrad of gregg: 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 ε lesion during delivery

Effects of syphilis on pregnancy & labour

- preterm labour, fetal death, fetal growth restriction, neonatal infection
- fetal hepatic ab(N) → anemia & thrombocytopenia
  → ascites & hydrops
- neonatal jaundice ε petechiae or purpuric skin lesions

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

- Rx:
  - penicillin (preferred)
  - erythro & azithro → curative to mother but not fetus
  - early syphilis → benzathine penicillin single dose
  - >1 yr duration → " " weekly → 3 dose
  - Neurosyphilis: aqueous crystalline penicillin iv

- X ray shows → moth eaten appearance of ~~syphilis~~ 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 gn preconceptionally & ↑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, ↓ed GI motility, antacid use, pregnancy hypervolemia, placental enzymes & induction of hepatic enzymes & ↑ed GFR

Pregnancy complications

- ↑ed Rlo CS & seizure disorder in baby
- ↑ed Rlo pre eclampsia & PPH

- untreated epilepsy is not also ↑ fetal malformation rate
- monotherapy is preferred ∈ lowest possible dose
- valproate, phenytoin & phenobarbitone → also ↑ Rlo NTD defects
- newer anticonvulsants are better
- M/m → targeted USG for women on anticonvulsant dr
- Contraception & breastfeeding
  - Breastfeeding is not contraindicated
  - ↑ed failure rates ∈ combined OCPs & POPs ∈ enzyme inducing anticonvulsant agents

Abnormal fetal presentation

- Incidence of malpresentation at term → 3 to 5%

Breech presentation:

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

Complete breech: - more common in multigravida
- PV → feel buttock & 2 feet of baby
- fully flexed posture
- legs flexed at both hips & knees

Incomplete breech: → frank breech, footling presentation, Knee presentation
- various degrees of deflexion present
- foot is presenting part (footling)

Frank breech:
- hips flexed & knees extended
- Presenting on

- chances of cord prolapse increase in footling &

Knee presentation

Causes of breech presentation

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

causes
- multiparity
- placentation high in fundus or low in pelvis
- uterine myomata (fibroid)
- intrauterine synechiae
- ~~aaaa~~ mullerian duct fusion abN
- prematurity & polyhydramnios
- ↓ed fetal muscle tone / activity
- CPD
- hydrocephalus
- oligohydramnios

Δ:
- clinical
- USG (gold std)
- position - * sacrum is denominator of breech.
  * 1st position → ① sacro anterior (LSA) - m/c

Mechanism of labour in breech

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

Pinard maneuver / Frank breech decompression:

- used to deliver the legs in frank breech

Lovset maneuver:

used to deliver the shoulder / arm in breech

Burns Marshall maneuver:

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

Mauriceau maneuver:

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

Delivery ~ piper's forceps:

- to deliver head

* The aftercoming head of breech, chin to pubis is delivered by
  - manual rotation & extraction by Pipers forceps
  - modified prague maneuver

* cause of death in breech delivery is
  - intracranial hemorrhage
  - atlantoaxial dislocation
  - asphyxia

- Breech presentation ~ hydrocephalus is managed by
  - CS
  - transabdominal decompression
  - PV ~~exam~~ decompression
  - craniotomy of aftercoming head (not done down)

Occipitoposterior position & deep transverse arrest:

- 90% corrects itself
- causes →
  - android or anthropoid pelvis (wide occiput in wider post. segment)
  - anterior attachment of placenta
  - high pelvic inclination
  - ab(N) uterine contraction

- long rotation (3/8°)
- short anterior rotation (1/8°)
- nonrotation
- short posterior rotation (1/8°)

} unfavourable → face to pubis

Deep transverse arrest :

- sagital suture is placed in transverse bispinous 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/n of deep transverse arrest

- CS
- vacuum extraction
- Kielland forceps (not (N) forceps)
- Manual rotation & forceps delivery

- baby at 90° to long axis of uterus → transverse lie

[45° to long axis of uterus is oblique lie]

- Presentation in transverse lie is shoulder presentation
- If labour progresses → hand prolapse, cord prolapse

Face presentation:

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

causes –
- preterm
- enlargement of neck
- coils of cord around neck
- hydramnios, contracted pelvis
- fetal malformations, anencephaly
- high parity – pendulous abdomen permit back to sag anteriorly in same direction of occiput

- diameter of engagement is submentobregmatic

Engagement diameters

- Brow presentation – mentovertical (14 cm)
- (N) flexed vertex presentation – submentobregmatic (9.5 cm)
  suboccipitobregmatic (9.5 cm)
- Breech (partially deflexed head) – suboccipitofrontal (10 cm)
- Fully deflexed head – occipitofrontal (11.5 cm)
- Face presentation – submentobregmatic (9.5 cm)

Brow presentation

- On pv examination, anterior fontanelle & supraorbital ridge is felt

- In (N) cephalic presentation, only posterior fontanelle is felt

Puerperium

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

Lochia

- vaginal discharge for first 15 days during peurperium
- offensive fishy smell
- Colour
  - lochia rubra (red) - 1 to 4 days
  - " serosa (yellowish/ pink/ pale brown) - 5 to 9 days
  - " 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 - 1000gm
  after 6 wks - 60 gm
- Carunculae myrtiformes → nodular tags of hymen after childbirth

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

Contraceptive effect of breast feeding:

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

Postpartum mastitis:

Rx: - analgesics, ABx, gentle hand expression, hot & cold compress
- do not stop BF

- m/cc is staph aureus especially MRSA
- source of infection → infant's nose & throat
- m/m → milk from affected breast for C&S
- dicloxacillin/ erythro

- Breast abscess:
  - suspected when a mass ± fluctuation is palpable
  - no defervescence within 48-72hrs of Rx
  - USG finding
  - Rx : ~~I&D~~ 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
- 50-400ml/day
- more protein, secretory Igs, more lactose, less fat

* Contraception ♀ is preferred in lactating mother is POP.

