O & G

OBSTETRICS

PHYSIOLOGICAL CHANGES IN PREGNANCY

	INCREASE	DECREASE	OTHER NOTES
CVS	* Cardiac output , Heart Rate, Stroke Volume. *Venous pressure >> varicose veins, hemorrhoid, lower limb edema	*Peripheral vascular resistance & blood pressure *venous return	*Hyperdynamic circulation. *Compression of IVC. *Peripheral vasodilation.
Hematologic	*WBC but impaired function *coagulation factors (X , IX, VIII , VII , II) & fibrinogen	*Apparent decrease in Hb & HCT. *Platelets (thrombocytopenia) *Hyponatremia (reset osmostat) *Fibrinolysis *Anti-coagualnts (anti-thrombin III , protein S)	*Hemodilution ->> physiological anemia *Hypercoagulable state – DVT & PE. Normal clotting test * *Venous stasis of lower limbs esp. Lf side
Respiratory	*Nasal congestion & epistaxis *O_2 consumption *minute ventilation & tidal volume >> hyperventilation >> resp. alkalosis *metabolic rate	-RR -TLC -FRC -RV -PaO_2 -Peak expiratory flow rate -Pul. Vascular resistance	-Elevated diaphragm -No change in VC & FEV!
GIS	Intra-abdominal pressure >>> GERD	GI motility (delayed emptying) lower esophageal pressure	-Gallstones -Constipation -Nausea & vomiting -Hemorrhoid Atypical appendicitis
GUS	Urinary frequency GFR & renal plasma flow Creatinine clearance Protein excretion	-Serum creatinine - Serum urea	-UTI –PN -Glycosuria (may be physiological) -Dilation of uterus &renal pelvis esp. Right Physiological Na & H2O retention

Nervous System	CTS (carpal tunnel syndrome) * *Bell's palsy		
Hepatobiliary	-Liver metabolism -Fibrinogen -Binding proteins (ceruloplasmin, TBG, Transferrin) - -ALP (3-4 x normal)	*Total serum protein *Albumin *Slight decrease ALT & AST	
Endocrine	-BMR (basal metabolic rate) -Total thyroxine (T3,T4) -TBG -Thyroid uptake from blood -Cortisol -Bone turnover (no loss of bone density) -Post prandial glucose -Glucose intolerance	Ca Albumin Fasting blood sugar Renal threshold for glucose Serum TSH Relative iodine deficiency	*Thyroid enlargement *Physiological insulin resistance *IGT *Anti-insulin hormone from placenta (HPL, Glucagon,Cortisol)
Pituitary & Adrenal	-Volume of anterior pituitary -Prolactin -Placental hormones -Free & bound cortisol -Angiotensin II -Plasma rennin -Plasma & urine Aldosterone	LH & FSH suppression	-NO change in ACTH -NO change in diurnal variation of cortisol
Skin	*Pigmentation of perineum & areola *Chloasma *Linea nigra *Spider angioma *Palmar erythema *Stria gravidarum *Post-partum hair loss *Pruritis		

ANTENATAL CARE

- **Definition :** Careful, systematic assessment and follow up of a pregnant patient to assure the best health of the mother and her fetus

Objectives of ANC	How often the visit?
-To prevent and identify maternal or fetal problems that adversely affect pregnancy outcome. -To educate the patient about pregnancy, labour-delivery, and parenting as well as about ways she can improve her health. -To promote adequate psychological support from her partner, family and caregivers.	-First visit in early pregnancy. -Then every 4 weeks until 28 weeks. -Then every 2 weeks until 36 weeks. -Then weekly until delivery. (For high risk patients, individualized and more visits)

FIRST VISIT	
HISTORY -**Medical problems** (DM, HT, others). -**Surgical**, previous operations, complications and need for transfusion. -**Family hx**- inherited problems (medical diseases, congenital anomalies, cystic fibrosis, hemophilia…..). -**Social hx**- psychosocial background and lifestyle, smoking (causes small fetus), alcohol (causes heart and facial anomalies) -**Obstetric**- EDC, recurrent problems (fetal & neonatal death, preterm deliveries, IUGR, macrocosmic babies, anomalies, abruptio, HT, PET, GDM, PPH, thromboembolism….). -**Gynaecologic**- infertility treatment, PID, ectopic pregnancy, STDs).	**PHYSICAL EXAMINATION** -General & full examination. -Obstetric examination. -Pelvic examination? Only If indicated **DETERMINING THE GESTATIONAL AGE** (Accurate estimation is vital & mandatory to avoid iatrogenic maturity) -First day of the **last normal menstrual period**. »» Regular and normal periods? »» Oral contraceptive pills? »» Lactation? **ULTRASONIC ESTIMATION OF EDC** (A) **1st trimester**: - The best & most accurate. - Measure crown-rump (CRL ± 5 days). (B) **2nd trimester:** - (BPD, HC, abdominal circumference (accurate for weight of fetus), FL ± 10 days). (C) **3rd trimester**: - Much less accurate.

ROUTINE LAB TESTS	-Hepatitis screen:
-Hemoglobin/ hematocrit (low, high - why?). -Blood type & Rh (Rh-negative women). -Antibody screen (Kell (may lead to hemolytic anemia in newborn), Duffy, E, S….). -Urinanalysis: screen for bacteruria. »» Urine culture, if indicated -Rubella titer: →Highly contagious disease. →Congenital rubella syndrome is now rare. →10-15% seronegative (should be immunized 2-3 months before conception/in postpartum period.	→Hepatitis B-sAg: transmit to the fetus mainly during birth. →Many of those babies become carrier & can develop chronic hepatitis. →Hep B Ig & vaccine within 12 hrs of life. →Hepatitis C. -Serologic tests for syphilis (VDRL). -HIV antibody (with consent). -Blood sugar, random. -Pap smear.

REVISIT	
HISTORY -Brief history to uncover any new problems. -Ask about pain, contractions, vaginal discharge, fetal movements… -Specific questions, those with medical problems or known complications. -Counseling for those desiring sterilization if patient has chronic problem	**PHYSICAL EXAMINATION** -Weight & blood pressure. -Examine the gravid uterus. -Measure fundal height (IUGR). -Determine fetal lie & presentation (3rd trim). -Estimate fetal weight (small vs large baby). -Auscultate fetal heart tones (sunicade). -Pelvic examination, if indicated.
LABORATORY TESTS -Hematocrit/ hemaglobin, »» Repeat at 28 & 36 weeks, or if indicated. -Urine dipstick on each revisit »» Presence of significant proteinuria (PET) »» Presence of glucosuria (GDM). »» Presence of leukocytes (UTI). Antibody screen, if Rh-negative women »» Repeat at 28 & 34 weeks, if negative »» Give Anti-D immune globulin. -Glucose screen, glucose tolerance test »» At 26-28 weeks. »» Repeat at 32 weeks, in high risk patients.	**ULTRASOUND DURING ANC** 1st trimester: »» Diagnose pregnancy (gestational sac+embryo,intra/extrauterine) »» Assure accurate dating. »» Fetal number (chroniocity/amniocity) »» Fetal viability. »» Adnexial mass. »» Screen for chromosomal anomalies, nuchal translucency & nasal bone. → Down's : redundant skin, fluid behind neck, hypoplastic/absent of nasal bone,

-Screening for group B streptococcus (GPS), »» Low vaginal swab (LVS) at 35-37 weeks. »» Significant reduction of early onset GBS neonatal infection.	2nd trimester: »» Detailed anomaly scan (18-20 weeks). »» Placental localization. 3rd trimester: »» When indicated (high risk pregnancy), »» Growth & fetal welfare parameters. Regular/ serial U/S: »» High risk pregnancy. »» Poor obstetric history. »» New problem during ANC (IUGR, PET, GDM…).
Then, Pregnancy is classified to be low or high risk. »» Scoring system for risk assessment	

IMPORTANT SIGNS !	**COMPLICATIONS WHICH CAN BE PREVENTED OR MINIMIZED BY GOOD ANC**
-Vaginal bleeding. -Abdominal or pelvic pain. -Uterine contractions from 20-36 weeks. -Leaking of fluid from vagina. -Decrease in fetal movements. -Severe headache or blurring of vision → pre-eclampsia -Persistent vomiting. -Fever. -Swelling of hands or face.	-Anemia due to iron or folic acid deficiency. -Urinary tract infections and pyelonephritis. -Pregnancy induced hypertension & PET. -Preterm labour and delivery. -Intrauterine growth restriction. Sexually transmitted diseases. -Rh isoimmunization. -Fetal macrosomia. -Hypoxia or fetal death from post-term birth. -Breech presentation at term.

PREGNANCY ISSUES	
Should I **take anything**? -Trace elements: Folate (start 3 months before conception), calcium, Iron (+ vit.C), multivitamins. -Dietary supplements : Protein drinks.	**Exercise?** # What is good about it? -Reduced weight gain. -More rapid weight loss after pregnancy. -Improved mood. -Improved sleep patterns.

Food to be **avoided**?
risk of LISTERIA infection which can lead to fetal baby death (chilled, ready -to-eat foods) :
-Soft cheeses.
-Takeaway chicken sandwiches.
-Cold meats.
-Pre-prepared or stored salads.
-Raw seafood.
-Smoked salmon & smoked oysters (can OK).

Fetal movement, what is the normal one?
- >10 times / day (depends on mother's condition)
-if less than that, go and check!

Some studies show,
-Faster labour.
-Less need for induction.
-Less likely to need epidural.
-Fewer operative births.

 # Take note!
Exercise does NOT increase risk of miscarriage.

Exercise common sense during pregnancy
-Take frequent breaks.
-Avoid exercise in extremely hot weather.
-Avoid unstable ground (joints more lax).
-Avoid contact sports.
-Avoid lifting weights over head.
-And weights that strain lower back muscles.

Can I **travel**?
-Travel must be completed by 36th week.
-Medical clearance needed for twins & complicated pregnancy.

Prevent DVT
-Support stockings.
-Hydration.
-Ankle rolls, walks around plane.
-Baby aspirin.

Vaginal **discharge**
-Normally increases with gestation.
-Exclude rupture of membranes.
-Canesten pessaries OK for thrush.

Stretchmark ☹
-Related to type of collagen *ie* genetic.
-May have link with pelvic floor & perineal "stretchiness"
-Goanna oil, emu oil, olive oil,vitamin E and other expensive topicals → found not to be effective

The uncomfortables ☹

Shoes won't fit, rings too tight
-85% of pregnancies have oedema.
-Rest and elevate
-risk of carpal tunnel.

Back is hurt ☹
-Posture:
→Don't slouch!, do not bend from waist.
→Choose chair with back support.
→Bra with support.
-Hot pack & panadol.
-Elastic brace supports.
-Physiotherapy review.

Sick of getting pregnant
-Check CTG & AFI when 7 days post EDC.
-Post dates IOL= 10 days after EDC.
-"Natural IOL" - does it work?
→Curry, chilli, castor oil, etc..
→Warm bath!
→Cervical sweep!

Is the baby too big?
-Fundal height = gestation +/- 2 cm.
-Engagement of fetal head.
-Liquor vs EFW.
-Assessing fetal size at term.

TYPES OF ABORTION		
	FEATURES	MANAGEMENT
THREATENED ABORTION	1. History → Mild vaginal bleeding. → No abdominal pain or mild abdominal pain 2. Examination → Good general condition. → The cervix is closed → The uterus is usually the correct size for date 3. U/S which is essential for the diagnosis Showed the presence of fetal heart activity	1. Reassurance If fetal heart activity is present, > 90% of cases will be progressed satisfactorily 2. Advice: Decrease physical activity, avoid intercourse 3. Hormones i.e. Progesterone & hCG to support pregnancy, (no proven value) 4. Anti- D: Adequate dose of anti-D should be given to all Rh –ve, non-immunised patients, whose husbands are Rh +ve 5. ANC as high risk patients Coz liable to late pregnancy complications such as APH and preterm labour.
INEVITABLE ABORTION INCOMPLETE ABORTION	1. History ➢ Heavy vaginal bleeding. ▪ with no passage of products conception (inevitable) ▪ with passage of products of conception (incomplete abortion) ➢ Severe lower abdominal pain which follows the bleeding 2. Examinations ➢ Poor general condition. ➢ The cervix is dilating and products of conception may pass thru the os ➢ The uterus may be the correct size for date (inevitable abortion) or small for date (incomplete abortion) 3. U/S → Fetal heart activity may or may not present in inevitable abortion or retained products of conception (RPOC) in incomplete abortion	1. CBC , blood grouping , XM 2 units of blood 2. Resuscitation → large IV line, fluids & blood transfusion 3. Oxytoxic drugs → Ergometrine 0.5 mg IM + Oxytocin infusion (20-40 units in 500 cc saline) 4. Evacuation & curettage. 5. Post-abortion management.
COMPLETE ABORTION	1. History ➢ Heavy vaginal bleeding → which has been stopped. ➢ Lower abdominal pain which follows the bleeding → which has been stopped. 2. Examination: The cervix is closed 3. U/S : Showed empty uterine cavity or PROP	1. Evacuation & curettage in the presence of RPOC. 2. Post-abortion management.

MISSED ABORTION	1. Most are diagnosed accidentally during routine U/S in early pregnancy. In some cases there may be a history of: ➢ Episodes of mild vaginal bleeding ➢ Regression of early symptoms of pregnancy. ➢ Stop of fetal movements after 20 weeks gestation. 2. Examination: The uterus may be small for date 3. U/S (essential for dx) diagnosed if two ultrasound (T/V or T/A) at least 7days apart showed an embryo of >7 wks gestation (CRL >6mm in diameter and gestational sac >20 mm in diameter) with no evidence of heart activity.	1. CBC , blood grouping, XM 2 units of blood 2. Platelets count: to exclude the risk of DIC NB : DIC does not occur before 5 weeks of missed abortion or IUFD and if occurred will be of mild grade 3. Options of treatment ➢ Conservative treatment: → if left alone spontaneous expulsion will occur ➢ Surgical evacuation of the uterus; by D & C: Indicated in 1st trimester missed abortion ➢ Medical termination of pregnancy: by Misoprostol (PGE1) Cytotec: Indicated in 1st & 2nd trimesters missed abortions. ➢ Cytotec vaginal (is the best) or oral tab. 200 µg, 2 tab/ 3 hrs/ up to 5 doses daily, which can be repeated next day if there is no response in the first day ➢ Subsequent surgical evacuation is needed in cases of RPOC ➢ Main side effects of cytotec: Nausea, vomit & fever. 3. Post-abortion management.
ANEMBRYONIC PREGNANCY (BLIGHTED OVUM)	➢ It is due to an early death and resorption of the embryo with the persistence of the placental tissue ➢ It is diagnosed if two ultrasound (T/V or T/A) at least 7 days apart showed after 7 weeks of gestation i.e. gestational sac >20mm, an empty gestational sac with no fetal echoes seen. ➢ It is treated in a similar way to missed abortion.	

SEPTIC ABORTION	Def: Incomplete abortion complicated by infection of the uterine contents. May be due to criminal interference. Features : Poor general condition ➢ Features of incomplete abortion: Severe vaginal bleeding with passage of product of conception, with/without hx of evacuation. ➢ Features of pelvic infection: Pyrexia , tachycardia , general malaise , lower ab pain, pelvic tenderness & purulent vaginal discharge. Bacteriology : Mixed infection ➢ The commonest organisms are : 1. Gram -ve: E.coli , strep & staph 2. Anaerobics: Bacteroides ➢ Rarely Cl. tetani - potentially lethal if not treated adequately. Types : ➢ Mild → the infection is confined to decidua : 80% ➢ Moderate →the infection extended to myometrium15% ➢ Severe →the infection extended to pelvis + Endotoxic shock + DIC 5%	1. Investigations : ➢ CBC, blood grouping, XM 2 units of blood. ➢ Cervical swabs (not vaginal) for culture and sensitivity ➢ Coagulation profile , serum electrolytes & blood culture if pyrexia > 38.5 2. Antibiotics: IV Cephalosporin + IV Metronidazole 3. Surgical evacuation of uterus → usually 12 hrs after antibiotic therapy (until reasonable tissue levels of antibiotics achieved) 4. Post-abortion management.

<table>
<tr>
<td>RECURRENT ABORTION</td>
<td>Definition: 3 or more consecutive spontaneous abortions
➢ It may presented clinically as any of other types of abortions .
Types :
➢ Primary: All pregnancies have ended in loss
➢ Secondary: One pregnancy or more has proceeded to viability (>24 weeks gestation) with all others ending in loss.
Incidence: Occurs in about 1% of women of reproductive age .
Causes
• Idiopathic recurrent abortion, in about 50%, in which no cause can be found .
• The known causes include the followings :
1. Chromosomal disorders:
➢ Fetal chromosomal abnormalities & structural abnormalities
➢ Parental balanced translocation
2. Anatomical disorders:
➢ Cervical incompetence: →congenital and aquired
➢ Uterine causes: → submucous fibroids, uterine anomalies & Asherman’s syndrome
3. Medical disorders:
➢ Endocrine disorders : diabetes , thyroid disorders , PCOS & corpus luteum insufficiency.
➢ Immunological disorders: Anticardiolipin syndrome & SLE.
➢ Thrombophilia: congenital deficiency of Protein C&S and antithrombin III, & presence of factor V Leiden.
➢ Infections
▪ ToRCH - CMV may be a cause of recurrent abortion, but ToRH are not causes of recurrent abortion.
▪ Genital tract infection e.g Bacterial vaginosis
➢ Rh – isoimmunization</td>
<td>Diagnosis :
1. History :
➢ Previous abortions : gestational age and place of abortions & fetal abnormalities.
➢ Medical history : DM , thyroid disorders, PCOS, autoimmune diseases & thrombophilia.
2. Examination :
➢ General : weight, thyroid & hair distribution
➢ Pelvic: cervix (length & dilatation) and uterine size.
3. investigations :
A. Investigations for medical disorders:
➢ Blood grouping & indirect Coomb’s test in Rh –ve women
➢ Endocrinal screening: Blood sugar , TFT & LH /FSH ratio
➢ Immunological screening: Anti anticardiolipin antibodies & lupus inhibitor.
➢ Thrombophilia screening: Protein C & S, antithrombin III levels, factor V leiden, APTT and PT.
➢ Infection screening
▪ High vaginal & cervical swabs
▪ ToRCH profile (which scientifically is unnecessary)
B. Investigations for anatomical disorders:
➢ TV/US: fibroids, cervical incompetence & PCOS.
➢ Hysteroscopy or HSG, fibroids, cervical incompetence, uterine anomalies & Asherman's syndrome
C. Investigations for chromosomal disorders:
➢ Parental karyotyping: Parental balanced translocation.
➢ Fetal karyotyping: Fetal chromosomal anomalies.</td>
</tr>
<tr>
<td></td>
<td colspan="2">Management</td>
</tr>
<tr>
<td></td>
<td>Idiopathic Recurrent Abortion</td>
<td>In the Presence of Cause</td>
</tr>
<tr>
<td></td>
<td>With support and good antenatal care , the chance of successful spontaneous pregnancy is about 60-70%</td>
<td>Treat the cause
➢ Endocrine disorders</td>
</tr>
</table>

	➢ Support : from husband, family & obstetric staff. ➢ Advice : stop smoking & alcohol intake, decrease physical activity ➢ Tender loving care ➢ Drug therapy • Progesterone & hCG: start from the luteal phase & up to 12 weeks. • Low dose aspirin (75 mg/day) start from the diagnosis of pregnancy & up to 37 weeks • LMWH (20-40 mg/day) start from the diagnosis of fetal heart activity & up to 37 ws	• Control DM and thyroid disorders before pregnancy • Ovulation induction drugs , ovarian drilling or IVF in PCOS. • Progesterone or hCG in corpus luteum insufficiency . ➢ In anti-cardiolipin syndrome: • Low dose aspirin (75 mg/day) & prednisilone (20-30 mg / day), starting when pregnancy is diagnosed till 37 weeks. • These drugs are not teratogenic. ➢ In thrombophilia: Low dose aspirin (75 mg/day) starting when pregnancy is diagnosed and low molecular weight heparin ie LMWH (20-40 mg/day) starting when fetal heart activity diagnosed & to continue both till 37 weeks . ➢ In uterine disorders • Cervical cerclage in cervical incompetence, best time at the 14 weeks of pregnancy. • Myomectomy in submucus fibroid, excision of uterine septum in septate & subseptate uterus & adhesolysis in Asherman's syndrome. ➢ In infection:: treatment of the genital tract infection. ➢ In Rh isoimmunization: Repeated intrauterine transfusion ➢ In parental balanced translocation • Explain the risk of fetal chromosomal disorders (about 30%) • Encourage to try again or adoption.

COMPLICATIONS OF ABORTION	POST-ABORTION MANAGEMENT
1. Haemorrhage . 2. Complication related to surgical evacuation ie E&C and D&C. – Uterine perforation- which may lead to rupture uterus in the subsequent pregnancy. – Cervical tear & excessive cervical dilatation – which may lead to cervical incompetence. – Infection – may lead to infertility & Asherman's syndrome. – Excessive curettage – which may lead to Adenomyosis 3. Rh- iso immunisation → if the anti –D is not given or if the dose is inadequate . 4. Psychological trauma.	In cases of incomplete, inevitable, complete, missed & septic abortions 1. Support: from the husband, family& obstetric staff 2. Anti D – to all Rh –ve, nonimmunised patients, whose husbands are Rh+ve 3. Counseling & explanation: A. Contraception (Hormonal, IUCD, Barrier) Should start immediately after abortion if the patient choose to wait , because ovulation can occur 14 days after abortion and so pregnancy can occur before the expected next period . B. When can try again : Best to wait for 3 months before trying again . This time allow to regulate cycles and to know the LMP, to give folic acid, and to allow the patient to be in the best shape (physically and emotionally) for the next pregnancy C. Why has it happened In the fiIn the majority of cases there is no obvious cause In the first trimester abortion, the most common cause is fetal chromosomal abnormality. D. Can it happen again As the commonest cause is the fetal chromosomal abnormality which is not a recurrent cause, so the chance of successful pregnancy next time in the absence of obvious cause is very high even after 2 or 3 abortions E. Not to feel guilty → as it is extremely unlikely that anything the patient did can cause abortion No evidence that intercourse in early pregnancy is harmful No evidence that bed rest will prevent it ..

<table>
<tr><td colspan="2">MISCARRIAGE
(ABORTION)</td></tr>
<tr><td colspan="2">Def: Expulsion or extraction of products of conception before fetal viability i.e. before 24 weeks of gestation.
<u>Incidence :</u>
✓ Is the commonest gynaecological & obstetric disorder
✓ About 15% of clinically recognized pregnancies end in abortion (this rise to 30% if unrecognized pregnancies are included).
✓ Most abortions occur between 8 and 12 weeks of pregnancy.</td></tr>
<tr><td colspan="2">ETIOLOGY</td></tr>
<tr><td>1st Trimester Abortion</td><td>2nd Trimester Abortion</td></tr>
<tr><td>1. Fetal chromosomal abnormalities - particularly trisomy, triploidy & monosomy
- is the commonest cause of abortion
- 50– 70 % of the first trimester abortions are due to chromosomal abnormalities
- the incidence of these abnormalities increased with the increase in the maternal age
2. Anembryonic pregnancy - Blighted ovum
3. Multiple pregnancy
4. Parental balanced translocation
5. Infections: genital tract infection , systemic infection with pyrexia & ToRCH syndrome
6. Endocrine disorders : Diabetes, thyroid disorders , PCOS & corpus luteum insufficiency
7. Uterine disorders: Uterine anomalies , submucus fibroid & Asherman’s syndrome
8. Thrombophilia: Congenital deficiency of protein C & S, & anti-thrombin III
9. Immunological disorders : Anticardiolipin syndrome and SLE
10. Cigarette smoking, anaesthetic agents & chemical agents.
11. Psychological disorders</td><td>1. Multiple pregnancy
2. Cervical incompetence (congenital & acquired)
3. Uterine anomalies and submucous fibroid
4. Genital tract infection and PROM</td></tr>
</table>

ECTOPIC PREGNANCY

INTRODUCTION

Def: Pregnancy that is implanted outside the uterine cavity
The incidence nowadays is triple the previously stated as 0.3%

SITES

- Tubes 95%, Rt. > L
  - 55% ampulla
  - 25% Isthmic
- Cervix, Rud. Horn
- Ovaries
- Peritoneal – Abdominal

CAUSES

Any delay in ovum passage until implantation stage.

- Tubal abnormalities: Diverticulae, False passages, Endosalpingitis
- ART
- Endocrine disorders: Delayed ovulation, Estrogen/Progesterone Ratio.
- Contraceptive failure: Minipill (4-6%), IUCD(4-9%), Tubal surgery (lig., constr.)
- Prior history (10-20% Recurrence)
- Pathology: PID, Endometriosis
- Others

COMPLICATION

-Tubal abortion
  - absorption in tube
  - Incomplete tubal abortion
  - Tubal blood mole
- Tubal rupture
  - intraperitoneal bleeding
  - Broad ligaments

MANAGEMENT

- I.V. line (wide bore)
- Blood, Hb, group, cross- matching
- Once diagnosed → laparatomy laparoscopy
  - Expression
  - Salpingostomy
  - Salpingectomy

DIAGNOSIS

PRESENTATIONS

- Silent –On routine examination or laparotomy
- Acute (often rupture)
- Subacute (Variable presentations

DDX

- Threatened miscarriage
- Corpus luteum cyst:

Amenorrhea, Unilateral pain, Spotting, No Pregnancy sx, Negative HCG, If ruptured – Same treatment.

- PID:

Bilateral, No amenorrhea or pregnancy sx, Signs of infection (50%)

- Acute appendicitis

No sx of pregnancy

- UTI

SYMPTOMS

Amenorrhea (6-10 wks)
Symptoms of pregnancy
Abdominal pain (99%): Generalised (45%), Unilateral (35%), Shoulder tip (25%)
Abnormal uterine bleeding (75%) Any form
Syncopal sx (35%)
Adnexal tenderness (96%)
Adnexal mass (90%)
Uterine size: Normal (70%)
- 6 to 8 wks 25%
D&C curettings: -Proliferative, secretory
- Arias-stella phenomenon

INVESTIGATIONS

- Pregnancy test
  - Serum βhCG correlates well with trophoblastCell mass.
  - Ectopic rarely ruptures when cell mass is small or βhCG levels are low.
  - In normal pregnancy values, serum βhCG > 1500 mlu/mL, must see gestational sac
  - In normal pregnancy, hCG Values doubles every 2.2 days. It doesn't occur in ectopic pregnancy.
- Blood Hb, grouping
- Transvaginal Ultrasound:
  - Intrauterine sac: - Pseudo sac (10-20%)
    - True sac (Yolk sac F. Poles)
  - Adnexial mass (90%): - Sac with fetus or no fetus
    - Echogenic mass (DDx. C.L)
  - Fluids in P.O.D: - in 80% of Ruptured ectopics
    - In 20% of normal pregnancy

** If Gest sac > 20 mm or (5-6 wk), must see yolk sac & fetal pole. if not seen, suspect either ectopic or blighted ovum

- Laparoscopy

GESTATIONAL TROPHOBLASTIC NEOPLASIA

Classification of GTN

BENIGN

- Hydatidiform mole
1. Complete mole
2. Partial mole

MALIGNANT

- Invasive mole
- Choriocarcinoma
- Placental-site trophoblastic tumor

Genetics of GTN

- Complete mole: the majority of HM are complete moles and have a 46XX karyotype, both X chromosomes are paternally derived, result from fertilization of an empty ovum by a haploid sperm
- Partial mole: the karyotype is usually triploid, often 69XXY, the remaining lesions are 69XXX or 69XYY
- Choriocarcinoma: usually aneuploidy or polyploidy typical for anaplastic carcinoma.

HYDATIDIFORM MOLE (HM)

COMPLETE HM

- Vaginal bleeding: the most common, 97%of cases
- Excessive uterine size: large for date is one of the classic signs, occurs in 50% of cases
- Toxemia: PET is observed in 27%, usually develops early in the pregnancy
- Hyperemesis Gravidarum: Occurs in 25% of cases
- Hyperthyroidism: clinically evident hyperthyroidism is observed in 7%
- Trophoblastic Embolization: respiratory distress develops in approximately 2%
- Theca lutein ovarian cysts: prominent cysts >6cm develop in 50% of cases
- Lower abdominal pain, expulsion of vesicles

PARTIAL HM

- Patients with Partial HM usually do not have the clinical features of complete HM
- In general, these patients present with signs and symptoms of incomplete or missed abortion
- The diagnosis may be made only after histologic review of the curetting.

DIAGNOSIS

- Ultrasonography is a reliable and sensitive technique of Complete HM, "Snow storm" pattern
- Serum B-HCG higher than normal pregnancy values
- Chest film
- Blood tests: FBC, BGRH, Coagulation profile, LFT, KFT

FEATURE	PARTIAL HM	COMPLETE HM
Karyotype	Most commonly 69,XXX Or 69,XXY	Most commonly 46,XX or 46,XY
PATHOLOGY		
Fetus	Often present	Absent
Amnion, fetal RBC's	Usually present	Absent
Villous edema	Variable, focal	Diffuse
Trophoblastic proliferation	Focal, slight to moderate	Diffuse, slight to severe
Clinical presentation		
Diagnosis	Missed abortion	Molar gestation
Uterine size	Small for date	50% larger for date
Theca lutein cysts	Rare	15-25%
Post molar malignant sequelae	<5%	9-20%

Treatment of HM

- SUCTION CURETTAGE

The preferred method of evacuation , regardless of uterine size in patients who desire to preserve fertility, it involves the following steps:

1. Oxytocin infusion- in the OR before the procedure
2. Cervical dilatation then Suction curettage followed by gentle sharp curettage
3. The specimens on suction and sharp curettage should be submitted separately for pathology

- HYSTERECTOMY

If the patient desires surgical sterilization, a hysterectomy may be performed with mole *in situ*. The ovaries may be preserved even though theca lutein cysts are present

- PROPHYLACTIC CHEMOTHERAPY
  - Controversial
  - Not indicated in patients with molar pregnancy because 90% have spontaneous remissions
  - may be useful in the management of high-risk complete HM, especially when hormonal follow-up is unavailable or unreliable

Follow-up of HM

- The B-HCG radioimmunoassay is the most reliable assay available for the management of patients with GTN
- Following molar evacuation or hysterectomy , patients should be followed by weekly determination of B-HCG levels until these are normal for 3 consecutive weeks and the by monthly determination until the levels are normal for 6 consecutive months

<u>CONTRACEPTION</u>

- Encourage to use effective contraception during the entire interval of follow-up.
- Intrauterine device should not be inserted until the patient achieves a normal B-hCG level
- If the patient does not desire surgical sterilization, the choice is either hormonal contraception or barrier methods

- **MALIGNANT GTN**

A) Non metastatic Disease

- ✓ Locally invasive GTN develops in 15% of patients after evacuation of a complete mole and infrequently after other gestations
- ✓ The trophoblastic tumor may perforate through the myometrium, causing intraperitoneal bleeding, or erode into uterine vessels, causing hemorrhage
- ✓ After molar evacuation, persistent GTN may exhibit features of eitherHM or choriocarcinoma
- ✓ After nonmolar pregnancy , persistent GTN always has the features of choriocarcinoma

B) Metastatic disease

- ✓ Metastatic GTN occurs in 4% of patients after evacuation of a complete mole and infrequent after other pregnancies
- ✓ Metastasis is usually associated with choriocarcinoma
- ✓ Trophoblastic tumor perfused by a network of fragile hemorrhagic vessels
- ✓ Symptoms of mets may result from spontaneous bleeding at metastatic sites

<u>Relative incidence of common metastatic sites</u>

Lungs	Vagina	Pelvis	Brain	Liver	Bowel,kidney,spleen	Other
80%	30%	20%	10%	10%	5%	5%

FIGO STAGING FOR GTN

Stage I: patients with persistently elevated hCG levels and tumor confined to the uterine corpus
Stage II: Patients with metastasis to the vagina or pelvis
Stage III: patients with pulmonary metastasis with or without uterine, vaginal or pelvic involvement
Stage IV: Patients with advanced disease and involvement of the brain, liver, kidneys, or GIT

REVISED FIGO SCORING SYSTEM

FIGO Score	0	1	2	4
Age (years)	<39	>39		
Antecedent pregnancy	HM	Abortion	Term pregnancy	
Interval from index Pregnancy(months)	<4	4-6	7-12	>12
Pretreatment hCG level (mIU/ml)	<1000	1000-10,000	>10,000-100,000	>100,000
Largest tumor size	3-4cm	5		
Metastatic site	lung, vagina	spleen, kidney	GI Brain, liver	
Number of mets	0	1-4	4-8	>8
Previous failed chemo			single drug	2 or more

The total score for a patient is obtained by adding the individual scoresfor each prognostic factor

Total score 0-6= low risk, 7 or more =high risk

Diagnostic evaluation

1. A complete history and examination
2. Measurement of the serum hCG value
3. Hepatic, thyroid and renal function tests
4. Complete blood count

Metastatic work-up

1. Achest X-Ray
2. CT scan of the abdomen and pelvis
3. CT or MRI scan of the head
4. Measurement of CSF hCG level if any metastatic disease is present and the head CT is negative
5. Selective angiography of abdominal and pelvic organs if indicated

Diagnosis of post HM trophoblastic neoplasia

1. Plateau of hCG lasts for four measurement over a period of 3 weeks or longer, i.e for days 1, 7,14 and 21
2. A rise in hCG level for three weekly consecutive measurements or longer, over a period of at least two weeks or more, i.e on day 1,7and 14
3. Histological diagnosis of choriocarcinoma
4. When hCG level remains elevated for 6months or more

Choriocarcinoma

- Highly malignant tumor with a predisposition to haematogenous spread
- Follows HM in 3-7% of cases
- The organs most frequently affected by mets are, lung, lower genital tract, brain, liver ,kidney and GIT
- Some cases manifested by intraperitoneal bleeding secondary to rupture liver or ruptured theca lutein cyst
- Patients with pulmonary mets may present with haemoptysis or respiratory failure
- CNS mets presents with neurologic signs resulting from spontaneous bleeding
- 50% of cases follow HM
- Treatment with EMA_CO

Placental Site Trophoblastic Tumor PSTT

- PSTT is a very rare and unique form of GTN, represent a neoplastic transformation of intermediate trophoblastic cells
- PSTT can occur after a normal pregnancy, abortion, term delivery, ectopic pregnancy or molar pregnancy
- Characterized by low B-hCG levels, expression of HPL is increased on histologic section and as well as in the serum
- Diagnosis is confirmed by dilatation and curettage and hysterectomy
- Most cases are confined to the uterus but mets has been reported
- Surgery is the primary treatment of choice
- Good prognosis is anticipated in cases localized to the uterus, with distant mets or delayed treatment the outcome is dismal

Management of malignant GTN

Nonmetastatic (Stage I)

1. **Chemotherapy:** Single agent chemotherapy to retain fertility
2. **Hysterectomy Plus Adjuvant Single Chemotherapy:** not preserve fertility

Protocol for treatment of stage I GTN

Initial : MTX-FA; if resistant, switch to Act-D or hysterectomy with adjuvant chemotherapy

Resistant: Combination chemotherapy or hysterectomy with adjuvant chemo, local uterine resection

Follow-up hCG: Weekly until normal for 3 week, then monthly until normal for 12 mo

Contraception: 12 consecutive mo of normal hCG values

Effective contraception during the entire interval of hormonal follow-up

Stage II and III

Low risk : primary single-agent chemotherapy

High risk : primary combination chemotherapy

Protocol for treatment of stage II and III GTN

LOW RISK:

Initial MTX-FA; if resistant , switch to Act-D

Resistant to both Combination chemotherapy

High risk

Initial Combination chemotherapy

Resistant Second-line combination chemotherapy

Follow up and contraception are the same as in stage I

Stage IV : Primary combination chemotherapy and the selective use of radiation therapy and surgery

Protocol for treatment of stage IV GTN

Initial

Brain Combination chemotherapy, Whole-head irradiation
Craniotomy to manage complications

Liver Resection to manage complications

Resistant Second-line combination chemotherapy
Hepatic arterial infusion

Follow-up hCG Weekly until normal for 3wk, then monthly until normal for 24mo

Contraception Until there have been 24 consecutive mo of normal hCG

Chemotherapy

Single-agent

1. ***Methotrexate :*** Usually given as a daily dose for 5 consecutive days for every other day for 8 days, alternating with folinic acid rescue (associated with less bone marrow, GIT, and liver toxicity)
2. ***Actinomycin D:*** Given for 5 consecutive days or every other week as a single dose

Combination chemotehrapy

EMA-CO Regimen for patients with GTN

Course 1(EMA)

Day 1 Etoposide, Actinomycin D and Methotrexate

Day 2 Etoposide, Actinomycin D and Folinic acid 24hr after start of MTX

Course 2 (CO)

Day 8 Vincristine and Cyclophosphamide

MAC III : Methotrexate-FA, Act-D, and Cyclophosphamide

Duration of treatment :

- Serum hCG is measured weekly after each course of chemotherapy
- Adequate response is defined as a falling off the hCG level by 1log after a course of chemotherapy
- Single and Combination chemotherapy should be given as often as toxicity permits until the patient achieves three consecutive normal hCG levels
- After normal hCG levels are attained, at least two additional courses of chemotherapy are undertaken to reduce the risk of relapse

Secondary Tumors: Leukemia, colon cancer, melanoma and breast cancer

Fertility outcome: No evidence that patients with GTN followed by chemotherapy have an adverse pregnancy outcome

With each subsequent pregnancy : Pelvic USS in 1st trimester to confirm normal gestation, histological review of the placenta, hCG measurement 6 weeks after completion of the pregnancy to exclude occult GTN.

PLACENTA PREVIA			
INTRODUCTION	PREDISPOSING FACTORS	CLASSIFICATION	PRESENTATION
● Incidence 1/250 deliveries 20-30% of APH ● Majority present as painless vaginal bleeding by 30 weeks of gestation ● 20% bleeding and abdominal pain ● Incidental discovery	✚ Multiparity ✚ Increased maternal age ✚ Previous placenta previa, recurrence rate 4-8% ✚ Multiple gestation ✚ Previous cesarean section ✚ Uterine anomalies	Related to internal os. ● Minor Grade I, Low lying placenta Grade II anterior, marginal ● Major Grade II posterior Grade III, partial Grade IV, central, complete.	• Painless vaginal bleeding, more severe with major degrees • Recurrent bouts of bleeding may be from early pregnancy • Malpresentation and high presenting part • Uterus is soft and not tender • Fetus is usually alive and well • More serious for mother than fetus
MATERNAL RISK	FETAL RISK	DIAGNOSIS	MANAGEMENT
● Maternal mortality 0.1% mainly from hemorrhage ● PPH ● Anesthesia ● Sepsis ● Air embolism ?? ● DIC, late occurring, late	● High perinatal mortality *** prematurity*** ● IUGR in 15-20% ● Congenital malformations doubled ● Umblical cord complication ● Malpresentation	Ultrasonography *Abdominal 95% accurate *Vaginal usually for post placenta difficult to define by abdominal ultrasound (done in hosp) * Double set up examination rarely needed in patients not actively bleeding	▪ Proper assessment of maternal condition and resuscitation ▪ In severe bleeding, emergency cesarean delivery irrespective of gestational age ▪ If bleeding after 36-37 weeks, deliver. ▪ If bleeding not severe and early pregnancy, expectant management, attempting to reach fetal maturity (36-38 wks) without risking maternal health

*Expectant Management

- Keep in hosp esp in major degree
- Steroids
- Correct anemia ? Blood transfusion
- Cross-matched blood should be available all the time
- Assess fetal well-being

*Delivery

- Delivery is by cesarean section
- ?? Anterior marginal placenta with lower margin >2cm from the internal os (by USS) may be delivered vaginally
- Observe for PPH
- Prophylaxis for Rh isoimmunization

<table>
<tr><th colspan="4">PLACENTA ABRUPTION</th></tr>
<tr><th>INTRODUCTION</th><th>PREDISPOSING FACTORS</th><th>CLASSIFICATION</th><th>CLINICAL FEATURES</th></tr>
<tr>
<td>Premature separation of the placenta (before delivery of the fetus)
Incidence: 0.5-1.5%</td>
<td>● Hypertension, mostly PET, in pregnancy
● Previous placental abruption, recurrence rate after one episode 8-17%, after two episodes 25%
● Trauma
● Polyhydromnios
● Premature rupture of memb.
● Short cord
● Smoking
● High parity and low social class
● Idiopathic</td>
<td rowspan="3">Grade 0. Asymptomatic, small retroplacental clot after delivery

Grade 1. *External vaginal bleeding
*Uterine tetany and tenderness may be present
*No signs of maternal shock
*No evidence of fetal distress

Grade 2. *External vaginal bleeding may or may not be present.
*Uterine tender and tetany
*No signs of maternal shock.
*Signs of fetal distress present.