Thromboembolism in pregnancy:

Virchow triad → stasis, local trauma, hypercoagulability

- RF
  - previous Hx thrombosis
  - twins
  - anemia
  - APH / PPH
  - CS foll by prolonged immobility
  - hyperemesis
  - obesity
  - multiparity
  - HTN
  - stillbirth
  - peripartum hysterectomy

DVT

- Iliofemoral > iliac > calf vein
- (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

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

Pulmonary TE:

- 1 in 7000 pregnancies
- CF: dyspnoea, pleuritic chest pain, cough
- Δ:
  - leg Sx → compression USG
  - CXR
  - CT pulm. angiography
  - V/Q scintigraphy
- m/m:
  - anticoagulants
  - vena caval filters in case CI is needed
  - thrombolysis
  - embolectomy

Obstetric neuropathies:

- m/c (N) injured during NVD → lateral femoral cutaneous (N) of thigh
- RF → nulliparity, prolonged 2nd stage, prolonged pushing in semi-Fowler position

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

Anemia in pregnancy

Physiological anemia of pregnancy:

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

Iron requirement in pregnancy

- It is 9 x higher
- ∴ 1000mg / pregnancy
  - 500mg for erythropoesis
  - 300mg for fetus & placenta
  - 200mg shed thro skin, urine & stool

WHO grading

mild 10-11 g/dl

moderate 7-10 "

severe <7 "

ICMR grading

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

CDC

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

Causes:

- Deficiency (mlc) → iron, folate, ↓t B12
- hemoglobinopathies
- aplastic anemia
- AIHA
- leukemia

Development of IDA: – 3 stages

① depletion of iron stores → ↓ ferritin

② iron deficient erythropoiesis

→ ↓ ferritin, ↓ sr. Fe, ↓ TSAT, ↓ Hct

→ no clinical Sx / morphology change

→ Ⓝ Hb

③ Frank IDA

→ change in RBC morphology

→ clinical Sx

→ ↓ Hb

Indicators :

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

Complications

In mother

During pregnancy
- pre eclampsia
- infection
- heart failure
- preterm labour

Labor
- uterine inertia
- PPH
- cardiac failure
- shock

Puerperium
- peurperal sepsis
- subinvolution
- lactation failure
- venous thrombosis
- pulm. thromboembolism (CPTE)

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 Program 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

- 60mg of elemental iron & 250 mcg folic acid for 6 months & continue 3 months postpartum

Rx :-

- Iron should be gn 2 hrs before or 4 hrs after ingestion of antacids

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

Preparations

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

- Dose calculation

Iron needed = Hb deficit x wt in kg x 2.21 + 1000 mg
(to replenish the stores)

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

Blood transfusion

- marked / severe anemia in T3
- APH
- packed cells in (N) anemia
- exchange transfusion in CLF

M/m during labour

1st stage

- propped up position
- analgesia
- O_2
- receive blood
- avoid fluid overload

2nd stage

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

- Pt who is severly anemia develops Sx by end of T2
- ↑ R/o CCF

Other types of anemias

SCA :

- m/c hemoglobinopathy seen in pregnancy
- AR inheritance
- In pregnancy maintain target Hct above 25%
- ↑ R/o thrombosis → m/m by thrombo Px, analgesics
- NVD is preferred
- POP & IUCDs 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 : desferoxamine after TI is advisable
- concurrent folic acid supplementation

PPH

- Blood loss > 500 ml in NVD & > 1000 ml in LSCS
- massive PPH > 2000 ml
- a drop in Hct > 10%
- Obstetric shock index - HR / systolic BP
  - * (N) value is 0.5 - 0.7
  - * values > 1 → massive hemorrhage

Types

- 1° → within 24 hrs, m/cc → atonicity
- 2° → 24 hrs to 12 weeks, m/cc → retained placenta

Causes: 4 T's of PPH

- Tone - 70% (over distension of uterus)
- trauma - 20% (hematoma, inversion, rupture, laceration)
- tissue (retained placenta → 10%
  invasive ")
- thrombin (coagulopathies) - 1%

Atonic PPH

- m/c
- causes → • over distended uterus
  - maternal anemia, malnutrition, multigravida
  - labour complications → prolonged 2nd stage, precipitate labour, APH

Degrees 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	(N)	↑	>30	>40
• BP	(N)	DBP↑, SBP (N)	SBP <100 mmHg	SBP <70 mmHg
• capillary refill	(N)	delayed	delayed	absent
• mental status	(N)	mild anxious	altered	altered consciousness, coma
• urine output	(N)	20–30ml/hr	<20 ml/hr	nil

Px :

- WHO recommendations for AMTSL, 2012 :

① use of uterotonics 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 tonus assessment for early id of uterine atony is recommended for all women

Medical m/m :

- DOC → oxytocin → 10 IU im/iv
  - → 3-5 min for onset of action & DOA is 2-3 hrs. by im
  - → ↑ contraction by ↑ing Ca^{2+}

- methergine → 0.2 mg im/IV ~~stat~~ every 2-4 hours - 5 doses
  → c/I in HTN, scleroderma & Raynaud's
- Carboprost (PGF 2α) → 0.25 mg i.m
  → c/I in asthma
  → can be repeated upto 8 doses
- Misoprostol (PGE1) → 800-1000 mcg
  → oral / SL / PR (preferred) / vaginal
  → A/E → fever, vomiting

Blood product utilisation

- Whole blood → 500 ml → ↑ Hct 3%
- PRBCs → 300 ml → ↑ HCT 3% less fever
- Platelets → 50 ml → ↑ PLT 10,000
- FFP → fibrinogen, AT III, clotting 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 → obstetrical silicone balloons (Bakri balloon - 500 ml)

→ foley's catheter balloons

→ sengstaken - Blakmore tube

→ Rusch catheter &

→ condom "

2) Compression sutures

commonly used → ~~•~~ • B Lynch sutures (m/c)

• hayman "

• cho "

3) stepwise devascularisation

• uterine A ligation

• int. iliac A "

• arterial embolisation

If all fails → hysterectomy - subtotal

APH

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

Causes:
- placenta previa (30-35%)
- abruptio placenta (35-40%)
- others – placenta accreta, vasa previa, etc

Placenta previa

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

Causes:

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

Management

PP
|
Mild (<150 ml blood loss) | moderate (150–500 ml) | severe (>500 ml)