Grade 3. &External bleeding may or may not be present.
*Marked uterine tetany.
*Persistent abdominal pain.
*Maternal shock.
*Fetal death or distress.
*Coagulopathy in 30%</td>
<td rowspan="3">● Vaginal bleeding, variable amount, no bleeding in concealed
● Abdominal pain, discomfort and backache in 65% of cases
● Uterine tetany and tenderness over placental site, more in concealed
● Normal lie and presentation
● High incidence of fetal distress and fetal death. Fetus is dead in 25-35% of cases at admission (perinatal mortality 4.4-67%)
● Blood pressure may be normal or elevated, protein urea (IUGR present in 80% of cases delivered after 36 weeks of gestation)
● Over distended uterus, rigid, difficult to feel fetal parts in concealed hemorrhage
● Evidence of skin ecchymosis in 13% of cases usually those admitted with fetal death</td>
</tr>
<tr><th>CLINICAL PRESENTATION</th><th>MANAGEMENT</th></tr>
<tr>
<td>Concealed 25-30%
Revealed 65-80%
Other:
Mild
Moderate
Severe abruption</td>
<td>▪ Ressuscitation, IV canula, IV crystalloid
▪ Cross match blood and FFP
▪ Assessment of mother, put fixed catheter, CBC, KFT, Urine for protein, and coagulation profile
▪ Assessment of fetal wellbeing, CTG
▪ Definitive treatment by delivery, assess for labour, do ARM and syntocinon infusion. Any fetal distress or deterioration of maternal condition deliver by C/S
▪ DIC, packed RBC and FFP
▪ Observe for PPH
▪ Observe urine output, risk of renal tubular or cortical necrosis</td>
</tr>
</table>

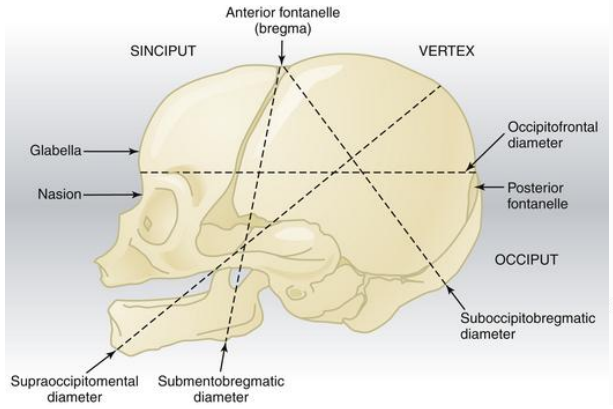
ANTEPARTUM HEMORRHAGE		
Vaginal bleeding after age of viability Blood loss is a major cause of maternal death Incidence 4%		
CAUSES		
○ Placenta previa	20-30%	
○ Abruptio placentae	15-20%	
○ Unclassified	50%	
–	Marginal separation	60%
–	Show	20%
–	Local causes	6%
–	Vasa previa	0.05%
–	Unknown cause	

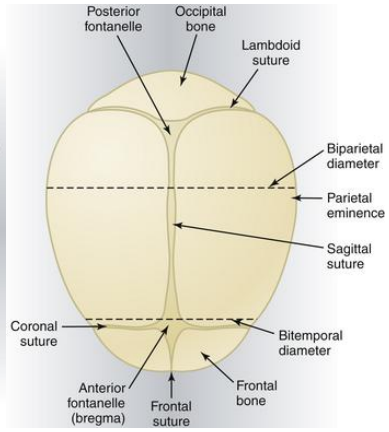
*****Vasa Previa: Fetal bleeding presented as acute fetal distress after membranes ruptured

LABOUR

Intrapartum : Phases of parturition.
PHASE 0 : Prelude to parturition
PHASE 1 (Activation) : Preparation for labour
✓ **Lightening :** 2 weeks **before** Labor , mostly *Primigravida* – Fetal Head settle in Pelvic Brim , Lightening may be **noted** by the mother as a flattening of the upper abdomen and increased prominence of the lower abdomen. ✓ **False Labor :** 4-8 weeks before Labor -"Braxton-Hicks" contractions , are - Painless, irregular , not associated with progressive cervical dilatations or effacement. ✓ **Cervical effacement :** it is **soften** due to increased water content and collagen lysis ***Thinning*** of the Cervix – As a Result , the mucus plug within the cervical canal may be released . Blood-tinged mucus from the vagina (" bloody show ") . ✓ **Rupture of membranes (ROM) :** leakage of amniotic fluid through the vagina .
PHASE 2 (Stimulation) : Process of labour
PHASE 3 (Inovulation) : Recovery

Anatomy of Fetal Head and Maternal Pelvic		
Fetal Head	the Largest & Least compressible Part of the fetus , the most important part weather cephalic or breech presentation	
	■ The skull consists of **Base** : non-compressible ,Protect the vital structures & **Vault** (cranium) : compressible , Overlap and change shape to conform the pelvis – a process known as *molding* . ■ Sutures & Fontanelles (Ant. & Post.). & Landmarks & Diameters .	
	Bregma — Anterior fontanelle Lambda — Posterior fontanelle Brow (sinciput) — Between Bregma and Glabella Glabella — Elevated area between orbital ridges Nasion — Root of Nose Occiput — Bony prominence behind lambda Vertex — Between fontanelles and Bounded Laterally by parietal eminences	





Pelvic Anatomy	■ The ***bony pelvis*** is made up of four bones: the sacrum, coccyx, and two innominates (composed of the ilium, ischium, and pubis). ■ The ***false pelvis*** is bordered by the lumbar vertebrae posteriorly, an iliac fossa bilaterally, and the abdominal wall anteriorly. Its only obstetric function is to support the pregnant uterus. ■ The ***true pelvis*** is a bony canal and is formed by the sacrum and coccyx posteriorly and by the ischium and pubis laterally and anteriorly. Its internal borders are solid and relatively immobile. *The posterior wall is twice the length of the anterior wall*. The true pelvis is the area of concern to the obstetrician because its dimensions are sometimes not adequate to permit passage of the fetus.
	PELVIC PLANES ▪ The **plane of the inlet** is bordered by the pubic crest anteriorly, the iliopectineal line of the innominate bones laterally, and the promontory of the sacrum posteriorly. The fetal head enters the pelvis through this plane in the transverse position. ▪ The **plane of greatest diameter** is the largest part of the pelvic cavity. It is bordered by the posterior midpoint of the pubis anteriorly, the upper part of the obturator foramina laterally, and the junction of the 2nd and 3rd sacral vertebrae posteriorly. The fetal head rotates to the anterior position in this plane. ▪ The **plane of least diameter** is the most important from a clinical standpoint because most instances of arrest of descent occur at this level. It is bordered by the lower edge of the pubis anteriorly, the ischial spines and sacrospinous ligaments laterally, and the lower sacrum posteriorly. Low transverse arrests generally occur in this plane.

- The **plane of the pelvic outlet** is formed by two triangular planes with a common base at the level of the ischial tuberosities. The anterior triangle is bordered by the subpubic angle at the apex, the pubic rami on the sides, and the bituberous diameter at the base. The posterior triangle is bordered by the sacrococcygeal joint at its apex, the sacrotuberous ligaments on the sides, and the bituberous diameter at the

AVERAGE LENGTH OF PELVIC PLANE DIAMETERS

Pelvic Plane	Diameter	Average Length (cm)
Inlet	True (anatomic) conjugate	11.5
	Obstetric conjugate	11
	Transverse	13.5
	Oblique	12.5
	Posterior sagittal	4.5
Greatest diameter	Diagonal conjugate	12.75
	Transverse	12.5
Midplane	Anteroposterior	12
	Bispinous	10.5
	Posterior sagittal	4.5-5
Outlet	Anatomic anteroposterior	9.5
	Obstetric anteroposterior	11.5
	Bituberous	11
	Posterior sagittal	7.5

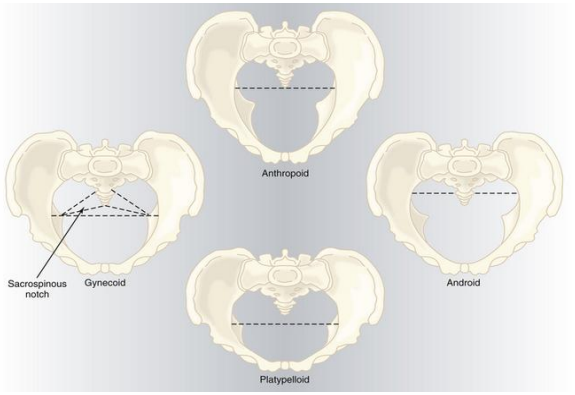
PELVIC SHAPES :

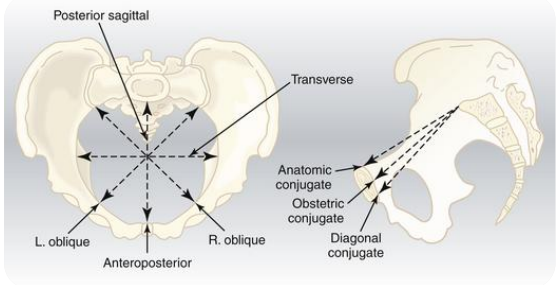
1) Gynecoid

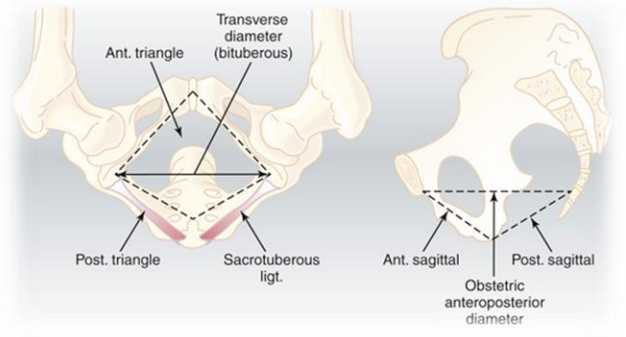
- The gynecoid pelvis is the classic female type of pelvis and is found in about 50% of women. It has the following characteristics:
- 1. Round at the inlet, with the widest transverse diameter only slightly greater than the anteroposterior diameter
- 2. Side walls straight
- 3. Ischial spines of average prominence
- 4. Large sacrospinous notch
- 5. Well-curved sacrum
- 6. Spacious subpubic arch, with an angle of about 90 degrees

These features create a cylindrical shape that is spacious throughout. The fetal head generally rotates into the occipitoanterior position in this type of pelvis.

	2) Android ■ The android pelvis is the typical male type of pelvis, and it is found in less than 30% of women and has the following characteristics: ■ 1. Triangular inlet with a flat posterior segment and the widest transverse diameter closer to the sacrum than in the gynecoid type ■ 2. Convergent side walls with prominent spines ■ 3. Shallow sacral curve ■ 4. Long and narrow (small) sacrospinous notch ■ 5. Narrow subpubic arch This type of pelvis has limited space at the inlet and progressively less space as one moves down the pelvis, owing to the funneling effect of the side walls, sacrum, and pubic rami. Thus, the amount of space is restricted at all levels. The fetal head is forced to be in the occipitoposterior position to conform to the narrow anterior pelvis. Arrest of descent is common at the midpelvis.
	3) Anthropoid ■ The anthropoid pelvis resembles that of the anthropoid ape. It is found in about 20% of women and has the following characteristics: ■ 1. A much larger anteroposterior than transverse diameter, creating a long narrow oval at the inlet ■ 2. Side walls that do not converge ■ 3. Ischial spines that are not prominent but are close, owing to the overall shape ■ 4. Variable, but usually posterior, inclination of the sacrum ■ 5. Small sacrospinous notch ■ 6. Narrow, outwardly shaped subpubic arch The fetal head can engage only in the anteroposterior diameter and usually does so in the occipitoposterior position because there is more space in the posterior pelvis
	4) Platypelloid ■ The platypelloid pelvis is best described as being a flattened gynecoid pelvis. It is found in only 3% of women, and it has the following characteristics: ■ 1. A short anteroposterior and wide transverse diameter creating an oval-shaped inlet ■ 2. Straight or divergent side walls ■ 3. Posterior inclination of a flat sacrum ■ 4. A wide bispinous diameter ■ 5. Long but small sacrospinous notch ■ 6. A wide subpubic arch The overall shape is that of a gentle curve throughout. The fetal head has to engage in the transverse diameter.







LABOUR : the process that permits a series of extensive physiologic changes in the mother, to allow the delivery of her fetus through the birth canal. This process is defined as a progressive cervical effacement and dilatation resulting from regular uterine contractions that occur at least every 5 minutes and last 30 to 60 seconds	
First stage	■ During the first stage, the progress of labor may be measured in terms of cervical ***effacement***, *cervical* ***dilation***, and ***descent*** of the fetal head. The clinical pattern of the uterine contractions alone is not an adequate indication of progress. ■ ***latent*** phase and ***active*** phase , The active phase begins when the cervix is 3 to 4 cm and ends on full dilatation (10 cm). ■ The minimal dilation during the active phase of the first stage is nearly the same for primiparous and multiparous women: **1** and **1.2** cm/hour, respectively. ■ during the first stage of labor, the entire cervical length is retracted into the lower uterine segment.

Management (stage 1)	■ MATERNAL POSITION : Ambulation , If she is lying in bed, the lateral recumbent position should be encouraged to ensure perfusion of the uteroplacental unit. ■ ADMINISTRATION OF FLUIDS : 16- to 18-gauge venous catheter hydrate the Patient with crystalloids and provide calories during labor, to administer oxytocin after the delivery of the placenta, and for the treatment of any unanticipated emergencies ■ ANALGESIA. ■ INVESTIGATIONS : CBC , Blood group , Rh type, HBV . ■ MATERNAL MONITORING . Vital signs every 2 h . ■ FETAL MONITORING. The fetal heart rate should be evaluated . ■ VAGINAL EXAMINATION : Cervical effacement and dilation, the station and position of the presenting part, and the presence of molding or caput in vertex presentations should be recorded. ■ AMNIOTOMY *. The artificial rupture of fetal membranes
Second stage	■ At the beginning of the second stage, the mother usually has a desire to ***bear down*** with each contraction. ■ MECHANISM OF LABOR ; Six movements of the baby enable it to adapt to the maternal pelvis: descent, flexion, internal rotation, extension, external rotation, and expulsion ■ VAGINAL EXAMINATION. Progress should be recorded about every 30 ***minutes*** during the second stage.
Management (stage 2)	■ MATERNAL POSITION : avoiding the supine position . ■ BEARING DOWN. ■ FETAL MONITORING. During the second stage, the fetal heart rate should be monitored continuously or evaluated every 5 minutes in patients with obstetric risk factors . ■ VAGINAL EXAMINATION. Progress should be recorded about every 30 minutes . ■ DELIVERY OF THE FETUS . “video” ■ To facilitate delivery of the fetal head, a ***Ritgen Maneuver*** may be performed . ■ Usually, the cord is clamped and cut within 15 to 20 seconds. Delayed cord clamping can result in neonatal hyperbilirubinemia as additional blood is transferred from the placenta to the newborn infant. ■ ***Episiotomy*** : It is a surgical incision through the perineum to enlarge the vagina and assist childbirth
Third stage	■ DELIVERY OF THE PLACENTA : Separation of the placenta generally occurs within 2 to 10 minutes of the end of the second stage of labor. ***Squeezing*** of the fundus to hasten placental separation is not recommended because it may increase the likelihood of passage of fetal cells into the maternal circulation. It is routine to add 20 U of oxytocin to the intravenous infusion after the baby has been delivered which reduce this bleeding
Management (stage 3)	■ Immediately after the baby’s delivery, the cervix and vagina should be thoroughly inspected for ***lacerations*** and surgical repair performed if necessary.

	■ Separation of the placenta generally occurs within 2 to 10 minutes of the end of the second stage of labor. ■ (1) a fresh **show of blood** from the vagina, (2) the umbilical cord lengthens outside the vagina, (3) the fundus of the uterus rises up, and (4) the uterus becomes firm and globular ■ It is routine to add 20 U of **oxytocin** to the intravenous infusion after the baby has been delivered.
Fourth stage	■ The ***hour immediately*** following delivery requires close observation of the patient. Blood pressure, pulse rate, and uterine blood loss must be monitored closely. It is during this time that **postpartum hemorrhage commonly occurs**, usually because of uterine relaxation, retained placental fragments, or unrepaired lacerations. Occult bleeding (e.g., vaginal hematoma formation) may manifest as pelvic pain. An increase in pulse rate, often out of proportion to any decrease in blood pressure, may indicate hypovolemia.
Management (stage 4)	■ Blood pressure, pulse rate, and uterine blood loss must be monitored closely. It is during this time that postpartum hemorrhage commonly occurs, usually because of uterine relaxation, retained placental fragments, or unrepaired lacerations

LABOR PAIN			
SOURCES OF PAIN			PATHWAYS OF PAIN DURING LABOR
1ST STAGE	2ND STAGE	3RD STAGE	• The body of uterus and cervix are supplied by autonomic nervous system (T10-L1) through Hypogastric and Pre-aortic plexuses. • Vulva, perineum are supplied by:- -somatic nerve, -Pudendal N S2-S4 -Genitofemoral N L1-L2 -Post. Femoral coetaneous N S1-S3.
✓ Uterine contractions which cause myometrial ischemia. The pain felt Through hypogastric plexuses, pre aortic plexuses to spinal cord through (T10-L1) ✓ Cervical dilatation. Felt through nerve entering to sacral root. ✓ At this stage this pain is visceral pain, diffuse, poorly localized, in lower abdomen radiated to the back.	1-Uterine contractions 2-Stretching of the vulval orifice 3-Pressure on the pelvic floor 2+3 felt through pudendal nerve (S2,3,4) mainly ilio-inguinal, genitofemoral, posterior femoral cutaneous nerve. This pain is somatic pain.	Is usually well tolerated with spontaneous placental delivery. Analgesia may be necessary for manual extraction.	

<table>
<tr><th colspan="3">ANALGESIA IN LABOR</th></tr>
<tr><th>NON-PHARMALOGICAL METHOD</th><th colspan="2">PHARMACOLOGICAL METHOD</th></tr>
<tr><td rowspan="2">➢ Psychoprophylaxis (Lamaze method): Emphasized relaxation coupled with a variety of patterned breathing techniques.
➢ Emotional support.
➢ Massage.
➢ Warm water baths.
➢ Transcutaneous Electrical Nerve Stimulation (TENS). (works on blocking pain fibers in the posterior ganglia of the spinal cord).
➢ Hypnosis.
➢ Acupuncture.

No definitive evidence.</td><td colspan="2">Best Analgesics those that provide rapid onset, with <u>Minimal to No</u> impact on: Progression of Labor, Maternal Vital Signs, Fetal Vital Signs, Maternal Passages, and Uterine contractions</td></tr>
<tr><td>INTRATHECAL ANALGESIA</td><td>INHALATIONAL ANALGESIA</td></tr>
<tr><td>Provide better analgesia than parenteral opioids:
▪ Provide rapid onset of relief.
▪ Can have a long duration of action.
▪ Typically cause no degree of motor blockade.
▪ Routinely cause no effect on the fetus.
Problems with Intrathecal Opiods.
✓ Pruritus (60-100%).
✓ Nausea (25-60%).
✓ Urinary retention (10-35%).</td><td>o More effective than opioids and is widely used.
o Most commonly used mixture is Entonox (an equal mixture of NO & Oxygen).
o Provides quick with short duration of effect.
o Not suitable for prolonged use from early labor, so most suitable is late in labor or while awaiting epidural analgesia.
o Adverse effects include nausea & light headedness.
o It is removed from the body unchanged via the lungs, and does not accumulate under normal conditions, explaining the rapid offset.
o Not suitable for prolonged use from early labor(bcoz hyperventilation may result in hypocapnea , tetany , fetal hypoxia, so most suitable is late in labor or while awaiting epidural analgesia.</td></tr>
</table>

ANALGESIA IN LABOR		
PHARMACOLOGICAL METHOD		
PARENTERAL NARCOTICS		
⦿ Easy administered. ⦿ Work best in early first stage, when pain is primarily visceral and less intense. ⦿ Cross placental barrier and so it affects fetus. ⦿ Narcotics can be given IV, IM or by Continuous infusion, in what is called PCA. ⦿ Naloxone is a narcotic antagonist, that is injected into the umbilical vein in Opioids toxicity cases		
PETHIDINE	FENTANYL	BUTORPHANOL
⦿ One of the most widely used systemic opioids for labor analgesia. ⦿ Work best in early 1st stage when pain is visceral. ⦿ T1/2(2-4 hour) ⦿ Usually given IM ⦿ Quick placental transfer ⦿ Pethidine does not inhibit uterine contraction so has less adverse effects on baby. ⦿ Diamorphine is better analgesic than pethidine but greater respiratory depressant effect on newborn so we don't use it	⦿ Synthetic, more potent than other opioids, so caution is necessary to avoid serious respiratory depression. ⦿ Rapid onset , Short action. ⦿ Continuous pulse oximetry monitoring and close nursing surveillance are also needed. ⦿ Side effect : nausea ,vomiting ,constipation ,drowsiness ,confusion. ⦿ Most serious side effect: delayed maternal gastric emptying (the danger to the mother when she require GA in the presence of full stomach under GA regurgitation and pulmonary aspiration can occur) tt by: no solid food during labor , antiemetic ,H2 blocker.	⦿ Synthetic analgesic. ⦿ 40 times more potent than Pethidine. ⦿ Duration 2-4 hours. ⦿ It antagonizes the narcotic effects of Pethidine, and therefore not given together. ⦿ Causes drowsiness and dizziness. ⦿ Less nausea and vomiting. ⦿ Less respiratory depression.

<table>
<tr><th colspan="3">ANESTHESIA IN LABOR</th></tr>
<tr><th>INTRODUCTION</th><th colspan="2">A] GENERAL ANESTHESIA</th></tr>
<tr><td rowspan="2">Anesthesia versus analgesia :
An analgesic drug such as aspirin may relieve pain but the person who takes aspirin still feels other physical sensation such as pressure , heat ,cold and vibration , in contrast anesthetic drugs blocks all these physical sensation</td><th>INTRODUCTION</th><th>BENEFITS OF GENERAL</th></tr>
<tr><td>⦿ GA isn’t used in vaginal delivery.
⦿ GA Used in C/S in certain circumstances.
• Extreme emergency situations .
• Any contraindication for regional anesthesia e.g infection at needle insertion site, prior back surgery, increase intracranial pressure, etc.
• Unexpected prolonged surgery.</td><td>⦿ Help the obstetrician to deliver the baby in fetal distress.
⦿ Situations where minutes may count for the fetus e.g. ruptured uterus, placental abruption, umbilical cord prolapse.
⦿ Situations where rapid induction maybe needed for maternal safety e.g. uncontrolled hemorrhage as in cases of: placenta previa, trauma, placental abruption, ruptured vessels.</td></tr>
</table>

ANESTHESIA IN LABOR

B] REGIONAL ANESTHESIA

SPINAL ANESTHESIA

INTRODUCTION	INDICATION	CONTRAINDICATION	COMPLICATION
⦿ Def: Introduction of a local anesthetic into the **subarachnoid space**. ⦿ A short procedure time. ⦿ Rapid onset of the block, and limited duration of action. ⦿ High success rate. ⦿ Used very late in labor.	(1) Operations below umbilicus (2) Any operation in perineum or genitalia (3) All possible operation on the leg except amputation (4) Very important to notice that spinal anesthesia is indicated for older patient with systemic disease .	⦿ Patient refusal, uncooperative patients ⦿ Hypovolemia ⦿ Clotting disorders ⦿ Septicemia ⦿ Anatomical deformities in the patient back ⦿ Neurological disease ⦿ Inadequate resuscitative drug and equipments	⦿ **Hypotension**, bradycardia if block reachs T2-T4. ⦿ **Post spinal headache (postural)** ⦿ Urinary retention ⦿ Failure of technique . ⦿ Epidural or subarachnoid hematoma ⦿ Spinal cord trauma or infection ⦿ Rarely, convulsions and blindness

EPIDURAL ANESTHESIA

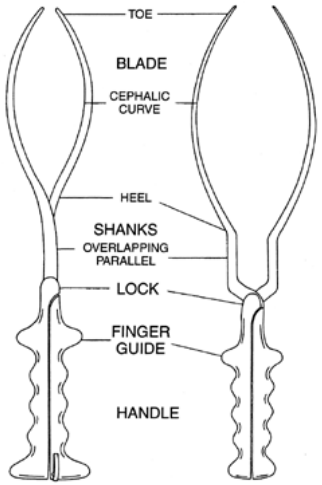
INTRODUCTION	INDICATION	COMPLICATIONS
⦿ Most effective form of labor pain relief, used in the first and second stages of labor. ⦿ No direct effect on fetus & doesn't cross placenta. ⦿ Injected into the epidural space between L2-L3 or L3-L4 interspace. ⦿ A test dose is given to confirm the catheter position, if no unwanted signs is observed after 5 min of injection, a loading dose can be administered. ⦿ Analgesia for the pain of labor and vaginal delivery necessitates a block from the T10 to the S5 dermatomes. ⦿ For cesarean delivery, a block extending from the T4 to the S1 dermatomes is desired. ⦿ The spread of the anesthetic depends upon the location of the catheter tip; the dose, concentration, and volume of anesthetic agent used, and whether the mother is head-down, horizontal, or head-up.	⦿ Pain. ⦿ Prolonged labor. ⦿ Maternal hypertensive disorder. ⦿ When there is high risk of operative intervention. ⦿ High risk fetus group. CONTRAINDICATION ⦿ Patient refusal. ⦿ Coagulopathy disorders. ⦿ Local or systemic infection. ⦿ Hypovolemia.	MATERNAL IMMEDIATE ⦿ Hypotension ⦿ Complete motor and sensory paralysis. ⦿ LA-induced convulsions, uncommon but serious. ⦿ LA-induced cardiac arrest. DELAYED ⦿ Postural puncture headache. ⦿ Backache; there is good evidence that epidural does not cause backache. ⦿ Urinary retention; To avoid this a catheter is placed early. ⦿ Tingling in hands or fingers. ⦿ Epidural abscesses; extremely rare. FETAL No direct adverse effect on fetus. Fetal distress can occur if maternal hypotension occurred.

SPINAL ANESTHESIA	EPIDURAL ANESTHESIA
ADVANTAGE	ADVANTAGE
⦿ Faster, Technically easier ⦿ More reliable - Defined endpoint, Minimal chance of patchy block ⦿ Denser block ⦿ Lower drug exposure for mother and fetus ⦿ No chance of systemic toxicity	⦿ Can tailor duration to need ⦿ Lower chance of postdural puncture headache ⦿ Beneficial in patients with cardiac and hypertensive disorders
DISADVANTAGE	DISADVANTAGE
⦿ Limited duration ⦿ Higher chance of postdural puncture headache	⦿ Slower onset ⦿ Higher risk of systemic toxicity ⦿ Risk of high spinal ⦿ Risk of "patchy block"

ANESTHESIA IN LABOR		
C] LOCAL ANESTHESIA		
PARACERVICAL BLOCK		PUDENDAL BLOCK
INTRODUCTION	COMPLICATION	⦿ Def: Bilateral transvaginal LA injection to block the pudendal nerve as it pass through the ischial spine (originate from S3-S4), ⦿ Relatively safe and simple method of providing analgesia for spontaneous delivery. ⦿ The successful pudendal block will allow pinching of the lower vagina and posterior vulva bilaterally without pain. ⦿ Pudendal block usually does not provide adequate analgesia when delivery requires extensive obstetrical manipulation. ⦿ Complications: Intravascular injection, hematoma, and rarely infection.
⦿ Def: Bilateral transvaginal local anestethetic injection to block the frankhouser's ganglion lateral to cervix. ⦿ Provides satisfactory pain relief during the first stage of labor. ⦿ Anesthetic agent is injected into the cervix laterally at 3 and 9 o'clock. ⦿ Has a short duration of action, so paracervical block may have to be repeated during labor. ⦿ Because the pudendal nerves are not blocked, another analgesic procedure may be needed. ⦿ Usually, we use: • Lidocaine • Chloroprocaine, 5 to 10 ml of a 1% solution. (Bupivacaine is contraindicated because of ↑ risk of cardiotoxicity.)	⦿ Fetal bradycardia (lasts up to 30 min) ⦿ Results from decreased placental perfusion, due to the drug-induced vasoconstriction and therefore should not be used in situations of potential fetal compromise.	

ASSISTED VAGINAL DELIVERY

<table>
<tr>
<td>Indication
- Maternal exhaustion
- Prolonged 2nd stage of labor >>
• nulliparous woman → this is defined as lack of continuing progress for 2 hours without regional/epidural anesthesia or 3 hours with regional/epidural anesthesia.
• multiparous woman → it's defined as lack of continuing progress for 1 hour without regional/epidural anesthesia or 2 hours with regional/epidural anesthesia.
- Fetal distress or compromise
- To shorten the 2nd stage of labor for maternal benefit; such as HTN, cardiac disorders, pulmonary disease, in which strenuous pushing –in the 2nd stage- is considered hazardous. Here we usually use forceps.
- To stabilize the after coming head during a breech delivery.</td>
<td>Requisition
- Complete cervical dilation & rupturing of membranes
- Well-trained obstetricians
- Baby is alive
- knowing fetal position & presentation which must be cephalic
- Engagement of the presenting part
- Proper analgesia
- Easy access to the operating theatre, or do it in the OR; to do C/S in case of assisted VD failure.
- Empty urinary bladder</td>
</tr>
<tr>
<td colspan="2">Forceps
- Instruments designed to provide traction & rotation of the fetal head when the expulsive efforts of the mother are insufficient to accomplish safe delivery of the fetus.
- Components: 2 blades, 2 shanks, lock & handle. Blades have cephalic curve –designed to accommodate the curvature of fetal head-, & pelvic curve. Note: in C/S forceps there is only cephalic curve
- Types:
• Classic/standard forceps >> used to help delivery by applying traction to the fetal skull. Ex: Simpson forceps.
• Specialized forceps >> Ex: Kielland forceps (rotational forceps) – not used now-, Piper forceps (for breech delivery of the after coming head)</td>
</tr>
</table>



1- Simpson's forceps

2- Kielland forceps

3- Piper forceps

Types of forceps operation	Application of forceps
-Outlet forceps "Wrigley forceps" >> scalp is visible at the introitus without separating the labia, fetal head is at perineum, fetal skull is at pelvic floor, sagittal suture is in antero-posterior or right/left occiput anterior or posterior position, & rotation of fetal head doesn't exceed 45 degrees. -Low forceps >> leading part of the fetal skull is at station +2 cm or more. -Mid forceps >> fetal head is engaged, but the leading point of the skull is above station +2 cm.	• Fetal sagittal sutures must be parallel to the shanks. • Hold one blade in each hand; left blade in the left hand (& this will be applied into the left side of maternal pelvis), & right blade in the right hand (& this will be applied into the right side of maternal pelvis). • Start by the left one guided by your hand in between vaginal mucosa & the presenting part sliding it on your hand to decrease soft tissue trauma. Then the same process is applied to the other one. • Pulling the forceps must be in down & backward direction during contraction & stop in between them • Pulling must be applied by the force of your forearm. • Episiotomy might or might not be needed

Vacuum

An instrument that uses a suction cup that is applied to the fetal head.

- Types : metallic, plastic, cylastic
  - these have different diameters.
  - plastic & cylastic ones are compressible, so if compared to metallic ones they have >> less incidence of laceration & easier to apply
  - BUT, risk of failure rate while pulling malpositioned fetus is higher in plastic & cylastic,
  - if fetus in occipitoposterior position, then it's better to use metallic vacuum
- After confirming that no maternal tissue is trapped between the cup & the fetal head , the vacuum seal is obtained using a suction pump
- Traction is applied with the help of maternal pushing efforts parallel to the axis of birth canal

Application of vacuum

- You should apply the cup into a leading point in between the anterior & posterior fontanel closer to the posterior one (3 cm from the anterior fontanel over the sagittal suture), otherwise, fetal head will be deflexed & acquire another larger diameter (other than the usual/normal suboccipitobregmatic one) to be the widest diameter that will pass through pelvic inlet. Also this might lead to fetal asynclitism.
- Vacuum creates negative pressure on the fetal scalp to pull it. The maximum pressure that is applied is 0.8 kg\m2 as a safety measure, if that pressure was exceeded, the cup will pop off to protect the fetus
- perform pulling of the fetus during contractions & keep holding up in between contractions

- ✓ Vacuum is contraindicated in preterm delivery; because the preterm fetal head & scalp are more prone to injury from the suction cup
- ✓ NEVER use vacuum for delivery of fetuses presented by face or breech presentations. But it's suitable for all vertex presentations
- ✓ stop the procedure when there is no progress; that's when the bony part is not going down.
- ✓ Maximum number of pop-offs allowed is 3-5, otherwise stop
- ✓ Maximum time allowed for vacuum is 20 min, then if failed do C/S
- ✓ the higher the number of pulls while the baby is still in, the lower the chance to succeed

How to choose what to use, Vacuum or forceps? This depends on:

1- attending obstetrician skills & well-training

2- position of the fetal presenting part.

- if the fetal presenting part needs rotation to a certain degree then it's preferable to use vacuum not forceps.
- if you expect the presenting part will need large angle of rotation, then you can allow spontaneous rotation of it while pulling it with the vacuum.

Complication → Forceps have more maternal complications, while vacuum have more fetal complications

FORCEPS	VACUUM
Maternal complications - trauma & laceration to vaginal & perineal tissue & upper genital tract - anal sphincter injury & damage	Maternal complications lacerations, rarely
Fetal complications - Soft tissue trauma - skull fracture - facial palsy; due to compression of facial nerve	Fetal complications - Cephalohematoma - shoulder dystocia - subgaleal hemorrhage - intracranial hemorrhage - skull fracture - retinal hemorrhage

- ✓ failure rate of forceps is less than vacuum; it's 7% for forceps & 12% for vacuum
- ✓ REMEMBER >> Your AIM always is to deliver a healthy baby with the least possible complications to both baby & mother

<table>
<tr><th colspan="4">PRETERM BIRTH</th></tr>
<tr><th colspan="2">CLASSIFICATION</th><th>ETIOLOGY</th><th>CLINICAL FEATURES</th></tr>
<tr><th>GESTATIONAL AGE</th><th>BIRTH WEIGHT</th><td rowspan="2">1) Infection – mostly subclinical infxn of choriodecidual space
2) Overdistension of uterus – polyhydromnios, multiple pregnancy
3) Vascular (placental abnormalities)
4) Intercurrent illness – pyelonephritis, pneumonia. By either direct spread or indirect chemical triggers eg endotoxins.
5) Cervical weakness
6) Idiopathic – esp in mild preterm births
7) Iatrogenic</td><td rowspan="2">1. Cramping lower ab pain that starts irregular & with time ↑ in freq & intensity & becomes more regular.
2. Low back ache
3. Bloody vaginal discharge</td></tr>
<tr><td>Mild preterm
• Late preterm: 34-36th wk
• Moderate preterm: 32-33rd wk
• Very preterm: 28-31st wk
• Extremely preterm: 24-27th wk</td><td>Low birth weight: <2.5kg
Very low birth weight: <1.5kg
Extremely low birth Weight: <1kg</td></tr>
<tr><th colspan="2">MANAGEMENT</th><th colspan="2">INVESTIGATIONS</th></tr>
<tr><td colspan="2">1. Admit at least 24h
2. According to GA
<34wks – antenatal steroids (given in 2 doses 12h apart)
3. Prophylactic antibiotics (erythromycin until culture indicates otherwise)
4. Tocolytics – for 48h until steroids works or till transfer to a hosp with good NICU facility</td><td colspan="2">1. Sterile speculum exam – amniotic fluid pooling
2. Transvaginal USS – asses cervical dilation.
Normal cervix ~3.5cm in length.
3. Vaginal swab for GBS
4. Urinalysis & culture (UTI main cause of preterm labor)
6. fetal fibronectin (fFN)
When to ask?
- cervical length >3.5cm& not in labor – no need
- cervical length <2n5cm & in labor – no need
- 2.5 < cervical length < 3.5cm – need fFN</td></tr>
</table>

"Braxton-Hicks contraction" @ False Labor @ Practice Contractions - Sporadic uterine contractions that start around 6 weeks, to aid body in its preparation in birth. Infrequent, irregular, only mild cramping.
(Preterm labor ↑ progressively & becomes more intense & regular with time)

<table>
<tr><th colspan="4">PREMATURE RUPTURE OF MEMBRANE</th></tr>
<tr><td colspan="4">Amniorrhexis (spontaneous rupture of membranes) before the onset of uterine contractions.
Preterm PROM (PPROM) – preterm (<37 wks) with ruptured membrane, with or without contractions.</td></tr>
<tr><th>RISK FACTOR</th><th>HX & PE</th><th>LAB TESTS</th><th>CONSIDERATIONS</th></tr>
<tr>
<td>o Hx of previous PPROM
o Vaginal & cervical infxn
o Antepartum bleeding
o Cigarette smoking have a particularly strong association with PPROM
o Abnormal memb physiology
o Incompetent cervix
o Nutritional deficiencies</td>
<td>Hx: Vaginal loss of fluid
R/O episodic urinary incontinence, leukorrhea or loss of cervical mucus plug.
PE: Pooling of amniotic fluid in posterior vaginal fornix.
Confirm by:
1) NItrazine paper (alkali → blue)
2) Microscopic exam (ferning)
Sterile vaginal speculum.
*No digital vaginal exam!!
USS: R/O fetal anomalies, assess GA & amniotic fluid volume.</td>
<td>Pulm maturation studies.
Gram stain & culture.</td>
<td>Management depends a lot to GA at time of memb rupture.
Also quantity of remaining amniotic fluid.

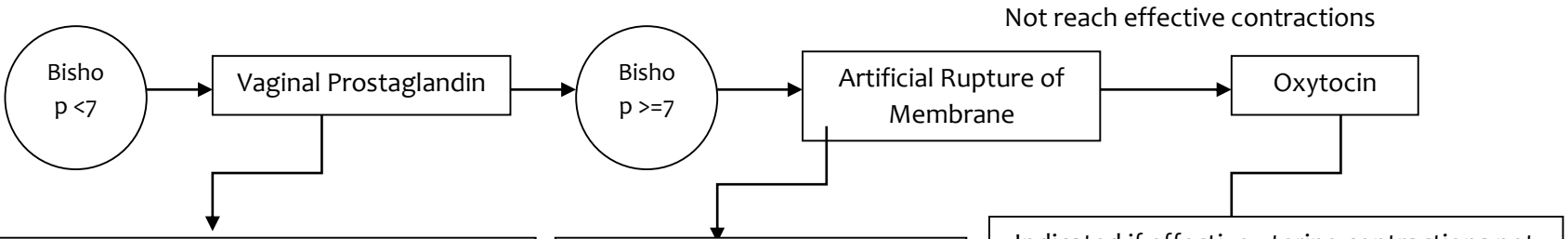
Amniotic fluid index (AFI):
Vertical axis of amniotic fluid present in four quadrants.
Abnormal is <5cm.</td>
</tr>
<tr><th colspan="4">MANAGEMENT</th></tr>
<tr><th>GA <24 wks (Pre-viable PROM)</th><th colspan="2">24wks > GA > 36wks (Preterm PROM)</th><th>GA 36wks or more (PROM)</th></tr>
<tr>
<td>Risk of dev of pulmonary hypoplasia – due to fetal crowding with thoracic compression, restriction of fetal breathing and disturbances of pulm fluid production & flow.
Positional skeletal abnormality eg talipes equinovarus.</td>
<td colspan="2">A] Conservative Expectant Management - To continue pregnancy until the lung profile is mature. Diagnose chorioamnionitis at an early stage to minimize fetal & maternal risks.
“Chorioamnionitis” – Maternal temp >38°C with no other sites of infxn, fetal tachy, tender uterus, uterine irritability on NST. Presence of bacteria by Gram/culture.
Ampicillin/erythromycin prolongs interval of delivery in pt with PPROM.
When diagnosed, ampicillin + gentamycin after taken enough cultures. Then induce labor.
C-section if cervix unfavorable, fetal involvement or presence of active genital herpes.
B] Tocolytic Therapy – To gain time for pulm maturation. Unsuccessful if with infection.
C] Cortisteroids Use - Given to pt with PPROM only up to 32 wks GA.
D] Outpatient Management
Can consider after inpatient observ for 2-3d without any evidence of infxn.
Rules of eligibility: Pt reliable, fully informed of risks, prepared to participate in her own care. Fetus vertex. Cervix closed.
Restrict physical activity. No coitus. Monitor temp at least 4x/d. Rush to hosp if >37.8°C. Seen weekly to measure temp, NST after 28wk, evaluate baseline fetal heart rate & AFI.
Contraindicated in oligohydramnios.</td>
<td>Induce labor after 6-12h if no spontaneous contractions occur.</td>
</tr>
</table>

INDUCTION OF LABOR			
Indicated in 10-20%			
INDICATION	CONTRAINDICATION		COMPLICATION
When delivery is safer to mother & fetus than continuation of pregnancy. 1. Postdate 2. Pre-eclampsia 3. PROM 4. Chorioamnionitis 5. IUGR 6. IUFD 7. Fetal anomalies 8. DM 9. Abruptio placenta 10. Rh isoimmunication	ABSOLUTE	RELATIVE	1. Hyperstimulation → fetal distress & uterine rupture. 2. Failed induction → increased incidence of C/S. 3. Prolonged labour → instrumental delivery & PPH. 4. More painful → more analgesia 5. Prematurity 6. Infection
	1. Placenta previa 2. Previous 2C/S, previous 1 due to recurrent cause, previous classical C/S 3. Abnormal antenatal CTG 4. Transverse/oblique lie 5. Absolute contracted pelvis 6. Active genital herpes infection. 7. Tumor occupies pelvis 8. Cervical carcinoma 9. Successful pelvic floor repair & successful surgical rx of stress incontinence.	1. Severe pre-eclampsia 2. Breech presentation. 3. Multiple pregnancy 4. Grand multipara 5. Polydroamnios 6. Presenting part above pelvic inlet.	

Bishop Score: Assess cervical condition & station of the head, in order to choose the best method for induction. Total score 13.

BISHOP SCORE	0	1	2	3
Cervical dilatation	Closed	1-2cm	3-4cm	>5cm
Cervical length	>2cm	2-1cm	1-0.5cm	<0.5cm
Cervical consistency	Firm	Medium	Soft	
Cervical position	Posterior	Central	Anterior	
Station of the head	-3	-2	-1-0	Below ischial spine
Bishop score <7 – unfavorable cervix. Bishop score 7 or more – favorable cervix				

METHOD OF INDUCTION



- PGE2 (Prostin) – most commonly used.

PGE1 (Misoprostol)

- Vaginal PGE2 pessory 3mg or

Intracervical PGE1 gel 0.5mg

- Dose can be repeated every 4-6h for a max dose of 3doses in 24h.
- Main complication: Uterine hyperstimulation → Uterine rupture & Fetal Distress.
- If cervix remains unfavorable despite max dose of PG (3doses/24h), re-evaluate pt & if there's no urgent indication for delivery, repeat the PG next morning.

Induces labor by

- ↑ PG release from fetal memb & decidua, & by mechanical descent of fetal head
- ↑ oxytocin from posterior pituitary (Ferguson reflex)

Complications: Cord prolapsed, placental abruption, infection.

Indicated if effective uterine contractions not obtained after 1-2 h of AROM.

Complications:

1. Uterine hyperstimulation → Uterine rupture & Fetal Distress
2. Hypotension if given IV bolus dose.
3. Neonatal jaundice – if total oxytocin >20 units.
4. Water intoxication – if total amt of fluids >1.5L → confusion, convulsion, coma, death.

How given:

Start 2mU/min, double the dose every 30min. Never exceed 32mU/min (multipara) & 64mU/min (primipara).

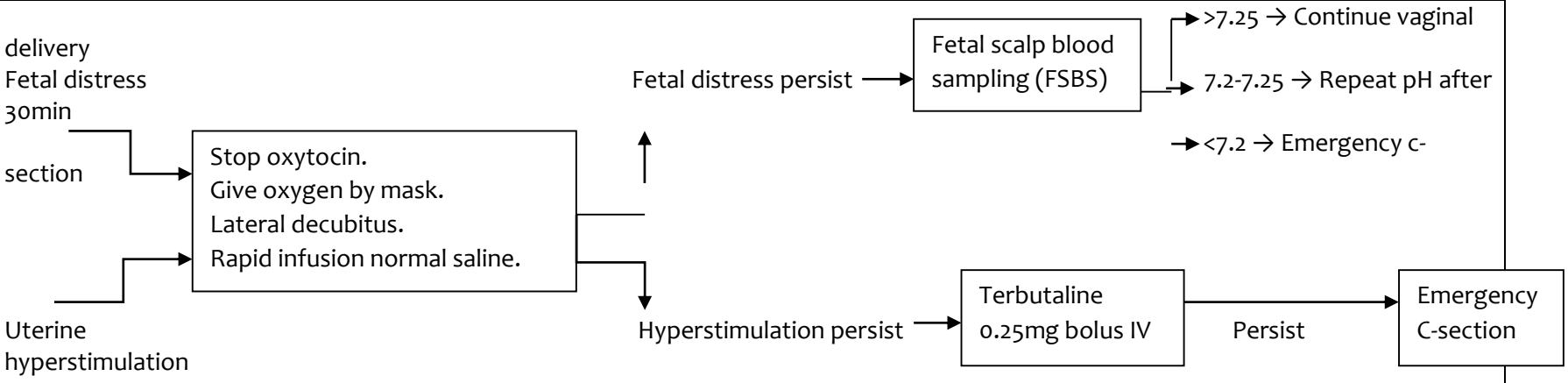
When effective contractions reach, keep lowest effective dose.

Maintain infusion after delivery until 3rd stage labor passed safely to prevent atonic PPH.

*Effective uterine contractions: 3-4 contractions, each lasting 50-60seconds in 10minutes.

MANAGEMENT OF PATIENTS FOR INDUCTION OF LABOR	
BEFORE INDUCTION	DURING INDUCTION
1. Counseling & explanation. 2. Hx – Assess GA & R/O contraindications of induction. 3. Obstetrics exam – Assess lie, presentation, engagement. 4. Vaginal exam: Assess Bishop score & pelvic adequacy. 5. Ultrasound: Assess fetal age, wellbeing, amt of liquor & placental site. 6. CTG: Assess fetal wellbeing.	1. Good selection of method of induction (Bishop score) 2. Proper dose of PG or Oxytocin. 3. Monitoring of labor – Fetal wellbeing, uterine activity, progress of labor & maternal wellbeing. 4. Adequate pain relief – Best epidural

TREATMENT OF HYPERSTIMULATION & FETAL DISTRESS



PROLONGED PREGNANCY

Post-date pregnancy: Continuation of pregnancy beyond 40 completed weeks
Post-term pregnancy: Continuation of pregnancy beyond 42 completed weeks
Incidence 5-10% pregnancies.

ETIOLOGY

Majority of cause – no underlying cause i.e. physiological continuation of the pregnancy.
Extremely rare cases may be due to anencephaly, fetal adrenal hypoplasia or to placental sulphatase enzyme deficiency.

RISKS

A] Placental insufficiency & hypoxia which leads to:
1. Increased perinatal mortality (PNM)
2. Meconium aspiration syndrome
3. Oligohydroamnios & cord compression

B] Increased fetal weight & ossification of skull with decreased moulding, which leads to:
1. Prolonged labour and failure to progress which leads to ↑ incidence of C/S.
2. Shoulder dystocia – with its neonatal & maternal risks.
   a) Maternal risks – vaginal & cervical lacerations & rupture uterus
   b) Neonatal risks: neonatal asphyxia & death, cervical cord injury, brachial plexus injury (erb's palsy in C5&C6, klumpk's palsy in C8&T1, phrenic nerve injury in C4), clavicular & humeral fractures.

MANAGEMENT

BEFORE DELIVERY	DELIVERY
1. Counseling & explanation: explain risks on the fetus. 2. Hx – for accurate assessment of GA and to exclude C/I for induction. 3. Obstetric exam: Assess lie, presentation & engagement. 4. Vaginal exam: Assess Bishop score & pelvic adequacy. 5. Ultrasound: at 40.41 & 42 wks, to assess amt of liquor, fetal wellbeing & w8. 6. CTG: every 3days after 40wks, to assess fetal wellbeing.	1. In uncomplicated postdate pregnancy, pt should be delivered at 41wks + 3-7days. 2. Method of delivery either induction of labour (method of induction depends on Bishop score) or by C/S if there is C/I for induction. 3. If delivery by induction of labour, a senior obstetrician should attend delivery due to risk of shoulder dystocia and a pediatrician should attend due to risk of meconium aspiration.

ASSESSMENT OF GA

ANTENATAL METHODS	POSTNATAL METHODS
1. First day of LMP – reliable in 50% of pregnancies. 2. Ultrasound – best is CRL between 7-13wks, then BPD & FL between 13-26wks & then BPD & FL after 26wks. 3. Clinical – onset of early pregnancy sx, early bimanual exam, quickening & serial fundal height.	1. Dubowitz score – include an assessment of the physical & neurological features of the newborn. 2. Farr score – which include an assessment of the physical features of the newborn.

POSTPARTUM HEMORRHAGE

Def1: Any blood loss of >500mL (vaginal) and >1000mL (caesarian) following delivery. (Blood loss estimation inaccurate)
Def2: 10% drop of PCV. (Depends on timing of test after onset)
Def3: Any bleeding which result in the signs & sx of hemodynamic instability. (Blood loss response differ from ppl – esp anemia, PET, cardiac diseases, dehydration)

INCIDENCE	HEMOSTATIC MECH	TYPE	RISK FACTORS
The leading cause of maternal mortality. Occur in 4% of deliveries.	At term, the estimated blood flow to the uterus 500-800mL/min. 10-15% of Cardiac output. Natural hemostatic mech: 1) Contraction & retraction of myometrial fibers 2) Hypercoagulable state in late pregnancy 3) Integrity of the genital tract	✓ Primary (Early) PPH: Excessive blood loss within 24h of delivery (esp 1st 6h). ✓ Secondary (Late) PPH: Excessive blood loss between 24h-6wks postpartum (esp 2nd wk)	• Previous PPH • Abruptio • Retained placenta • Failure to progress during 2nd stage • Placenta accreta • Laceration • Instrumental delivery • LGA • HTN disorders • Induction & Augmentation of labor

ETIOLOGY

Bleeding mostly from endometrial spiral arterial arterioles & decidual veins that supplied & drain intervillous spaces of placenta.
Causes of primary PPH: 1) Tone; 2) Trauma; 3) Tissue; 4) Thrombosis; 5) Uterine Inversion

TONE (ATONY, 75-80%)	TRAUMA	TISSUE	THROMBOSIS	UTERINE INVERSION
Failure of contraction & retraction of myometrial muscle fibers. Predisposing factors: 1) Uterus overdistention – Multiple gestation, LGA, Polyhydroamnios. 2) Prolonged labor → fatigue. 3) Drugs – Halogen anest, nitrate, NSAID, $MgSO_4$, Nifedipine. 4) Placenta previa - ↓content of musculature of the wall. 5) Bacterial toxins – chorioamnionitis, endometritis & septicemia. 6) Fibroid esp intramural. 7) Grand multipara >5 8) Precipitous labor 9) Abruptio esp concealed coz interstitial bleeding.	Vascular beds in genital tract are engorged during pregnancy. 1) Laceration in cervix & vagina – spontaneous/ instrumental/ manipulation of fetus/ LGA/ precipitous labor. 2) Uterine rupture → intraperitoneal bleeding. 3) Hematoma – perineal, vaginal or broad ligament hematoma.	- Retention of part of placenta. 50% cases of 2ndary PPH. - Incomplete separation of accreta or precreta.	Preexistent: ITP, TTP. Acquired: 1- Abruptio (Leak AF) 2- HELLP (low Plt) 3- Sepsis → DIC 4- Dilutional coagulaopathy 5- Amniotic fluid embolism – intravascular infusion of small amt of AF.	Just after 2nd stage of labor, due to uterine atony, cervix is open & placenta attached. Inexperienced doc exert fundal pressure while pull umbilical cord before complete placental separation. Inversion → Traction of peritoneal structure → Vasovagal response → Vasodilation → Hypovolemic shock.

CLINICAL MANIFESTATION	COMPLICATION	DDx OF CAUSES
▪ Heavy vaginal bleeding ▪ ↓ BP ▪ ↑ HR ▪ ↓ RBC count (Hct) ▪ Swelling & pain in tissues in the vaginal & perineal area.	Anemia Hypotension & hypovolumic shock Renal failure Risks of blood transf Surgery complications & sepsis Sheehan syndr Venous thrombosis & embolic effects (coz of surgery, bed rest & hypercoagulable state)	Hx: Ask risk factors of uterine atony & coagulopathy. Exam: Soft boggy & large uterus with profuse vaginal bleeding? Atony. Bright red bleeding b4 separation of placenta? Trauma. Abdominal / vaginal mass increasing in size? Hematoma. Bleeding severe, bright red, no clots but uterus contracts well? Coagulopathy. Cupping of fundus or non-palpable fundus? Uterine inversion. Fever & tenderness? Endomyometritis. If no cause identified → Manual exploration of genital tract.

MANAGEMENT OF PRIMARY POSTPARTUM HEMORRHAGE

Identification of those at risk of PPH. Start prophylactic measures during labor to minimize maternal mortality.
1. CBC – Hb & Plt (correct anemia if present). 2. Blood typing & Ab screening (Xmatch 2-6units blood) 3. Insert large bore IV line.

ROUTES OF MANAGEMENT

RESUSCITATION	EVALUATE	SURGICAL	PROGNOSIS
Oxygen by mask. 2 large bore IV lines. Central venous line. Draw blood – CBC, coagulation screen, urea, creatinine, electrolyte. Immediate fluid replacement with NS or RL. Transfuse RBC as available & appropriate. FFP if abnormal coagulation test results & sites oozing. Cryoprecipitate if abnormal coagulation tests not corrected with FFP. Plt concentrates if plt count <50x10/L & bleeding continues.	Monitor pulse, BP, Urine output, blood gases, level of consciousness. Order regular CBS counts & coagulation tests to guide blood component therapy.	1. Laparotomy to drain free blood & inspect any injury & repair. 2. Uterine artery ligation 3. Internal iliac (inferior hypogastric) artery ligation. 4. Total hysterectomy 5. Selective arterial embolization	Depends on cause of PPH, duration, amount of blood loss & effectiveness of rx. Prompt dx & rx.

MANAGEMENT OF UNDERLYING CAUSE

TONE	TRAUMA	TISSUE	COAGULOPATHY
Assess uterine size & tone. Bimanual – express clots, stimulate uterine contractions. Empty bladder. Uterine artery ligation, selective arterial embolization or subtotal/ total hysterectomy. Uterotonic drugs.	Cervical & vaginal lacerations → absorbable continuous stitch. Large lower genital tract, unstable broad lig & retroperitoneal hematoma → incision, drain, ligation, packing. Uterus rupture → sub/TAH.	Removed even if bleeding stopped with use of uretrotonics. Diffcult without GA. Resuscitate adequately. Manual explore. Vaginal hand in situ to ↓ discomfort, infxn, trauma. Adherent? Curettage.	Confirm by risk factors & abnormal coagulation test. FFP: 1U=1g fibrinogen Cryoprecipitate:VIII, XiII & fibrinogen. Plt concentrate: 1u ↑ 20-25k plt. Packed RBC: 1U ↑ 1g/dL

SECONDARY POSTPARTUM HEMORRHAGE		
OVERVIEW	ETIOLOGY	RISK FACTORS
Commonly presents as prolonged or excessive bleeding once woman has returned home after 24h-6wks postpartum. Most commonly at 2nd wk. 2ndary to sloughing of eschar on placental site - ↑amt of bleeding. Extent of bleeding less than primary PPH.	Retained products of conception (RPOC) – most common cause of 2ndary PPH Infection – often 2ndary to RPOC. 1-3% after spontaneous vaginal delivery. Most common cause of postnatal morbidity between day2-10. Lacerations – includes episiotomy Trauma – Rupture of vulval hematoma Pre-existing uterine disease – Fibroids Others (rare) – Blood disorders, Carcinoma of cervix.	C-section Prolonged ROM Prolonged labor with multiple exams. Manual removal of placenta Mother's age at extremes Low socioeco status Maternal anemia Internal fetal monitoring Severe meconium staining in liquor Prolonged surgery

ASSESSMENT	INVESTIGATIONS	MANAGEMENT
Dx obtained clinically. [Hx: Crampy ab pain? Passage of bits of placenta? Sx of infxn. Duration of labor. Smooth? Instrumental? Placenta complete? Complication?] Exam: Check temp, HR, BP. Assess uterine size. Larger than appropriate? Assess clinical signs of blood loss. Estimate total blood loss. Establish IV line as indicated. Oxygen via face mask as indicated.	Crossmatch 2-4units of blood if marked bleeding. Coagulation profile as indicated. Speculum exam – check status of cervical os & obtain endocervical swab. US – may be used if RPOC suspected. Blood clots may resemble RPOC. CBC	Mainstays: Bed rest & IV antibiotic therapy. Curettage not performed outinely (risk of uterus perforation) No oxytocin. Gentle digital evacuation of uterus under GA performed with antibiotic coverage. Iron supplement if Hb low.

DIABETES IN PREGNANCY

CLASSIFICATION	DIABETOGENIC EFFECTS OF PREGNANCY	DEFINITIONS	PRESENTATION
• Diabetes mellitus type I --- insulin dependent (Ketosis-prone) • Diabetes mellitus type II--- non-insulin dependent (Ketosis-resistant) • Impaired Glucose Tolerance and Gestational Diabetes (IGT)	• Insulin resistance • Increased lipolysis • Altered maternal gluconeogenesis	• DM= Fasting venous glucose concentration $\geq$ 8.0 mmol/l and 2 hrs (75 gm load) $\geq$ 11.0 mmol/l (or) one of the above + Symptoms • IGT = Fasting < 8.00 mmol/l, but 2 hr (75 gm load) = (9.0-10.9)	-Symptoms -Risk factors (hx & examination) -Blood tests--screening

SCREENING	RISK FACTORS
Why. • 30% have none of the Above risk factors • Not all DM, IGT, have persistent glucosuria • 50% of pregnant women have glucosuria at some time	• Age > 30 • Family history of DM • Past history of: Diabetes in a previous pregnancy, Unexplained IUFD, Neonatal death, Congenital abnormalities, Recurrent abortions, Large babies > 90th centile • Obesity • HTN in multipara • Polyhydramnios • Recurrent infections: Urinary, Fungal • Significant Glycosuria

COMPLICATIONS

MATERNAL	FETAL	NEONATAL
• Obstetric: Polyhydramnios, pre-eclampsia (10-15%) • Diabetic Emergencies: Hypoglycaemia, Ketoacidosis, Diabetic coma • Vascular & End-Organs: Renal, Ophthalmic, Peripheral vascular • Neurologic: Peripheral neuropathy GI disturbance • Infections: Urinary	(1) Macrosomia & Traumatic delivery (30% in seemingly controlled) (2) Delayed organ maturity (RDS) 6 times (3) Congenital malformations: • Cardiovascular: Transposition of great vessels, VSD, ASD, Aortic coarctation • Central Nervous system: Anencephaly, Holoprosencephaly, Encephalocele • Skeletal & spinal - Caudal regression • Genitourinary - Renal agenesis, ureteral dupliction • Gastrointestinal - anal atresia	Hypoglycaemia RDS Hypocalcaemia Polycythaemia

	** Note: when a two-vessel cord is found, suspect a high incidence of congenital anomalies (4) Intrauterine fetal Death (5) Growth restriction (in advanced DM)	

MANAGEMENT

- Start in preconception time
- Specific during pregnancy

SPECIFIC	CONTROL
Diet: 16 x Wt. (pounds) + 300 = CALORIES Carbohydrates 60% Fat 20% Protein 20% Insulin: – Regiment A * 3 times sol.-with meals + lnt. Evening Or - Regiment B * 2 types (short & intermediate) Twice Daily Dose (daily) = wt. (kg) x 0.6 first x 0.7 second x 0.8 third 2/3 in A.M. 2/3 1nt + 1/3 short 1/3 in P.M. 1/2 1nt + ½ short.	Control : • Fasting < 5.0 mmol/1 • 2 hrs P.P. < 7.0 mmol/1 • Adjustment when necessary • Glycosylated Hb A1c (retrospective) ≤ 6 Fetal well being: • AFP 16-18 wks • Detailed scan 19-20 wks • Biophysical assay from 28 wks • Fetal wt. & growth two weekly (3rd) Delivery: - Timing depends on: (Around 38 wks) • Maternal factors • Biochemical control • Fetal status - Method --- LSCS in any medical or obstetric complication. **Insulin dose adjusted on hourly basis with caloric requirements intravenously.

<table>
<tr><th colspan="5">HYPERTENSIVE DISORDERS IN PREGNANCY</th></tr>
<tr><th colspan="3">PREGNANCY-INDUCED HTN</th><th rowspan="2">PREGNANCY-AGGRAVATED HTN</th><th rowspan="2">CHRONIC HTN WITH PREGNANCY</th></tr>
<tr><th>GESTATIONAL HTN</th><th>PRE-ECLAMPSIA</th><th>ECLAMPSIA</th></tr>
<tr>
<td>BP reading ≥ 140/90 mmHg for the first time after 20 weeks of pregnancy
• No proteinuria
• BP returns to normal within 12 weeks postpartum
(if the BP elevation persists, the woman is diagnosed as having chronic hypertension)
• Final dx is only made postpartum
• May even be associated with an increased birth weight</td>
<td>Mild pre-eclampsia
• BP ≥ 140/90 mmHg after 20 weeks' gestation
• Proteinuria ≥ 1+ by urine dipstick or a total protein level ≥ 300 mg/24 hours
- Pre-eclampsia can develop before the 20 week of gestation in hydatidiform mole and in the presence of lupus anticoagulant

Severe pre-eclampsia
• BP ≥ 160 mmHg systolic or 110 mmHg diastolic
• Proteinuria ≥ 2+ by urine dipstick or a total protein level of 2 gm/24 hours</td>
<td>CNS involvement with the occurrence of convulsions that cannot be attributed to other causes</td>
<td>Development of PE or eclampsia in pre-existing hypertension detected by a further increase of
> 30 mmHg in SBP or >15 mmHg in DBP
or
New onset proteinuria of >300 mg/24 hours in hypertensive women but with no proteinuria before 20 weeks gestation
or
A sudden increase in proteinuria or a drop in platelet count to <100,000/mm3 with HTN and proteinuria before 20wk gestation</td>
<td>Hypertension is present before pregnancy or detected before 20 weeks Or
Hypertension first diagnosed after 20 weeks gestation and persisted after 12 weeks postpartum</td>
</tr>
<tr><th colspan="4">PREDISPOSING FACTORS</th><th>FACTORS THAT ↓ PET</th></tr>
<tr>
<td colspan="4">Age: PG<20, all women > 35
Parity: PG have double incidence
Lower socio-economic status
Genetic predisposition: Recessive trait
Obstetric complications: multiple and molar pregnancy, fetal hemolytic diseases (hydrops fetalis), polyhydramnios
Existing medical conditions: chr BP, ren D, DM, SLE, antiphospholipid AS.
Previous PE. The recurrence rate for PE with the same male partner is ~ 20%, and usually becomes apparent at later gestation than in the first pregnancy
Obesity: 4.3% for a women with a body mass index <19.8 kg/m2 to 13.3% for those > 35 kg/m2</td>
<td>Prolonged exposure to paternal Ag
Smoking, but if PE occurs then perinatal mortality triples
Placenta previa</td>
</tr>
</table>

PRE-ECLAMPSIA

CAUSE (UNKNOWN)

THEORIES
(I) Defective trophoblastic invasion of the spiral arteries
(II) Immunologic factor
(III) Increased pressor responses
(IV) Prostaglandins imbalance
(V) Genetic predisposition
(VI) Inflammatory factors

Ischemia could be due to:
- Underlying vascular changes (HTN/ Failure of physio chgs of spiral arteries)
- Increased myometrial resistance of the myometrial vessels, which could be related to a heightened myometrial tension produced by the large fetus in a primiparous women, by twins, or by polyhydramnios
The poorly perfused trophoblast release "factor X" which enters maternal circulation and causes endothelial dysfunction → Imbalance between different classes of locally produced vasoconstrictor and vasodilators.

PATHOPHYSIOLOGY

VASOSPASM

Excess production/ sensitivity to vasoconstrictors	Decreased production/sensitivity to vasodilators
Angiotensin II Serotonin Endothelin	Prostacyclin Nitric oxide

INTRAVASCULAR COAGULATION
Platelet activation
Thrombocytopenia
Reduced production of anti-thrombin III

PLASMA VOLUME CONTRACTION
Redistribution of fluid from the intra-vascular to interstitial fluid spaces so that total ECF volume remains unaltered

ORGAN HYPOPERFUSION
Kidney: ↓ GFR, proteinuria, hyperuricaemia, oliguria
Liver: ↑ SGOT, ↑ SGPT, epigastric pain
Brain: visual scotomata due to occipital lobe ischemia, headache, convulsions (eclampsia)
Placenta: IUGR, placental abruption

Stimulation of the maternal immune system by the early conceptus → Produce blocking Ab to antigenic sites on the placenta, thus preventing the rejection of the fetus and placenta. Hypoimmune response results in damage of the placenta and subsequent PE.
PE less common in previously stimulated immunity conditions as: Previous pregnancy, Consanguineous marriages, Increased maternal anti-HLA Ab.

<table>
<tr><th colspan="7">PRE-ECLAMPSIA</th></tr>
<tr><th colspan="2">DIAGNOSIS</th><th>INDICATIONS OF SEVERITY OF PRE-ECLAMPSIA</th><th colspan="3">INDICATIONS TO TERMINATE PREGNANCY IN PRE-ECLAMPSIA</th></tr>
<tr><th>HISTORY</th><th>INVESTIGATION</th><td rowspan="2">Presence of symptoms
- Persistent headache or other cerebral or visual disturbances
- Persistent epigastric pain
Diastolic BP ≥ 110 mmHg
Proteinuria of 2+ or more by urine dipstick or a total protein level of ≥2 gm/liter in a 24-hour urine sampling
Abnormal lab findings
Obvious fetal growth restriction
Neurological effects:
• Scotomata, hyperreflexia
• Eclamptic convulsions</td><th>MATERNAL FACTORS</th><th>FETAL FACTORS</th></tr>
<tr><td>Personal hx
Past hx
Menstrual hx
Obstetric hx
Complaints and hx of present pregnancy

PHYSICAL EXAM
Hypertension
Edema
Obstetric exam</td><td>CBC: ↑ PCV and thrombocytopenia
SGOT, SGPT: Elevated liver enzymes
Creatinine clearance, serum creatinine, uric acid, total protein in urine, and blood urea: impaired
Coagulation profile: PT, PTT, clot. and bleed. time → in DIC. Fibrinogen and FDP to diagnose DIC
Fetal Surveillance</td><td>Completed 37 weeks
Abnormal liver/ renal functions
Severe preeclampsia/ eclampsia regardless of the gestational age
• In selected cases of severe PET, where the expertise and equipment are available, expectant management may be undertaken for fetal indications</td><td>Obvious IUGR
Oligohydramnios, IUFD
Presence of congenital fetal malformations incompatible with life</td></tr>
<tr><th colspan="7">MANAGEMENT</th></tr>
<tr><th>CONVULSION PROPHYLAXIS</th><th>ANTIHYPERTENSIVES</th><th>TIMING OF DELIVERY</th><th>FLUIDS</th><th colspan="3">END-ORGAN COMPLICATIONS</th></tr>
<tr><td>Magnesium sulphate ($MgSO_4$)
Blocks neuromuscular conduction through blocking the release of acetylcholine at NMJ.
Toxicity: Neurotoxicity (Loss of patellar reflex) , Resp depression (RR <12/min), Cardiac toxicity (cardiac arrest)
Antidote: Calcium gluconate</td><td>Hydralazine
Nifedipine
Labetalol
Sodium nitroprusside</td><td>Once the dx of severe preeclampsia has been established, termination of pregnancy becomes mandatory after patient stabilization
Mode of delivery depends upon whether: The woman is in labor, The progress of labor, The cervix is fit for induction.</td><td>Careful. Colloids.</td><td colspan="3">Consumption coagulopathy: Administer platelets/FFP only if clinical bleeding.
Intracranial hemorrhage:Severe headache in the post partum with pre~ or eclampsia. CT scan
Rx: Combined obstetric and neuro in 3° hosp
Sub capsular hematoma:Severe epigastric pain+hepatomegaly. US. Rx:Correct coagulopathy. If liver rupture, transfusion + liver surgery.
HELLP syndrome: Abnormal vascular tone, vasospasm and coagulation defects.
Rx:Delivery regardless GA. Steroids. Platelets only if <20k/ml. Csection in platelets >50k/ml.</td></tr>
</table>

ECLAMPSIA		
CLINICAL FEATURES	MANAGEMENT	
▯ Premonitory phase ▯ Tonic phase ▯ Clonic phase ▯ Coma	EMERGENCY TREATMENT 1. Clear airway passages 2. Apply an oxygen mask 3. Protect the patient from harm during seizures 4. Control convulsions with $MgSO_4$ 5. Control high BP to avoid fatal complications 6. Deliver fetus 7. Avoid diuretics and hyperosmotic agents 8. Close observation	MONITORING DURING HOSPITAL STAY ▯ Close observation: • Every 30 minutes assess pulse, BP and respiratory rate • Maintain a fluid balance chart monitoring fluid intake and urinary output ▯ Limit IV fluid administration unless excessive blood loss ▯ If convulsions occur despite $MgSO_4$ therapy: • CT scan should be performed ▯ If severe respiratory insufficiency occurs: • ICU admission and ventilation • Measuring blood gases and blood pH levels

CHRONIC HYPERTENSION		
DIAGNOSIS		
HISTORY	EXAM	INVESTIGATIONS
▯ Past history: • Hypertension treated before pregnancy with various antihypertensive medication • Renal problems ▯ Obstetric history: • Hypertension during previous pregnancies • Previous superimposed pre-eclampsia • Previous IUFD, IUGR and abortions ▯ Family history of hypertension	▯ Hypertension: Presenting before 20 weeks' gestation ▯ Cardiac enlargement: May be present ▯ Edema: Occurs when pre-eclampsia or heart failure occurs as a complication of hypertension	▯ Urine analysis: Proteinuria indicates occurrence of superimposed pre-eclampsia ▯ Renal function: Serum creatinine, uric acid and BUN ▯ Fundus examination: Changes indicating chronic hypertension
MANAGEMENT		
ANTIHYPERTENSIVE THERAPY		
- Antihypertensive therapy recommended when SBP ≥ 160-170 and DBP ≥ 105-110, as such treatment decreases the maternal cerebral and cardiovascular complications. - In acute control of severe HTN, the objective of therapy is a gradual reduction of blood pressure to a level of about 140-150 / 90-100 mmHg BP should be assessed every 15 minutes initially, once the blood pressure is stabilized the interval can be lengthened to 30 minutes - Lowering the blood pressure rapidly over minutes → abrupt and profound ↓ in BP → ↓ cardiac output to the uterus with possible fetal hypoxia. Continuous fetal heart rate monitoring should be performed during initial therapy to detect such effect and allow early remedial action		
HYDRALAZINE (Vasodilator), NIFEDIPINE (Ca Channel Blocker), LABETOLOL (Beta-blocker) - most commonly used. Labetalol should be avoided in asthma. Atenolol, ACE inhibitor, ARB and diuretics should be avoided ▯ Diuretics are not recommended ▯ Angiotensin converting enzyme inhibitors are contraindicated with pregnancy ▯ Termination if: • Fetal maturity reached • Fetal distress and severe IUGR • Additional complications occur (severe preeclampsia, abruptio placentae)		

ESSENTIAL HYPERTENSION

CLASSIFICATION	MATERNAL RISK	FETAL RISK
According to Severity: A] Mild B] Severe (>160/110) According to Complications A] Uncomplicated B] Complicated. Any of the following: Cardiomyopathy; Age >40; Duration of HTN >15yrs; Renal disease; DM, CT disease or coarctation of aorta; Previous hx of perinatal loss.	Superimposed PET. Abruptio. Cerebral hemorrhage Exacerbation of HTN Renal failure Congestive heart failure.	IUGR. IUFD. Prematurity. PNMM directly related to HTN severity.

MANAGEMENT

PRENATAL	ANTENATAL	DURING LABOR	POSTNATAL
Assess cause & severity. Look for risk factors. Review drugs. Establish baseline – CBC, urine analysis, 24h urine collection test, KFT, Echo, ECG, CXR.	Mild uncomplicated HTN, stop drugs & observe 3months. If BP stayed mild, don't give drugs. If BP >160/110, give drugs. Observe fetal growth (IUGR/IUFD) Observe maternal condition. No indication to induce labor before 41wks.	Observe BP. Strict fluid control. Continue AHD if taking them. Continuous fetal heart monitoring.	Observe BP esp 48h postpartum. Observe for pulm edema. Observe for hypertensive encephalopathy. Reassess cardiac & renal f(x).

CARDIAC DISEASES

Complicates 1% of pregnancies.
Complications during pregnancy: HF, arrhythmia, stroke & death.
Assessment before pregnancy is essential.
Can be congenital, rheumatic & ischemic.

Predictors (NYHA Functional Classification)	# predictors	Risk of complications
Cardiac dysf(x) – EF>60 or <40	0	3%
Presence of pulm HTN	1	30%
Presence of cyanosis (low Hb saturation)	2	60%
Presence of aortic or mitral valve stenosis		
Hx of CHF, arrhythmia or TIA		

MANAGEMENT

PRENATAL	ANTENATAL	DURING LABOR
Classify patients (predictors of complications). Investigations. Review drugs. Give predictions (safe to go thru pregnancy?)	Monitor signs & sx of HF. Serial Echo. Serial fetal monitoring.	Avoid pain coz pain ↑ EF → acute HF. Avoid hypotension. Left lateral position with oxygen mask. Continuous saturation monitoring. Shorten 2nd stage labor (instrumental?), Prophylactic Abx. Careful fluid therapy. Continuous CTG. Continuous ECG.

HYPERTHYROIDISM	
OVERVIEW	MANAGEMENT
95% cases due to Graves. 50% have +ve family hx. Clinical picture similar to non-pregnant. Most discriminatory features in pregnancy are weight loss, tremor & lid lag. Thryotoxicosis usually improves in pregnancy as other autoimmune. Exacerbations may occur esp in 1st trimester (α subunit of βhCG resembles TSH) Thyrotoxicosis associated with infertility, recurrent pregnancy loss, IUGR, preterm labor, higher PNMM.	Carbimazole & PTU are most commonly used drugs. Both cross placenta, to a lesser extent does PTU, so even in moderate doses may cause fetal hypothyroidism. Both not teratogenic. β blockers used in thyroid crisis. Thyroidectomy rarely indicated in pregnancy. Radioiodine contraindicated in pregnancy (also 4months before) & during lactation. Check for neonatal & fetal thyrotoxicosis.

HYPOTHYROIDISM	
OVERVIEW	MANAGEMENT
Commoner than thyrotoxicosis. Associated with other autoimmune like DM & pernicious anemia. Clinical picture similar to non-pregnant. Most discriminatory feature are cold intolerance, slow pulse & delayed relax of tendon reflexes. Pregnancy itself has no effect on hypothyroidism. Associated with infertility, miscarriages, anemia, fetal loss, IUGR & PET. Association b/w untreated hypothyroidism & reuced IQ & neurodev delay in offspring.	Thyroxine supplements are the only replacement therapy. Crosses placenta but only very little amount will reach fetus, so fetus not at risk of dev hyperthyroidism.

EPILEPSY	
OVERVIEW	MANAGEMENT
Most cases are idiopathic and no underlying cause. 30% familial. All types of seizures can occur in pregnancy. Most AED are teratogenic. In majority of cases, frequency of seizure are not altered by pregnancy. Risk of seizures are highest in peripertum period. 5-10% risk of transmitting epilepsy. The fetus is relatively resistant to short episodes of hypoxia.	The lowest effective dose. Try with 1 drug & give full dose of folic acid 5mg. Try stopping AED, if she is fit free for >2yrs. Detailed anomaly scan at around 20wk gestation. Vit K supplement for the last 4wks of pregnancy for pts taking valproic acid. Avoid pain & long course of labor. Major malformations caused by AED are NTD, orofacial defects & congenital heart defects. Minor malformations are dysmorphic features, hypertelorism, hypoplastic nails & digits.

ANEMIA IN PREGNANCY

PHYSIOLOGICAL CHANGES IN PREGNANCY

- Progressive increase in plasma volume up till 32-34 weeks, (50%).
- Progressive increase in Red Cell mass, although the pregnancy, (25%).
- Maximum physiological anaemia occur at 32-34 weeks gestation.
- MCV, MCHC stay constant, i.e. dilutional anaemia.
- Progressive fall in platelet count, Low platelets only if Platelets are <100 or pathologically reduced count. 5-10% will be 100-150*109/l
- There is 2-3 fold increase in Iron requirements in pregnancy
- Hypercoagulable state.

ANEMIA

- Lower Hb normal values:
  - Non-Pregnant 11.5-12 g/dl
  - Pregnant, change with gestation, but generally 10.5 g/dl.
- Clinical features:
  - Mostly detected on routine testing.
  - Tiredness.
  - Lethargy.
  - Dizziness.
  - Fainting.

IRON DEFICIENCY ANEMIA

- The commonest in pregnancy.
- Increased demand by the developing fetus, leads to increased absorption and increased mobilisation from stores.
- IDA is more common in multiple pregnancies.
- Blood loss at delivery will further increase maternal anaemia, so it is not only a problem confined to pregnancy period.

Anemia results if

- Stores are depleted.
- Iron intake is poor.
- Absorption is poor.
- Utilisation is reduced.
- Demand is increased:
  - Multiple gestations.
  - Chronic blood loss.
  - Haemolysis.
- A lot of patients start pregnancy with already depleted stores.
  - Menorrhagia.
  - Inadequate diet.
  - Previous recent pregnancies
  - Conception while breast feeding.

FOLATE DEFICIENCY ANEMIA

- Second commonest in pregnancy.
- The normal dietary Folate intake is inadequate to prevent megaloblastic changes in the bone marrow in 25% of pregnant ladies.
- Prevalence varies according to :
  - Social class.
  - Nutritional status.
- Factors increasing the risk of FDA:
  - Anticonvulsant therapy.
  - Haemolytic anaemia.
  - Thalassemia.
  - Hereditary spherocytosis.

DIAGNOSIS		MANAGEMENT
IRON DEFICIENCY ANEMIA	FOLATE DEFICIENCY ANEMIA	☐ Routine iron supplement, as demand is rarely met by normal iron intake. ☐ Oral supplementation is not without side effects: ■ Constipation. ■ Taste. ■ Diarrhoea. ■ Nausia and vomiting. ☐ Alternate routes are available: ■ IM. ■ IV. ■ The maximum rate of rise in Hb is around 1g/dl/week. ☐ Severe anaemia diagnosed in the later stages of pregnancy may need transfusion. ☐ Preconception advice for all women is to take folate supplement of 0.4mg/day to reduce the risk of NTD, this will increase to 5mg/day in cases of previous NTD baby, or in case of intake of anti-folate medications
■ As it is the commonest, it is always presumed to be the diagnosis, but should always be confirmed. ■ Changes in the indices as follows: ☐ MCV reduced. ☐ MCH, MCHC reduced. ☐ Serum iron fall, <12mmol/l (normally falls in pregnancy). ☐ Total iron binding capacity increased, saturation <15% indicate anaemia. ☐ Serum ferritin, fall.	■ MCV increased. ■ Megaloblastic changes in the bone marrow. ■ Reduced serum and red cell folate.	