Mild:
|
>36 wks → LSCS
<36 → expectant m/m

moderate:
|
>34 wks → LSCS
<34 wks → resuscitate, steroids
|
stable → expectant m/m
unstable → LSCS

severe → resuscitate & LSCS

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 1° CS for previa
- Transabdominal USG → simplest, safest
- PV is C/I in previa
- painless bleeding diff previa from abruption
- Type 2b → dangerous → posterior placenta previa
  ↓
  might not be detected in routine USG
- Stallworthy's sign → slowing of fetal HR on pressing the head down into pelvis is seen in posterior placenta previa
- CS is indicated in all previa

- Leaking hemorrhages → seen in T2 or early T3, not severe & settles by itself

Abruptio placenta

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

Classifications

① Sheris 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

IV a → without DIC
IV b → with DIC

classification

ges

agnosis purely on pathology, no sx

uiescent form ε a live baby, mild bleeding

ld form ε onset of clotting problems & fetal distress

severe form ε coagulation defects & fetal death in utero

- same as previa ←
  - previous Hx abruption, smoking, cocaine abuse
  - 3Ps → polyhydramnios, PROM, preeclampsia
  - 2Ts → trauma, thrombophilias
  - ↑ed AFP in T2

of delivery

Abruption

Couvelaire uterus

- aka uteroplacental apoplexy
- rare complication in severe abruption
- d/t vascular damage within placenta ⇒ causes hemorrhage that progresses 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 mg/dl of plasma) & ↑ FDP, D dimers & variable ↓ in other coagulation factors → seen in 30% of abruptio placenta severe enough to kill fetus
- Release of thromboplastin in abruption d/t DIC
- M/m → FFP, cryoprecipitate
- Plt transfusion if plt < 50,000

Placenta accreta

- morbidly adherent placenta
- d/t absence of decidua basalis & Nitabuch's layer
- < increta → invade upto serosa, but doesn't breach it
  percreta → breach the serosa & may involve bladder etc.
- placental localisation using USG & plan for hysterectomy c LSCS

Vasa previa

- d/t velamentous insertion of cord (ie cord uncovered into fetal membrane)
- Blood loss is of fetal origin → ↑ed fetal mortality
  - maternal mortality is not ↑ed
- can be diagnosed antenatally by doppler USG
- when bleeding occurs → sinusoidal fetal HR pattern seen
- Δ at time of bleeding by - Singer alkali denaturation test / Apt test
- M/m - emergency CS
- on staining blood c Wright stain, if RBC's appear nucleated the blood is of fetal origin

IUD

- WHO → death of fetus that has reached wt of ≥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 (N) labor, rupture uterus

- Fetal causes - (m/c)
  - chr. ab (N), structural anomalies, chorioamnionitis, IUGR, Rh-incompatibility, non immune hydrops fetalis, intra partum asphyxia

- placental → 1) ~~Accidental abruption~~ > previa
  2) cord prolapse, true knot, cord around neck
  3) TTTS
  4) placental insufficiency
- Iatrogenic → ECV, drugs
- Idiopathic

Pathology :

- dead fetus undergoes aseptic degenerative process termed maceration
- epidermis is 1st structure to undergo maceration that appears 12-24 hrs after death
- Blistering & peeling of skin occurs. Fetus becomes swollen & looks dusky red
- gradually aseptic autolysis of ligamentous structures & liquefaction of brain matter & other viscera occur, producing characteristic radiological signs

Δ :

- Sx – loss of fetal movement
- Signs → regression of breast changes of pregnancy
  - PA → fundal height smaller than POG
  - FHS absent
  - Egg shell crackling feel of fetal head – late feature

- USG – earlier diagnosis
- evidence of IUD → lack of fetal movement & cardiac activity for 10 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 (N) parallelism

Complications

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

Evaluation

- ABO & Rh
- Blood sugar
- urine ex
- VDRL
- RFT, TFT
- TORCH screening
- lupus anticoagulant, anticardiolipin Abs
- sr. fibrinogen level
- aPTT, when fetus is retained for > 2 wks

M/m

- Expectant → in 80% upontaneous expulsion occurs in 2 weeks of death
  → sr. fibrinogen estimation – done weekly
  current → terminate pregnancy once IUD is Δed
- Mode of delivery → always by medical induction
  - oxytocin infusion – if Cx favourable
  - PGs – vaginal (PGE_2) gel, if Cx unfavourable
  - Misoprostol (PGE_1) 25–50 µg vaginal or oral every 4 hr
- ARM in C/I d/t R/o infections
- exclude 2° abdominal pregnancy if repeated attempts at induction fails to start labour

Examination of fetus for

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

Ix for autopsy & chromosomal studies

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

Ix for CS in IUD ?

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

Contracted pelvis & CPD

- 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 tiradiate 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 → trial labour
  - major → elective LSCS
  - minor + obstetric complications → elective LSCS

Trial of labour

- in minor CPD
- during labour there occurs an adjustment bw 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 ~~with~~ partogram
- FHS monitored ~~with~~ continuous CTG
- Platypelloid pelvis → is favourable as pelvis is contracted at one diameter & only one level (cavity/ brim / outlet)

Obstructed labour:

- inspite 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 ascite

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 btw 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

Dx:
- 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
  - sacroiliac jt – synovial
  - sacrococcygeal jt – synovial hinge joint

True pelvis

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

False pelvis

- above brim of pelvis

Pelvic inlet

- almost round (in gynecoid) ∴ AP diameter being shortest
- in erect posture, tilted forward, making an angle of inclination of about 55°
- ↑ in angle of inclination
  - d/t sacralization of L5 vertebra
  - delay in engagement because uterine axis fails to coincide ∴ inlet
  - favour occipito posterior
  - difficulty in descend of head d/t long birth canal & flat sacrum interfering ∴ 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 <10cm
- obstetric conjugate = diagonal conjugate − 1.5 cms
- 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
- Bispinous diameter → 10cm
- aka plane of least pelvic dimensions
- ⊛ in labour
  - internal rotation occurs at this level
  - beginning of forward curve of pelvic axis
  - deep transverse arrest occurs here
  - 0 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 " → 13 cm
  subpubic angle → 85 degrees
  ↓
  If <75 degrees → lead to dystocia