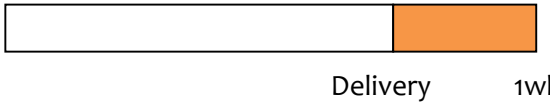
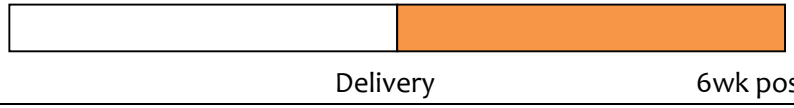
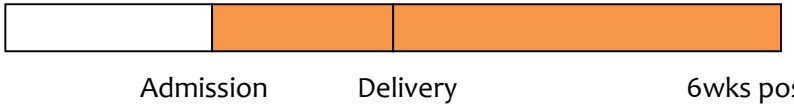
PHYSIOLOGICAL CHANGES IN COAGULATION & FIBRINOLOYTICS SYSTEMS IN PREGNANCY	
• Coagulation system : Increase in levels of coagulation factors, mainly fibrinogen and factor VII, VIII, IX and X – from the beginning of the second trimester. • Fibrinolytic system : Inhibition of the system, due to increase in the levels of plasminogen inhibitors. • Platelet count : No change • Anticoagulant system : ↓ levels of anti-thrombin III, no change in the levels of protein C & S	– The result of these changes is hypercoagulable state in pregnancy – The benefit of these changes is protection of mother from severe haemorrhage after delivery – The risk of these changes is the increased risk of thromboembolism

DISSEMINATED INTRAVASCULAR COAGULOPATHY		
MAIN CAUSES IN PREGNANCY	DIAGNOSIS	MANAGEMENT
• Placental abruption • Preeclampsia & eclampsia • Endotoxic shock – septic abortion, chorioamnionitis and puerperal sepsis • Amniotic fluid embolism • Prolonged shock • Prolonged retention of dead fetus – missed abortion or IUFD for >5 weeks	• Clinical observation – Vaginal bleeding, oozing from venepuncture sites & surgical incisions. – History of any of the above risk factors. • Whole blood clotting time – prolonged (Normal 5-10 minutes) • Coagulation profile : – Platelet count – reduced (Normal 150,000 – 350,000) – Fibrinogen level – reduced (Normal 2-4 gm/L) – APTT – prolonged (Normal 35-43 seconds) – PT – prolonged (Normal 10-14 seconds) – TT – prolonged (Normal 10 seconds) – FDPs levels – increased	• Insertion of at least 2 large infusion lines – Send at least 20ml of blood for cross-matching & coagulation profile, and request at least 6 units of blood & 4 units of FFP. • Correct hypovolaemia – by fluids & whole blood • Correct coagulation disorder – by FFP **NB:-** platelet transfusion only indicated in presence of active bleeding if platelet count is less than 50,000 , and if no active bleeding, if platelet count less than 20,000. • Empty the uterus as rapidly as possible – by delivery or D&C. **NB-** DIC is always a 2ry phenomenon to an underlying stimulus, and is usually self-limiting if the stimulus producing it is removed – therefore the uterus should be emptied as rapidly as possible

<table>
<tr><th colspan="3">THROMBOEMBOLISM</th></tr>
<tr><th>RISK FACTORS</th><th colspan="2">DIAGNOSIS</th></tr>
<tr><td rowspan="2">• Pregnancy – hypercoagulate state .
• Maternal age > 35 years.
• Parity > 4
• Obesity > 80 kg.
• Caesarean section, particularly emergency C.S
• Previous history of thromboembolism
*the risk of recurrence of thromboembolism is 12%, & the majority occur after delivery .
• Prolonged hospital stay.
• Family history of thromboembolism.
• Thrombophilia ie. Congenital deficiency of antithrombin III, protein C or protein S, & presence of factor V leiden.
• Anti – cardiolipin syndrome or presence of lupus inhibitor
• Cardiac disease – such as valvular prosthesis or atrial fibrillation.
• Sickle cell disease.
• Gross varicose veins.
• Blood groups other than O
• Suppression of lactation with estrogen</td><td>DVT</td><td>PE</td></tr>
<tr><td>1- Sx: Pain & swelling in the leg
2- Signs: ↑ temp of the leg, tender calf muscles, a difference of >2cm in the circumference at identical sites of legs, and +ve Homan's sign.
*in 50% of patients there are no clinical symptoms & signs referable to the limbs & pulmonary embolism may be the 1st indication of thromboembolism .
*Over 80% of DVT are left-sided.
3-Investigations :
I. Doppler U/S – Noninvasive, safe, accurate in 95%, more accurate if DVT above the knee due to absence of collaterals than if DVT below the knee due to presence of collaterals.
II. Ascending venography – Contraindicated in the 1st trimester, and maybe indicated after that if Doppler U/S is not informative.</td><td>*There may be (50%) or may be not (50%) prior clinical evidence of DVT.
1. Sx: Sudden onset of dyspnoea, chest pain, cough & haemoptysis, and sudden collapse in massive PE
2. Signs: Cyanosis, rapid breathing & jugular veins distention.
3. Investigations:
• CXR: May be helpful ie. Consolidation, infarction & elevated hemidiaphragm on affected side, but can be totally normal.
• ECG: Usually normal except when the embolus is large.
• Respiratory alkalosis and low Pco2 - due to hypoxia → hyperventilation with blowing off of CO2 and respiratory alkalosis.
• Ventilation & perfusion lung scan
• Pulmonary angiography.</td></tr>
</table>

<table>
<tr><th colspan="3">ANTICOAGULANTS</th></tr>
<tr><th>LMWH HEPARIN</th><th>HMWH HEPARIN</th><th>ORAL WARFARIN</th></tr>
<tr><td>Low molecular weight heparin (LMWH) e.g Enoxaparin sodium (Clexane).
• The preferred type used now.
• Given by S.C route. The therapeutic dose is 2 mg/kg/day, given in two divided doses.</td><td>@ High molecular weight heparin
• Rarely used (if LMWH is not available).
• Given by I.V route. The therapeutic dose is 10,000 units bolus dose , followed by 24,000 units / day, given via infusion pump.</td><td>The dose is 2.5-5 mg/twice daily
Monitored by International Normalized Ratio ie. INR (which should be around 2) & by PT (which should be 2-2.5 times of control.)
MOA: Inhibition of the synthesis of Vit-K dependent factors: II, VII, IX & X.</td></tr>
<tr><td colspan="2">• Monitored therapeutic dose (1mg/kg/day 2x/day) by APTT; should be 2 times of control.
• Monitor maintenance (40mg/ml 1x/day) & prophylactic (20-40mg/ml 1x/day) dose by plasma heparin level; should be 0.2-0.4 units/ml.
• The action of therapeutic heparin is inhibition of thrombin & factors IX, X, XI & XII.
• Heparin DON'T cross the placental barrier & so can be used safely during pregnancy
• The side effect is bleeding.
• If bleeding occur, stop the treatment, and in practice this is enough. Rarely if bleeding not stopped, give specific antidote ie. Protamine sulphate.
The advantages of heparin – compared to warfarin - are:-
1. Does not cross the placenta
2. Small dose prophylaxis- no haemorrhagic hazard
3. Easily and rapidly reversed as heparin disappears from the circulation in 6 hours.
The disadvantages:-
1. Osteoporosis if given for more than 6 months
2. Thrombocytopenia.</td><td>Disadvantages of warfarin are:
1. Bleeding
2. Teratogenic - if given in 1st trimester during the period of organogenesis coz warfarin cross the placenta: chondrodysplasia punctata, cerebral haemorrhage, calcification & microcephaly.
3. Fetal & Neonatal cerebral haemorrhage if given after 36 weeks.
4. Effect not easily or rapidly reversed as warfarin disappears from the circulation in 3 days.
• In case of bleeding, antidote is FFP.
*Neither heparin nor warfarin are excreted in breast milk, so that they are safe to be used during lactation.</td></tr>
</table>

<table>
<tr><td colspan="3">MEDICAL TREATMENT OF THROMBOEMBOLISM</td></tr>
<tr><td colspan="3">ACUTE PHASE</td></tr>
<tr><td colspan="3">Therapeutic dose of heparin. Either LMWH or HMWH.
LMWH's duration of acute phase therapy 2 – 3 months.</td></tr>
<tr><td>LMWH (Clexane)</td><td colspan="2">HMWH</td></tr>
<tr><td>The therapeutic dose is 2 mg/kg/day, given in two divided doses</td><td colspan="2">The therapeutic dose is 10,000 units bolus dose, followed by 24,000 units / day, given via infusion pump.</td></tr>
<tr><td colspan="3">LONG TERM THERAPY</td></tr>
<tr><td colspan="3">Either LMWH (S/C) heparin or Oral Warfarin</td></tr>
<tr><td colspan="3">- If thromboembolism occurs during pregnancy – the best after acute phase therapy for 2-3 months, is to give S/C heparin in the maintenance dose for the rest of pregnancy (in order to avoid side effects of warfarin during pregnancy) & for 12 weeks postpartum of either S/C heparin or warfarin.
- If thromboembolism occurs after delivery – S/C heparin in therapeutic dose for 4 weeks, <u>then</u> either S/C heparin in maintenance dose or oral warfarin , for 3 months.</td></tr>
<tr><td colspan="2">HEPARIN</td><td>WARFARIN</td></tr>
<tr><td colspan="2">Given by SC route. The best is LMWH, if not available, HMWH.
Dose of LMWH (Clexane) 40mg/ 1x/day. HMWH 5000IU 2x/day.
Monitored by plasma heparin level; should be between 0.2-0.4 units/ml.
**</td><td>Given orally.
Dose is 2.5-5mg 2x/day</td></tr>
</table>

COUNSELING AFTER AN ATTACK OF THROMBOEMBO DURING PREGNANCY	
1. Explain the risk of recurrence in future pregnancies– which is about 12 % 2. Explain the need for prophylactic anticoagulatnts in future pregnancies 3. Avoid the use of combined contraceptive pill. 4. Consider screening for thrombophilia – in absence of other risk factors	
POLICY OF PROPHYLAXIS	
No hx. Did c-section. With 1 risk factor.	
1 hx of DVT No risk factor.	
1 hx of DVT. Other risk factor (e.g. admitted to hospital)	
Hx of 2DVT or 1PE or anticardiolipin or lupus inhibitors presence or cardiac indication for prophylactic anticoagulants (eg AF, valvular prosthesis)	
*The policy of prophylactic anticoagulant during pregnancy in previous history of pulmonary embolism or DVT or thrombophilia is by LMWH – once daily throughout pregnancy up to 12 hours before delivery . *The policy of prophylactic anticoagulant during pregnancy in cardiac indication : warfarin in the 1st trimester & up to 36 weeks and then by HMWH, I.V, 6000 units/6hrs up to 12 hours before delivery. *S/C heparin is not effective in prevention of cardiac thrombosis, and so warfarin given in the 1st trimester in spite of it's known teratogenic effects.	

MULTIPLE PREGNANCIES

DEFINITION : Presence of more than one fetus in the gravid uterus

- 2 fetuses : twins
- 3 fetuses : triplets
- 4 fetuses : quadruplets
- 5 fetuses : quintuplets
- 6 fetuses : sextuplets

INCIDENCE : Hellin's law

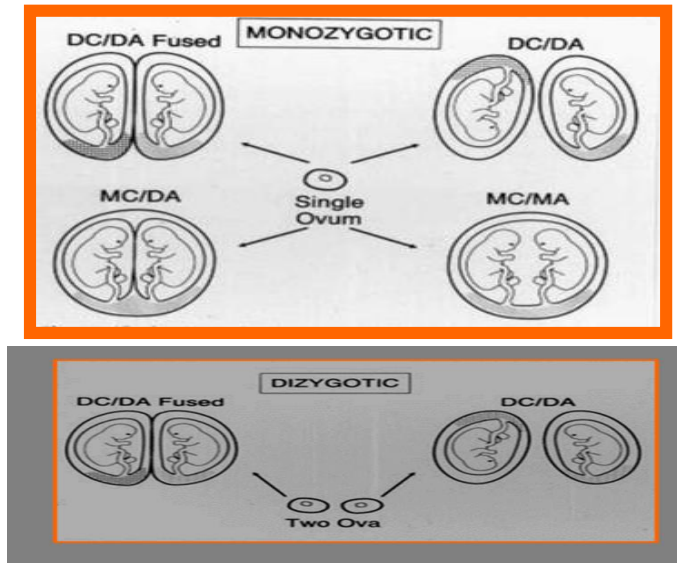
- Twins : 1:89
- Triplets : 1:892
- Quadruplets : 1:893
- Quintuplets : 1:894
- Conjoined twins : 1:60000
- Worldwide incidence of monozygotic : 1 in 250
- Incidence of dizygotic varies and increasing

PREDISPOSING FACTORS :

- Race: most common in negroes
- Age : > increased maternal age
- Parity : more common in multipara
- Family history of multifetal gestation
- Nutritional status : well-nourished women
- ART : ovulation induction with clomiphene citrate, gonadotropins and IVF
- Conception after stopping OCP

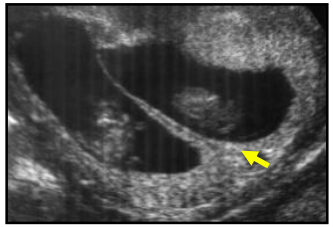
TYPES :

DIAZYGOTIC	MONOZYGOTIC
- 2/3	- 1/3
- Same or discordant sex	- Concordant sex & genetic material (look alike)
- Fraternal, binocular	- Identical, uniovular
- Fertilization of 2 ova by 2 sperms	- Fertilization of 1 ovum with same sperm
- Always dichorionic (50% fused placentae)	- 3 placental configurations
- Never anastomoses	- anastomoses

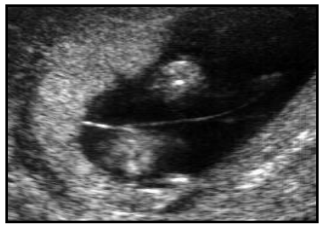


CHORIONICITY DETERMINATION :

- is the main factor determining the pregnancy outcome and what can be done for complications
- All risks increased with monochorionicity
- 100% accuracy in 1st trimester



DIZYGOTIC 80%



MONOZYGOTIC 20%

DIAGNOSIS

HISTORY :
- Family hx, late childbearing, black race
- ART : clomiphene citrate, gonadotropins, IVF
- Hyperemesis gravidorum
- Cardio-respiratory embarrassment : palpitation, SOB
- Leg swelling, varicose veins, hemorrhoids, excessive abdominal enlargement
- Excessive fetal movements

PE:

1) Palpation :
- Large fundal height
- Many fetal parts
- Two fetal heads

2) Auscultation :
- Two distinct feral heart sounds
- Unusual weigh gain, not explained by pre-eclampsia or obesity
- Evidence of pre-eclampsia (25%) is a common association

FETAL PRESENTATION :

- Longitudinal lie 90%
- Vertex 74%
- Both twins vertex 40%
- First twin presents breech 20%
- Both twins breech 10%
- Other (transverse/oblique) 6%

DDX of increased fundal height

- Full bladder (1st trimester)
- Macrosomia
- Fibroid with pregnancy
- Ovarian tumor with pregnancy
- Adnexal mass with pregnancy
- Wrong dates
- Hydramnios
- Ascites withpregnancy
- Molar pregnancy

COMPLICATIONS

MATERNAL :
1. Hyperemesis gravidorum
2. Anaemia
3. Pre-eclampsia 25%
4. Polyhydramnios 10%
5. Gestational DM 2-3x
6. Antepartum hemorrhage : placental previa & placental abruption
7. Cholestasis of pregnancy
8. Malpresentation
9. Mechanical distress : palpitation, dyspnea, varicosities, hemorrhoids
10. Obstructive uropathy
11. Miscarriages

FETAL : more with monochorionicity

1) MISCARRIAGE
- Overall risk : 5%
- 12x higher than MC than in singletons
- 20% have vanishing twin phenomenon
- Twins diagnosed 2x more in 1st trimester than at birth

2) CEREBRAL PASLSY
- CP increased 12 folds in twins
- 47 folds in triplets

3) PERINATAL MORTALITY
- Increased 5-7x with twins
- 3-4x more in MC>DC
- Mainly due to prematurity

4) PRETERM LABOUR AND BIRTH

- Media GA at delivery in twins at 36-37 w (36w for MC)
- 34 w for triplets & 29-31 w for quads
- Severe preterm delivery before 32 w is 2x more in MC
- Delivery after 32w → almost 100% survival
- Delivery at 24-32 w → high chance of NND or handicap

Prevention : bed rest, tocolysis, cervical cerclage
Prediction : cervical length at 20-24w, <2.5cm significantly associated with preterm birth

5) GROWTH RETARDATION

- Main factors for fetal growth, genetic and placental
- IUGR is higher than in singletons (4x more in MC)
- 25% of twins are small for gestational age at birth

6) STRUCTURAL DEFECTS

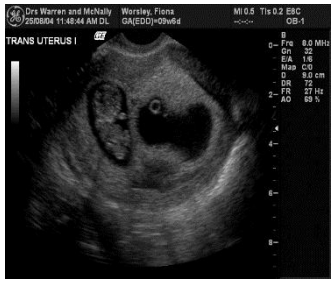
- Congenital heart disease CHD and NTD are common in MP
- DZ twins, same prevalence per fetus as in singletons
- MZ twins, the rate is 3-4x higher per fetus, mainly CHD
- Usually discordant (one fetus is affected)
- Concordance is uncommon (10% in DC & 20% in MC)

7) CHROMOSOMAL DEFECTS

- DZ twins, each fetus has the same MA-related risks as in singletons
- MZ twins, the risk is the same as in singletons
- In MZ, the vast majority of cases are concordant
- Occasionally discordant (Turner & normal male / female)
- Screening by NT & NB only
- Dx by CVS, Amnio

8) SINGLE FETAL DEMISE

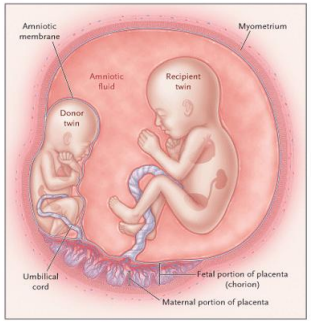
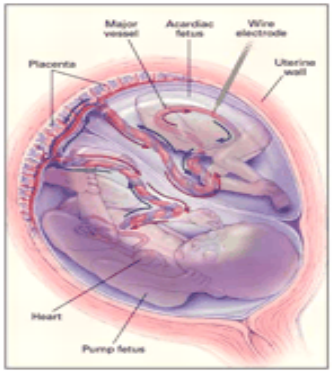
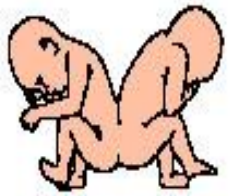
- 4x more in MC
- Risk is entirely dependent on chorionicity and stage of gestation
- 1st trimester :
  Risk of demise of the co-twin is 2-3x as high as normal twin
  No increased risk on survivors
- 2nd or 3rd trimester :
  If DC – preterm labor
  If MC – death, neurological, renal, ischemic lesions in survivors, in addition to preterm labor

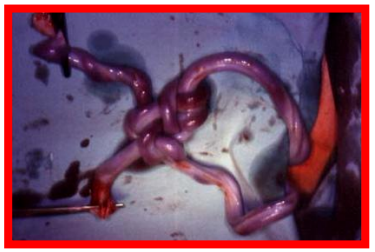


single fetal demise in 1st trimester

9) DURING LABOUR

- Prelabour rupture of the membranes
- Cord prolapse
- Incoordinate uterine contractions
- Increased operative interventions
- Placental abruption after delivery of 1st baby
- Postpartum hemorrhage

10) TWIN-TWIN TRANSFUSION SYNDROME (TTTS)	
- Normally 100% vascular anastomoses inMC - Therefore, intertwin transfusion is a normal event when balanced - TTTS --- unequal sharing and transfusion - Mortality >90% if untreated and neurological damage up to 30% - Diagnosis → poly/oligo sequence - 5 stages : liquor, bladder, Doppler, hydrops, demise - Treatment : Aggressive amnioreduction for stage 1&2 (survival rate up to 60%) Selective laser surgery for stage 2 and above (survival rate up to 80%)	
11) TWIN REVERSED ARTERIAL PERFUSION SEQUENCE (TRAP, Acardiac Twin)	
- Dead twin is perfused by live twin (pump) thru large umbilical a-a anastomoses in retrograde fashion - Trunk & lower limb best perfused, head and upper limb usually involute - Mortality >70% normal twin - Always single umbilical artery	
12) CONJOINED TWINS	
- 1:70000 births - Classified according to the site of union - Thoracopagus is the commonest - Craniopagus is the least common - High perinatal mortality, 50% stillborn - Surgical separation is possible	

13) MONOAMNIOTIC TWINS	
- Sudden, unexpected and non-preventable fetal death intrauterine - High perinatal mortality (50%), largely <32w - Mainly due to cord entanglement, fetal anomalies, preterm labour and hemodynamic effects - Delivery after 32-34w after steroids	cord entanglement 

ANTENAL MANAGEMENT :	
- Diet : Additional 300kcal/day, increased protein, extra protein 80-100g/d 60-100mg of iron and 1 mg of folic acid - Frequent and regular antenatal visit - Fetal surveillance by US - Corticosteroids – only in threatened preterm labour	Indications for c-sec: - Non cephalic presentation of first baby - Monoamniotic twins - Conjoined twins - Locked twins - 2nd twin – incorrectible lie, closure of cervix
Place of delivery : tertiary level hospital 1st stage : cross-matched blood, intrapartum fetal monitoring 2nd stage : Delivery of 1st baby as in singleton pregnancy No oxytocin after delivery of 1st baby Secure cord clamping at 2 places before cutting Ensure labelling of 1st baby Delivery of 2nd baby: - FHA of 2nd baby - Lie and presentation of 2nd twin - Wait for uterine contractions - Conduct delivery	High order multiples: - Preventable, stricter control of IVF and induction - Restrict no of embryos, 2 are good - Perinatal risk increases exponentially with fetal number - ANC in tertiary centre with MFM service - 1 out 5 triple pregnancies result in at least one child with a major long term handicap
MULTIFETAL PREGNANCY REDUCTION	
- Reduce risk of severe PT birth - Best at 11-13 weeks, usually to twins	

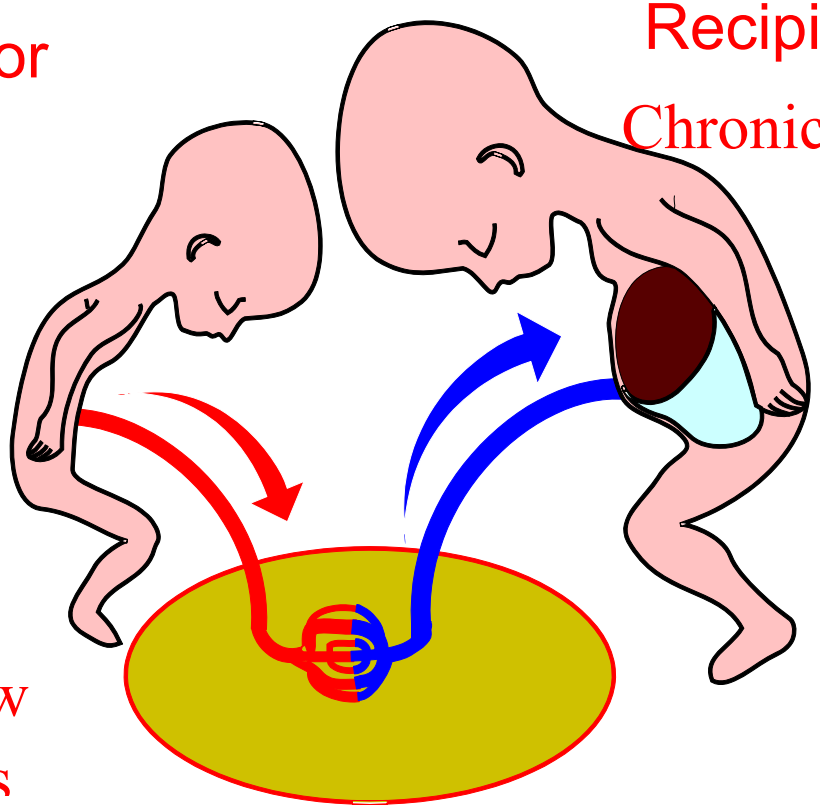
- Which fetus to kill? CRL, NT, gross anomalies
- But if all same and normal, choose the furthest away from the complication
- Inject KCL (intracardiac or thorax) if DC
- Miscarrige rate 5-10%

MC is problematic, needs more invasive, risk more

Donor

Chronic blood loss

Hypovolemia
Anemia
Hypoxia
IUGR
Decreased renal flow
⇒ Oligohydramnios



Recipient

Chronic blood gain

Hypervolemia
Polyhydramnios
Polycytemia
Embolization
Hypertension
Cardiac failure

SMALL FOR GESTATIONAL AGE				
INTRODUCTION				
- Fetal growth is dependent on genetic, placental and maternal factors. - Fetal growth restriction is the second leading cause of perinatal morbidity and mortality.				
ASSESSING FETAL GROWTH				
HISTORY	PHYSICAL EXAM	USES OF ULTRASOUND	FETAL VIABILITY	
- Mother's age - Accuracy of LMP date - Infections during pregnancy - Multiple pregnancy - ANC and visits, Supplements - Past obs. Hx, Past Med Hx, Drug Hx, Family Hx, Socioeconomic Hx.	a) General Exam b) Obstetrical Exam Uterine Fundal Height - Obtaining serial uterine fundal height measurements. - The "Mcdonalds rule" in pregnancy is a rough determination of fetal age in weeks Uterus size: by pelvic examination in the first trimester and subsequent antenatal visits. Misleading in: Full bladder, obesity, deep masses, uterine fibroids & multiple pregnancy	- Diagnosis and confirmation of viability in early pregnancy - Determination of gestational age and assessment of fetal size - Intrauterine or Ectopic pregnancy. - Multiple pregnancy - Diagnosis of fetal abnormalities - Placental localization - Assessment of fetal well-being	Detection of : » Gestational sac (4-5wks) » Yolk sac (5wks) » Embryo (5-6wks) » Visible heart beat (6wks).	
DETERMINATION OF GA AND ASSESSMENT OF GROWTH				
UP TO 13TH WEEKS GA	FROM 16 – 24 WEEKS GA			
CROWN-RUMP LENGTH (CRL)	BIPARIETAL DIAMETER (BPD)	HEAD CIRCUMFERENCE (HC)	FEMUR LENGTH (FL)	ABDOMINAL CIRCUMFERENCE (AC)
▪ From Crown to Coccyx (Rump) (longitudinal axis). ▪ Accurate up to 14 wks (1st TM). ▪ It is the most accurate parameter. ▪ Accuracy of +/- 5 days from the GA.	▪ The transverse width of the head at its widest (the distance between the parietal bones eminence of the skull). ▪ Accurate up to16-24 wks. ▪ Accuracy of +/- 7 days. ▪ It is affected by the shape of the head.	Not affected by the shape of the head.	- Better than BPD in accuracy and timing. - Accurate only when the image shows two blunted ends of the femur.	▪ Made at the widest points in the abdomen. ▪ Most accurate single predictor of fetal weight.

SMALL FOR GESTATIONAL AGE (SGA)

Small for gestational age is defined as a fetal birth weight below the 10th centile for the stated gestational age.
The incidence of SGA fetuses is 5-10%

- SGA
  - CONSTITUTIONAL (70%)
  - PREMATURITY (10%)
  - INTRAUTERINE GROWTH RESTRICTION (20%)
    - SYMMETRICAL IUGR (20%)
    - ASYMMETRICAL IUGR (80%)

A] CONSTITUTIONALLY SMALL FETUS

- Unfortunately, it can be concluded that a fetus is constitutionally small only after pathologic processes have been excluded.
- Therefore, identification of a constitutionally small infant is usually made in retrospect, after the infant is born.

Causes (Multifactorial): Race, Geographical area, Sex (M>F), Maternal age, Maternal weight and height, Socioeconomic status

B] INTRA-UTERINE GROWTH RESTRICTION

Failure of the fetus to achieve its growth potential

INCIDENCE	TYPES
• 3 - 10 % of all pregnancies. • 20 % of stillborns are growth retarded. • 9 - 27 % have anatomic and/or genetic abnormalities. • Perinatal mortality is 8 - 10 times higher for these fetuses.	• Symmetrical growth restriction: fetus whose entire body is proportionally small. (20%) • Asymmetrical growth restriction: Decrease in subcutaneous fat and abdominal circumference with relative sparing of head circumference and femur length. (80%)

CAUSES OF IUGR

MATERNAL PHYSIOLOGICAL CAUSE	MATERNAL PATHOLOGICAL CAUSES	FETAL PHYSIOLOGICAL CAUSES	FETAL PATHOLOGICAL CAUSES	PLACENTAL CAUSES
• Multiple pregnancy • Short stature • Younger or older age (<15 and >45) • Low socioeconomic class • Primiparity • Grand multiparity • Low pregnancy weight • Previous h/o preterm IUGR baby	• ↓ Uteroplacental blood flow: - PET/eclampsia - chronic renovascular disease - Chronic HTN • Maternal malnutrition • Maternal hypoxemia - Hemoglobinopathies - High altitudes • Drugs - Cigarettes, alcohol, heroin, cocaine - Teratogens, antimetabolites and therapeutic agents such as trimethadione, warfarin, phenytoin. • Chronic illness (DM, renal failure, cyanotic heart disease etc.)	• Genetic Factors: • Race, ethnicity, nationality • sex (male weigh 150 -200 gm more than female) • Parity (primiparous, weigh less than subsequent siblings)	• Genetic disorders (Achondroplasia, Russell - silver synd) • Chromosomal anomalies: - Chromosomal deletions - Trisomies 13,18 & 21 • Congenital malformations: Ex: Anencephaly, GI atresia, Potter's syndrome, and pancreatic agenesis. • Fetal Cardiovascular anomalies • Congenital Infxn: mainly TORCH. • Inborn error of metabolism: - Transient neonatal diabetes - Galactosemia - PKU	• Placental insufficiency (most imp in 3rd trimester) • Anatomic problems: - Multiple infarcts - Aberrant cord insertions - Umbilical vascular thrombosis & hemangiomas - Premature placental separation - Small Placenta

DIAGNOSIS

- History, Physical examination, Investigations
- Ultrasound
- Abdominal circumference is the single most effective parameter for predicting fetal weight because it's reduced in both symmetrical & Asymmetrical IUGR .

In the presence of normal head and femur measurements, abdominal circumference (AC) measurements of less than 2 standard deviations below the mean appear to be a reasonable cutoff to consider a fetus asymmetric.

- Asymmetrical growth restriction : BPD is normal in the 3rd trimester , whereas ratio of HC/AC is abnormal .
- Symmetrical growth restriction : HC/AC may be normal .
- Amniotic fluid volumes (oligohydramnios is associated with IUGR) .

Umbilical artery & fetal artery dopplar assessments : increased resistance is associated with a greater risk of IUGR as pregnancy progresses.

COMPLICATIONS		MANAGEMENT	
ANTENATAL	NEONATAL	PRE-PREGNANCY	ANTEPARTUM
• Metabolic changes (acidosis etc). • Oligohydramnios (80%) • Abnormal fetal heart patterns. • Abnormal Doppler studies. • IUFD **INTRAPARTUM** • Abnormal CTG. • Fetal death. • Meconium stained liquor. • ↑ incidence of instrumental and caesarean deliveries.	1- Related to hypoxia and acidosis: a- Meconium aspiration. b- Persistent fetal circulation. c- Hypoxic ischemic encephalopathy. 2- Metabolic: Hypoglycemia, HypoCa, Hypothermia, Hyperviscocity syndrome 3- Related to the etiology: a- Chromosomal abnormalities. b- Infection. c- Congenital anomalies.	• Modify lifestyle habits. • Detect and treat medical disorders.	• Regular antenatal care. • Serial fetal growth assessment. • Serial fetal wellbeing assessment 1- Biophysical profile 2- Computerized CTG 3- Umbilical artery Doppler • Timing of delivery. • Mode of delivery. Time & Mode of delivery governed by: Maternal age, Past obs. History, GA, Fetal well being, Status of cervix, Availability of direct monitoring during labor (Ex: scalp PH sampling). Mode of Delivery ▪ Cesarean delivery without a trial of labor: 1. in the presence of evidence of fetal distress 2. for traditional obstetrical indications for cesarean delivery ▪ Induction of labor Continuous heart rate monitoring and scalp pH monitoring optimize success of vaginal delivery

RHESUS ISOIMMUNIZATION

FETOMATERNAL HEMORRHAGE
Leakage of Rh+ve fetal cells in Rh-ve maternal circulation during pregnancy
Examples: Spontaneous abortion, Induced abortion, APH, E.C.V, Cordocentesis, CVS, amniocentesis, Severe preeclampsia, Ectopic pregnancy, Caesarean section, Manual removal of placenta, Silent feto-maternal hemorrhage

1- If ABO is incompatible:
RBC easily destroyed, so not reaching enough immunological component to cause antibody response and reaction
2- If ABO is compatible:
Rh +ve fetal cells → remain in circulation (life span) until removed by R.E.S → destroyed → liberating Ag (D) → isoimmunization.

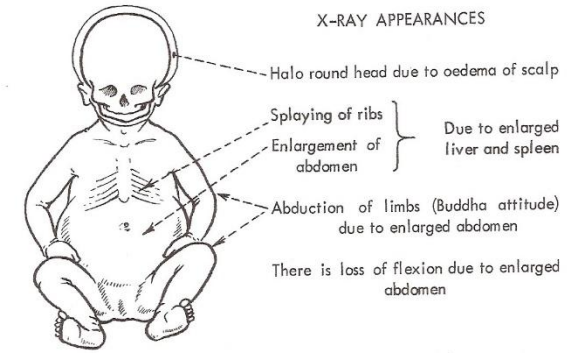
It takes time:
1st pregnancy is almost always not affected:
1% during labour or 3rd stage
10% 6 months after delivery
and15% by the 2nd pregnancy

Mild Cases:
Fetal RBC destruction from IgG anti D → anaemia → compensating hemopoiesis → excess of unconjugated bilirubin.
Severe Cases:
Excessive destruction of fetal RBC → severe anemia → Tissue Hypoxia → Cardiac / circulatory failure → Generalized edema → Heart Failure → Ascites -→Intrauterine Fetal Death

With excessive unconjugated bilirubin >310-350 mol/L → Passes BBB → kernicterus → permanent neuro and mental disorders.

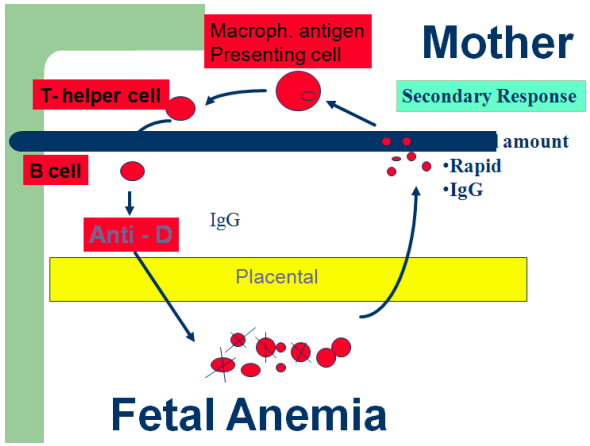
FETAL & NEONATAL EFFECTS
- Haemolytic anaemia of newborn Hb 14-18g/dl
- Icterus gravis neonatorum Hb 10-14g/dl

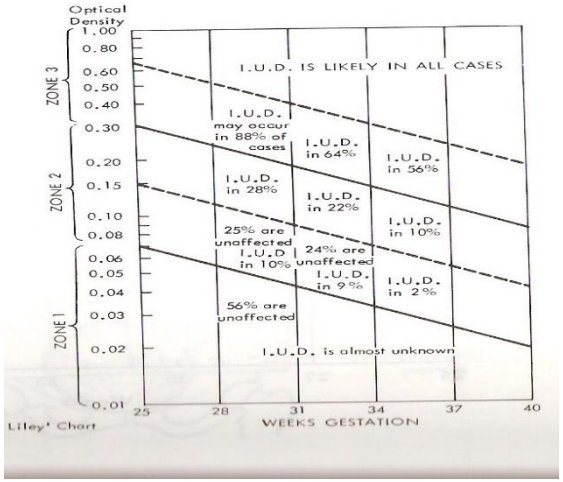
Factors Affect Dev of Rhesus antibodies:
1- Inborn ability to respond
2- Protection if ABO incompatible1\10
3- Strength of Rh Ag stimulus (CDe=R1)
4- Volume of leaking feta blood (0.25ml)
IgM (7 days) doesn't cross placenta, then IgG 21 days - crosses placenta)

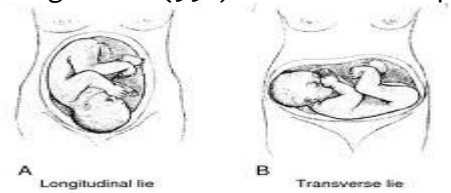


INTRODUCTION	MANAGEMENT
Rhesus factor: Agglutinogen (C,D,E) mainly D C,D,E dominant antigen d,e recessive antigen - Rh +ve about 85% (homozygous DD35% or heterozygous Dd 50%, - Rh negative about 15%. Incidence of Rh -ve in far east is about 1% Examples of Rh factor: (CDe=R1) , (Cde=r) (cDE=R2) Other systems: kell-antikell, luther, Duffy, etc . Introduction of foreign protein (Ag) → production of Ab to neutralize the Ag. In ABO and other non Rh-incompatibility: It usually causes mild anemia, mainly as there is no intrapartum boosting . In Rhesus isoimmunization: mainly (D), but C,E can produce antibodies. Kleihauer-Betke technique (acid elution test): Measure amount of feto-maternal haemorrhage. If 0.1- 0.25ml of fetal blood leaks (critical volume) this will produce isoimmunization represented by 5 fetal cells in 50 low power microscopic field of peripheral maternal blood. So 1ml is represented by 20 fetal cells	A) Prophylaxis 1- Prevention of Rhesus isoimmunization: Anti D (RhoD IgG) Standard dose for >20w, and ½ standard dose for <20weeks. Given within 72h of the incident. SD: I.M. injection: 500 iu = 100 ugm (UK) - neutralize 5ml (4ml + 1ml) = 100fetal cells SD in USA 300ugm=1500iu - neutralize 15ml K-B test if large amount of leaking → another SD if mother is Rh -ve, baby Rh +ve with no isoimmunization (checked by indirect or direct coomb's test) 2- A.P administration of anti D SD at 28w or at 28 and 36w will reduce Rh isoimmunization B) 1- Antibody Screening: for all pregnant women in ANC for irregular antibodies (mainly for Rhesus Negative women) then start at 20w, and every 4 weeks 2- Management following detection of Rh antibodies - Should be treated in specialized centers - Quantitative measures of antibodies + husband genotype - Repeat titration (indirect coomb's, detecting of antibodies) titer or specific enzymes for antibodies IU - Amniocentesis once necessary - Obstetrical management based on timing of I.U trans-fusion (Now cordocentesis + fetoscopy) versus delivery 3- Amniocentesis: should be performed under ultrasound guidance if titer > 1\16 = 0.5-1 ugm = > 2.5-5 I.U - timing: 1st amniocentesis 10 weeks before previous IUFD - Start from 20-22 weeks, 2-4 weekly or more frequent if needed - Amniotic fluid analysis: spectrophotometry: optical density at the height of optical density deviation at wave length 450 nM.

	IU transfusion (cordocentesis, in the past intraperitoneal transfusion) versus delivery of the baby: - Using Lily's chart - Prediction chart (Queenan curve) - Whitefield's action line -Alternatively follow up with doppler study for the fetal middle cerebral artery. Prognosis depends on: Obstetric hx, paternal genotype, maternal history (blood transfusion, antibody titre) amniocentesis results. Delivery: Vaginal versus Cs Intensive plasmaphoresis: when severe cases anticipated, using continous flow cell separator, as early as 12 w Postnatal management: for the neonate - direct coomb's test, Blood group, Rh type, Hb, bilirubin . - Mild cases: phototherapy, correction of acidosis - Severe cases :exchange transfusion





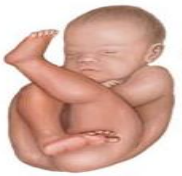
<table>
<tr><th colspan="2">BASIC CONCEPTS</th></tr>
<tr><th>PRESENTATION</th><th>STATION</th></tr>
<tr><td>Part of the fetus occupying the lower segment of the uterus
Cephalic (Head, 95%), Breech (Buttocks, 3-4%), Shoulder, Cord, Compound.
Malpresentation is any presentation other than cephalic.</td><td>Relation between the lowest bony part of the presenting part to the imaginary line between two ischial spines
-3, -2, -1, 0, +1, +2, +3 [if above line: -ve, if below line: +ve]
Assessed through vaginal exam.</td></tr>
<tr><th>LIE</th><th>ENGAGEMENT</th></tr>
<tr><td>Relation of longitudinal axis of fetus to the mother's longitudinal axis
Longitudinal (99%) Transverse Oblique
 
</td><td>Passage of the widest diameter of the presenting part through the pelvic inlet.
If <2 fingers needed to palpate fetal head in pelvic grip: Engaged.

*Pelvic inlet: Upper border of symphysis pubis (ant), sacral promontory (post), ileopectineal line of iliac crest (lateral).</td></tr>
<tr><th>POSITION</th><th>FETAL ATTITUDE</th></tr>
<tr><td>Relation between the dominator (bony landmark of the fetus) to the internal pelvis.
8: Anteriorly (left, right, direct), Posteriorly (left, right, direct), Transverse (left, right)
Most common: Left occipito-anterior.
Malposition is any position other than occipito anterior.
*Bony landmarks:
Vertex: Occipital Face: Mental
Brow: Frontal Shoulder: Scapula
Breech: Sacrum</td><td>Relation of fetus parts to each other
Well-flexed (normally), Extended</td></tr>
</table>

BREECH PRESENTATION

DEFINITION	CAUSES	DIAGNOSIS
Buttocks occupying lower segment of uterus. Commonest malpresentation. Commonest cause is prematurity.	Maternal Causes: Nulliparity, Old age, Fibroid, Polyhydroamnios, oligohydroamnios, Bicornuate / septate uterus, Hx of breech, Uterine/pelvis tumors. Fetal Causes: Prematurity, IUGR, Large babies, Multiple gestations, Fetal abnormalities, Congenital anomalies, Short umbilical cord, Cephalo-pelvic disproportion.	Hx: Subcostal pain (coz fetal head) Abd exam: Leopold maneuver –solid ballotable rounded mass in fundus; soft irregular mass in pelvis. Vaginal exam: 2 ischial tuberosities & tip of sacrum. US: Confirm dx, look placental site, uterine abnormality, # of babies, liquor amount, estimate fetal weight.

TYPES

 Frank breech	FRANK @ EXTENDED Hips flexed, knees extended. Primigravida.	 Complete breech COMPLETE Feet present beside buttocks. Both hips & knees flexed. Multipara.
 Incomplete breech	FOOTLING Hip & knee extended in one or both sides. Preterm singleton.	

A] EXTERNAL CEPHALIC VERSION (ECV)

NOTES	METHOD	CONTRAINDICATIONS	COMPLICATIONS
Done at term (>37th wks) coz may revert to breech, or induce labor. Success rate 60%.	Prep her as if to do C-section – NPO, cannula, conduct in OT, ultrasound, CTG. Rotate the baby by direction of his nose until it becomes cephalic. Repeat CTG. If Rh-ve mother, give antiD coz risk of fetomaternal hemorrhage. Discharge waiting for spontaneous vaginal delivery.	Absolute: HTN, PET, Previous 2 C-sections, Multiple gestation with 1st twin breech, Abnormal CTG, HSV, Previa, Pt refusal. Relative: IUGR, Polyhydroamnios, oligohydroamnios, fetal anomaly.	Labor, PROM, Placental abruption, Cord compression & prolapsed, Fetal bradycardia.

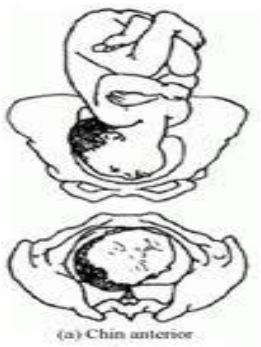
B] ASSISTED VAGINAL BREECH DELIVERY

CRITERIA	ASSISTANCE
Normal baby weight 2.5-3.5kg >36wks GA. Good pelvimetry (roomy pelvis). Fetal head flexed, Breech type Frank or Complete. Experienced obstetrician. Anethetist (Epidural), No other indications for C-section, Multiparous,	After delivery of buttocks, → 1) Episiotomy 2) Keep fetal back anterior After appearance of scapula → 3) Rotate 90 to deliver anterior shoulder & vice versa. After visible hair line → 4) Either Liverpool maneuver, or Mauriceau-Smellie-Veit maneuver or forceps delivery.

C] CAESEREAN SECTION

Absolute Indications:
Prematurity, Footling breech.
Superior to vaginal breech delivery.

Normal labor progress, Uncomplicated pregnancy.		

<table>
<tr><td colspan="3">FACE & BROW PRESENTATIONS</td><td colspan="2">SHOULDER PRESENTATION</td></tr>
<tr><td colspan="2">FACE</td><td rowspan="2"></td><td>BROW</td><td>Transverse or oblique lie.</td></tr>
<tr><td colspan="2">Vaginal Exam: Nose, cheeks, mouth.
Commonly misdiagnosed as breech.</td><td>Vaginal exam: Orbital ridges & nose.</td><td>Abd Exam: Head in one flank & buttock in another. No vaginal exam till R/O previa.</td></tr>
<tr><td>TYPES</td><td>MANAGEMENT</td><td>CAUSES</td><td rowspan="4"> 
Presenting diameter: 13cm.
C-section. Never vaginal delivery.</td><td>CAUSES</td></tr>
<tr><td>MENTO-ANTERIOR
 
(a) Chin anterior
Presenting diameter: 9.5cm.</td><td>If fully dilated → Allow vaginal delivery.
If not fully dilated → Can augment labor with oxytocin.

COMMON NOTES
Never attempt to convert face to vertex.
Never apply vacuum.
Can use forceps.
Can augment with syntocinon.
Large episiotomy.</td><td rowspan="2">Grand multiparous
Neck swelling (cystic hygroma, goiter etc)
Anencephaly</td><td>Placenta previa, Prematurity, Polyhydroamnios, Multiple pregnancy, Abnormal uterus, Fibroid, Cervical cancer, Contracted pelvis, Relaxed ab wall.

MANAGEMENT: C-section</td></tr>
<tr><td>MENTO-POSTERIOR
 
(b) Chin posterior
Presenting diameter: 13cm.</td><td>If fully dilated → C-section.
If not fully dilated → Monitor for conversion into vertex presentation.
If fetus dead → Craniotomy.</td></tr>
</table>

UMBILICAL CORD PROLAPSE			
DEFINITION	RISK FACTORS	CONSEQUENCES	MANAGEMENT
Umbilical cord as the presenting part, below any part of the fetus. Coz ill-fitting presenting part into lower segment of uterus → cord can go into space below presenting part mainly when footling breech or AROM.	Multiparity Prematurity Macrosomia Breech Polyhydroamnios	Cord compression & Vasospasm → Cutting blood from fetus → Fetal distress. Outcome depends on GA & duration of compression.	Emergency C-section Vaginal delivery only if delivery is imminent (when vaginal quicker than c-section: fully dilated & presenting part very low) While waiting for C-section - Fill bladder with 1L - Push presenting part up - Tredelenburg position

SHOULDER DYSTOCIA		
Def: Difficult delivery of the shoulder. Arrest of normal labor after the delivery of the head by the impaction of anterior shoulder against symphysis pubis. Posterior shoulder may also be obstructed by sacral promontory.	Risk Factors **1. Fetal macrosomia** **2. Maternal DM** 3. Others: - Antepartum: Obesity, Multiparity, Post-term gestations, Short stature, Previous hx of macrosomia, Previous hx of shoulder dystocia - Intrapartum: Labor induction, epidural analgesia, Prolonged labor, Operative vaginal delivery	Clinical Manifestations Fetal shoulder fails to deliver after delivery of fetal head despite routine maneuvers. Impaction of fetal shoulder behind pubic symphysis. "Turtle sign": Retraction of fetal head into perineum after its delivery and before the shoulders can be delivered.

Complications		Pregnancies at Risk
Fetal risks	Maternal risks	Cannot accurately predict (50% without risk factors) ACOG recommends CS for EFW ≥5kg in nonDM mothers and ≥4.5kg in DM mothers.
- Asphyxia - Birth trauma: Brachial plexus injuries (Erb's & Klumpke's), Frx humerus & clavicle - Death	Genital tract trauma ↑ risk of PPH Uterine rupture	

HELPER

H: Call for **Help**.

E: Evaluate for **Episiotomy**
Midline episiotomy.
F(x): Facilitates delivery of posterior shoulder.

L: **Legs @ McRobert's Maneuvre**
Hyperflexion, abduction & external rotation of maternal legs.
F(x): Straightens maternal lordosis, remove sacral promontory as obstr, open pelvis to max dimension, pelvic inlet into plane perpendicular to max expulsive force.

P: Suprapubic **Pressure @ Mazzanti** Technique
Direct suprapubic pressure against fetal anterior shoulder to dislodge from under symphysis pubis.

E: Enter @ **Rubin** & **Wood-Corkscrew** Maneuvres
Rubin: Digital pressure to the posterior aspect of anterior shoulder, pushing towards fetal chest.
Woods: Digital pressure applied to anterior aspect of posterior shoulder, pushing towards fetal back.
Can use both hand-in-hand. If unsuccessful, try **reversed Woods-Corkscrew** method.

R: Release of posterior shoulder @ **Jacquemier Maneuvre**
Hand inserted into vagina, and the posterior arm is grasped and pulled resulting delivery in posterior shoulder & displacement of anterior shoulder.

Gaskin All-Fours
Put patient on all fours (knee-chest position) and repeat maneuvers.

Last Resorts:
Zavanelli Maneuvre: Replace head back into pelvis & deliver CS.
Deliberate fracture of clavicle.
Symphysiotomy

<table>
<tr><td colspan="2">PUERPERIUM</td></tr>
<tr><td colspan="2">PHYSIOLOGIC CHANGES DURING PUEPERIUM</td></tr>
<tr><td colspan="2">The period following delivery of the baby and placenta to about 6-8 weeks postpartum.
During that the reproductive organs and the maternal physiology return toward the non-pregnant status.</td></tr>
<tr><td colspan="2">LOCAL</td></tr>
<tr><td colspan="2">UTERUS</td></tr>
<tr><td>Uterine involution.
➢ Structure
- autolysis of excess muscle fibers
- obliteration & thrombosis of blood vessels, then degeneration & transformation into elastic tissue
- separation of the decidua

➢ Weight - reduced (1kg after delivery → 70-100g end of puerperium)

➢ Size
- after delivery the length of the uterus is 20 cm and felt at the level of umbilicus
- after one week it is midway between umbilicus and symphysis pubis
- after 2 weeks it is at the level of symphysis
- by the end of the 6th week it is 7.5 cm long

➢ Uterine ligaments - are involuted
Subinvolution predisposes to prolapse and retroversion.

Endometrial lining rapidly regenerates, so that by the 7th day the endometrial glands will be evident. By the 16th day, the endometrium is restored throughout the uterus, except at the placental site.

The contractions of the arterial smooth muscle and compression of the vessels by contraction of the myometrium result in homeostasis. The size of the placental bed decreases by half, and the changes in the placental bed result in the quantity and quality of the lochia that is experienced.</td><td>“Lochia”
Bloodstained uterine discharge that is composed of cervical mucous, vaginal transudate, and products of necrosis and sloughing of the superficial layer of the decidua

- Lochia rubra (red): consists mainly of blood and decidua. It lasts for 5 days.
- Lochia serosa (pale): due to relative ↓ in RBCs and predominance of leukocytes. It lasts for 5 days.
- Lochia alba (white): consists mainly of leukocytes and mucus. It lasts for 5 days.

- Persistence of red lochia means subinvolution.
- Offensive lochia means infection.
- In severe infection with septicaemia, lochia is scanty and not offensive
- The period of time the lochia can last varies, although it averages approximately 5 weeks.
- Often, women experience an increase in the amount of bleeding at 7-14 days secondary to the sloughing of the eschar on the placental site.</td></tr>
</table>

<table>
<tr><th colspan="3">PUERPERIUM</th></tr>
<tr><th colspan="3">PHYSIOLOGIC CHANGES DURING PUEPERIUM</th></tr>
<tr><th colspan="3">LOCAL</th></tr>
<tr><th>CERVIX</th><th>OVARIES</th><th>PERINEUM</th></tr>
<tr><td>The cervix begins to rapidly revert to a non-pregnant state (less elastic, more firm) but it never returns to the nulliparous state.
By the end of the first week, the external os is closed to the extent that a finger could not be easily introduced.
By the end of the second week the internal os should be closed.</td><td rowspan="3">The resumption of normal function by the ovaries is highly variable and is greatly influenced by breastfeeding the infant.
The woman who breastfeeds her infant has a longer period of amenorrhea and anovulation than the mother who chooses to bottle-feed.
Mother who does not breastfeed may ovulate as early as 27 days after delivery.
Most women have a menstrual period by 12 weeks; the mean time to first menses is 7-9 weeks.</td><td>The perineum has been stretched and traumatized, and sometimes torn or cut, during the process of labor and delivery. The swollen and engorged vulva rapidly resolves, and swelling and engorgement are completely gone within 1-2 weeks.
Most of the muscle tone is regained by 6 weeks, with more improvement over the following few months. The muscle tone may or may not return to normal, depending on the extent of injury</td></tr>
<tr><th>VAGINA</th><th>ABDOMINAL WALL</th></tr>
<tr><td>In the first few days the streched vagina is smooth & edematous, but by the end of the third week rugae begin to appear, but never completely return to its pre-pregnant size</td><td>The abdominal wall remains soft and poorly toned for many weeks. The return to a prepregnant state depends greatly on exercise</td></tr>
</table>

<table>
<tr><th colspan="5">PHYSIOLOGIC CHANGES DURING PUERPERIUM</th></tr>
<tr><th colspan="5">GENERAL</th></tr>
<tr><th>WEIGHT</th><th>BREAST</th><th>BOWEL</th><th>BLOOD</th><th>CARDIOVASCULAR</th></tr>
<tr><td>Decreased due to
- evacuation of uterine content.
- more fluids loss through urine and sweat</td><td rowspan="3">Changes that prepare the mother for breastfeeding occur throughout pregnancy:
- Lactogenesis is triggered initially by the delivery of placenta
- Falling level of Estrogen and Progesterone
- Continue presence of PRL
- If mother is not lactating, the Prolactin level ↓ and return to normal within 2-3 weeks
- Colostrum secreted during the first 3 days

“Colustrum” - yellowish, high protein concentration (IgA), protects against infection, replaced by milk at the 3rd-4th day postpartum

- Engorgement of the breast
- Large and painful breast
But, suckling relieve the discomfort
Suckling → PRL (milk secretion) + Oxytoxin (milk ejection)</td><td>Tendency to constipate
Causes :
- Atony of bowel
- Laxity of ab and perineum
- Anaroxia
- Loss of fluids
- Fear of evacuation: Pain from stretched perineum, prolapse hemorrhoid, or anal fissure</td><td>Increase coagulability of blood continue during the first 2 weeks despite of decrease in a number of coagulation factor This may increase incidence of PE and DVT.
Hb concentration tends to fall in the first 2-3 days</td><td rowspan="3">- Immediately following delivery, there is marked increase in peripheral vascular resistance due to the removal of the low-pressure utero-placental circulatory shunt
- The cardiac output and plasma volume gradually return to normal during the first two weeks of puerperium
- Pulse is normal but may increase if there is infection or hemorrhage</td></tr>
<tr><th>TEMPERATURE</th><th colspan="2">URINE</th></tr>
<tr><td>A reactionary increase may occur following difficult delivery, but it does not exceed 38˚C and drops within 24 hrs.
A slight rise may occur in the 3rd day due to breast engorgement.</td><td colspan="2">Increase in urine production by the 2nd-4th day
Retention of urine is not uncommon
Causes:
- Atony of bladder neck
- Laxity of ab
- Compression of urethra by vulval edema or hematoma</td></tr>
</table>

<table>
<tr><th colspan="3">PATHOLOGIC CHANGES DURING PUERPERIUM</th></tr>
<tr><th colspan="2">POST PARTUM HEMORRHAGE</th><th>PSYCHOLOGICAL</th></tr>
<tr><td colspan="2">It is an excessive (>500 mL at vaginal and >1000 mL at cesarean) blood loss after delivery.
■ First 24 h→ primary
■ Up to 6 weeks→ secondary</td><td>POSTPARTUM BLUES
▪ Complicates 50% of deliveries
▪ Mild, transient, self-limited disorder.
▪ Mood swings, with change in appetite and sleep.
▪ First 2 weeks after delivery, often resolves by postpartum day 10.
▪ No pharmacotherapy is indicated.</td></tr>
<tr><th>PRIMARY</th><th>SECONDARY</th><th>POSTPARTUM DEPRESSION</th></tr>
<tr><td>Causes:
1) Uterine atony
Risk Factors: chorioaminionitis, multiple gestations, macrosomic fetus, fibroid.
Rx: Massage and bimanual compression, then oxytocin infusion, ergometrin, prostaglandin F2
If bleeding persists despite uterine contraction, suspect
2) Genital tract trauma
Cervical and vaginal lacerations</td><td>Causes:
1) Retained products of conception
Management:
Heavy bleeding→IV infusion and X-match of blood.
Syntocinon (synthetic oxytocin).
Examination under anesthesia.
Evacuation of the uterus.
Antibiotics given if placental tissue is found, even without evidence of overt infection.
If blood loss is not excessive, use pelvic US to exclude retained products.
2) Endometritis
3) Bleeding disorders</td><td>- Complicates 5% of pregnancies
- Sx: Sadness, fatigue, changes in sleeping and eating patterns, reduced libido, crying episodes, anxiety, and irritability.
- Usually in the first few months, and may last up to several months or even a year.
- Treatment is recommended for 9-12 months beyond remission of symptoms.
<u>POSTPARTUM</u> PSYCHOSIS
▪ Rare but serious.
▪ Sx: Restless agitation, confusion, delusions, hallucination, thoughts of self harm.
▪ Rarely presents before the 3rd postpartum day but usually does so before 4 weeks.
▪ Recovery occurs over 4-6 weeks.
▪ Patient should be referred urgently to a psychatrist and will usually require admission to a psychiatric unit.
Pathophysiology
▪ Poorly understood, but may be due to rapid changes in estrogen, progesrtone and prolactin in postpartum patients.
▪ Stress - Responsibilities of child rearing
▪ Postpartum thyroid dysfunction (psychiatric disorders).
▪ Seen in higher rates in patients with history of depression or other mental illnesses.</td></tr>
</table>

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<tr><th colspan="3">PATHOLOGIC CHANGES DURING PUERPERIUM</th></tr>
<tr><th>ENDOCRINE</th><th>NEURO – OBSTETRIC PALSY</th><th>BLADDER PROBLEMS</th></tr>
<tr><td>SHEEHAN'S SYNDROME
Anterior pituitary gland enlarges during pregnancy due to an increase in the size and the number of prolactin secreting cells
If significant hemorrhage occur during the peripartum period, ischemic necrosis of the gland will happen.
Full clinical picture apparent after 95% destruction
1) ↓ PRL → Failed lactation
2) ↓ FSH & LH → Anovulation & 2ndary amenorrhea
3) Hypothyroidism → Brady, cold intolerance, constip
4) ↓ ACTH → Hypotension & ↓ weight
Rx: Replacement therapy
-cortisone & thyroxine for life.
-HMG for induction of ovulation if pregnancy is desired.</td><td rowspan="3">One or both of the lower limbs may develop signs & sx of motor and/or sensory neuropathy following delivery.
Causes:
1- Compression or stretching of the lumbosacral plexus as it crosses the sacroiliac joint during decent of the fetal head.
2- Herniation of the lumbosacral discs (L4/L5) may also occur in the exaggerated lithotomy position and during instrumental delivery.

Features: Sciatic pain, unilateral foot drop, hypoaesthesia, muscle wasting.

-Most cases resolve spontaneously in a matter of days or weeks, or managed orthopaedically.</td><td>*Urinary retention, voiding difficulty, and bladder over-distension are common.
*The baby's exit may have traumatized it leading to temporary paralysis.
*Loss of bladder sensation due to regional anesthesia.
*Swelling and pain in the perineal area.
*Psychological (fear) factors

Over-distention will lead to :
1. Dampen bladder sensation, render it hypo-contractile and lead to fibrous replacement of smooth muscle.
2. Over flow incontinence.

It's important to urinate within 6-8h of delivery.
This ↓ rate of UTI and prevent any damage and bleeding that can happen when bladder gets overly full.
If the female didn't urinate during this period, we insert a catheter.</td></tr>
<tr><td>POSTPARTUM THYROIDITIS
Transient destructive inflammation of Thyroid gland occuring within the 1st year after delivery.
Believed to result from modif to the immune system necessary in pregnancy.
2phases: 1) Thyrotoxicosis 2) Hypothyroidism
The process is normally self-limiting, but when conventional antibodies are found there is a high chance of this proceeding to permanent hypothyroidism.</td><td>URINARY INCONTINENCE
Vaginal delivery strongly implicated in the development of stress incontinence.
- Delivery weakens muscles around the bladder and pelvis, which makes it harder to control when urine starts.
- Hormonal changes</td></tr>
<tr><td>POSTPARTUM GRAVE'S DISEASE
✓ Less common than PPT.
✓ Similar to Grave's disease in other settings.
✓ Autoimmune Ab against TSH receptors → Excess production of thyroid hormones.
✓ Needs treatment, like any other grave's disease.</td><td></td></tr>
</table>

PATHOLOGIC CHANGES DURING PUERPERIUM		
BOWEL FUNCTION DISORDERS	THROMBOEMBOLISM	PUERPERAL PYREXIA
CONSTIPATION It is a common problem during puerperium periods. There are three main causes: 1. Interruption of normal diet, dehydration during labor. 2. Fear of evacuation due to pain from sutured perineum, prolapsed hemorrhoid, anal fissures. 3. Atony of intestinal, abdominal, perineal muscles. Management: *Adequate fluid intake and increase in fiber intake. *Stool softener (laxatives) if necessary. Laxatives: lactulose, ispaghula ANAL INCONTINENCE & FECAL URGENCY Incontinence of stool and flatus are frequent complications of childbirth. Anal incontinence is associated with: - Anal sphincter damage especially operative delivery. - Following repair of third or fourth degree tear. - Stool impaction. - Dev of the anovaginal or rectovaginal fistula. - Pelvic floor dysfunction	* The risk of thromboembolic disease rises 5 fold during pregnancy * The majority of deaths occur in the puerperium and are more common after caesarean section. * If DVT or pulmonary embolism is suspected, a full anticoagulant therapy should be commenced & a bilateral venogram or lung scan should be carried out within 24 – 48 hours.	Def: Temperature of 38°C or higher on any two of the first 10 days postpartum, exclusive of the first 24h measured orally by a standard technique Causes of pyrexia: * Genital tract infection; upper tract (bulky tender uterus) , or perineal infection only * UTI. * Breast infections e.g. mastitis. * Resp tract infection - more common after anesthesia. * Thrombophlebitis and deep vein thrombosis. * Wound infections; Anemia. * Non-puerperal related causes e.g. appendicitis.

<table>
<tr><th colspan="3">PUERPERAL INFECTIONS</th></tr>
<tr><td colspan="3">Infections are among the most prominent puerperal complications.
Fever remains the hallmark of puerperal infection, and the patient with fever can be assumed to have genital infection until proven otherwise.</td></tr>
<tr><th>ENDOMETRITIS</th><th>MASTITIS</th><th>URINARY TRACT INFECTION</th></tr>
<tr>
<td rowspan="2">Def: Infection of the endometrium or decidua, with extension into the myometrium and parametrial tissues.
Usually develops on the 2nd or 3rd postpartum.
Etiology: PROM > 24 hours, Cesarean section, Chorioamnionitis, Excessive number of digital pelvic exam, Prolonged labor > 12 hour, Low socioeconomic status, Toxemia, intrauterine monitoring devices, Pre-existing vaginitis and cervicitis.
Bacteriological findings: Anaerobic strep, Gram negative coliforms, Bacteroid spp., Aerobic strep, Chlamydia and Mycoplasma (difficult to culture)
DDx: UTI, Acute pyelonephritis, Lower genital tract infection, Wound infection, Atelectasis, Pneumonia, Thrombophlebitis, Mastitis, Appendicitis.
Management:
Admit, Evacuation of retained products of conception under antibiotics cover, Parenteral broad-spectrum antibiotics, usually stopped once the patient is afebrile for 24-48 hours, tolerating a regular diet, and ambulating without difficulty.</td>
<td>Def: Inflammation of the mammary gland
Could be :
- Congestive mastitis (breast engorgement).
- Infectious mastitis
Both are more common in Primigravidas.
Infectious mastitis and breast abscess are uncommon complications of the breastfeeding.
Almost certainly occur as a result of trauma to the nipple and the subsequent introduction of the Organisms from the infant’s nostrils.
Most common m/o: Staph aureus.

Rx: isolation. Cease breast feeding from affected breast. Expression of milk. Culture & sensitivity. Flucloxacillin commenced while awaiting sensitivity result. 10% with breast abcess need surgical incision & drainage.</td>
<td rowspan="2">2-4 % of women dev UTI postpartum.
Causes: Bladder and the lower urinary tract remains hypotonic, Catheterization, Birth trauma, Conduction anesthesia, Frequent pelvic exam.
Commonest m/o: E.coli and Proteus spp.

Sign & sx: Dysuria, Frequency, Loin pain if pyelonephritis, Systemic sx, May be asymptomatic and recognized on routine mid-stream urine (MSU) sample.
(performed on all patients who have been catheterized in labor)
Exam: Raised temperature, Supra-pubic and/or renal angle tenderness.
Investigation: MSU, White cell count, Nitrites and leucocytes on dipstick.

Rx: Broad-spectrum antibiotics until the results of culture and sensitivity are known, then be specific. Bed rest. High fluid, light solid diet.</td>
</tr>
<tr>
<td>
<table>
<tr><th>INFECTIOUS</th><th>CONGESTIVE</th></tr>
<tr><td>1 wk or more after delivery.
Usually one breast is involved.
Tender, redness, swollen and hot.
Pt is febrile and appears ill.
Purulent discharge may be present.</td><td>Usually occur on the 2nd or 3rd postpartum day.
Breast swollen, tender, tense and warm.
Temp may be mildly elevated.
Axillary adenopathy can be seen.</td></tr>
</table>
</td>
</tr>
</table>

PUERPERAL PYREXIA

Elevated temperature of more than 38.2°C for a whole day within the first 10days postpartum.

History

Introduction
Permission
Complete patient profile: GA, GravidaPara, Address, Occupation, Blood group
Chief Complain
Analysis of chief complaint: Duration, onset, documented/not
R/O Endometritis: Offensive vaginal discharge, Ab pain
R/O Mastitis: Lactating or not, Abnormal breast discharge
R/O UTI: Urinary sx, Nausea, vomit, chills, rigors
R/O Wound infxn: Wound pus, bleeding from site
R/O URTI: Cough, SOB
R/O DVT: Leg swelling or redness
R/O Bacterial vaginosis: Vaginal discharge during pregnancy
Intrapartum Hx: Vaginal delivery/CS? Elective or emergency CS? Induction of labor? Instrumental delivery? PROM? Hx of blood transfusion? Urine catheterization?
Hx of pregnancy: Anemic? Bacterial vaginosis during pregnancy?
Hx of the same problem in previous pregnancies?
PMH/PSH
Drug Hx. Family Hx. Social Hx.

Physical Examination:

Introduction

Permission

Privacy. Lighting. Hygiene.

General Exam:

- Cannula site (superficial thrombophlebitis)
- Catheter (uti)
- Signs of anemia (anemia per se is risk factor)
- Vital signs

Breast Exam (mastitis)

Chest Exam (urti)

Thyroid Exam

Abdominal Exam:

- Uterine tenderness (endometritis)
- Uterine subinvolution (endometritis)
- Inspection of wound (wound infxn)

Pelvic Exam:

- Cervix is open (endometritis)
- Offensive vaginal discharge (endometritis)

Lower Limb Exam

- Leg circumference (dvt)
- Doppler (dvt)

checking whether or not the fetal growth is appropriate to the gestational age

Aim :

1-Confirming gestational age.

2-Detecting fetal growth abnormalities.

3-Early identification of congenital malformations.

4-Detecting acute & chronic fetal hypoxia.

Determinants of fetal growth

Fetal growth depends on

- Oxygenation through placenta
- Nutrients
- Fetal hormones

Adequate oxygenation and nutrition both depend on

- Maternal nutrition
- Placental perfusion

Fetal hormones affect:

- The metabolic rate
- Growth of tissues
- Maturation of the individual organs

Hormones:

- IGF → in late gestation
- Thyroxine → for skeletal and cerebral maturation
- Insulin
- Cortisol → surfactant, digestive enzymes, villous proliferation

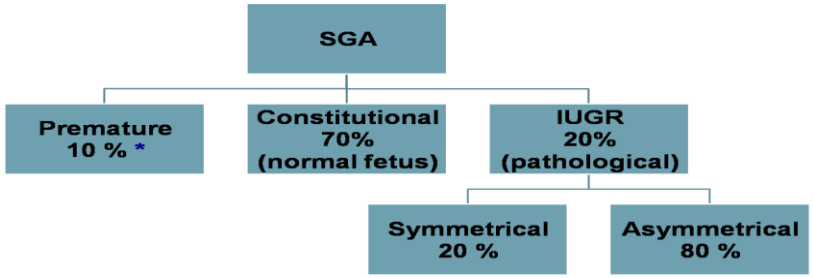
Maternal physiological factors affecting normal fetal growth

1. Sex of the fetus :male > female 200 grams
2. Maternal booking weight
3. Maternal height
4. Maternal age (Teenage pregnancies)
5. Ethnicity (South Asians – LBW)
6. Maternal parity (increased BW)

Small for Gestational Age (SGA)

- (SGA) babies are those whose birth weight lie below the 10th percentile for that gestational age. This is not always pathological.
- Not all IUGRs are below 10th centile ,and not all those below the 10th centile are IUGRs.

Why is the fetus small?



IUGR "Intrauterine growth restriction"

Failure of the fetus to achieve its growth potential

Significance of IUGR

- Major cause of neonatal morbidity and mortality.
- Significant cost in terms of the management.
- There is a growing appreciation that certain adult diseases (including hypertension and diabetes) are related to birth weight

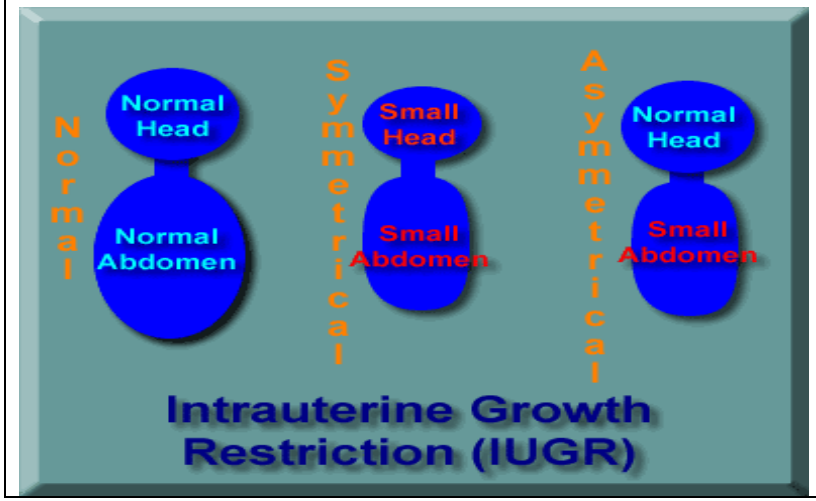
Types of IUGR

1. Asymmetrical (brain sparing) (80%)

- Normal HC, and abnormal AC
- The fetus is usually with normal potential
- Mostly caused by fetal hypoxia

2. Symmetrical (all parameters involved) (20%)

- Usually the fetus loses its potential
- Mostly caused by chromosome abnormalities and viral infections



Maternal causes of asymmetrical IUGR

- Cigarette smoking
- High blood pressure
- Chronic kidney disease
- Advanced diabetes
- Heart disease.
- Chronic respiratory disease
- Malnutrition, anemia
- Autoimmune disorders (SLE ,thrombophilia, APA)
- Drugs (substance abuse, ACE inhibitors)

Placental causes of asymmetrical IUGR

- Placenta previa.
- Abnormal placentation.
- Circumvallate placenta
- Placental tumours.

Fetal causes of symmetrical IUGR

- Multiple gestation (twins ,triplets, etc)
- Infection(TORCH)
- Congenital malformations
- Chromosomal abnormality(triploidy, trisomy 18,21,13)

Complications of growth restriction	Possible long term complications
Antenatal Complications ▪ Metabolic changes in fetus (acidosis,hypoxia). ▪ Oligohydramnios (80%) ▪ Abnormal fetal heart patterns. ▪ Abnormal Doppler studies. ▪ Intra uterine fetal death Intrapartum complications ▪ Abnormal CTG. ▪ Meconium stained liquor. ▪ Increased incidence of instrumental and caesarean deliveries. ▪ Fetal death Neonatal complications 1- Related to hypoxia and acidosis: a- Meconium aspiration. b- Persistent fetal circulation. c- Hypoxic ischemic encephalopathy. 2- Metabolic: a- Hypoglycemia b- Hypocalcaemia c- Hypothermia d- Hyperviscocity syndrome 3- Related to the etiology: a- Chromosomal abnormalities. b- Congenital anomalies. c- Fetal infection	▪ Lower IQ ▪ Learning and behavioral problems . ▪ Neurological deficits(Cerebral palsy) ▪ Hypertension and Ischemic Heart Disease. ▪ Metabolic disorders (type 2 D.M).

Methods of assessing the fetal well-being ▪ History ▪ Physical examination ▪ Investigations	History Assure accurate dating. Confirm gestational age through: 1- LMP The first day date of the last period Ask if is the lady sure about the date? Amount of blood? Length of period? 2-Regularity of the last 3 cycles 3-lactation in the last 3 months 4-Oral combined contraceptive pills use 5-Quickening: primigravida feels it btw 18-20 weeks multigravida feels it btw 15-17 weeks Current pregnancy history: ▪ Mother's age. ▪ Exposure to X-Ray. ▪ Infections during pregnancy. ▪ Multiple pregnancy: lead to SGA due to inadequate nutritional supply . ▪ Antenatal care and visits. ▪ Supplements Past obs. History: Previous deliveries of preterm ,low birth weight , complications, miscarriage or live and well babies Past medical history: some diseases like sickle cell anemia and HTN and DM and anti phospholipids syndrome can lead to IUGR and SGA Drug history: cocaine, heroin, marijuana, immunosuppressive, anti convulsant agents and SLE drugs can lead to fetal growth retardation. Family history: Inherited diseases ,any congenital deformities or handicapped Socioeconomic history : maternal under nutrition Chronic diseases: (DM, HTN, chronic RF)

Physical examination

General examination:

vital signs ,skin, head & neck, chest, abdomen and extremities

Obstetric examination:

-Symphysis-Fundal height (primary screening tool starting 20 weeks)

→ up to 3cm variation

-Size of uterus by pelvic examination/abdominal palpation

→ Misleading in: Full bladder, obesity, deep masses, uterine fibroids &multiple pregnancy

Investigation

Ultrasound is used to

- Diagnose pregnancy.
  - Detection of gestational sac: 5 wks.
  - Detection of fetal heart activity: 6 wks.
  - Detection of fetal pole: 7 wks.
- Estimate the Gestational age (GA).
- Intrauterine or Extrauterine (Ectopic) pregnancy.
- Multiple pregnancy.
- Check for fetal viability
- Check for chromosomal anomalies or structural abnormalities
- Locate the placenta (placenta previa, marginal placenta)
- Measure the amniotic fluid volume (Polyhydramnios ,Oligohydramnios)
- Assess fetal growth and estimate fetal weight

Multiple Pregnancy

- ❖ Monochorionic : thin inter-twin septum
- ❖ Dichorionic : thick inter-twin septum, lambda sign (tongue of placenta tissue is seen within the base of dichorionic membrane)

Crown-Rump Length (CRL)

- From Crown to Coccyx (Rump) (longitudinal axis).
- Accurate up to 14 wks (1st TM).
- It is the most accurate parameter.
- Provides accuracy of +/- 5 days from the GA.

Biparietal Diameter (BPD)

- The transverse width of the head at its widest (the distance between the parietal bones eminence of the skull).
- Accurate up to16-24 wks.
- Provides accuracy of +/- 7 days.
- It is affected by the shape of the head.

Femur length (FL)

- Better than BPD in accuracy and timing.
- Accurate only when the image shows two blunted ends of the femur.

Head Circumference (HC)

- Not affected by the shape of the head.
- Formula=(BPD+APD)/2 *3.14

Abdominal Circumference (AC)

- It is made at the widest points in the abdomen.
- It is the most accurate single predictor of fetal weight.
- Small abdominal circumference in comparison with normal head and femur length indicates asymmetrical growth retardation

HC to AC Ratio, and the GA ▪ HC:AC > 1 (HC is bigger) → the GA < 35 wks. ▪ HC:AC = 1 (Equal) → the GA = 35 wks. ▪ HC:AC < 1 (AC is bigger) → the GA > 35 wks	Assessing fetal well being • Amniotic Fluid Volume • Cardiotocograhphy (CTG) • Doppler Investigation Umbilical Artery • Biophysical Profile

Amniotic fluid volume

Two indications of amniotic fluid volume:

1.Maximum vertical pool

- Measured after a general survey of the uterine contents
- Measurements of less than 2 cm suggest oligohydramnios
- Measurements of more than 8 cm suggest polyhydramnios

2. Amniotic fluid index (AFI)

- Measured by dividing the uterus into 4 quadrants.
- A vertical measurement of the deepest cord free pool in each quadrant is taken and the results summated.
- In the third trimester the AFI should be between 5 and 25 cm

Management

-No effective drug therapy for IUGR has been yet found, except in some conditions when the cause is controllable Ex: Thrombophilia and Anti-phospholipid syndrome

-The goal is to deliver the fetus as mature as possible in the best physical conditions.

-Management principles

- Pre-pregnancy
- Antepartum
- Labor & delivery

Pre-pregnancy

- Modify lifestyle habits.
- Balanced nutrition
- Magnesium & Foliate supplements decrease rate of SGA
- Quit smoking, alcohol, & drug abuse
- Detect and treat medical disorders
- Correction of anemia.
- Control any chronic illnesses (*anti-phospholipids syndrome , sickle cell disease, DM, HTN, thyroid dysfunction*)

Antepartum

- Regular antenatal care: assess Fetal heart beat and fetal movement
- Serial fetal growth assessment.
- Fetal surveillance & serial US measurements at three weekly intervals are indicated
- Fetal weight every 2 weeks
- Serial fetal wellbeing assessment.

1-Biophysical profile
2-Computerized CTG
3-Umblical artery Doppler

- Bed rest to maximize uterine blood flow
- Betamethasone administration between GA 30-35weeks
- Timing of delivery : to maximize gestation without the fetus suffering any neurological abnormality, and increasing maturity as possible before delivery.
- Mode of delivery.

Labor & Delivery
Time & Mode of delivery governed by:

- maternal age
- past obs. History
- gestational age
- fetal well being
- Bishop score
- status of cervix
- availability of direct monitoring during labor Ex: scalp ph sampling.

Cesarean delivery without a trial of labor:
1. in the presence of evidence of fetal distress
2. for traditional obstetrical indications for cesarean delivery
Induction of labor : continuous heart rate monitoring and scalp pH monitoring optimize success of vaginal delivery

-Postpartum

- The infant should be carefully examined for any congenital anomalies and infections.
- Monitor blood glucose, hypoglycemia is a common finding.
- optimized nutrition may help the baby to catch up height and weight

-Prognosis

- Main danger is neurological injury
- Some will suffer morbidity or die as a result of prematurity .
- But long-term prognosis is good with low incidence of mental or physical handicap.
- Height and weight curves remains slightly below 50th centile .
- Infants with IUGR secondary to placental insufficiency show "catch up" growth after delivery when feeding is optimized
  While IUGR related to chromosomal abnormality or congenital infection the development depend on abnormality present.
- New researches suggest a link between IUGR and birth weight and increased incidence of HTN and diabetes in adults

BIOPHYSICAL PROFILE

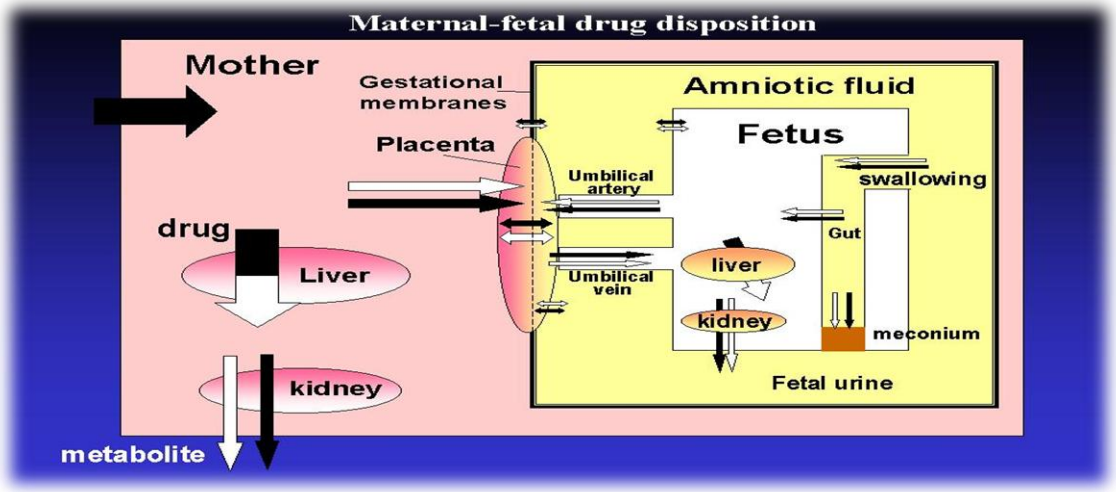
variables	normal score = 2	abnormal score = 0
fetal breathing movements	≥1 episodes in 30 min each lasting ≥30 sec	absent or no episode ≥30 sec in 30 min
gross body movements	three or more discrete body or limb movements in 30 min (episodes of active continuous movement = a single movement)	less than 3 episodes of body or limb movements in 30 min
fetal tone	≥1 episodes of active extension with return to flexion of fetal limb(s) or trunk; opening and closing of hand is considered normal tone	slow extension w/return to flexion, movement of limb in full extension, or fetal movement absent
reactive fetal heart rate	≥2 episodes of accelerations (≥ 15 beats/min) in 20 min, each lasting ≥ 15 sec and associated with fetal movement	< 2 episodes of accelerations or acceleration of < 15 beats/min in 20 min
qualitative amniotic fluid volume	≥1 pockets of fluid measuring > 1 cm in 2 perpendicular planes	pockets absent or pocket < 1 cm in 2 perpendicular planes

	score	notes
normal	8 – 10 (if amniotic fluid index is adequate)	CNS is functional & fetus is not hypoxemic
equivocal	6	
abnormal	< 4	along w/oligohydramnio⟶ labor induction

DRUGS IN PREGNANCY

Drug Epidemiology

- More than 50% of pregnant women take prescribed or non-prescribed (OTC) drugs or use social drugs (such as tobacco and alcohol) or illicit drugs at some time during pregnancy.
- In general, drugs should NOT be used during pregnancy unless absolutely necessary because many can harm the fetus.
- About 2-3% of all birth defects result from drugs that are taken to treat a disorder or symptom.