Caldwell Moloy classification of pelvis

Gynaecoid pelvis

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

- ischial spines are not prominent
- sacrosciatic 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 anteriorposteriorly oval
- only pelvis ī 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°)

Platypelloid pelvis
- least common type
- flate female pelvis
- inlet is transversely oval
- subpubic angle is wide

Outcomes

- Gynaecoid → • m/c & ④ type
  - diameter of engagement : transverse / oval
  - no difficulty in engagement
  - ① occipitoanterior (LOA) / ② occipito transverse (LOT) are m/c position
- 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 superparieto - subparietal diameter (8.5 cm)

Dystocia dystrophia syndrome

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

- asynclitism → sagital suture deflected from transverse diameter of pelvis
- anterior asynclitism → Nagele's obliquity
  posterior " → Litzman "

Fetal skull

- Sinciput → btw glabella & bregma
  (root of nose) (ant. fontanelle)
- BPD → most ~~imp~~ for engagement → 9.5 cm
- Bimastoid diameter → 7.5 cm
  ↓
  cannot be reduced
- Biparietal diameter → 8.5 cm

- Engagement → BPD of fetal head is through pelvic inlet

Diagnosis in obstetrics

Maternal serum AFP (MSAFP)

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

- Elevated levels
  - multifetal gestation
  - fetal death
  - neural tube defect
  - gastroschisis, omphalocele
  - cystic hygroma
  - maternal hepatoma/teratoma
  - underestimated GA
- Low levels
  - Obesity
  - DM
  - Trisomies 21 or 18
  - GTD
  - fetal death
  - overestimated gestational age
- gastroschisis > omphalocele/spina bifida occulta

Screening tests

- T1 → CVS
- T2 → amniocentesis
- [illegible] → cordocentesis → 18 wks
- In Downs syndr → ↓ AFP & ↑ hCG
- β-hCG is ↑ in Rh incompatability
- ↑acetyl cholinesterase in amniotic fluid is present in open neural tube defect

Procedures for prenatal genetic diagnosis:

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

Amniocentesis

- It is transabdominal withdrawal of amniotic fluid
- performed btw 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
- Early amniocentesis – btw 11 to 14 weeks
  - a/w more fluid leakage, fetal loss & talipes equinovarus

CVS:

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

PUBS / cordocentesis / funipuncture

- performed after 18 wks
- m/c Ix → fetal malformation detected during T2 & T3
  → Rh isoimmunisation to detect fetal HCT.
- Differentiate btw fetal blood & maternal blood:
  - Fetal blood cells (140fL) are larger than maternal cells (80fL)
  - 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

MCQs

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

Best marker of GA

- in T1 → CRL
- in T2 → BPD
- T3 → femur length

- Doppler USG detects 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

Atencephaly:

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

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

Spina bifida:

- defect in vertebrae in dorsal arch ∴ exposure of meninges & spinal cord
- open spina bifida → defect includes skin & soft tissues
- Types
  - occulta → skin/soft tissue not involved, small defect covered by tuft of hair/lipoma
  - aperta → covered by thin membr, giving appearance of cystic tumor
- affects lumbosacral region in 63%.

- ventral defects of spine are extremely rare
- Banana sign → compressed cerebellum
- lemon sign → indentation of frontal bone

Anterior abdominal wall defect

① Omphalocoele :

- Sac covering the content is very thin consists of 3 layers → outer amniotic membr, middle Wharton's jelly & inner peritoneal layer. Sac may get ruptured during birth
- often a/w cardiac & genitourinary anomalies ~70%

② Gastroschisis :

- small defect in ant. 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 T1 & who regularly smoke & take alcohol
- not a/w other anomalies

Down syndrome

Etiology

- 95% → trisomy 21 / non disjunction during meiosis
- 3-4% → robertsonian translocation
- 1-2% → isochromosome or mosaicism
- Risk ↑ ε ↑ maternal age, > 35 yrs
- But 70% of Down synds are seen in women <35yrs
- m/c nonlethal trisomy
- seen in 1 in 500 recognised pregnancies, &
  1 in 740 live births
- adult women ε Down synds are fertile & a third of their offspring have Down synds.
- Risk factors
  - Age : advanced maternal age
  - prior pregnancy ε autosomal trisomy / triploidy
  - women or partner ε balanced chromosomal translocation

T1 screening

- btw 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 → ↑ed
  - PAPP-A → ↓ed
  - hCG → ↑ed

T2 screening

- lower maternal serum AFP, ↑ hCG, ↓ unconjugated estriol (uE3) levels
- dimeric inhibin α → ↑ in Down

Triple test → MSAFP + βhCG + uE3

Quad test → MSAFP + βhCG + uE3 + dimeric inhibin α

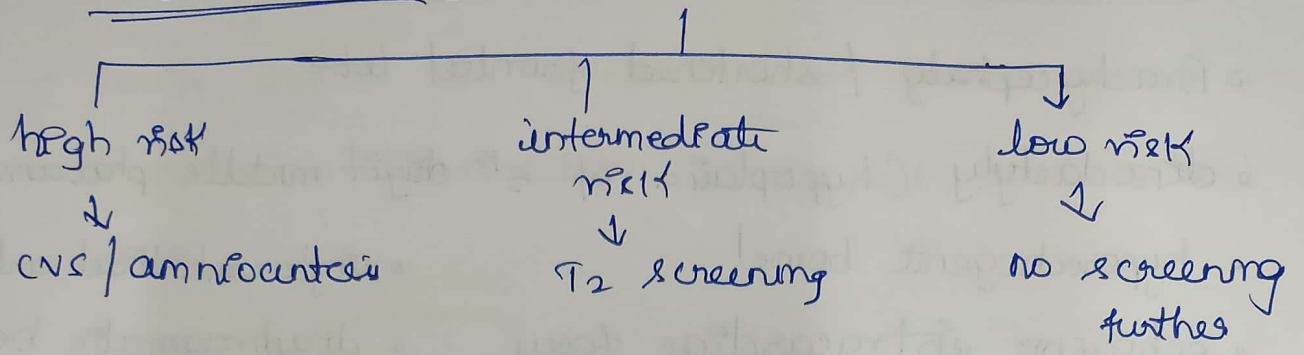
Abs low uE3 levels

- seen in smith lemli opitz syndrome & X linked ichthyosis

Combined T1 & T2 screening

- Integrated screening → all pts undergo T1 & T2 screening
- Sequential screening
  - → stepwise sequential screening
  - → contingent " "

Contingent screening → 1st 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 ≤0.5%
  - uses the tech of massively parallel sequencing or chromosome selective sequencing
  - Recommendations:
    - women > 35 yrs
    - USG finding of ≥ 1 fetal anomaly
    - risk factor for Down

Sonographic markers (soft markers) for Down syndrome in T2

- Brachycephaly / shortened frontal lobe
- clenodactyly (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 w/ high risk of aneuploidy

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

Recurrence risk of Down

- 100% chance of recurrence in ~~pati~~ parents ∵ Robertsonian translocation (balanced translocation)

IUGR

- Birth wt below 10th percentile of average for G.A

Symmetrical	Asymmetrical
• uniformly small	• head larger than abdomen
• ponderal index (Birth wt / crown heel length) — (N)	• low
• HC : AC & FL : AC ratio (N)	• elevated
• etiology → genetic disease or infection (intrinsic to fetus)	• chronic placental insufficiency (extrinsic to fetus)
• Total cell no. — less cell size — (N)	• (N) smaller
• Neonatal course → complicated ∴ poor prognosis	• uncomplicated ∴ good prognosis
• less common	• more common

* amniotic fluid volume is ↓ed in IUGR
* smoking, alcohol, cocaine cause IUGR
* IUGR babies are prone for hypoglycemia

MTP

- MTP act 1971 → MTP Ix in base of therapeutic (mother is sick), eugenic (anomalous baby), humanitarian, social (failure of contraception in married)
- who can terminate
  - * one who has assisted in atleast 25 MTP in authorized centre & having a certificate
  - * one who has 6 months house surgeon training in OBG
  - * one with diploma or degree in OBG
- can be done only in government established or approved hospitals
- consent → consent of woman is sufficient
  written consent of gaurdian in minor or mentally ill
- pregnancy <12 wks → opinion of single doctor
  12-20 wks → 2 doctors
- MTP 4 I in >20 wks
- T1 abortion

Medical

- mifepristone
- mifepristone & misoprostol (PG E1)
- M+x & misoprostol
- Tamoxifen & u

Surgical

- menstrual regulation
- vacuum aspiration
- suction evacuation &/or curettage
- Dilatation & evacuation
  - rapid method
  - slow method

- PGs → PGE1 (misoprostol), 15 methyl PGF2α (carboprost), PGE2 (dinoprostone) & their analogs
- Dilatation & evacuation (13-14 wks)
- Intra uterine installation of hyperosmotic solution
  - * extra amniotic → ethacridine lactate, PGs (PGE2, PGF2α)
  - * extra amniotic saline infusion (isotonic) ± a transcervical catheter balloon
  - * intra amniotic hypertonic urea, saline (not used now)
- oxytocin infusion – high dose ± either of above 2 methods
- hysterotomy (abdominal) – less commonly done

T1 MTP

(1) mifepristone (RU-486) & misoprostol
↓
progesterone antagonist
→ effective upto 63 days & highly successful within 49 days of gestation

(2) oral mifepristone 200mg (1 tab) ± vaginal misoprostol 800μg (4 tab, 200 μg each) 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 obstetrics

ECV:

- conversion of non cephalic to cephalic presentation
- done ideally at 37 weeks
- Ix → non cephalic presentations
- C/I → conditions where NVD is C/I as in placenta previa / abruption (APH)
  - → twin pregnancy (can be done in 2nd twin if intact membranes)
  - → oligohydramnios
- previous 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 pregnancy

Oxytocin

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

Methergin :

- syntometrin → oxytocin 5U + ergometrine 0.5U
- Ix → • control bleeding foll. abortion & evacuation of molar pregnancy
  - PPH
- C/I → pre eclampsia & eclampsia, heart disease, twin pregnancy following delivery of 1st twin, Rh incompatability, labour induction / augmentation
- Causes tetanic uterine contractions
- Ad/E → ↑ in BP, N & V
- PV (fastest) / im / oral

Misoprostol → PG E1 analogue

→ 25, 50, 100, 200, 400 mcg tablets

- uses → abortion, induction of labour, PPH
- oral / SL / vaginal / rectal

- Dosage : 1) abortion - 200 [illegible] mcg 4 hrly
  2) induction - 25-50 mcg @ 4-6 hrs
  3) PPH - 800-1000 mcg 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 (PGE2)

- gel contains 0.5 mg of PGE2
- Ix → cervical ripening, induction of labour
- Dosage → intracervical application - 0.5 mcg
- Ad/E → fetal distress, rupture of uterus

Carboprost (PGF2α):

- each ampule - 250 mcg/ml
- Ix → 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

- PMTP → misoprostol + mifepristone
- control of bleeding in abortion → misoprostol, oxytocin, methergin
- induction < Cx unfavourable → PGE2, PGE1 (Bishop score <6)
  Cx favourable (Bishop score >6) → oxytocin & PGs
- augmentation → oxytocin is DOC

AMTSL → oxytocin 10 U IV or IM

- control of PPH → oxytocin, ergometrine (syntocinon), misoprostol, carboprost

Antihypertensives in pregnancy

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

α methyl dopa

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

Labetalol :

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

Nifedipine :

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

Anticonvulsants

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

$MgSO_4$

- anticonvulsant in eclampsia
- Tx in severe pre-eclampsia & "
- Tocolysis – in preterm labour
- MOA →
  - suppress motor end plate sensitivity to Ach & reduces release of Ach from N endings
  - competitive ⊖r of Ca^{2+}
  - direct depressive action on uterine muscle
  - reduces cerebral edema

Dosage → PRITCHARD Regimen

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

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

- CCB – nifedipine
- $MgSO_4$
- β mimetics → terbutaline, ritodrine, salbutamol
- NSAIDs – indomethacin, sulindac
- atosiban – oxytocin antagonist
- Glyceryl trinitrate – NO donor

Anticoagulants :