Maternal Pharmacokinetics

- Changes in body fluid volume
- Changes in CVS parameters
- Changes in pulmonary function
- Alterations in gastric activity
- Changes in serum binding protein concentrations and occupancy
- Alterations in kidney function

Fetal Pharmacokinetics

- Plasma binding proteins differ from maternal.
- Drugs transferred across placenta undergo 1st pass through the fetal liver.
- Liver expresses metabolizing enzymes, but capacity less than mother.
- Fetal kidney immature.

Placental Pharmacokinetics

- Blood flow through the placenta (maternal side) increases during gestation
- Transfer of flow-limited drugs affected by placental flow
- Compounds that alter blood flow alter maternal drug disposition and placental transfer
- Placental metabolism (dealkylation, hydroxylation, demethylation) affects drug transfer across the placenta
- At term, the surface area of the placenta is at its maximum and nearly all substances can reach the fetus

Drug Transfer

- Most drugs have a molecular weight below 1000 daltons (D)
- Drugs < 1000 D cross the placenta (< 500 D cross easily)
- Main determinant of the drug concentration in the embryo/fetus is the mother's blood concentration
- Other factors:-
  - lipid solubility & protein binding
  - degree of ionization at physiologic pH
  - placental blood flow & surface area available for transfer

The Processes That Govern The Passage Of A Drug Into Milk Are Similar To The Placenta

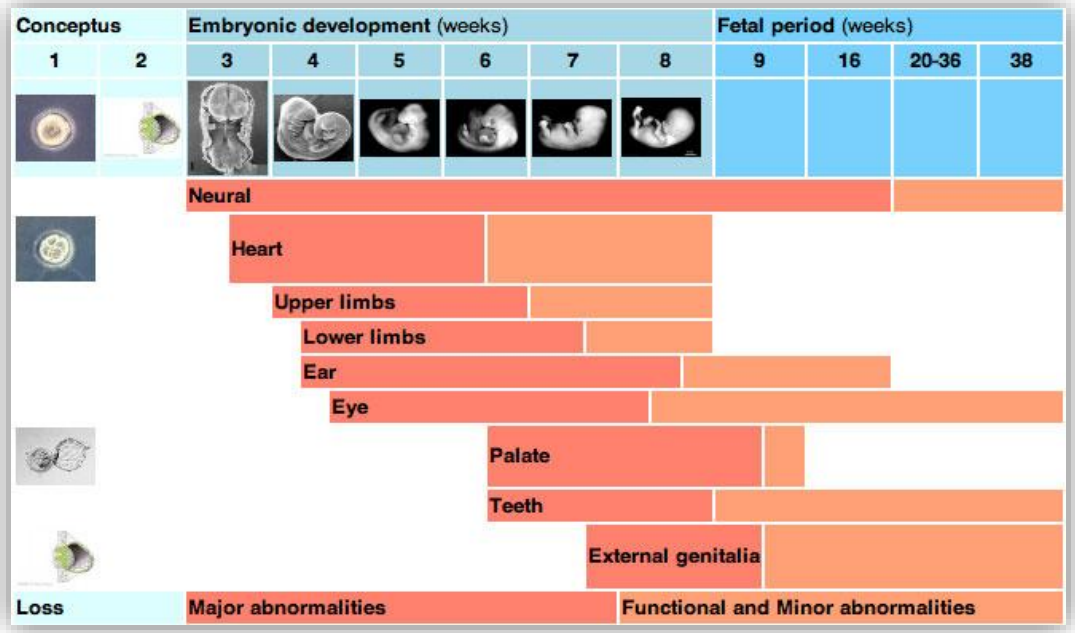
- **Maternal serum concentration is the main determinant**
- **The milk pH is slightly acidic in comparison to serum pH; so weak bases could become trapped in milk (ion trapping).**

Type Of Effects

- **Teratogenicity** (e.g. thalidomide) - detected at, or shortly after, birth .
- **Long term latency** (e.g. DES - increased risk of vaginal adenocarcinoma after puberty, or abnormalities in testicular function and semen production) .
- **Predisposition to metabolic diseases** (e.g. Barker hypothesis - low birth weight associated with increased risk of diabetes, hypertension, heart disease in adulthood).
- **Impaired intellectual or social development** (e.g. exposure to phenobarbitone- alters programming of brain)

Fetal Age Affects The Type Of Drug Effect:

- **Before the 20th day after fertilization :**
  - ✓ (all-or-nothing effect), Teratogenesis is unlikely during this stage.
- **During organogenesis (between 20 and 56 days after fertilization):**
  - ✓ Teratogenesis is most likely at this stage, spontaneous abortion, gross anatomic defect (true teratogenic effect), or the drugs may have no measurable effect.
- **After organogenesis (in the 2nd and 3rd trimesters):**
  - ✓ Teratogenesis is unlikely, but drugs may alter growth and function of normally formed fetal organs and tissues



TERATOGENESIS

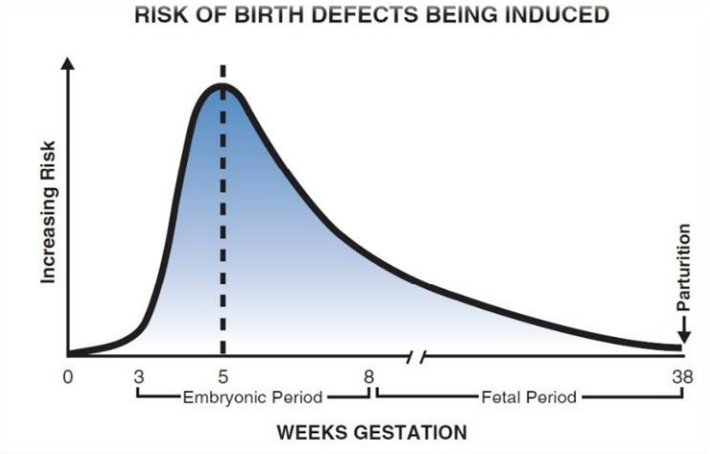
- It is defined as structural or functional dysgenesis of the fetal organs.
- Typical manifestations include
  - congenital malformations with varying severity
  - intrauterine growth restriction
  - carcinogenesis
  - fetal demise
- In humans, the critical time for drug-induced congenital malformations is in the first trimester

Malformations

- The overall incidence of
  - major congenital malformations is around 2-3%
  - minor malformations is 9%
  - 25% are due to genetic or chromosomal abnormalities
  - 10% due to environmental causes including drugs
  - 65% of unknown aetiology
- The part played by drugs is probably small

Organogenesis

- The critical time for drug-induced congenital malformations is usually the period of organogenesis
  - about 20 to 55 days after conception
  - about 34 to 69 days (5-10 weeks) after the first day of the LMP
- If a drug is given after this time it will not produce a major anatomical defect, but more of a <u>functional one</u>



Pregnancy Risk Categories – FDA

- **Category |A|** Safety has been established using human studies, no fetal risk.
- **Category |B|** Presumed safety based on animal studies, but no well-controlled human studies.
- **Category |C|** Uncertain safety. Animal studies show an adverse effect, no human studies.
- **Category |D|** Evidence of fetal risk, but benefits outweigh risks.

Category |X| Highly unsafe. Risk outweighs any possible benefit

Antibiotics

<u>Category |B|</u>

- ❖ Penicillin one of the safest antibiotics that could be used in pregnancy
- ❖ Cephalosporin one of the safest antibiotics in pregnancy
- ❖ Macrolides; erythromycin& azithromycin can be used.
- ❖ Nitrofurantoin; Commonly used in pregnancy to treat UTI should not be given to women in late pregnancy due to the potential risk of hemolytic anemia in the newborn.
- ❖ Metronidazole; not recommended for lactation
- ❖ Vancomycin (oral); possible fetal ototoxic effect

<u>Category |C|</u>

- Aminoglycoside [neomycin – tobramycin]
- Quinolones [ciprofloxacin – levofloxacin]; There are safety concerns of fluoroquinolone use during pregnancy and, as a result, are contraindicated except for when no other safe alternative antibiotic exists.
- Trimethoprim; can affect folate metabolism, so; relatively contraindicated during pregnancy, especially the 1st trimester.
- Chloramphenicol; Gray Baby Syndrome

<u>Category |D|</u>

- Tetracycline; use during tooth development can cause permanent discoloration & enamel hypoplasia.
- Aminoglycosides [streptomycin – gentamicin]; hearing deficit & 8th cranial nerve damage

Antivirals

- Acyclovir |B| recommended for treatment of Varicella during pregnancy, especially during the 2nd and 3rd trimesters
- Amantadine|C| CHD; tetralogy of Fallot / single ventricle with pulmonary atresia

Anti-retroviral agents|B|

- [Didanosine – Etravirine – Ritonavir – Enfuviritide – Maraviroc]

Anti-retroviral agents|C|

- [Lamivudine – Delaviridine – Indinavir]

Thalidomide

Potent Teratogen |X|

was used against nausea and to alleviate morning sickness in pregnant women.

- ❑ Meromelia
- ❑ CHD
- ❑ Eye abnormalities
- ❑ Facial Palsy

Antifungals

Category |B|

- Amphotericin b remains the drug of choice for systemic fungal infections in pregnancy despite its serious side effects i.e. renal toxicity
- Terbinafine; approved for the treatment of onychomycosis

Category |C|

- Ketoconazole; inhibits placental microsomal aromatase & cytochrome P-450

Category |C/D|

- Fluconazole; depends on doses & duration of use

Category |X|

- ❖ Griseofulvin; contraindicated during pregnancy & pregnancy should be avoided for 1 month after treatment

Antimalarial

- ❖ Chloroquine**; drug of choice for the prophylaxis and treatment of sensitive malaria species during pregnancy.

Cytotoxic Drugs	❖ Methotrexate |X|; Potent teratogen that produces major congenital anomalies. ❖ Cyclophosphamide – Chlorambucil |D|; Teratogenic: - growth restriction - ear and facial abnormalities - absence of digits - hypoplastic limbs ❖ Azathioprine|D|; can cause birth defects ❖ Cyclosporine|C|; does not appear to be a major human teratogen; but could cause complications like: - Preeclampsia - Eclampsia - Oligohydramnios	**Anticoagulants**	❖ **Warfarin** • Adverse effects when given during the 1st trimester, fetal warfarin syndrome (e.g. nasal hypoplasia, epiphyses stippling, bilateral optic atrophy, various degrees of intellectual disability) • Adverse effects when given during the 2nd or 3rd trimester, optic atrophy, cataracts, intellectual disability, microcephaly, microphthalmia, and fetal and maternal hemorrhage. • FDA Pregnancy category |X/D| for women with mechanical heart valves who are at high risk for thromboembolism. ❖ **Heparin** • Heparins are used for the management of venous thromboembolism in pregnancy because they do not cross the placenta. • FDA Pregnancy category: Low molecular weight heparin: |B| Unfractionated heparin: |C|
Anti-inflammatory Drugs	NSAIDs: ❖ Aspirin |D| in 3rd trimester Ibuprofen – diclofenac - celecoxib |D|; >30 weeks could cause premature closure of DA	**Insulin & Hypoglycemic Drugs**	▶ insulin is the treatment of choice for diabetes during pregnancy. ▶ Neonates born to mothers with diabetes who are taking oral hypoglycaemics in pregnancy may have hypoglycaemia. Metformin is FDA pregnancy category |B|.
Anticonvulsants	Phenytoin & Carbamazepine |D|; Potent teratogen - Fetal Hydantoin Syndrome 5-10% - IUGR - Craniofacial anomalies - Developmental delay - Mental retardation Valproic Acid|D| - neural tube defects - cognitive impairment - dysmorphic features - risk of autism	**Vitamin A Analogues**	❖ Isotretinoin |X|; Potent teratogenic - Severe birth defects - Neuropsychological impairment - Spontaneous abortion - Premature birth - Fetal death - Internal abnormalities

Diethylstilbestrol [Des]	Human teratogen |X| - Vaginal adenosis - Cervical erosions - Transverse vaginal ridges - Vaginal adenocarcinoma	**Progesterone**	▶ Danazol, Synthetic progestin (but not the low doses used in oral contraceptives), when given during the first 14 wks., masculinization of a female fetus's genitals. FDA pregnancy category |**X**| ▶ Progestin exposure is associated with an increased prevalence of cardiovascular abnormalities. Combined Oral contraceptive pills, when taken during the early stages of an unrecognized pregnancy, are believed to be teratogenic agents.
Lithium |D|	▶ Neonatal lethargy, hypotonia, poor feeding, hypothyroidism, goiter, and nephrogenic diabetes insipidus ▶ Increased risk of Ebstein's anomaly when it is used in early pregnancy.		
Cardiovascular drugs	❖ **ACE inhibitors, ARBs** ▶ Contraindicated in pregnancy. |C| for the 1st trimester of pregnancy and |D| during the 2nd and 3rd trimesters**.** ▶ Prenatal exposure to an ACE inhibitor (e.g. enalapril) or to an angiotensin II receptor antagonist (e.g. losartan) during the 2nd or 3rd trimester of pregnancy is associated with an increased risk for fetal hypotension, renal failure, and oligohydramnios leading to fetal growth restriction, joint contractures, pulmonary hypoplasia, & stillbirth or neonatal death. ❖ **β-Blockers** |C| ▶ Can cause Fetal bradycardia, hypoglycemia, & possibly fetal growth restriction ❖ **Ca channel blockers** l C l ▶ When given during the 1st trimester, possibly phalangeal deformities ▶ When given during the 2nd or 3rd trimester, fetal growth restriction ❖ **Amiodarone** |D| ▶ should only be given during pregnancy when there are no alternatives and benefit outweighs risk.	**Antithyroid drugs**	❖ Carbimazole - Propylthiouracil (PTU) Both drugs cross the placenta and may cause fetal hypothyroidism in high doses. PTU is preferred for new cases as there is less transfer across the placenta.
		Corticosteroids |B|	▶ When used during the 1st trimester, possibly orofacial clefts ▶ Hydrocortisone and prednisolone are largely (90%) metabolized by placental dehydrogenase, but fluorinated corticosteroids (e.g. betamethasone) and dexamethasone are not, thus making them the drugs of choice when treating the fetus is the aim of therapy, such as for fetal lung maturation.
		GI drugs	❖ Omeprazole does not seem to be teratogenic, but less is known about other PPIs during pregnancy. ❖ Ranitidine crosses the placenta. Although the manufacturer advises use should be avoided during pregnancy, epidemiological study reveals no increased prevalence of adverse fetal outcomes. Rodent teratogenicity studies are reassuring. ❖ Metoclopramide l B l

	❖ **Thiazide diuretics**|D| ▶ Can cause neonatal hyponatremia, hypokalemia, & thrombocytopenia ❖ **Methyldopa** |B| ❖ **Statins** |**X|** ▶ statins should be avoided during pregnancy – congenital anomalies have been reported. ▶ Neonatal lethargy, hypotonia, poor feeding, hypothyroidism, goiter, and nephrogenic diabetes insipidus ▶ Increased risk of Ebstein's anomaly when it is used in early pregnancy.	**Benzodiazepines** |D|	▶ If benzodiazepines (especially those with a long half-life) are taken in late pregnancy, they can cause neonatal respiratory depression, poor temperature regulation, poor feeding and hypotonicity. ▶ Risk of neonatal withdrawal symptoms and craniofacial anomalies. ▶ Avoid regular use and use only if there is a clear indication such as seizure control.
Caffeine	▶ Consuming caffeine in small amounts (e.g. 1 cup of coffee/day) appears to pose little or no risk to the fetus. ▶ Some data, suggest that consuming large amounts increases risk of stillbirths, preterm deliveries, low birth weight, and spontaneous abortions.	**Opioids**|C|	▶ Codeine, Meperidine, Morphine ▶ In neonates of women addicted to opioids, withdrawal symptoms possibly occurring 6 h to 8 days after birth ▶ With high doses given in the hour before delivery, possibly neonatal CNS depression and bradycardia
Aspartame (artificial sweetener)	▶ Use during pregnancy is often questioned. The most common metabolite of aspartame, phenylalanine, is concentrated in the fetus by active placental transport; toxic levels may cause intellectual disability. However, when ingestion is within the usual range, fetal phenylalanine levels are far below toxic levels. Thus, moderate ingestion of aspartame (e.g. no more than 1 liter of diet soda per day) during pregnancy appears to pose little risk of fetal toxicity	**Respiratory drugs**	❖ There is no convincing evidence that any of the drugs commonly used to treat respiratory disorders cause particular problems during pregnancy. ❖ Pseudoephedrine: possible risk of gastroschisis. FDA pregnancy category |C| ❖ Loratadine; Possible risk of hypospadias. FDA pregnancy category |B|

Smoking	▶ Carbon monoxide and nicotine in cigarettes cause hypoxia and vasoconstriction, increasing risk of spontaneous abortion, fetal growth restriction, abruptio placentae, placenta previa, premature rupture of the membranes, preterm birth, chorioamnionitis, and stillbirth. ▶ Neonates whose mothers smoke are also more likely to have anencephaly, congenital heart defects, orofacial clefts, sudden infant death syndrome, deficiencies in physical growth and intelligence, and behavioral problems. ▶ Smoking during pregnancy is linked to childhood asthma.	**Vaccines**	❖ Killed virus, toxoid, or recombinant vaccines may be given during pregnancy. ❖ Live attenuated vaccines (varicella, measles, mumps, polio, and rubella) should be given 3 months before pregnancy or postpartum. ❖ **Live virus vaccines are contra-indicated in pregnancy** secondary to the potential risk of fetal infection.
		SSRI	❖ Fluoxetine (category |C|) is the SSRI with lowest known risk in pregnancy. ❖ Paroxetine is category |D| ▶ SSRIs should not be used during pregnancy unless the potential benefit outweighs the risk. ▶ There is a small increased risk of congenital heart defects when SSRIs are taken during early pregnancy. ▶ If SSRIs are used during the third trimester there is a risk of neonatal withdrawal symptoms, and persistent pulmonary hypertension in the newborn has been reported.
Alcohol	▶ Increases risk of spontaneous abortion. ▶ Decreases birth weight by about 1 to 1.3 kg if regular drinking. ▶ Binge drinking in particular can cause fetal alcohol syndrome. This syndrome may include fetal growth restriction, facial and cardiovascular defects, neurologic dysfunction, Vision or hearing problems, behavioral, and intellectual disabilities. ▶ It can cause neonatal death due to failure to thrive (FTT)	**Tricyclic antidepressants**	❖ Tricyclic antidepressants (amitriptyline, imipramine, & nortriptyline) have lower known risks than other newer antidepressants.

GYNECOLOGY

<table>
<tr><th colspan="3">HUMAN PAPILLOMAVIRUS (HPV)</th></tr>
<tr><th>OVERVIEW</th><th>PREVALENCE & TRANSMISSION</th><th>CLINICAL PRESENTATION</th></tr>
<tr>
<td>• There are about 200 HPV genotypes, 30 to 40 types of HPV are typically transmitted through sexual contact and infect the anogenital region.
• The most clinically evident results of infection with human papillomavirus (HPV) are condyloma acuminata or genital warts.
• Majority of genital warts are caused by HPV types 6 and 11 which have little oncogenic potential.
• HPV types 16 and 18 may cause flat warts and have been linked with the dev of cervical ca.</td>
<td>• Common viral STI with an estimated 20 million infected persons in the US and 5 million new cases every year.
• About 75% of sexually active adults will be infected sometime in their life.
• Transmission of HPV can occur even when there are no visible lesions (latent).
• During pregnancy, condylomata may increase in number and size, however, transmission from mother to infant is very rare.</td>
<td>• Mostly asymptomatic; no visible lesions (latent infection).
• Characterized by readily visible "warty" growths (condylomata acuminata) on the vulva, vagina, cervix, urethra, and perianal area, that can be pruritic or cause bleeding.
• HPV infection usually clears spontaneously within 2 years, but recurrences are common.</td>
</tr>
<tr><th colspan="2">DIAGNOSIS</th><th>TREATMENT</th></tr>
<tr>
<td colspan="2">• Clinical exam
• Biopsy of the lesion (uncertain of diagnosis or for lesions that are unresponsive to therapy)</td>
<td>• Provider-applied topical therapies include: podophyllin resin 10% to 25% in tincture of benzoin and trichloroacetic acid (TCA) or bichloracetic acid (BCA) 80% to 90%.
• Patient-applied topical therapies include: podofilox solution or gel and imiquimod cream.
• Surgical therapies include: cryotherapy, manual excision, electrocautery, laser vaporization, and intralesional interferon.
• Reasons for treating visible genital warts are to relieve symptoms (pain and/or bleeding) and sometimes for cosmetic concerns of the patient</td>
</tr>
</table>

HERPES SIMPLEX VIRUS (HSV)			
OVERVIEW	PREVALENCE	TRANSMISSION	COMPLICATION
• STD caused by the HSV type I or type 2. • HSV-1 is most commonly associated with oral lesions (cold sores), but about 30% of primary GH is due to HSV-1. • HSV-2 is the cause of 70% of primary GH and 95% of recurrent GH. • Characterized by repeated eruptions of small, painful blisters on the genitals, around the rectum, or covering adjacent areas of skin (**genital ulcer**).	• Genital herpes (GH) is the most prevalent STD in the US. • Although only about 5% of women report a history of genital herpes infection, as many as 25% to 30% have antibodies on serologic testing (asymptomatic). • Transmission from an infected male to his female partner is more likely than from an infected female to her male partner.	• Virus enters body thru mucosa or microabrasions in the skin and follows the sensory nerves to the dorsal spinal ganglion where it remains dormant until reactivated. • Transmission occurs through intimate genital, oral or anal contact. • An infected mother can transmit the virus to her infant during delivery resulting in significant fetal mortality and morbidity.	• Psycho distress. (counseling) • Neuro involvement (aseptic meningitis, transverse myelitis or autonomic neuropathy). • Herpes keratitis (corneal scarring and blindness). • ↑ risk of HIV infection • Neonatal herpes (vertical transmission)

PRIMARY	RECURRENT	NEONATAL
• Typically asymptomatic • Presents up to 3 weeks after acquisition • Begins with flulike symptoms (malaise, myalgias, nausea, diarrhea, and fever). • Vulvar burning and pruritus followed by multiple bilateral vesicles that appear next and usually remain intact for 24 to 36 hours before evolving into painful genital ulcers. • Inguinal adenopathy, dysuria and acute urinary retention may occur. • Require a mean of 10 to 22 days to heal, with no scarring.	• When the virus becomes reactivated and travels down the sensory nerve to the mucoepithelial surface. • Can occur as frequent as 1-6x/yr. • Trigger factors include fever, menses, emo stress, or local trauma. • Usually occur in the same area, unilateral, may be less painful than those of the first episode but can still be uncomfortable. • Systemic sx are uncommon with recurrences. • By the 7-9th day, most lesions are healed without scarring.	• 85% of cases occur during birth, 5% are infected in utero, and10% of cases are acquired postnatally. • Risk of transmission to the newborn is 30-57% in cases where the mother acquired a primary infection in the 3rd trimester of pregnancy, risk of transmission by a mother with existing antibodies for both HSV-1 and HSV-2 is about 1-3%. • Neonatal herpes may take three forms: 1) **Disseminated** (with the high morbidity and mortality despite appropriate treatment) 2) **CNS** only (high morbidity, some mortality despite treatment) 3) **Skin/Eye/Mouth** involvement only (low morbidity and almost no mortality with treatment)
DIAGNOSIS	TREATMENT	MANAGEMENT FOR NEONATAL HERPES
• Clinical diagnosis • Viral culture from vesicular fluid (the gold standard; low sensitivity) • DNA PCR assays for HSV (experimental) • A Tzanck smear (cytology) • Type- specific antibodies for HSV-1 and HSV-2 IgG (diagnose the primary infection and the serotype of the causative organism).	• The goals of treatment for GH are sx relief, acceleration of lesion healing, and a ↓ in frequency of recurrences. • Antiviral - acyclovir, famciclovir, and valacyclovir are safe and effective for treating primary and episodic outbreaks, and suppressive therapy for patients with chronic disease. • No treatment completely eradicates virus. • Education and supportive counseling.	• Acyclovir may be administered orally to pregnant women with mild or moderate outbreaks, but for primary infections or severe recurrent outbreaks, IV therapy should be administered. • At the onset of labor, all women should be questioned carefully about symptoms of genital herpes, and should be examined carefully for herpetic lesions. • Patients with typical prodromal symptoms or active lesions in labor should be delivered by cesarean section. • Preventing is difficult because 70-80% of afflicted newborns are born to mothers with no hx of prior infection or signs or sx at or around the time of delivery.

<table>
<tr><th colspan="3">CHLAMYDIA TRACHOMATIS</th></tr>
<tr><th>INTRODUCTION</th><th>CLINICAL FEATURES</th><th>SCREENING</th></tr>
<tr><td>

Most common bacterial STI in US.
Obligate intracellular bacteria that grows in vitro only in tissue culture.
Infects columnar epithelium of endocervix, urethra, endometrium, fallopian tubes & rectum.
No vaccine. Antibodies doesn't protect against reinfection.

</td><td rowspan="2">Hx : Most (70% women + 50% men) are asymptomatic!

Mucopurulent cervicitis or mucopus
Acute urethritis with dysurea but minimal frequency & urgency & negative urine culture.

Examination: Mucopurulent cervical discharge & Cervical Erythema

Lab tests

Tissue culture - expensive
Antigen tests
DNA hybridization & nucleic acid amplification tests NAATs (PCR or ligase chain reaction)

</td><td>Selective screening.

All sexually active female <26yrs
All women with risk factors (unmarried, multiple sexual partners, inconsistent use of barrier contraception, previous hx of STI, pregnant)

DNA amplification test</td></tr>
<tr><th>RISKS</th><th>TREATMENT</th></tr>
<tr><td>For pregnant women: Preterm labor, Chorioamnionitis, Postpartum endometritis. 30% untreated chlamydial cervicitis progress to PID.
For infant: Neonatal conjuntivitis & pneumonia</td><td>1. Presumptive treatment with appropriate antibiotics

Azithromycin 1g orally single dose, or
Doxycycline 100mg 2x/day for a week
Pregnant? Amoxicillin or erythromycin

2. Treat all sexual contacts within the past 60 days of diagnosis. PDPT.
3. Testing for other STI
4. Abstinence from sexual contact for 7 days after starting treatment.</td></tr>
</table>

<table>
<tr><th colspan="3">GONORRHEA</th></tr>
<tr><th>INTRODUCTION</th><th>SIGNS & SYMPTOMS</th><th>DIAGNOSTIC TESTS</th></tr>
<tr>
<td>

Gram negative diplococcus Neisseria Gonorrhea
Infects cuboidal & columnar epithelium in endocervical & urethral mucosa.
Also rectal & nasopharyngeal mucus memb.
Coinfection with Chlamydia & Trichomonas.
No vaccines.
15% untreated gonococcal cervical infxn progress to PID.

</td>
<td>

Asymptomatic (Most women, but 5% men)
Increased vaginal discharge with lower ab/pelvic pain.
Dysurea with urethral discharge.
Proctitis with rectal bleeding, discharge, pain.
Endocervical mucopurulent discharge & contact bleeding.
Mucopurulent urethral discharge.
Pelvic tenderness with cervical excitation.

</td>
<td>

Gram staining: Gram –ve diploccocci in leukocytes.

Very sensitive in men. In women, 50% sensitive.

Culture: Thayer-Martin or Transgrow media. Sensitive & specific but takes time.
Nucleic acid amplification tests (NAATs): PCR & LCR. More expensive but rapid & high sensitivity & specificity.
Nucleic acid hybridization tests

</td>
</tr>
<tr><th>RISKS</th><th colspan="2">TREATMENT GUIDELINES</th><th>TREATMENT</th></tr>
<tr>
<td>To pregnant women: Preterm labor & delivery, chorioamnionitis, postpartum endometritis.
To infants: Neonatal conjunctivitis (ophthalmia neonatorum).</td>
<td colspan="2">1. Treatment with appropriate antibiotics.
2. Simultaneous treatment for chlamydia (1g azithromycin in single oral dose)
3. Treatment of all sexual contacts within 60days before diagnosis.
4. Abstinence from sexual activity for 7 days.
5. Testing for other STIs.
6. Counselling regarding long-term complications: Chronic pelvic pain, tubal infection, subfertility.</td>
<td>

Single oral dose of cefixime, or
Single IM dose of ceftriaxone, or
Single IM dose of spectinomycin, or
Single oral dose of ciprofloxacin, or
Single dose of oral (Ampicillin 2g or Amoxycillin 1g) + Probenicid 2g

Pregnant? Penicillin & cephalosporin are safe!
</td>
</tr>
</table>

SYPHILIS

INTRODUCTION

- Treponema pallidum, motile anaerobic spirochete.
- Can be eradicated, yet can reinfect.
- Spread: STD or Congenital

DIAGNOSIS

- History and physical examination
- Lab investigations: Blood tests
  - Non-specific – VDRL, rapid plasma reagin test
  - Treponema-specific – TPHA, FTA-Abs
- Dark-field microscopy

TREATMENT

- Mode of delivery: Vaginal delivery; C-section only for obstetric indications.
- STD Prevention: Avoid multiple sexual partners, promote use of barrier contraceptives.
- Treatment: Benzathine penicillin (G) in pregnancy
- Allergy? Full penicillin dose with oral desensitization regimen under controlled conditions.

PRIMARY SYPHILIS

- 1st stage after infxn. Appear 2-3wks after contact.
- "Chancre" - Firm, painless, non-itchy ulcers with rolled edges most commonly on vulva, vagina or cervix.
- Spontaneously disappear.

SECONDARY SYPHILIS

- 2-3 months after contact
- Systemic sphirochetemia.
- Fever, malaise, general adenopathy, maculopapular skin rash "money spots". Broad exophytic excrescences "condyloma lata" on vulva.
- Spontaneously disappear.

LATENT SYPHILIS

Absence of sx or physical findings.

TERTIARY SYPHILIS

- Necrotic, ulcerative nodules "gumma"
- Sx dependent on which organ affected.

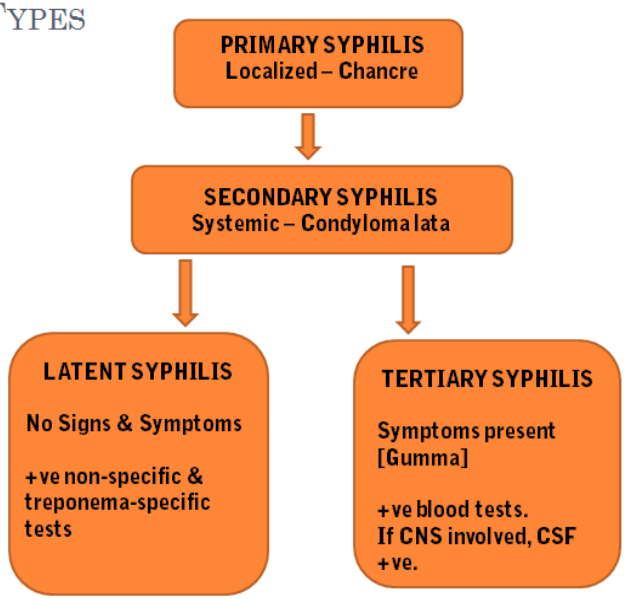
CVS: Aortitis, saccular aneurysm
CNS: Meningitis, Tabes dorsalis, Dementia, Ataxia
MSS: Osteitis

CONGENITAL SYPHILIS

Transmission: Transplacental passage from mother to fetus during delivery. Or, at birth.
Birth outcomes in congenital syphilis: Low birth weight, prematurity, congenital anomaly, miscarriage or death of baby.

Early Manifestation (<2 yrs)	Late Manifestation (>2 yrs)
Non-immune hydrops, macerated skin, anemia, thrombocytopenia, hepatosplenomegaly. Fetal death rates high – Perinatal mortality rate 50%. Placenta typically large & edematous.	"Hutchinson" teeth "mulberry" molars "saber" shins "saddle" nose 8th nerve deafness.

Types



	Primary	Secondary	Latent	Tertiary
VDRL	-	+	+	+
FTA-Abs	+	+	+	+
Dark field	+	+	+	+
CSF				+ if CNS involved

CONTRACEPTION

<table>
<tr><th colspan="2">FEMALE CONTRACEPTION</th></tr>
<tr><td colspan="2">1. BARRIER METHODS
Methods that physically or chemically block sperm from reaching an egg AND provide a BARRIER between direct skin to skin contact.
⦿ Diaphragm
⦿ Cervical cap
⦿ Female condom</td></tr>
<tr><td>Female Condom
-A polyurethane sheath intended for one-time use, similar to the male condom.
-2 flexible rings and measures 7.8 cm in diameter and 17 cm long.
-ring at the closed end of the sheath serves as an insertion mechanism and internal anchor that is placed inside the vaginal canal. The other ring forms the external patent edge of the device and remains outside of the canal after insertion
-79 to 95 percent effective
-Worn by the woman, barrier to the passage of semen into the vagina
-It can be inserted up to eight hours before sexual intercourse</td><td>Advantages
• Provides some protection to the labia and the base of the penis during intercourse.
• The sheath is coated on the inside with a silicone-based lubricant.
• It does not deteriorate with oil-based lubricants.
Disadvantages
• The lubricant does not contain spermicide.
• The device is difficult to place in the vagina.
• The inner ring may cause discomfort. Some users consider the female condom cumbersome.
• The female condom may cause a urinary tract infection if left in vagina for a prolonged period</td></tr>
<tr><td>Diaphragm
-A shallow latex cup with a spring mechanism in its rim to hold it in place in the vagina.

-manufactured in various diameters. A pelvic examination and measurement of the diagonal length of the vaginal canal determines the correct diaphragm size. It is inserted before intercourse so that the posterior rim fits into the posterior fornix and the anterior rim is placed behind the pubic bone.

-Spermicidal cream or jelly is applied to the inside of the dome, which then covers the cervix.
- provides effective contraception for 6 hours
-If a longer interval has elapsed without removal of the diaphragm, fresh spermicide is added with an applicator. After intercourse, the diaphragm must be left in place for at least 6 hours

-Prevents pregnancy by acting as a barrier to the passage of semen into the cervix</td><td>Efficacy
• Effectiveness of the diaphragm depends on the age of the user, experience with its use, continuity of use, and the use of spermicide. The typical-use failure rate within the first year is estimated to be 20%.
Advantages
• Does not require hormonal usage. Contraception is controlled by the woman.
• may be placed by the woman in anticipation of intercourse.
Disadvantages
• Prolonged use during multiple acts of intercourse may increase the risk of urinary tract infections.
• Usage for longer than 24 hours is not recommended due to the possible risk of toxic shock syndrome (TSS).
• The diaphragm requires professional fitting. Poorly fitted diaphragms may cause vaginal erosions.
• Diaphragms have a high failure rate. Use of a diaphragm requires brief, formal training.
• may develop an odor if not properly cleansed.</td></tr>
</table>

Cervical Cap -A cup-shaped latex device that fits over the base of the cervix. -The cap must be filled one third full with spermicide prior to insertion. -It is inserted as long as 8 hours before coitus and can be left in place for as long as 48 hours. -A cervical cap acts as both a mechanical barrier to sperm migration into the cervical canal and as a chemical agent with the use of spermicide -Effectiveness depends on the parity of women due to the shape of the cervical os. With perfect use in the first year, the failure rate for nulliparous women is 9%, as opposed to 20% in parous women.	Advantages • It provides continuous contraceptive protection for its duration of use regardless of the number of intercourse acts. Unlike with the diaphragm, additional spermicide is not necessary for repeated intercourse Disadvantages • Cervical erosion may lead to vaginal spotting. • A theoretical risk of TSS if it is left in place longer than the prescribed period. • Requires professional fitting and training for use. • Severe obesity may make placement difficult. • It has a relatively high failure rate. • Candidates must have history of normal results on Papanicolaou (Pap) tests.

2. SPERMICIDES

- Creams, Films, Foams, Jellies, Pessaries, Sponges
- nonoxynol-9 or octoxynolx
- must be inserted into the vagina prior to each coital act.

Spermicides prevent sperm from entering the cervical os by attacking the sperm's flagella and body, reducing their mobility, and disrupting their fructolytic activity, thereby inhibiting their nourishment.

Advantages • The lubrication provided by spermicides may heighten satisfaction in both partners. • The ease of application. Either partner can purchase and apply spermicide because it is easily accessible, available over the counter, and inexpensive. • Applying spermicide requires minimal patient education. It augments contraceptive efficacy of the cervical cap and diaphragm. Spermicides produce no adverse systemic effects.	Disadvantages • Spermicides provide minimal protection from STDs. • Insertion may be uncomfortable for some couples. • Vaginal irritation is possible, and spermicides may cause an allergic reaction.