- I_x → venous TE, APLA, AF, prosthetic valves, cardiomyopathy

Heparin

- MOA → ⊖ action of thrombin
  enhances action of AT III
- dose → 5000 – 10,000 units SC every 12 hrs (↓ aPTT monitoring)
- Ad/E → maternal – hemorrhage, thrombocytopenia, osteoporosis
  fetal → no effects as it doesn't cross placenta
- antidote → protamine sulphate

LMWH :

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

Warfarin :

- more effective than heparin in preventing TE
- Ad/E → . warfarin in T1 causes warfarin embryopathy
  . ↑ r/o abortion, still birth & fetal bleeding
- BF is not C/I ↓ warfarin therapy
- vit K & FFP are gn in overdose

Protocol for anticoagulation:

- 6 to 12 wks → heparin is gn
- 12 – 36 wks → warfarin or continue heparin
- >36 wks → heparin – stopped at onset of labour restarted thru following NVD & 24 hrs foll CS
- warfarin is started in postnatal period overlapping ~ heparin till desired INR is achieved & heparin is stopped

→ heparin therapy : aPTT is kept 2 times the control
warfarin " : INR btw 2·5 – 3·5

Teratogens

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

FDA risk catagories:

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

X drugs → alcohol, ACE ors, Li, M+x, mifepristone, danazol, isotretinoin, radioactive Iodine

Drugs & fetal effects

Analgesics

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

Anticoagulants

- warfarin → chondrati syndrome / chondrodysplasia punctata

Antihypertensives

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

Others

- carbimazole → fetal aplasia cutis, choanal atresia, esophageal atresia
- Li → ebstein anomaly, fetal goitre / hypothyroidism, fetal DI
- DES → vaginal clear cell Ca, T shaped uterus, vaginal adenosis
- Isotretinoin → b/l microtia, hypertelorism, outflow tract defects, hydrocephalus
- chloramphenicol → gray baby syndrome (peripheral vascular collapse)
- Sulphonamides → neonatal hemolysis, jaundice
- corticosteroids → high dose - 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, craniofacial defects – microcephaly, microphthalmia, flat nasal bridge, hypoplastic philtrum, behavioural anomalies
- Smoking → IUGR, LBW
- Cocaine → microcephaly, cerebral infarct, abruption, LBW

Abnormal uterine action

(N) uterine contractions

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

Stages of labour

- preparatory division → latent phase + acceleration phase
- dilatory " → phase of maximum dilatation
- pelvic division → 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 → montevideo units

1MV = intensity of contraction (mm Hg) × no. of contraction in 10 min

Parameters

- (N) basal tone – 5 to 20 mm Hg
- peak pressure – 60 mm Hg
- adequate uterine contractions → 3 in 10 min lasting for 45 sec
  → causing intrauterine pressure of 65–75 mm Hg or 220 MV units
- Tachysystole – >5 contractions in 10 min
- Hyperstimulation – tachysystole causing fetal distress

Dysfunctional labour:

- more common in primi
- (N) Cx dilatation in primi – 1 cm/hr
  multigravida – 1.5 cm/hr
- 1° dysfunctional labour – Cx dilates <1 cm/hr following (N) latent phase of labour; mostly gets corrected by amniotomy/oxytocin augmentation

• 2 arrest of dilatation – (x dilatation stops or slows after start of (R) active phase of labour

Abnormal uterine ~~cont~~ action

coordinated uterine action → polarity is maintained
incoordinate uterine action → polarity lost

Co-ordinated

① Hypotonic
– uterine inertia

② Hypertonic
(i) no obstruction – precipitate labour
(ii) obstruction – generalised tonic contraction & retraction
pathological retraction ring

In co-ordinate

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

Hypotonic uterine contractions

• m/c disorder
• < 3 contraction in 10 min, each < 45 sec / < 180 MV units
• occur at any stage of labour
• polarity maintained, FHS good
• M/m → rule out CPD, malpresentation, malpositions
→ labour augmentation ε ARM + oxytocin

Precipitate labour:

- Rapid expulsion of baby d/t hyperactive uterine contraction
- combined duration of stage I & II < 2hr
- Cx dilatation ≥5 cm/hr in nulliparous & 10 cm/hr in multi
- common in multipara, recurrence is common
- C/C → injury to Cx, vagina, perineum
  - PPH, uterine rupture / inversion, infections
  - fetal → ICH

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

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

End result

- primi → further retraction stops — uterine exhaustion
- multi → progressive thinning of LUS → uterine rupture

CF

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

M/m → analgesics, correct dehydration & acidosis
rule out uterine rupture
CS done

Prevention → partogram

Spastic lower segment

- Reversed polarity, lack of fundal dominance
- LUS contractions stronger
- Basal tone ↑ed
- 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 jn of upper & LUS, usually around neck of fetus in cephalic presentation; abnormal polarity (X)
- usually reversible
- causes → injudicious oxytocics
  → 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 contraction)
  → PV = no features of obstruction
- maternal condition not much affected
- fetus may be in distress, usually FHS good
- uterus never ruptures
- No alteration in position of ring

M/m - ◦ delivery by CS

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

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

Generalised tonic contraction (tetany)

◦ contraction involve whole of uterus upto internal os

◦ no diff btw upper & LUS

• tonic muscular spasm of uterus ± fetus inside

◦ no uterine rupture

◦ cause → CPD, methergine, injudicious oxytocics

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

◦ M/m → * in hypercontractility induced by oxytocics, oxytocin stopped. Terbutaline gn

* in CPD & fetal distress → CS

Cervical dystocia

◦ Failure of cervical dilatation

◦ cause → inefficient ut. contractions, malpresentation, malposition

◦ Cx spasm < 1° - primi, Cx rigid & fails to dilate
2° → scarring of Cx from previous surgery or d/s

• M/m ⇒ ◦ malpresentation / position → CS

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

◦ Duhrssen's incision at 2 & 10 o clock position

Abnormalities of amniotic fluid

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

* golden yellow - RH incompatibility (bilirubin)
* dark coloured - abruption
* dark brown - IUD

o Functions

* NOT MUCH NUTRITIVE VALUE
* shock absorber
* temp. maintanence
* movement of fetus - prevents adhesion
* growth of fetus
* during labour → bag of membrane helps in dilatation of cx

o Assessment

* USG → antepartum fetal surveillance (APFS)
* AFI → sum of 4 quadrant measurement of largest vertical pocket
  → (N): 5 - 25
* Single deepest pocket (SDP) • measurement : (N) → 2-8 cms (or SVP)