3. HORMONAL METHODS

Oral contraceptive - Combined estrogen/ progestogen
- Progestogen only

Depot progestogens – Injections
- Subcutaneous silicone implants

Vaginal - Silicone rings releasing estrogen & progestogen

Two types of estrogens are used

- ethinyl estradiole & mestranol. Mestranol is converted in the body to ethinyl estradiole
- Several progestins of varying potency are used in the combined OCP

Types of progestins in COCP

- Estrane ➡ Norethindrone, ethynodiol diacetate
- Gonane ➡ Levonorgestrel, desogestrel, norgestimate (gonans more potent)

PROGESTINS IN COCP

- Progestins are also classified to 1st, 2nd, 3rd, generation progestins
- 2nd ➡ levonorgestril
- 3rd ➡desogestril & gestodene
- Norgestimate ➡partly converted to levonorgestril ➡included in 2nd or 3rd gp
- Newer progestins ➡ desogestril & norgestimate have little or no androgenic activity
- VTE is 2 folds higher in preparation containing 3rd generation progestins when compared to 2nd generation

Dosage & regimen

- Estrogen ➡ 20-35µg/ day
- Better cycle control with higher estrogen dosage but the efficacy is the same
- Used for 3 wks with one wk gap when menstruation occurs

Formulations

- Monophasic ➡ contains fixed amount of estrogen & progestin
- Biphasic ➡ a fixed amount of estrogen, while the progestin increases in the 2nd half of the cycle
- Triphasic ➡ the amount of estrogen may be fixed or variable, while the amount of progestin increases in 3 equal phases

Efficacy

- COCP is highly effective 99.9% in preventing pregnancy.
- 30% of women miss 3 or more pills in the 1st cycle of use
- 47% miss 1 or more pills
- ↑ body Wt may ↓ the efficacy of the pills (not proven)

Indication

- Any women seeking a reversible, reliable, coitally-independent method of contraception, in the absence of contraindications

ESTROGEN IN COCP

-reduction in the dosage of ethinyl estradiol to 20 mcg to improve the safety and reduce adverse effects → decrease in the incidence of estrogen-related adverse effects, such as weight gain, breast tenderness, and nausea

Mechanism of action

- Suppression of gonadotropin secretion ➡ inhibition of ovulation (main mechanism)
- Development of endometrial atrophy making it unreceptive to implantation
- Production of viscous Cx mucous that impede sperm transport
- Possible effect on the secretions & peristalsis of the fallopian tube interfering with ovum & sperm transport

COMBINED ORAL CONTRACEPTIVE PILLS	
Absolute contraindications • < 6 Wk postpartum if breastfeeding • Smoker , > 35 Y of age • HPT systolic ≥ 160 mm Hg or diastolic ≥ 100 mm Hg • Current or past Hx of venous thromboembolism VTE • Ischemic heart disease • Hx of cerebrovascular accident • Complicated valvular heart disease (pulmonary HPT, atrial fibrillation, subacute bacterial endocarditis) • Migraine headache with focal neurological symptoms • Current breast cancer • Diabetes with retinopathy/ nephropathy/ neuropathy • Severe liver cirrhosis • Liver tumour (adenoma or hepatoma) Relative contraindications • Adequately controlled HPT • HPT systolic 140-159 mm Hg, diastolic 90-99 mm Hg • Migraine headache > 35 Y of age • Currently symptomatic gallbladder disease • Mild liver cirrhosis • Hx of COCP related cholestasis • Medications that might interfere with OCP metabolism Incidence of pill failure that results in pregnancy is approximately 1-2% per year (1-2 pregnancies per 100 women per year of use)	Side effect COCP Minor side-effects commonly occur during the 1st 3 cycles & may lead to unnecessary discontinuation 1. Irregular bleeding (breakthrough bleeding/ spotting) • 10-30% in the 1st month of use • improves with time over 3 cycles • amenorrhea 2-3% of the cycles 2. Breast tenderness & nausea • Improve with time • Less with lower estrogen dosage 3.Wt gain • Placebo controlled trials have failed to show any association between wt gain & COCP 4.Mood changes • Women report depression & mood changes • Placebo controlled trials have failed to show any significantly increased risk of mood changes with COCP

<table>
<tr><td>Non-contraceptive benefits
• Cycle regulation
• ↓↓ menstrual flow ➡ ↓↓ anemia
• ↓↓ dysmenorrhea
• ↓↓ acne
• ↓↓ hirsutism
• ↓↓ ovarian ca 50% ↓↓ after 5 Y of use
• ↑↑ bone mineral density
• reduce and sometimes eliminate mittelschmerz.
• ↓↓ endometrial ca 50% ↓↓
• ↓↓ risk of fibroids
• Possibly ↓ ovarian cysts
• Possibly ↓ benign breast disease
• Possibly ↓ colorectal ca
• ↓↓ incidence of salpingitis
• ↓↓ incidence or severity of premenstrual syndrome
• ↓↓ peri-menopausal symptoms
• Ectopic pregnancies are prevented by the cessation of ovulation.</td><td colspan="2">Oral contraceptives
1. prevent epithelial ovarian and endometrial carcinoma, 40% reduced risk of malignant and borderline ovarian epithelial cancer. This protection appears to last for at least 15 years following discontinuation of use and increases with duration of use.
2. 50% reduction of risk of endometrial adenocarcinoma. Protection appears to persist for at least 15 years following discontinuation of use.
3. Hepatocellular adenoma, these tumors are histologically benign, their danger lies in the risk of rupture of the capsule of the liver, leading to extensive bleeding and, possibly, death. With low-dose oral contraceptive combination, the risk for liver tumors is much lower.
4. lower risk of thromboembolism
5. dose-related effect on blood pressure
6. minimal risk increases the risk of cervical neoplasia, women who use oral contraceptives should have annual Pap tests
7. does not lead to coronary atherosclerosis.
8. a woman's habits are more significant than the use of oral contraceptives in determining her risk for cardiovascular disease. The patient who is sedentary, is overweight, smokes heavily, is hypertensive, is diabetic, or has hypercholesterolemia is clearly at risk to get CVD.</td></tr>
<tr><td>Misconceptions
-Women on COCP should have periodic pill breaks
Fact ➡this would ↑↑ risk of unwanted pregnancies & cycle irregularities

-COCP affects future fertility
Fact ➡ fertility restored 1-3 M after stopping the pills

-COCP causes birth defects if a woman becomes pregnant while taking it
Fact ➡There is no evidence that it causes birth defects

-COCP must be stopped in all women >35 Y
Fact ➡Healthy non-smoking women can continue taking the pills until menopause

-COCP causes acne
Fact ➡ it improves acne due to ↓ circulating free androgens</td><td>Initiation
Patient assessment
• A thorough Hx to exclude contraindications, smoking & medications
• BP
• Pelvic exam not mandatory before prescribing COCP
• No routine lab screening is required
Counselling
• Instructions on how to use the pills
➡ To start in the 1st 5 days of the cycle
➡Quick start method ➡ any day of the cycle
➡ requires the use of back up method of contraception for the 1st wk</td><td>Counselling
• Women who use 21 –day preparation should be cautioned not to exceed the 7 day pill-free interval between packs
• Discussing what to do if a pill is missed
• Information about side-effects, risks & non-contraceptive benefits of COCP
• Discussing warning signs & when to come to the hospital
• The use of COCP in a continuous fashion
• COCP must be stopped 4 wks prior to major surgery or users should be given antithrombotic prophylaxis</td></tr>
</table>

Risks of COCP

1-Venous thromboembolism

- VTE 3-4 X higher in users than nonusers
- Absolute risk of VTE in COCP users – 1-1.5/10 000/year
- Risk of VTE is higher during the 1st year of use than subsequent years
- Incidence of VTE in nonpregnant women is 0.3/ 10000/year at 20-24 Y------0.6 at 40-44 Y
- Incidence of VTE in pregnancy is 13/ 10000 deliveries
- The risk is attributed to the estrogen component of the pill & decline with lower dosage

2- Myocardial infarction

- In the past with pills containing >50µg ethinyl estradiole --- 3X ↑↑ in MI
- Recent studies with pills containing < 50µg ethinyl estradiole ----- No significant ↑↑ risk

3-Stroke

- Some studies showed 2X ↑↑ risk of stroke
- Smoking & HPT ↑↑ risk of stroke

4-Gallbladder disease

- COCP ↑↑ secretion of cholic acid in bile ➡↑↑ incidence of gallstone formation

5-Breast cancer

- Still controversial, the risk is small and the resulting tumors spread less aggressively than usual.
- A large meta-analysis 1996 ➡ significant ↑ risk of breast ca in women currently taking the COCP & in the 1st 10 Y after discontinuing it
- Cumulative breast ca risk up to age 35 is 2 / 1000, with COCP 3 / 1000
- It is not known whether this ↑ is due to the pills or due to delaying the 1st full term birth
- More recent study > 9000 women ➡➡ no significant ↑↑ in breast ca risk

➡➡No ↑↑ risk with different dosage of estrogen, longer periods of use, or with different progestin components

➡➡No ↑↑ risk in Pt with family Hx of breast ca

➡➡No ↑↑ risk in Pt who started using the pills at an earlier age

➡➡↑↑ risk in Pt who carry BRCA1, BRCA2 genes

6-Cervical cancer

- One study ➡↑↑ risk of Cervical ca in long term COCP users who are HPV positive
- A review of 28 studies of women with Cervical ca ➡↑↑ risk of Cervical ca with ↑↑ duration of COCP use
- Probably due to ↑↑ risk of HPV (a major risk factor for cervical ca) that might be related to sexual behavior which differs in users & non users of COCP
- Another study HPV + ve women followed up for 10 years showed no increased risk

Troubleshooting

1-Breakthrough bleeding

- To continue on the same pills with the expectation that it will improve with time (rather than switching to another preparation)
- If bleeding persists beyond 3 M (or new onset of bleeding in a long term user) rule out other causes of bleeding:

-irregular taking of the pills
-pregnancy
-infections
-uterine or Cervical pathology
-malabsorption/ diarrhea , vomiting
-concomitant use of medications

Management of breakthrough bleeding

- Oral estrogens: premarine 1.25 mg or estradiole -17 ß /7 days
- Change the another preparation with different progestin

2-Missed pills

- <12 hrs Take the pill as soon as you remember (this means taking 2 in 1 day)
- >12 hrs use another method for 1 week
- If 2 pills in a row missed in the 1st 2 wks of the pack ➡ take 2 /day for 2 days
- If 2 pills in a row missed in the 3rd wk of the pack ➡ through the remainder of the pack & start a new one / use back up contraception in the first 7 days of the new pack
- If 3 pills in a row missed ➡ follow steps above
- If intercourse occurred after missing a pill ➡ use emergency contraception

3-Amenorrhea

- It occurs in 2-3% of COCP users
- Pregnancy should be ruled out
- It is not dangerous ➡ no need for Rx
- If not acceptable by Pt ➡ change preparation

➡Add oral estrogen for 10 days

4-Chloasma

- Darkening of the facial skin
- Changing to another preparation will not help
- It may never completely disappear
- Use of sunscreen to prevent further darkening

5-Breast tenderness & galactorrhea

- Often resolves with continued use
- ↓ caffeine intake may help
- ↓ estrogen content
- Galactorrhea is rare ➡ if it happens ➡ check prolactin level

6-Nausea

- ↓ with time
- Taking the pill with food or bedtime
- ↓ estrogen content
- If it occurs in a long time user ➡rule out pregnancy

7-Pregnancy

- Pills must be stopped immediately
- There is no ↑ risk of birth defects

Metabolism & Drug interactions

-Ethinyl estradiole is metabolized at several sites:

1-Sulphated at the intestinal wall

2-Hydroxylated in the liver then conjugated with glucuronides & pass to enterohepatic circulation

-Anticonvulsants (phenytoin or carbamazepine)

➡ women should use 50 µg E estradiole pill

➡Monitor phenytoin level as COCP may inhibit its metabolism

-Rifampicin & griseofulvin ➡contraceptive failure

-Other antibiotics do not appear to affect the efficacy of COCP

TRANSDERMAL CONTRACEPTIVE PATCH

- Delivers 150µg norgestimate & 20 µg E estradiole daily
- One patch is applied weekly for 3 wks followed by one patch-free-wk
- Pearl index with perfect use ➡ 0.7
    - with typical use ➡ 0.88
- Women weighing more than 90 kg ➡↑ risk of pregnancy
- Mechanism of action similar to COCP
- Irregular bleeding in the 1st M of use is more 18% for the patch than COCP 11% / Amenorrhea is rare
- Breast symptoms are more 22% in the 1st 2 cycles of the patch use than COCP use
- Local skin reaction 20%
- This skin patch is worn on the lower abdomen, buttocks, or upper body

Advantages

- greater compliance
- decreased adverse effects, such as nausea and vomiting, due to the avoidance of the first-pass effect.

Disadvantages

- may cause skin irritation
- removed unnoticed, such as from showering, this may compromise efficacy

COMBINED INJECTABLE CONTRACEPTION

- Monthly injectable contraceptive composed of 5 mg estradiole cypionate & 25 mg medroxyprogestrone acetate
- Less breakthrough bleeding
- Amenorrhea 14.6% compared to 3.3 in COCP users
- Wt gain 4 pounds/year

VAGINAL CONTRACEPTIVE RING

- A flexible transparent ring 54 mm diameter /4 mm cross-sectional diameter
- Releases 15µg E estradiole & 0.12 mg of desogestrel (etonogestrel)/ day
- Ring is used for 3 wks continuous followed by one ring-free wk
- Irregular bleeding 6.4 % less than COCP especially in the 1st cycle
- Headache 11.8%, nausea 4.5%, breast tenderness 2.8%
- Vaginitis 13.7% (5% Rx related), coital problem or expulsion 1-2.5%
- The ring contains 11.7 mg of etonogestrel and 2.7 mg of ethinyl estradiol. It releases 120 mcg of etonogestrel and 15 mcg of ethinyl estradiol each day. The hormones are released slowly and are absorbed directly by the reproductive organs
- The ring can be inserted any time during the first 5 days of the menstrual cycle.
- The ring should be placed in the vagina even if the woman has not finished bleeding, and she should use a backup contraceptive method for 7 days.
- A new ring should be inserted each month. If the ring comes out during the first 3 weeks of use, it should be washed with lukewarm water and replaced.
- If the ring-free interval is more than 3 hours, a backup contraceptive method should be used for 7 days.
- The ring should never be left in the vagina for more than 4 weeks. If left in for more than 4 weeks, pregnancy should be excluded before inserting a new ring and a backup contraceptive method should be used for 7 days after inserting a new ring

Advantages

- NuvaRing is highly effective because it results in complete suppression of ovulation. The steady release of hormone provides exceptional cycle control. The ring is a very effective reversible method of birth control.
- The ring delivers the lowest dose of ethinyl estradiol compared with other combined hormonal contraceptives. Unlike combined oral contraceptives, the adverse effects of nausea and vomiting are avoided with ring use
- Because daily intake is not a component of NuvaRing contraception, because it is easily inserted and removed by the woman herself, and because return of fertility is rapid upon discontinuation, NuvaRing is a highly acceptable method for women and their partners.
- Because the hormones are absorbed directly into the blood through the vaginal mucosa, the hepatic first-pass metabolism of progestin is prevented.

Disadvantages

- Adverse effects include headaches and vaginal irritation or discharge.
- The ring may accidentally slip out during intercourse and either the user or the partner may feel the ring during sexual intercourse.
- Contraindications are similar to those of combined oral contraceptive

  - It is 91 to 99 percent effective at preventing pregnancy
  - Ring goes inside vagina up around the cervix
  - This method does not protect from HIV or other STDs.

<table>
<tr><th colspan="2">PROGESTIN ONLY HORMONAL CONTRACEPTION</th></tr>
<tr><td>INJECTABLE PROGESTIN [DEPOT MEDROXYPROGESTRONE ACETATE]
• Highly effective with a failure rate < 0.3% / year
Mechanism of action
• Inhibiting the secretion of pituitary gonadotropins ➡ suppression of ovulation *1ry mechanism*
• ↑↑ viscosity of Cervical mucous
• Induces endometrial atrophy</td><td>DMPA INDICATIONS
• Any women seeking reliable, reversible, coitally independent method of contraception in the absence of contraindications
• Women who have difficulty complying with other methods / it does not require daily attention
• Women with contraindication to estrogens
• Women >35 Y who smoke
• Women with migraine headache
• Women who are breastfeeding
• Women with endometriosis
• Women with sickle cell disease
• Women taking anticonvulsant medications
• Mentally handicapped women

DMPA CONTRAINDICATIONS
Absolute contraindications
• Pregnancy
• Unexplained vaginal bleeding
• Current breast ca
Relative contraindications
• Severe liver cirrhosis
• Active viral hepatitis
• Benign hepatic adenoma</td></tr>
<tr><td>DMPA NON-CONTRACEPTIVE BENEFITS
• Amenorrhea (55-60% at 12 M / 68% at 24 M) with subsequent reduction in dysmenorrhea & anemia
• the failure rate is 0.3%.
• ↓↓ risk of endometrial ca
• ↓↓ symptoms associated with endometriosis, PMS, & chronic pelvic pain
• ↓↓ incidence of seizures
• Possible ↓↓ risk of PID
• Possible ↓↓ incidence of sickle cell crisis</td><td>Advantages
• DMPA does not produce the serious adverse effects of estrogen, such as thromboembolism.
• The risks of endometrial and ovarian cancer are decreased.
• It contains no estrogen, thus making it suitable for women who cannot or will not take estrogen products.
• It also is safe for breastfeeding mothers</td></tr>
</table>

DMPA SIDE-EFFECTS	DMPA RISKS
1- Menstrual cycle disturbance • Irregular bleeding ➡ ↓ in frequency & amount over time • Abnormally heavy or prolonged occurred only in 1-2% • Amenorrhea 55-60% at 12 M 68% at 24 M approximately 70% of former users desiring pregnancy conceive within 12 months, and 90% of former users conceive within 24 months. 2-Hormonal side effects • Headache 17% • Acne • ↓↓ libido • Nausea • Breast tenderness 3-Weight gain • 56% ↑↑ Wt (mean gain 4.1 kg) ➡ possibly through appetite stimulation & a mild anabolic effect - 2.5 kg in 1st Y -3.7 kg in 2 Y -6.3 kg in 4 Y • 44% ↓ Wt or maintained (mean loss 1.7 kg) 4-Mood effects • Prospective studies did not demonstrate ↑ depressive symptoms • Some women discontinue use because of mood changes	1-Delayed return of fertility • An average of 9 months delay before restoration of full fertility after last injection • Rate of conception 50% at 10 M, 90% at 24 M • adverse effects, such as weight gain, depression, and menstrual irregularities, may continue for as long as 1 year after the last injection. 2-Reduction in bone mineral density • A mean loss of BMD at the lumbar spine 0.87-3.5% • Does not induce osteoporosis • It improves after discontinuation of use • bone loss from using Depo-Provera "may not be completely reversible" even after stopping the drug. 3-VTE, CVD, Stroke ➡ No ↑ risk

Depo-Provera

-Not to use Depo-Provera on a long-term basis unless all other methods were inadequate.

-A subcutaneous version of the drug is now available (depo-subQ provera 104) that delivers a lower dose of medroxyprogesterone acetate (MPA) than does the intramuscular formulation (104 mg vs 150 mg).

-home self-injections, and the lower dose could decrease suppression of pituitary function and ovarian estradiol production. Further study is needed.

DMPA DOSAGE & ADMINISTERATION

- ⦿ 150 mg IM every 12 Wks
- ⦿ Started during the 1st 5 days of menses or within 5 days of stopping COCP
- ⦿ Effective within 24 hrs of injection if given during the 1st 5 days of the cycles
- ⦿ If given later than D5 of the cycle ➡ back up method of contraception must be used for 1 wk

DMPA TROUBLESHOOTING

1- Menstrual cycle disturbance

- If irregular bleeding persists after the 1st 6 M of use ➡ rule out other causes of abnormal bleeding

Management options

- ↑↑ DMPA dosage ➡ 225-300 mg for 2-3 injections
- ↓↓ interval between dosage
- Supplemental estrogen therapy :

➡0.625 conjugated equine estrogen po –28 days

➡1-2 mg 17ß-estradiole po –28 days

➡Transdermal estrogen 50-100 µg 17ß-estradiole patch for 25 days

- Nonsteroidal anti-inflammatory ➡ ibuprofen 400-800 mg bd for 10 days
- Adding COCP for 1-3 M

2-Late injection

- <14 wks since last injection it can be given
- ≥ 14 wks since last injection

-ve serum ß hcg, no intercourse for last 10 days

➡ give the injection

➡ back up contraception must be used for 2 wks

- ≥ 14 wks since last injection

-ve serum ß hcg, intercourse within the last 10 D

➡ give the injection

➡ back up contraception must be used for 2 wks

➡Repeat serum ß hcg –2 wks

➡Not teratogenic if inadvertently given during pregnancy

ORAL PROGESTINS PROGESTIN ONLY PILL / MINIPILLS

- Package contains 28 tab
- Started on the 1st day of the menstrual cycle/ or any day if pregnancy excluded
- Must be used at the same time every day within 3 hrs
- A back up contraception must be used for 7 days
- Norethindrone 0.35 mg ➡ micronor
- Must be used continuously ➡ no pill-free interval
- Perfect use failure rate ➡ 0.5%
- Typical use failure rate ➡ 5-10% (It must be taken the same time every day)
- It can be used immediately postpartum with no effect on lactation
- lower doses of progestin than combined oral contraceptives. One formulation contains 75 mcg of norgestrel. The other has 350 mcg of norethindrone.

Indications

- It can be used for any women seeking reliable, reversible, coitally independent method of contraception in the absence of contraindications
- Women with contraindication to estrogen
- Women > 35 Y who smoke
- Women having migraine headache with neurological symptoms
- Women who have unwanted side-effects of COCP
- Breast-feeding women

Mechanism of action

1-Main mechanism is alteration of Cervical mucous

- ↓↓ volume of mucous
- ↑↑ viscosity
- alter its molecular structure

➡Little or no sperm penetration

➡Sperm motility is impaired ➡ ↓↓ fertilization

2- Ovulation is suppressed in 60% of the women. suppression of ovulation (not uniformly in all cycles)

a reduction in cilia motility in the fallopian tube, thus slowing the rate of ovum transport

#Unlike DMPA, fertility is immediately reestablished after the cessation of progestin-only oral contraceptives.

Absolute Contraindications

- Pregnancy
- Current breast cancer

Relative Contraindications

- Active viral hepatitis
- Liver tumors

Non contraceptive benefits

- ↓ menstrual flow
- 10% amenorrhea
- ↓ dysmenorrhea, PMS

Side-effects

- Irregular bleeding

➡ spotting –12% ➡ 1st month

--3% ➡ 18 months

➡40 % continue to have regular cycles

- Hormonal side-effects
- Headache, bloating, acne, breast tenderness, nausea

Risks • Not associated with any major morbidity • No ↑ risk of VTE, stroke or MI Myths & misconception -It can only be used with breast feeding Fact ➡ It can be used in any women seeking reliable, reversible method of contraception -POP is not an effective method of contraception Fact ➡ When used correctly it is safe & effective with a failure rate of only 0.5%	Troubleshooting 1-Irregular bleeding • A common side effect • Pregnancy, infection & genital pathology must be ruled out Rx options • Non steroidal anti-inflammatory for 10 days • Switching to COCP • Adding a short course of estrogen ➡ 0.625 mg conjugated equine estrogen (premarine) for 28 days ➡1-2 mg micronized 17ß-estradiole—28 days ➡Transdermal 50-100 µg 17ß-estradiole patch –25 days Antiprogestinic agents ➡ mifepristone 2-Missed pill • To be taken as soon as possible • Next pill to be taken at the regular time • If delayed > 3hrs ➡ use back up contraception for 48 hrs • If 2 or more pills missed in a row ➡ 2 pills/day for 2 days ➡back up contraception for 48 hrs • Emergency contraception must be used if intercourse occurred after a missed pill 3- Drug interactions ➡ anticonvulsants may ↓↓ effectiveness of POP

PROGESTIN IMPLANTS

- NORPLANT ➡ Levonorgestril
- Implanon ➡ Etonogestrel
- Highly effective failure rate 0.1% / year
- NORPLANT 6 rods implanted under the skin ➡ effective for 5 years
- Implanon One rod ➡ effective for 3 years
- Reversible contraception
- Mechanism of action

➡ Suppression of ovulation
➡ Endometrial atrophy
➡ Rendering Cervical mucous impermeable to sperms
Prolonged irregular bleeding the major side effect

Norplant

- This method consists of 6 silicone rubber rods, each measuring 34 mm long and 2.4 mm in diameter and each containing 36 mg of levonorgestrel.
- The implant releases approximately 80 mcg of levonorgestrel per 24 hours during the first year of use-.
- Contraceptive protection begins within 24 hours of insertion if inserted during the first week of the menstrual cycle.
- The rods are inserted subcutaneously, usually in the woman's upper arm, where they are visible under the skin and can be easily palpated
- The mechanism of action is a combination of suppression of the LH surge, suppression of ovulation, development of viscous and scant cervical mucus to deter sperm penetration, and prevention of endometrial growth and development.

Efficacy

- The contraceptive efficacy of the method is equivalent to that of surgical sterilization. Overall, pregnancy rates increase from 0.2% in the first year to 1.1% by the fifth year.

Advantages

- The longevity of its effectiveness.
- Its effectiveness is not related to its use in regards to coitus.
- Exogenous estrogen is absent.
- Prompt return to the previous state of fertility occurs upon removal.
- No adverse effect on breast milk production occurs.

Disadvantages

- A minor surgical procedure is necessary for insertion.
- Difficulty in removal.
- Menstrual irregularities are common along with other adverse effects, including headaches, mood changes, hirsutism, galactorrhea, and acne.
- Appropriate candidates are women who are postpartum or breastfeeding, women who have difficulty with contraceptive compliance, women in whom pregnancy is contraindicated due to a medical condition, and patients with contraindications to the use of estrogen.
-

Absolute contraindications

- active thrombophlebitis or thromboembolic disease
- undiagnosed genital bleeding
- acute liver disease
- benign or malignant liver tumors
- known or suspected breast cancer
- history of idiopathic intracranial hypertension.

Relative contraindications

- heavy cigarette smoking
- history of ectopic pregnancy
- diabetes mellitus
- hypercholesterolemia
- severe acne, hypertension
- history of cardiovascular disease
- severe vascular or migraine headaches
- severe depression

Implanon • Implanon is a single-rod implant that is 4 cm long and 2 mm in diameter. It consists of 68 mg of etonogestrel in an ethylene vinyl acetate copolymer core. • Etonogestrel is a biologically active metabolite of desogestrel. Desogestrel is significantly more potent than levonorgestrel; a serum concentration of 0.09 ng/mL can inhibit ovulation in most women. • Serum concentrations are adequate for contraception coverage for approximately 3 years. • etonogestrel implant may be less effective in women who are overweight.	• Compared with the Norplant system, Implanon is associated with a higher frequency of amenorrhea and oligomenorrhea, a decrease in the prevalence of frequent and prolonged bleeding, and a decrease in the frequency of adverse effects such as weight gain, headache, and acne. • When the rod is removed, the return to fertility is rapid, with the return of ovulation within 3 weeks. • Implanon is not associated with loss of bone mineral density (BMD).

4. INTRA UTERINE DEVICES • Inert • Copper bearing • Progestogen releasing • Nonmedicated IUCD (Multiload) • Copper IUD(Nova T) • Levonorgestrel – releasing IUD (Mirena)	Efficacy • Failure rate of Nova T ➡ 1.26 % /year, Mirena ➡ 0.09 % /year • Ectopic pregnancy rate ➡0.25 %/year, Mirena ➡0.02 %/year • Effective for 5 years • failure rate is 2% with Progestasert (the progesterone form) • 0.6% with the Copper T380, 0.1 % with Mirena
Inert IUDs • Inert IUDs are IUDs with no bioactive components; they are made of inert materials like stainless steel or plastic • They are less effective than copper or hormonal IUDs, with a side effect profile similar to copper IUDs. • Their primary mechanism of action is inducing a local foreign body reaction, which makes the uterine environment hostile both to sperm and to implantation of an embryo.	**Progestasert** • The T-shaped progesterone-releasing IUD Progestasert, contains 38 mg of progesterone and minimal amounts of barium sulfate for greater visibility on x-ray films. • The vertical limbs are 36 mm long, and the horizontal arms are 32 mm wide. It has a pair of dark-blue double-strings that hang from the lower limb. • Approximately 65 mcg/d of progesterone is released from the progesterone form from a reservoir in its stem. This is a sufficient amount of hormone to last for 400 days; therefore, this IUD must be replaced yearly

Copper T380 • The Copper T380 was introduced in 1988. • The T-shaped IUD is made of polyethylene with fine copper wire wrapped around the vertical stem. • The string is clear or white and hangs from the lower limb of the IUD. This device consists of 308 mg of copper covering portions of its stem and arms. • Contraceptive effectiveness continues for 10 years, after which time it must be replaced	**Mirena** • Mirena is similar in shape to the Copper T380 in that it also consists of a small T-shaped frame with a reservoir that contains levonorgestrel, a progesterone. • This intrauterine system releases 20 mcg of levonorgestrel per day into the uterine cavity for as long as 5 years. • It consists of a polyethylene frame with a cylinder containing a polydimethylsiloxane-levonorgestrel mixture enveloping the vertical arm. The cylinder is coated with a membrane that regulates the release of the hormone. This model is also visible on x-ray films. • The Mirena device now has FDA labelling for treating menorrhagia as well
IUDS Advantages • IUDs produce no adverse systemic effects. • Ectopic pregnancies are reduced overall; however, the ratio of extrauterine to intrauterine pregnancy is increased if conception does occur. • Menstrual blood loss and dysmenorrhea are decreased with Progestasert. • Twenty percent of women experience amenorrhea with Mirena.	IUDS Disadvantages • Risk of uterine perforation at the time of insertion. • Increased dysmenorrhea occurs with the Copper T380. • Increased menstrual blood loss occurs in the first few cycles with use of the Copper T380 and Mirena IUDs. • Whether IUDs increase the risk of PID is controversial. • IUDs may be expelled unnoticed, and they do not protect against STDs.
IUCD Mechanism of action • Prevention of fertilization ➡the chief mechanism • Inhibition of implantation • Presence of foreign body & copper ➡biochemical & morphological changes in the endometrium ➡adversely affect sperm transport • Copper ion have direct effect on sperm mobility ➡ ↓↓ in its ability to penetrate Cervical mucous • Levonorgestrel releasing devices ➡ weak foreign body reaction & endometrial decidualization & glandular atrophy ➡estrogen & progestrone receptors are ↓↓ ➡Cervical mucous becomes thick & impermeable to sperms ➡ovulation may be inhibited in some women	Indication for IUCD • In the absence of contraindications may be considered for any woman seeking a reliable, reversible, coitally independent method of contraception • Women seeking long term birth control • A method requiring less compliance • Women with contraindications to estrogen • Breast feeding women • Copper IUCD used for postcoital contraception within 7 days • LNG- IUCD ➡↓↓ menstrual flow & cramping ➡suitable for women with menorrhagia & dysmenorrhea Copper IUD: Can stay for up to 10 years • Interferes with sperm, fertilization, and prevents implantation Hormonal IUD: Can stay for up to 5 years • It releases a small amount of hormone each day •

IUCD Contraindication- Absolute contraindications • Pregnancy • Current, recurrent or recent (within 3 M) PID or STD • Puerperal sepsis • Immediate post septic abortion • Severely distorted uterine cavity • Unexplained vaginal bleeding • Cervical or endometrial ca • Malignant trophoblastic disease • Copper allergy/Wilson disease, ➡Copper -IUCD • Breast ca ➡ LNG -IUCD	Relative contraindications • Risk factor for sexually transmitted diseases or HIV • Increased susceptibility to infection (eg, those with leukemia, diabetes, valvular heart disease, or AIDS,-women on corticosteroid Rx • 48hrs- 4 wks postpartum • Ovarian ca • Benign gestational trophoblastic disease • History of ectopic pregnancy
IUCD myths & conception -IUCD ➡↑↑ risk of ectopic Fact ➡ IUCD work primarily by preventing fertilization Ectopic in IUCD users : nonusers 0.02-0.25/100WY:0.12-0.5/100WY -IUCD ➡↑↑ risk of infertility Fact ➡ Women who discontinue IUCD use conceive at the same rate of women who never used IUCD -IUCD ➡↑↑ risk of long term PID Fact after the 1st M the risk of infection is not higher than non users/ PID < 2/ 1000 year of use -IUCD are not effective contraceptives Fact LNG –IUCD as effective as tubal ligation 9	Initiation - Counselling • Inserted any time during a menstrual cycle once pregnancy excluded • During menses ➡ exclude pregnancy & mask insertion related bleeding • Infection & expulsion ↑ with insertion during menses • It can be removed any day of the menstrual cycle • If there is mucopurulent discharge Cervical swabs must be taken & insertion delayed • Antibiotic prophylaxis is not indicated Follow up • A follow up visit must be scheduled in 6 wks then yearly • Women must be instructed to come if: – IUCD thread cannot be felt – She feels the lower end of the IUCD – Pregnant – Abdominal pain, fever or unusual discharge – Pain or discomfort during intercourse – Sudden change in menstrual period – Wants to remove the device or conceive

Troubleshooting

1-Lost string

- Speculum exam
- Exclude pregnancy
- Cervical canal explored
- U/S
- Plain X ray

2- Pregnancy

- Exclude ectopic
- If she wishes to continue the pregnancy ➡ remove IUCD
- If string missing ➡ u/s ➡ if in the uterus ➡ no attempt to remove it

3-Amenorrhea /delayed menses

- Exclude pregnancy
- 35% of LNG –IUCD users have amenorrhea

4-Pain & abnormal bleeding

- Exclude pregnancy, partial expulsion, perforation
- NSAID may help
- Bleeding ↓ overtime
- If it persists or worsen ➡ removal

5-Difficulty removing IUCD

- Cervical dilatation
- U/S
- Hysteroscopy

6-Sexually transmitted disease with IUCD in situ

- Antibiotics
- Removal

7-Actinomycosis on PAP smear

- It is a vaginal commensal
- 20% in Cervical smears of copper IUCD users
- 3% in LNG-IUCD users
- Removal is not necessary if asymptomatic
- If symptomatic ➡ remove IUCD after starting antibiotics / continue Ab Rx

EMERGENCY CONTRACEPTION

- Also known as the "morning after pill"
- Copper IUCD can be inserted up 7 days after intercourse
- Levonorgestrel 0.75 mg , 2 doses 12 hrly or 1.5 mg single dose ➡ similar efficacy
- Yuzpe method ➡ 2 doses 100µg E estradiole & 500 µg levonorgestrel (Ovral)
- Hormonal contraception must be started as soon as possible max 5 days
- Women should be evaluated for pregnancy if menses does not occur after 21 days
- Mechanism of action ➡Hormonal contraception ➡interferes with ovulation
  ➡other mechanisms could be interference with sperm mobility or transport, endometrial receptivity, fertilization or zygote development
- Most studies cite an effectiveness rate of 55-94%, with the true effectiveness rate likely to be approximately 75%.

Disadvantages

Adverse effects include nausea and emesis, minor changes in menses, breast tenderness, fatigue, headache, abdominal pain, and dizziness.

Ectopic pregnancy is possible if treatment fails

Copper T380 Intrauterine Device

- The Copper T380 IUD can be inserted as many as 7 days after unprotected sexual intercourse to prevent pregnancy.
- Insertion of the IUD is significantly more effective than either the ECP or MECP regimen, reducing the risk of pregnancy following unprotected intercourse by more than 99%.

Effectiveness

- Yuzpe ➡ 75% reduction in pregnancy, pregnancy rate 3.2%
- LNG ➡89% reduction, pregnancy rate 1.1%
- Effectiveness ➡↓↓ with ↑↑ delay between intercourse & contraception
- IUCD more effective ➡98.7%

Side effects

- LNG have lower incidence of nausea(23 vs 50%), vomiting (5.6vs 18.8%), dizziness (11.2vs16.7%), fatigue (16.9vs28.8%)than Yuzpe

Candidates for emergency contraception include

- Reproductive aged women who have had unprotected sexual intercourse within 72 hours of presentation independent of the menstrual cycle.
- should only be used after no birth control was used during sex, or if the birth control method failed, such as if a condom broke
- Should NEVER be used as regular birth control
- No known absolute contraindications to any of these methods have been described because exposure to the high dose of hormones is short lived. However, cases of deep vein thrombosis have been documented in women using the ECP method.

ECP

- The ECP mode is marketed as Preven. It consists of 2 pills, which each contain of levonorgestrel and 100 mcg of ethinyl estradiol, ingested 12 hours apart for a total of 4 pills. The first dose should be taken within the first 72 hours after unprotected intercourse; however, studies demonstrate effectiveness if the pills are taken after that period.

PLAN B

- Only the progestin levonorgestrel has been studied for the use in MECM.
- It is marketed as Plan B.
- Its treatment schedule comprises 1 dose of 750 mcg levonorgestrel taken as soon as possible and no later than 48 hours after unprotected intercourse and a second dose taken 12 hours later.

5. NATURAL METHODS

- Breast feeding (while baby is totally breast fed)/ Lactational amenorrhea
- Coitus interruptus
- Natural family planning/Rhythm

-One of the most widely used methods of fertility regulation, particularly for persons whose religious or cultural beliefs do not permit devices or drugs for contraception.
-This technique involves periodic abstinence, with couples attempting to avoid intercourse during a woman's fertile period, which is around the time of ovulation

Natural family planning
Techniques to determine the fertile period include
-the calendar method
-cervical mucus method
-the symptothermal method

1. The calendar method is based on 3 assumptions:

(1) A human ovum is capable of fertilization only for approximately 24 hours after ovulation
(2) spermatozoa can retain their fertilizing ability for only 48 hours after coitus
(3) ovulation usually occurs 12-16 days before the onset of the subsequent menses

-The menses is recorded for 6 cycles to approximate the fertile period.
-The earliest day of the fertile period is determined by the number of days in the shortest menstrual cycle subtracted by 18.
-The latest day of the fertile period is calculated by the number of days in the longest cycle subtracted by 11.

2. cervical mucus method

Under the influence of estrogen,

a) The mucus increases in quantity and becomes progressively more elastic and copious until a peak day is reached.
b) This is followed by scant and dry mucus, secondary to the influence of progesterone, which remains until the onset of the next menses.
c) Intercourse is allowed 4 days after the maximal cervical mucus until menstruation

3. The symptothermal method

-The basal body temperature of a woman is relatively low during the follicular phase and rises in the luteal phase of the menstrual cycle in response to the thermogenic effect of progesterone.
-The rise in temperature can vary from 0.2-0.5°C. The elevated temperatures begin 1-2 days after ovulation and correspond to the rising level of progesterone. Intercourse can resume 3 days after the temperature rise.

Efficacy

- The failure rate in typical use is estimated to be approximately 25%.

Advantages

- No adverse effects from hormones occur. This may be the only method acceptable to couples for cultural or religious reasons.
- Immediate return of fertility occurs with cessation of use

Disadvantages

- This is most suitable for women with regular and predictable cycles.
- Complete abstinence is necessary during the fertile period unless backup contraception is used.
- This method requires discipline. The method is not effective with improper use.
- The failure rate is relatively high.
- This method does not protect against STDs

Coitus Interruptus -involves withdrawal of the entire penis from the vagina before ejaculation. Fertilization is prevented by lack of contact between spermatozoa and the ovum. Efficacy The failure rate is estimated to be approximately 4% in the first year of perfect use. In typical use, the rate is approximately 19% during the first year of use Advantages • Include immediate availability, no devices, no cost, no chemical involvement, and a theoretical reduced risk of transmission of sexually transmitted diseases (STDs). Disadvantages • The probability of pregnancy is high with incorrect or inconsistent use.	**Lactational Amenorrhea** -Elevated prolactin levels and a reduction of gonadotropin-releasing hormone from the hypothalamus during lactation suppress ovulation. -This leads to a reduction in luteinizing hormone (LH) release and inhibition of follicular maturation. The duration of this suppression varies and is influenced by the frequency and duration of breastfeeding and the length of time since birth. -Mothers only need to use breastfeeding to be successful; however, as soon as the first menses occurs, she must begin to use another method of birth control to avoid pregnancy. Efficacy • The perfect-use failure rate within the first 6 months is 0.5%. The typical-use failure rate within the first 6 months is 2%. Advantages • Involution of the uterus occurs more rapidly. Menses are suppressed. can be used immediately after childbirth. facilitates postpartum weight loss. Disadvantages • Return to fertility is uncertain. • Frequent breastfeeding may be inconvenient.

6. SURGICAL METHODS / STERILIZATION

-Laparoscopic sterilisation - Fallope Rings , Clips, bands, segmental destruction with - Bipolar diathermy, Laser

-Tubal ligation: suture ligation with partial salpingectomy.

VASECTOMY

- This operation is done to keep a man's sperm from going to his penis, so his ejaculate never has any sperm in it that can fertilize an egg.
- Operation is more simple than tying a woman's tubes
- Vasectomy involves incision of the scrotal sac, transection of the vas deferens, and occlusion of both severed ends by suture ligation or fulguration
- The procedure is usually performed with the patient under local anesthesia in an outpatient setting.
- Complications include hematoma formation and sperm granulomas.
- Spontaneous resolution is rare. After sterilization, remnant sperm remains in the ejaculatory ducts.
- The man is not considered sterile until he has produced sperm-free ejaculates as documented by semen analysis. This usually requires 15-20 ejaculations.

Efficacy

- The failure rate is approximately 0.1%.

Advantages

- Vasectomy involves no hormones, is permanent, is an outpatient procedure, is quick, and carries minimal risk with regard to the procedure.

Disadvantages

- Patients may regret their decision after the procedure. Alternative contraception is required until the ejaculate is deemed free of sperm. Vasectomy does not prevent STDs. Short-term discomfort occurs

TUBAL LIGATION / TYING TUBES

- A woman can have her fallopian tubes tied (or closed) to stop eggs from being fertilized
- Over time, the ends of the fallopian tubes could fuse back together, and it may be possible to get pregnant
- failure rates vary according to the procedure performed.
- The cumulative 10-year failure rate with each method of tubal ligation is as follows: spring clip method, 3.7%; bipolar coagulation, 2.5%; interval partial salpingectomy, 2%; silicone rubber bands, 2%; and postpartum salpingectomy, 0.8%

Advantages

- Female sterilization does not involve hormones. It is a permanent form of contraception.
- No data indicate that change in libido, menstrual cycle, or lactation occurs.
- Female sterilization is usually a same-day procedure.
- It can be performed with laparoscopy, laparotomy, or colpotomy

Disadvantages

- Female sterilization is a procedure that involves general or regional anesthesia.
- It is permanent contraception, and patients may regret the decision later, especially women younger than 30 years

Essure system

- The latest form of female permanent sterilization is the Essure system.
- prevents fertilization by interrupting the fallopian tubes; however, the Essure system does not require surgical incisions and can be performed with the patient under local anesthesia.
- It is performed hysteroscopically, and a microinsert is placed directly into the fallopian tubes.
- During the first 3 months after the procedure, the fallopian tube and the microinsert create a tissue barrier that prevents sperm from reaching the egg.
- After the 3-month period, patients must undergo a hysterosalpingogram to ensure placement.

MALE CONTRACEPTION		
⦿ Condom ⦿ Vasectomy ⦿ Male oral contraception with androgens and with cotton seed oil ⦿ Immunological contraception	**Male Condom** • Male condoms are 82 to 98 percent effective at preventing pregnancy • Condoms can only be used once • When putting a condom, make sure the rolled up ring is on the outside, squeeze tip of condom so no air is trapped inside • Do not use oil-based lubricants such as massage oils, baby oil, lotions, or petroleum jelly. They will weaken the condom, causing it to tear or break. • Good choices: Latex condoms and polyurethane • provides the most effective protection of the genital tract from sexually transmitted diseases (STDs)	Efficacy • The failure rate of condoms in couples that use them consistently and correctly during the first year of use is estimated to be approximately 3%. • The true failure rate is estimated to be approximately 14% during the first year of typical use. This marked difference of failure rates reflects errors in usage. • Common errors with condoms usage include failure to use condoms with every act of intercourse and throughout intercourse, improper lubricant use with latex condoms (eg, oil-based lubricants), incorrect placement of the condom on the penis, and poor withdrawal technique. Advantages • Condoms are readily available and are usually inexpensive. This method involves the male partner in the contraceptive choice. Condoms are effective against both pregnancy and STDs. Disadvantages • Condoms possibly decrease enjoyment of sex. Some users may have a latex allergy. Condom breakage and slippage decrease effectiveness. Oil-based lubricants may damage the condom

ENDOMETRIOSIS

Def: Presence of endometrial glands and stroma outside the endometrial cavity and walls.
Deposits proliferate during the menstrual cycle, break down & bleed, causing local inflammatory reaction.
Fibrosis & distortion of the tissue affected with dense scarring.
Benign.