Polyhydramnios

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

Causes

- 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 incompatability
- Placental → chorioangioma of placenta
- Idiopathic (m/c cause) - 60%

Clinical types

(i) Acute type →
- usually before 20 wks
- very rare
- a/w TTTS, chorioangioma of placenta

(ii) chronic type → m/c type

Dx:

- FH (fundal ht) > POG, Tender abdominal girth
- difficulty in palpating fetal parts, presentation, position, FHS

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

Complications

- Maternal

Antepartum	intrapartum	post partum
○ pre eclampsia	○ early ROM	○ subinvolution
○ malpresentations	○ cord prolapse	
○ PROM	○ uterine inertia	
○ preterm labour	○ operative interferences	
○ abruption	○ PPH	

- Fetal → Ted morbidity & mortality d/t congenital anomalies & prematurity

Management

- Mild → no Rx 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 slow ROM, allow slow escape of liquor
  - * P/v following spontaneous ROM to rule out cord prola
  - * AMTSL
  - * examine newborn for anomalies

Oligohydramnios

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

- **cause**

i) fetal → * renal agenesis (m/c)
* fetal anomalies, obstructive uropathy (post. urethral valve),
* IUGR, post maturity

ii) maternal → * hypertensive disorder (uteroplacental insufficiency)
* PROM
* dehydration

iii) placental → amnion nodosum, ACE ors.

iv) idiopathic → m/c cause of mild oligohydramnios

Δ

- FH < POG, red abd. girth
- uterus feels 'full of fetus'
- USG

Complications

- Maternal – red operative interference
- Fetal → * fetal pulm. hypoplasia
* fetal deformities d/t intra amniotic adhesions - alteration in shape of skull, CTEV, amputation of limb
* cord compression
* meconium aspiration

M/m → rule out anomalies
→ isolated oligohydramnios in T3 – conservative m/m ± APFS upto term → 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 min

Ix of amnio infusion

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

Variable deceleration

- not in relation ↄ uterine contraction
- seen in cord prolapse

Early deceleration

- seen in head compression
- starts ↄ uterine contraction & passes of before contraction passes

Late deceleration:

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

PIH

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

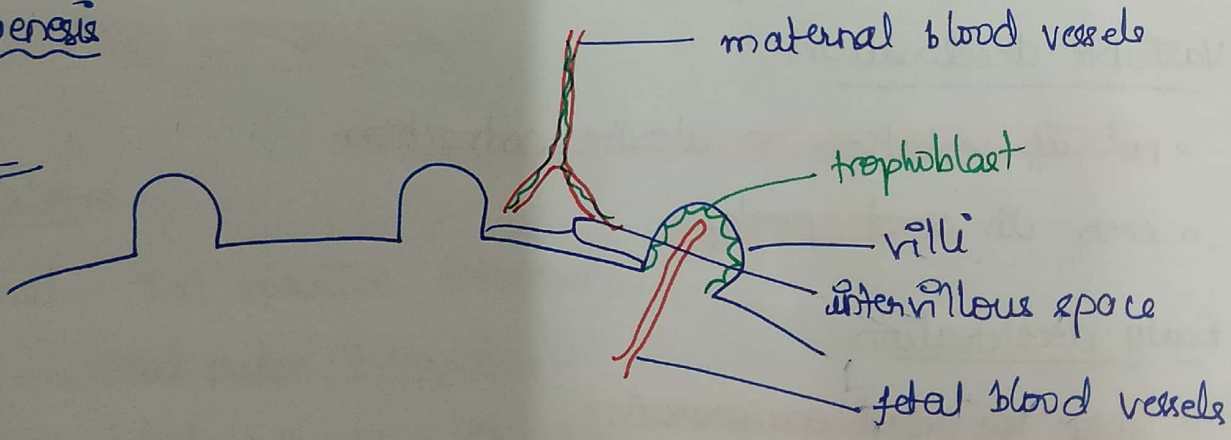
Measurement of BP :- sitting / lateral lying down position
- manometer at level of heart
- Korotkoff's sound V is taken as DBP

Chronic HT during pregnancy
- ↑ in BP before 20 wks (1% chr. HT)
- BP doesn't come back to (N) after 12 wks of delivery

PIH :
- ↑ in BP after 20 wks of pregnancy
- BP comes back to (N) before 12 wks of pregnancy

Pathogenesis

(N)



- Trophoblast replaces maternal lining, ∴ spiral arteries become low (R) vessels

i.e trophoblastic invasion
- step I → 10 to 12 wks
  - trophoblast replaces lining of arteries upto decidua
- step II → 16 to 18 weeks
  - replaces upto myometrium

incomplete trophoblastic invasion l/t high ® & ↑ BP
⇒ PIH

incomplete trophoblastic invasion → ↓ blood supply to placenta → placental ischemia → inflammatory mediator release → endothelial injury

endothelial injury → leaky capillaries → movement of fluid into extracellular space → edema
movement of fluid into extracellular space → • hemoconcentration in intravascular space
• platelet fn & no is defective
↓
end organ failure/damage

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

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

Types of PIH

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

Proteinuria

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

Signs of end organ damage

1) Plt count < 1 lakh
2) Sr. creatinine > 1.1 mg/dl
3) ↑ liver enzyme to more than 2 times (N)
4) evidence of pulm edema
5) presence of visual / cerebral Sx

Grade of preeclampsia

- Mild → preeclampsia without severe features / simply preeclampsia
  - BP > 140/90 mm Hg but < 160/110 mm Hg
- Severe preeclampsia → BP > 160/110 mm Hg or signs of end organ damage

Criteria is removed to classify btw mild & severe
→ • amount of proteinuria
• oliguria
• IUGR

- In PIH, as $P \propto \frac{1}{V}$ ⇒ fetal blood flow is ↓ed → IUGR (brain sparing effect)
  → fetal oliguria → oligohydramnios

Chronic HTN + superimposed pre eclampsia :-

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

RF : * previous H/o pre eclampsia
* primi
* chr. HT
* diabetes
* obesity
* chr. renal d/s
* extremes of maternal age <18 / >40

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

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

Changes in Kidney in PIH:

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

Other changes:

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

Eclampsia

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

Signs of impending eclampsia:

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

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

HELLP syndrome

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

Criteria for Δ of HELLP ⇒ Tennessee criteria

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

Mississippi classification of HELLP syndrome :

- class 1 (most severe) – plt count < 50,000
- class 2 → plt count btw 50000 – 1 lakh
- class 3 → 1 lakh – 1.5 lakhs, but AST & LDH ↑ed

M/m :

- Immediate termination of pregnancy if $MgSO_4$ > 34 wks
- If btw 27 – 34 wks, give corticosteroid & deliver after 24 hrs of last dose of corticosteroid

Corticosteroid

- DOC → Betamethasone – 2 doses of 12 mg 24 hrly im worldwide
- In India → dexamethasone 6 mg, 12 hrly 4 doses i.m

D/D of HELLP :

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

Predictors of pre-eclampsia :

- Uterine artery doppler →
  - shows diastolic notch
  - It disappears by 24 weeks in (N) pregnancy
  - persists after 24 weeks in PIH

Predictors no longer used

- hemoconcentration
- uric acid levels
- hypocalciuria
- Ted homocysteine levels
- grant roll over test

Basis of grant roll over test → supine hypotension syndrome / or / IVC syndrome

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

done btw 28-32 wks

Drugs to prevent pre eclampsia:

- Best - aspirin - 50 - 150 mg 1 day, continued throughout pregnancy & stopped 7 days before labour
- Calcium supplementation — only in hypocalcemics

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 mm Hg
  as per NICE " → >150/100 mm Hg
  → labetalol, hydralazine, α methyl dopa, nifedipine, nitroglycerine, sodium nitroprusside

- Anti HT of choice in PIH / preeclampsia / chronic HTN / acute HT / hypertensive crisis → labetalol
- Target BP in pre eclampsia – SBP ⇒ 130 - 140mm Hg
  DBP ⇒ 80 - 90mm Hg
- in chr HT during pregnancy → systolic 140 - 150 mm Hg
  DBP 90 – 100 mm Hg

Labetalol

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

Antihypertensive d/I in pregnancy

- ACE OR
- losartan
- diazoxide
- diuretics → d/I only as antihypertensive, but if pt has CHF, diuretic can be given

M/m of mild pre eclampsia / gestational HT

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

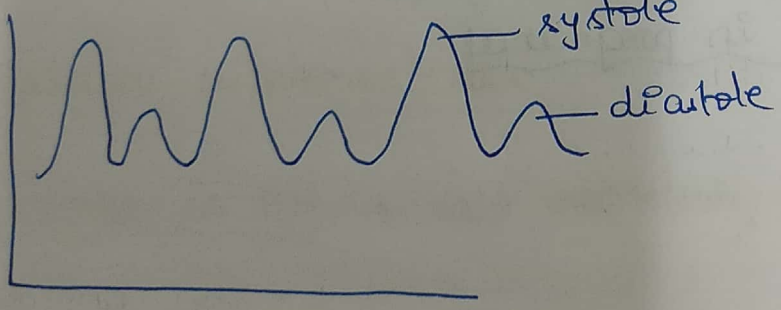
M/m of severe pre eclampsia

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

Ix for immediate termination irrespective of GA :

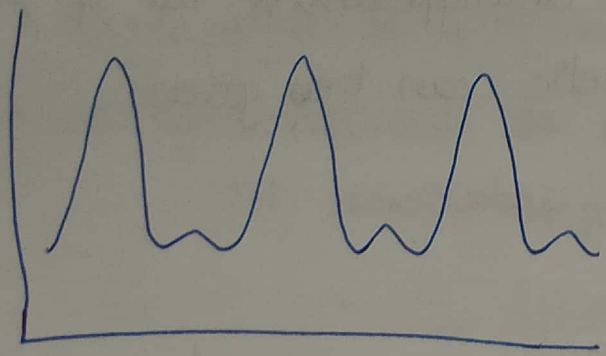
- impending eclampsia
- eclampsia
- fetal distress / abruptio placenta
- uncontrolled BP or rising S creatinine levels
- HELLP syndrome
- reversal of end diastolic flow in umbilical artery doppler

Umbilical artery doppler



in (N) pregnancy

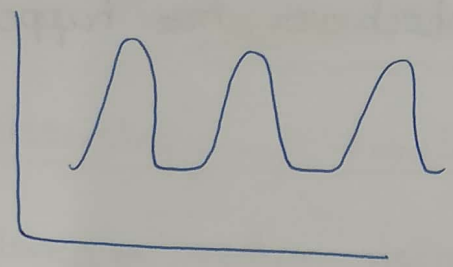
(R) of blood vessels ↓
↓
Diastolic flow ↑
∴ S/D ratio = ↓



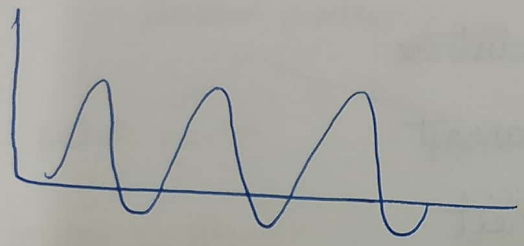
in PIH

diastolic flow ↓ d/t ↑(R)
∴ S/D ratio = ↑

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 → airway management
- then ↳ convulsion → $MgSO_4$ & antihypertensive → I.V labetalol
- Definitive m/m → terminate pregnancy irrespective of GA
- Mode of delivery → try vaginal - if it occurs in 24 hrs of last convulsion, otherwise cesarean section

* DOC for status epilepticus in pregnancy → thiopentone sodium > phenobarbitone

$MgSO_4$:

- MOA → blocks NMDA receptors in brain, causes cerebrovasodilation, peripherally it blocks calcium channels (use cautiously ∈ CCBs)

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

$MgSO_4$ toxicity

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

Pt is kla **Sibai regime**

→ loading dose – 6mg iv
→ then 2g/hr iv infusion
→ if convulsion recure – 2g iv is given again

Uses of $MgSO_4$:

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