EPIDEMIOLOGY

- Disease of reproductive age group
- Affect 5-15% of women
- Diagnosed in 20-30% of women investigated for infertility
- More in women whose first degree relative have the disease
- Often diagnosed incidentally
- High social class women in their thirties and infertile!
- Can be diagnosed in any type of women and all age groups

OVERVIEW

- Hormone dependant
- Responds to estrogen
- Regress after menopause, oopherectomy and during pregnancy

ETIOLOGY

- Unknown
- Theories
  - Retrograde menstruation
  - Coelomic epithelium transformation
  - Lymphatic and vascular spread
  - Genetic and immunologic factors

SITES

More commonly in the dependant part of the pelvis

- Ovaries (2/3 of women)
- Broad ligament
- Peritoneal surface of Cul-de-sac and uterosacral ligaments
- Rectovaginal septum
- Rectosigmoid colon
- Distant and laparatomy scars

PATHOLOGY

- Gross
  - Hemorrhagic vesicle — Free
  - Papule and later nodule — Enclosed
  - White nodules or flattened fibrotic scar — Healed
- Ovarian endometrioma is an enclosed hemorrhagic cyst of variable sizes

HISTOPATHOLOGY

- Active endometrial glands and stroma
- Blood filled cystic lesions
- Fibrosis with glands only no stroma
- Adhesion formation

<table>
<tr><td colspan="4">SYMPTOMS</td></tr>
<tr><td colspan="4">• According to site
• No relation between extent of the disease and severity of the symptoms
• Often discovered incidentally</td></tr>
<tr><td>FEMALE REPRODUCTIVE TRACT</td><td colspan="3">Dysmenorrhea, Lower abdominal and pelvic pain, Dyspareunia, Accident to endometriotic cyst, Low back pain, Infertility, Menstrual irregularity</td></tr>
<tr><td>URINARY TRACT</td><td colspan="3">Cyclical haematuria / dysuria, Ureteric obstruction</td></tr>
<tr><td>GIT</td><td colspan="3">Dyschezia, Cyclical rectal bleeding, Intestinal obstruction</td></tr>
<tr><td>SURGICAL SCAR & UMBILICUS</td><td colspan="3">Cyclical pain and bleeding</td></tr>
<tr><td>LUNGS</td><td colspan="3">Cyclical haemoptysis, Haemopneumothorax</td></tr>
<tr><td>CLNICAL FINDINGS</td><td>INVESTIGATIONS</td><td>DDx</td><td>DIAGNOSIS</td></tr>
<tr><td>• Often Negative
• Suggested by
– Thickening and nodularity of uterosacral L.
– Tenderness in POD
– Ovarian mass/ masses
– Fixed retroverted uterus
– Tender nodule in the cervix, umbilicus or scar</td><td>• Ca 125 often elevated
• Ultrasonography for ovarian cyst
• MRI</td><td>• All causes of chronic pelvic pain
• Acute conditions
– Ectopic pregnancy
– Acute PID
– Complicated ovarian cyst
– Acute appendicitis and other surgical emergencies</td><td>• Direct visualization of the lesion
– Laparascopy
– Laparatomy
• Histopathology to confirm the diagnosis</td></tr>
</table>

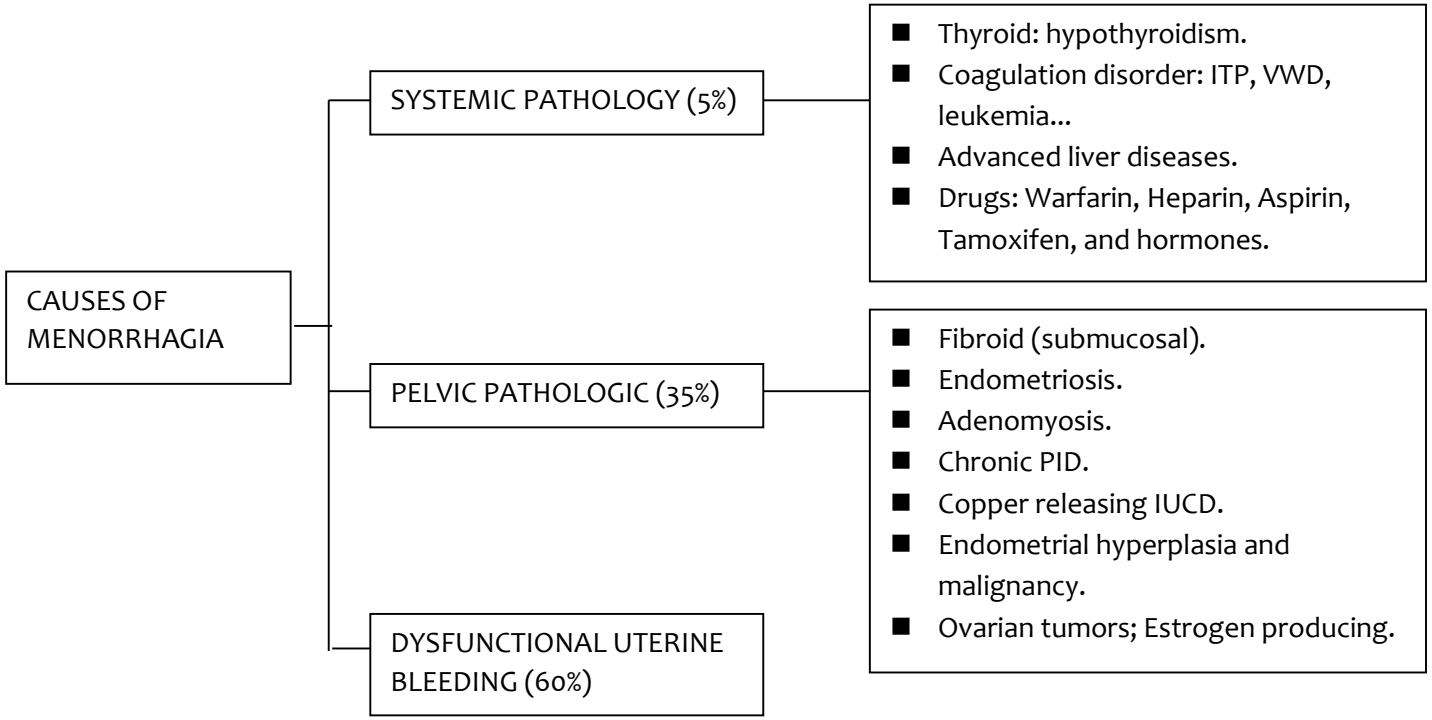
<table>
<tr><td colspan="4">TREATMENT</td></tr>
<tr><td colspan="4">NSAIDS</td></tr>
<tr><td>PSEUDO-PREGNANCY</td><td colspan="3">- Combined OCP continuous
- Cyclical ?? of limited value
Side effect
- Synthetic progestogens: Medroxyprogesterone acetate and dydrogesterone high doses continuous Side effect
- Levonorgestrel-releasing system reduces dysmenorrhoea and regress POD implants</td></tr>
<tr><td>PSEUDO-MENOPAUSE</td><td colspan="3">– Danazol androgen derivative 6-9 months
– Gestrinone, androgen derivative } Both drugs have androgenic side effects
– GnRH agonists - Menopausal symptoms, Osteoporosis
? Add back therapy</td></tr>
<tr><td rowspan="2">SURGERY</td><td>CONSERVATIVE</td><td colspan="2">Young patient, women seeking pregnancy, cysts >3cm in diameter
Surgical excision, Laser</td></tr>
<tr><td>HYSTERECTOMY & BSO</td><td colspan="2">Radical/Definitive surgery</td></tr>
<tr><td colspan="2">FACTORS TO CHOOSE TREATMENT</td><td colspan="2">ENDOMETRIOSIS & INFERTILITY</td></tr>
<tr><td colspan="2">• Certainty of diagnosis
• Severity of symptoms
• Extent of the disease
• Fertility
• Age
• Damage to other organs</td><td colspan="2">• Ovarian function
• Tubal function
• Coital function
• Sperm function
• Early pregnancy failure</td></tr>
</table>

ADENOMYOSIS	
• Endometrial glands deep within the myometrium • Unknown etiology • Different type of patient and presentation	
RISK FACTORS	TREATMENT
• Multiparous women • Late thirties or early forties • Severe spasmodic dysmenorrhea • Menorrhagia • Bulky uterus • Diagnosis often histological on examination of hysterectomy sample	• Induce amenorrhea - sx recur once treatment is stopped. • Hysterectomy is the only definitive treatment

MENORRHAGIA

- It is the most common gynecological case seen in the clinic.
- It is the most common cause of anemia in developed countries.
- And the 2nd common cause of iron deficiency anemia after poor diet in the developing countries.
  - Subjective: Prolonged or heavy Regular menstrual bleeding.
  - Objective: menstrual blood loss more than 80 ml (more accurate), but not used in practice , just in researches)

***** Normal menstrual blood loss range from 20 - 80 ml with average of 35 ml.



<table>
<tr><th colspan="4">DYSFUNCTIONAL UTERINE BLEEDING</th></tr>
<tr><th>DEFINITION</th><th colspan="2">FACTORS OF BLEEDING AMONG MENSES</th><th>ETIOLOGY</th></tr>
<tr><td>Menorrhagia in the absence of organic (pelvic, systemic) pathology.
Is a diagnosis of exclusion.</td><td colspan="2">1- PG E2 and PG F2α.
2- Fibrinolytic system.
3- Blood Vessels of the endometrium.
■ The most important is prostaglandin release and Fibrinolytic system → any disturbance in them → bleeding.
■ Disturbance in prostaglandin release such as if PGE2 increased (it is a vasodilator) will lead to bleeding and increased PG F2α which will cause spasmodic or primary dysmenorrhea.
Also, if too much fibrinolytic system activity→ menorrhagia.</td><td>1. Endometrial dysfunction (Ovulatory DUB):
- PG's imbalance (dec PGF2a : inc PGE2 ratio).
- Increased fibrinolytic activity.
- Ineffective contraction of myometrial vessels.
2. Hypothalamic – Pituitary – Ovarian hormonal axis: (Anovulatory DUB)
- Most common age at presentation is less than 20 and more than 40.</td></tr>
<tr><th colspan="2">HISTORY</th><th>PHYSICAL EXAM</th><th>INVESTIGATION</th></tr>
<tr><td colspan="2">- Complaint: Assess the amount of blood loss.
- Associated Gynecological problem: Congestive dysmenorrhea, deep dyspareunia, chronic pelvic pain, pressure symptoms, and vaginal discharge.
- Gynecological & Menstrual Hx: Last cx smear, previous gynae surgery, contraception, IMB, and PCB.
- Medical Hx: Thyroid symptoms, Hematological disorders…
- Medications: The previous 4 drugs.
- Previous investigations and treatment.</td><td>- General examination: General condition (does she look pale or not?), Vitals, Weight, Thyroid, Lymph nodes (axillary and inguinal), Breast, Abdomen (Pelviabdominal mass/ ascites).
- Pelvic examination: Speculum examination, Bimanual examination.</td><td>- CBC: Hb and platelets.
- Pelvic ultrasound: Uterus; size, shape, masses, and endometrial thickness. Adnexia
- Cervical smear.
- Office biopsy: Pipelle, Novak…
- Hysteroscopy and endometrial biopsy: Mandatory
for women older than 40 years.
- Others, according to the suspected problem.</td></tr>
</table>

TREATMENT					
LESS THAN 20 YEARS OLD					
Menorrhagia is a common cause of Gyn clinic visit in teenager, mainly due to DUB. (delayed maturation of HPO axis) Treatment is simple and for short duration (few months) till the hormonal axis becomes mature. Lines of management: a) Reassurance and explanation. b) Correction of anemia if present. c) Medical treatment.					
NON-HORMONAL		HORMONAL			
ANTI-PROSTAGLANDIN	ANTI-FIBRINOLYTICS	PROGESTOGENS	COMBINED OCP	DANAZOL	GnRH ANALOG
Most commonly used. **Mefenamic acid (Ponstan):** - Is the most common drug used by adolescent female; for dysmenorrhea as well. - 3 capsules daily, from day 1 to day 5 of the cycle. - ↓ menstrual blood loss by 25%. -S/E: gastritis, gastric ulcer.	**Tranexamic acid**: - 3 capsule daily, from day 1 to day 5 of the cycle. - ↓ menstrual blood loss by 50%. - Main S/E: nausea and vomiting, ~ 25% of patients stop it because of these side effects. - Rarely, it may cause cerebral thrombosis, so it is contraindicated in patient with risk factors for thromboembolism.	**Norethisterone and Medoxyprogesterone acetate.** - Most common drug used for DUB. - 5 mg twice daily, from day 5 to day 25 of the cycle. - ↓ menstrual blood loss by 25%. - No serious S/E. - Safe to use.	- 1tab daily for 21 days, from day 5. - ↓ menstrual blood loss by 50%. - Less commonly used due to its side effects. - Minor S/E: Nausea, vomiting, headache, irritability, ↑ in weight... - Major side effects: HT, thromboembolism, cardiovascular…	- It is an androgen analogue (17-α–ethinyl testosterone). - Also antiestrogentic & antiprogestrogenic. - Depression of the HPO- axis and has a direct suppressive effect on endometrium. - ↓ menstrual blood loss by 80 – 100%. - S/E: Hoarseness of voice. Hirsutism and acne. ↑ muscle mass. Cliteromegaly. Breast atrophy. Hypooestrogenic Menopausal sx.	- 3.75mg IM monthly, for 4 months. - ↓ Menstrual blood loss by 80-100%. - Depression of the HPO- axis; Menopausalsx. - Major risk: Osteoporosis if used more than 6 months. *Although, these drugs are extremely effective, but they are no more used nowadays due to their serious side effects.

BETWEEN 20 & 40 YEARS OF AGE	ABOVE THE AGE OF 40
Two lines of management: A] Medical: same as for the teenagers. B] **Levonorgestrol releasing IUCD (Mirena)** → they desire contraception; very effective. - 20 mcg of levonorgestrol daily. - It decreases menstrual blood loss by 80–90 %. - ~30% of women are amenorrhoeic after one year of insertion. - It decreases the incidence of PID. - Doesn't increase risk of ectopic pregnancy. - Side effects: breakthrough bleeding & spotting for the first 3-6 months after insertion.	Three lines of management: A] Medical: Same, not OCP. B] Mirena: Safely used. C] Surgery: 1) **Endometrial resection and ablation**: - Day case surgery under GA. - Short stay in hospital, rapid recovery. - Cure rate of 70–80%. - Risk of recurrence 20–30%; Risk of endometrial cancer exists. 2) **Hysterectomy:** Is the best, 100% cure rate. No recurrence of the problem.

POSTCOITAL BLEEDING	
Def: Bleeding during or after coitus. The cause is almost always cervical:- - Cervical ectropion, the commonest cause. - Cervical ulcer, cervicitis. - Cervical polyps. - Cervical cancer. » How to diagnose? - History - Examination: General and pelvic, speculum. » Treatment: Should be directed toward the cause; Pap smear is mandatory before treatment.	"Cervical Ectropion" It is normal, physiological, it is not an ulcer. - Occurs in high estrogenic state: Pregnancy or COCP users. - The estrogen will cause overgrowth of the columnar epithelium of the endocervix into ectocervix → postcoital bleeding. - C/C: PCB and excessive mucoid secretions. - In menopause, there will be inversion (the stratified layer of the ectocervix will move inwards). - During pregnancy; Conservative treatment, after delivery it usually improves spontaneously. - If on COCP, stop it, reassess again.

AMENORRHEA

Primary No secondary sexual characteristics (*Growth spurt, Breast development, Pubic hair growth, Menstruation, Axillary hair growth*), by the age of 14, or no menstruation by the age of 16 **Secondary** No menstruation for more than 6 months in a normal female of reproductive age that is not due to pregnancy.	Menorrhagia: heavy and prolonged Hypomenorrhea: light Polymenorrhea: < 21 days Oligomenorrhea: > 35 days Metrorrhagia : irregular
Etiology **A) Reproductive outflow tract abnormalities** 1. **Vaginal disorders** **- Imperforate hymen** **- Transverse vaginal septum** **- Absent vagina and functional uterus** **- Müllerian agenesis:** complete or partial absence of the uterus and variable malformations of the vagina **(**Mayer-Rokitansky-Küster-Hauser Syndrome**).** Common 2. **Uterine disorders** **- Cervical stenosis** **- Asherman Syndrome** **- TB of the uterus** 3. **Ovarian disorders** **- Premature ovarian failure** **- Resistant ovary syndrome** **- PCOS**	B. **Hypothalamic/pituitary causes** Constitutional delay Kallmann Syndrome (deficiency of GnRH, with anosmia, and hypogonadotropic hypogonadism) Wt loss, exercise and psychological stress Craniopharyngioma, Glioma, germinoma, dermoid cyst Head injuries Infection, TB, Sarcoidosis Idiopathic Hyperprolactinaemia Empty sella syndrome Hypopituitarism C. **Drug induced hyperprolactinaemia** -Tranquilizers - Tricyclic antidepressants -Narcotics

Other causes

A) Chromosomal

1. **Turner Syndrome** 45X and 45X/46XX (mosaics)
-Lymphoedema in newborn

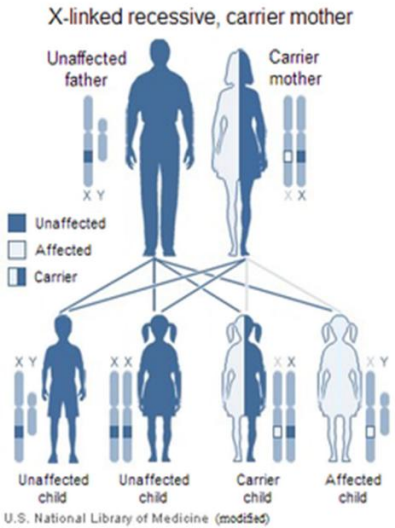
2. **Swyer Syndrome** 46XY (complete gonadal dysgenesis)
3. **45X/46XY** (Mixed gonadal agenesis/dysgenesis)
with absence of TDF or its receptor

- failure of testicular development, with no androgen or MIF, therefore Wolffian structures regress and Mullerian structures persist, with normal female phenotype
- normal or excessive height (delayed epiphyseal closure due to low androgens or oestrogens)
- Menses will occur with oestrogens

4. **Gonadal agenesis**: complete failure of development of the gonad (These girls may be with 46 XX or 46 XY)
47XXX, 48XXXX, 49XXXXX
-The gonad is abnormally formed
- Turner syndrome is commonest (45X or 45X/46XX)
- Short stature as the gene for height is on the short arm of the X chromosome

Basis of human development
46 XY - male
46 XX - female
The presence or absence of the Y chromosome will determine if the gonad becomes a testis or an ovary
The Y chromosome contains a gene sequence on its short arm (Yp). This gene is the Sex Determining Region on the Y (SRY gene), and encodes for Testis Determining Factor (TDF)
TDF induces differentiation through the cell surface antigen (H-antigen) found in individuals with the Y chromosome

B) Genetic
Androgen insensitivity - Testicular feminization syndrome



- X-linked trait
- Absent cytosol receptors
- Normal breasts but no sexual hair
- Normal looking female external genitalia
- Absent uterus and upper vagina
- Karyotype 46 XY
- Male range testosterone level
- Gonadectomy after puberty + HRT
- Vaginal creation (dilatation, Vaginoplasty)

Androgen Sensitivity : has breast, but no uterus and sexual hair
vs
Swyer syndrome : no breast, but has uterus and pubic hair

Systemic causes
- Chronic debilitating illness
- Wt loss
- Endocrine (thyroid, Cushing etc...)

Cushing syndrome
-Hirsutism, truncal obesity, purple striae,↑ BP
-If suspicion is high:
Dexamethasone suppression test (1 mg PO 11 pm))
and obtain serum cortisol level at 8 am. < 5 µg/dl excludes Cushing's
-24 hours total urine free cortisol level
- 2 forms: 1. Adrenal tumour
2. ACTH hypersecretion (pituitary or ectopic site)

Heterosexual development

1. Congenital adrenal hyperplasia
   - Autosomal recessive trait
   - Most common form is due to 21-hydroxylase deficiency
   - Mild forms closely resemble PCO
   - Severe forms show signs of severe androgen excess
   - High 17-OH-progesterone levels
   - Rx:cortisol replacement and ? corrective surgery

2. Arrenoblastoma (Sertoli–Leydig cell)
   Ovarian tumor that release male hormone testosterone
   Tumor of the sex cord stromal

3. 5 alpha reductase deficiency (XY female)
   - XY gene type
   - Failure of testosterone to change into DHT
   - Girls have no uterus or cervix
   - Girls have vagina which is sometimes smaller than usual
   - Some treatment may be needed to enlarge vagina
   - Gonads are usually in the abdomen
   - Removal of gonads and HRT needed for girls
   - Carrying a pregnancy not possible

4. True hermaphrodite

5. Absent Mullerian Inhibitor

History

Developmental history, age of menarche
Medical and surgical history.
Chronic illness. Wt loss. Exercise
Anosmia
Menopausal symptoms
Medications
Family history of premature menopause
Virilizing signs
Gallactorrhoea
Psychological history
Stress

Clinical examination

Height
Secondary sexual characteristics
Visual fields and papilloedema
Pelvic examination

Investigations

A) Pregnancy test, Prolactin, TFT, LH/FSH, Testosterone (<5 nmol/l)

B) Progesterone challenge test : 10 mg for 5 days
   - Bleeding : ovulation disorder

C) If PCT(Progesterone Challenge Test) is negative (no bleeding):
   21 days of oestradiol(estrogen) 2mg/d for 21 days followed by progesterone 10 mg for 5 days as in PCT
- No bleeding : Outflow abnormality
- Bleeding: HPO (hypothalamus, pituitary, ovaries) problem

D) Repeat LH and FSH 6 weeks after tests
If elevated: Ovaries problem continue by karyotyping
If LH and FSH not elevated: hypothalamic or pituitary problem

E) Do headCT/MRI : see pituitary tumor
- If normal : hypothalamic disorder (diagnosis of exclusion)

This should determine compartment
Depending on results karyotyping may be appropriate

PCOS is a syndrome of ovarian dysfunction along with hyperandrogenism and polycystic ovary morphology
Prevalence : 5-10 %, On ultrasound 25%

Etiology : unclear

1. Increased ovarian androgen production
2. Disordered ovarian cytochrome p450 activity
3. Increased LH production
4. Insulin resistance
5. Family clusters : gene or cluster of gene

Clinical features
Oligo/amenorrhoea
Hirsutism
Subfertility
Recurrent miscarriage
Acanthosis nigricans

Laboratory tests
Elevated T levels
Decreased SHBG
Elevated LH levels
Elevated LH/FSH ratio
Increased fasting insulin levels
Eight or more subcapsular cysts <10 mm and increased stroma

Treatment :

1. Weight loss
2. Progesterone
3. COC
4. Metformin

Long Term Sequele : DM and CVD

<table>
<tr><th colspan="4">INFERTILITY</th></tr>
<tr><th>INTRODUCTION</th><th colspan="3">PHYSIOLOGY</th></tr>
<tr><td>Def: Involuntary failure to conceive within 12 months of commencing regular unprotected intercourse. (Old definition within 24 months)
• Primary Infertility: No previous pregnancy
• Secondary Infertility: With previous pregnancy (whatever the outcome)

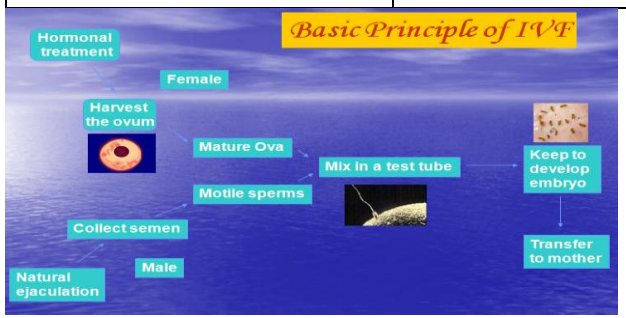
Incidence:
• 10-15% of married couples
• About 75% of couples conceive by 12 months,
• About 85% of couples conceive by 24 months.</td><td colspan="3">Conception requires :
• Oocyte, sperm, at optimal stage
• Needs transportation
• A receptive place for implantation
• Intact Male and Female reproductive systems</td></tr>
<tr><th colspan="4">POSSIBLE CAUSES OF INFERTILITY</th></tr>
<tr><th>FEMALE</th><th>MALE</th><th colspan="2">IDIOPATHIC</th></tr>
<tr><td>• Ovulatory failure factor: (anovulation)
High centres - hypothalamic-pituitary axis
Thyroid
Adrenal
Ovarian - PCO(20%), premature failure(>30IU/L)
Genetic & Chromosomal
• Tubal factor: PID,T.B
• Uterine factor: Asherman’s, congenital anomaly.
• Cervical factor: infection, immunological
• Endometriosis:
• Coital factor: aparunia, dysparunia, vaginismus
• Psychosomatic: ? Neurohormones</td><td>Coital factor: (psychological or organic)
Spermatogenesis problem: Azospermia, oligospermia
Ductus problem: Azospermia due to obstruction (infection)</td><td colspan="2">About 15-30% of Infertility. It is a definition by exclusion, and that depends on the standard investigations used.
(Ovulatory, Tubal, Male)</td></tr>
</table>

INFERTILITY WORK UP				
HISTORY			EXAM	
• Male: Age, history of mumps, occupation, drugs, chemical, irradiation, hernia operations, varicocele • Female: Age, menstrual cycle (regularity) - previous pregnancies, Abortions and TOP, galactorrhoea - 1°/2° - contraceptions , Hirsutism. • Both: Coital history:- S.T.D - past med & surgical hx, smoking and taking drugs			• Male: exam.vas deference -size of testicles , varicocele, endocrine stigmata • Female: B.P - Thyroid - galactorrhoea – hirsutism. Abdominal and pelvic exam genitalia (External & Internal)	
SPECIAL INVESTIGATIONS				
OVULATION	CERVICAL FACTOR	TUBAL & UTERINE FACTORS	MALE FACTOR	OTHERS
1. B.B.T.chart: Biphasic an increase of 0.5C, progesterone effect 2. Cx mucus alteration: Mittelschmerz pain 3. Hormonal assay: S. progesterone d21 (20-30nmol/L), S. prolactin <20ng/ml S. FSH and LH first days of period 4. Endometrial biopsy -d 21- secretory endometrium. i.e.T.B 5. U.S.S - monitoring of follicles 18-22 mm, d12=12mm 6. Laparascopy - laparatomy - incidental findings 7. LH peak (LH home kits): 26h later ovulation occur 8. Pregnancy	- Cx score (amount, spinnbarkeit, ferning, os) - Cervical mucus alteration: thin-clear-watery–elastic (subjective) - Elasticity (spinnbarkeit) - Fernning (arborization or crystallization) of NaCl due to unopposed action of estrogen - P.C.T:(post coital test): positive if: more than 5-10 sperm in (H.P.F) alive forward progressive motility after 6-12 hours of sexual intercourse at time of ovulation. Repeat 3 times if Negative (wrong timing, cervical hostility due ?antibodies, severe oligo or azospermia) - Cross hostility test (Kremer test) if P.C.T is negative - Antisperm antibody titre - and MAR (mixed agglutination reaction)	(CILIA, FERTILIZATION, TRANSPORT) - Tubal insufflation (Rubin's test) obsolete nowadays - Hysterosalpingogram: using radioopaque water soluble, no G.A - Laparascopy + Methylene blue dye test: under G.A, checks for endometriosis and D&C in the same sitting (when done in luteal phase) - Hysteroscopy - in Ashemann's synd. congenital anomaly to visualize the uterine cavity (using CO_2 or glyceine solution) - Hy-co-sy (hysterosalpingo-contrast-sonagraphy) using Echovist	Semen analysis: by coitus interruptus or masturbation after abstinence of 3-5 days - delivered within 2 hours to lab. Normal values: by WHO criteria Volume: >2ml - liquification in 20- 30 minutes Density: >20-250 Mil/ml Motility: > 50% forward motility within 2 h. Morpho: > 30% normal forms Seminal fluid 90% of ejaculate: 2\3 from seminal vesicles (fructose), 1\3 from prostate (zinc & acid ph.) In azospermia + oligospermia: hormonal FSH,LH, prolactin & karyotyping	CBC Urine analysis S.T.D – Chlamydia Rubella titer TFT Skull x ray CT Scan X ray chest.

<table>
<tr><th colspan="3">INFERTILITY MANAGEMENT</th></tr>
<tr><th>INDUCTION OF OVULATION</th><th>TUBAL FACTORS</th><th>CERVICAL FACTOR</th></tr>
<tr><td rowspan="2">A] Fertility agents:
• Oral agents: 90% induce ovulation, but 60% pregnancy
-In cases of hyperprolactinaemia :
Bromocriptine (ergot alkaloids, dopamine agonist)
Lisuride 1x1
Cabergoline(Dostinex) 1mg weekly
-Clomiphene citrate (clomid): 50mg _ 200mg _ (d2 - d6), for 6 cycles. It is oral cyclical, synthetic, nonsteroidal, weak estrogen with antiestrogenic activity
-Tamoxifen : 10-40mg (d2 - d6) for 6 cycles, in PCO
-Cyclofenil : 200mgb.d for 10 days
In PCOS :metformin (oral insulin sensitizers)
Side effects: ovarian cysts, twins 5%, hair loss, GIT, rarely hyperstimulation syndrome (OHSS)

• Injectable agents
Gonadotrophin therapy: urinary extracts, now recombinant
- HMG: (FSH+LH) (Humegon-pergonal) injections 1-3 ampules daily or every other day till follicular maturation, about 5-10 injections
- FSH: only (metrodin) in P.C.O
- H.C.G: (pregnyl,profasi) - 5000 - 10000 unit after follicular maturation - to release oocyte
- L.H.R.H-a: - (Busserlin-Zoladex-superfact-Decapeptyl) continuous (nasal or s.c. 4-6 times daily or depot IM) to deplete endogenous FSH,LH
- L.H.R.H-a: - pulsatile infusion every 90 minutes, 15ug
- L.H.R.H.antagonist
Side effects: multiple pregnancy 25% - hyperstimulation syndrome (if severe) -ascitis, large ovarian cysts, hydrothorax, thromboembolic disease, multiorgan failure
• B- Surgical: Ovarian drilling, wedge resection(obsolete)
Options of treatment: Oral fertility agents → Injectables → SIUI,SIVF</td><td>• Selective Hysterosalpingogram
• Surgery: microsurgery: after falloposcopy:
• Salpingolysis, salpingostomy, excision & reanastom. (success from 10-40%)
• Uterine anomalies:
Myomectomy for fibroids
Metroplasy in certain cases
• I.V.F program</td><td>• improve cervical score: Treat infection, Cryocautery, Estrogen
• immunological - ?corticosteroids for the male during the luteal phase of female ,Male use of condom for 6 months
• SIUI and SIVF</td></tr>
<tr><th>ENDOMETRIOSIS</th><th>MALE FACTOR</th></tr>
<tr><td>• Danazol, LHRH a treatment (medical)
• Conservative surgery:
• I.V.F. program</td><td>▶ Treatment directed towards cause.
▶ Advice: stop smoking and alcohol, avoid tight underwear, take regular cold baths, improvements in coital practice.
▶ Psychological therapy: for sexual dysfunction
▶ In severe oligospermia and azoospermia, check for karyotype (Klinefelter's syndrome, testicular atrophy)
▶ hMG for hypothalamic-pituitary failure.
▶ Bromocriptine for hyperprolactinemia.
▶ Surgical treatment in vasectomy reversal.
▶ Varicocele ligation in varicocele.
▶ ART: SIUI,ICSI (by TESE or MESA)</td></tr>
</table>

ASSISTED REPRODUCTION TECHNIQUES (ART)			
ARTIFICIAL INSEMINATION	IVF + ET (EMBRYO TRANSFER)	VARIANTS OF IVF	RESULTS OF IVF
• Artificial insemination (AI) • AIH: intravaginal, intracervical and pericervical, intrauterine, intraperitoneal • AIH(DI) • IUI and SIUI • The mostly used nowadays: is SIUI (stimulated intrauterine insemination: - Proper selection of the cases - Controlled ovarian stimulation - Preparation of semen - Timing of insemination	✓ Up to 35% could benefit from infertile couples ✓ Candidates: Tubal factor , endometriosis , oligospermia ,unexplained infertility ✓ It is expensive, requires sophisticated lab. facilities, highly skilled medical, nursing, scientific and tech. personnel	○ G.I.F.T: Gamete intra fallopian transfer (indicated: unexplained infertility, oligospermia, endometriosis. (C.I.tubal damage) ○ Procedure: Laparascopy at ovulation - retrieve oocycte mix with prepared semen - deposit both in tube ○ Z.I.F.T ○ SUZI subzonal injection ○ ICSI intracytoplasmic sperm injection ○ PESA percutaneous epididymal sperm aspiration ○ MESA ○ TESE	▪ E.T (1) - 10% chance of single pregnancy ▪ E.T (3) - 25-30% chance of single pregnancy. 5% twins, 1%triplets ▪ Efficiency: 25-35% for each cycle - Take home baby 15-20% ▪ According to the infertility factor and the centre
		OUTCOMES OF IVF PREGNANCIES	RISKS OF IVF
		25% risk of miscarriage 2-5% risk of ectopics, heterotropic pregnancy 25% risk of multiple pregnancy Increase risk of prematurity, low birth wt, Cs	Psychological trauma if failed (OHSS) ovarian hyperstimulation syndrome Multiple pregnancy



<table>
<tr><th colspan="5">PELVIC INFLAMMATORY DISEASE</th></tr>
<tr><td>● Endometritis
● Salpingitis
● Oophoritis
● Parametritis
● Peritonitis</td><td>PATHOLOGY</td><td>EPIDEMIOLOGY</td><td colspan="2">CLINICAL FEATURES</td></tr>
<tr><td>DDx
▪ UTI
▪ Early pregnancy complication, Ectopic pregnancy
▪ Appendicitis, diverticulitis
▪ Complicated ovarian tumor
▪ Other causes of lower ab pain</td><td>• Infection ascend to the uterus resulting in endometritis with plasma cell infiltration
• Tubes, mucosal inflammation with swelling, redness and deciliation, polymorph infiltration of the submucosa, exudate fills the lumen, adhesions and spread to the serosal surface, pus from the fimbria
• Ovaries and formation of tubo-ovarian abscess or mass</td><td>✓ Incidence variable 10-13%, underestimated, increasing
✓ Age, teens
✓ Sexual activity, early coitus, multiple partners, often proceeded by STD (chlamydia, GC) with 2ndary invasion with anaerobes
✓ Contraception
Pills? Protective
IUCD Cu releasing↑ risk
Barrier protective
✓ Parity 75% are nulliparous</td><td>○ Lower ab pain and tenderness
○ Deep dysparunea
○ Abnormal vaginal or cervical discharge
○ Cervical excitation and adnexal tenderness
○ Fever >38°C
○ Adnexal mass in 20% of cases
○ Raised ESR, WBC, CRP
○ Excess of Leukocytes in the vagina
○ General symptoms depend on severity</td><td>○ Non specific, lack sensitivity and specificity
○ Positive predictive value of clinical diagnosis is 65-90% as compared to laparoscopy
○ Excess of WBC in the vagina present in lower genital tract infection also
○ Endometrial biopsy not recommended as routine investigation</td></tr>
<tr><td>COMPLICATIONS</td><td>INVESTIGATIONS</td><td colspan="3">TREATMENT</td></tr>
<tr><td>▪ Tuboovarian abscess, polymicrobial, 50% bilateral
▪ Tubal damage, infertility and future ectopic.
▪ Fitz-Hugh-Curtis syndr
▪ Reinfection is 25%
▪ Chronic PID, adhesions, pelvic pain, dysparunea, dysmenorrhea</td><td>● CBC, CRP, Urine analysis and culture
● HVS, Endocervical swab
● USS, adnexal mass or abscess
● Laparoscopy if there is doubt about diagnosis or no improvement after 24-48 hours of proper treatment
● BhCG</td><td>Mild cases : Ambulatory Antibiotic
Doxycycline 2x/day/14d + metronidazole 2x/day/5d (if GC suspected add ciprofloxocine single dose)

Or
Ofloxocine + metronidazole</td><td>Severe cases: Admit!

Indications to Admit:
Surgical emergency cannot be excluded,
Clinically severe disease, Tuboovarian abscess,
PID in pregnancy, Lack of response to oral therapy,
Intolerance to oral therapy</td><td>IV cephalosporin + metro till 24 h, improvement → oral doxycycline and metronidazole

Clindamycine
Ofloxocine+ metronidazole
Clindamycine

Supportive therapy</td></tr>
</table>

NOTES

- Patients not improving on antibiotic therapy should have laparoscopy
- IUCD may be left in situ in women with mild PID but should be removed in sever cases
- Tuboovarian abscess may be drained abdominally, lapaporoscopic or USS guided aspiration on pelvic collection

*TUBERCULOSIS

- Rare
- Often secondary to pulmonary TB
- May present as pelvi-abdominal mass
- Systemic symptoms, menstrual disturbance, amenorrhoea, infertility
- Treated by antituberculous drugs and surgery

<table>
<tr><th colspan="5">GENITAL PROLAPSE</th></tr>
<tr><td colspan="5">Genital prolapse is the downward descent of the uterus and /or the vagina towards or through the introitus
Occurr in about 10-30% of multiparous women and in 2% of nulliparous women .</td></tr>
<tr><th colspan="2">TYPES</th><th>PELVIC SUPPORT</th><th>ETIOLOGY & PATHOPHYSIO</th><th>PREVENTION</th></tr>
<tr><th>UTERINE</th><th>VAGINAL</th><th>UTERUS</th><td rowspan="2">Weakening of and damage to the supporting structures of the pelvic organs:
1. Childbirth: (most imp)
Increasing parity, prolonged labour, bearing down before full cervical dilatation and difficult instrumental deliveries.
2. Chronic elevation in intra-abdominal pressure: Obesity, smoking, chronic cough, chronic constip, heavy lifting at work, abd masses and ascites .
3. Menopause: Weakness of the pelvic support due to ↓ collagen and weakness of the connective tissue
4. Pelvic surgery :
➢ Abd / vaginal hysterectomy → Vault prolapse
➢ Composuspension → Rectocele and enterocele.
5. Congenital prolapsed - congenital ↓ amount of collagen and weakness of connective tissue of the pelvic support. For the prolapse in 2% of nulliparous women .
6. Racial variation .
Common in Caucasian women , less common in Asians , and rare in Blacks .
Variation in the amount of collagen and connective tissue in the pelvic support.</td><td rowspan="2">Genital prolapse is a preventable disease
1. Prevention during childbirth:
Good labor management, postnatal pelvic floor exercises, family planning.
2. Avoiding ↑ intra-abd pressure
Obesity , smoking, chronic cough and chronic constipation
3. Prevention postmenopausal:
Balanced diet, exercise, calcium & by the increased use of HRT.</td></tr>
<tr><td rowspan="2">▪ 1st degree: Descent of the cervix within the vagina.
▪ 2nd degree: Descent of the cervix thru the introitus.
▪ 3rd degree (Procidentia): Descent of the cervix and the whole uterus through the introitus.</td><td rowspan="2">▪ Cystocele: Prolapse of the upper 2/3 of the anterior vaginal wall and the bladder.
▪ Urethrocele: Prolapse of the lowest 1/3 of the anterior vaginal wall and the urethra.
▪ Rectocele: Prolapse of the posterior vaginal wall and the rectum.
▪ Enterocele: True hernia of the pouch of Douglas through the posterior vaginal fornix - may contain bowel or omentum.
▪ Vault prolapse: Inversion of the vaginal apex which occur after abdominal or vaginal hysterectomy.</td><td>i) Transverse @ Cardinal @ Mackenrodt's cervical ligaments.
ii) Uterosacral ligaments
iii) Pubocervical ligaments

Don't give support to the uterus, ie broad ligaments, round ligaments and the levator ani muscles

VAGINA
Pelvic floor muscles (the levator ani muscles "mainly" and the superficial and deep transverse perineal muscles) and by the pelvic floor fascia.</td></tr>
</table>

<table>
<tr><td colspan="4">DIAGNOSIS</td></tr>
<tr><td>HISTORY
Sx depends on the site, type & degree of the prolapsed.
• A feeling of something coming down below or a lump within the vagina or protruding from the introitus - worse at the end of the day, ↑ on standing and coughing, and ↓ by lying down.
- Other sx, depends on the organ which prolapsed into the vagina.
• Uterine prolapse: Low backache - central, worse at the end of the day, ↑ on standing and ↓ by lying down.
• Cystocele: Urinary sx, pt has to reduce the cystocele to empty her bladder.
• Rectocele: Constipation, incomplete rectal evacuation and the patient has to reduce the rectocele digitally to empty her rectum.
• Procidentia: Ulcer, blood stained or purulent vaginal discharge.
Coital problems - uncomfortable or difficult intercourse –in uterine and vaginal prolapse.</td><td colspan="2">EXAMINATION
- Inspection of the vulva with cough and straining – demonstrate severe prolapse and may demonstrate stress incontinence (provided the bladder is full)
- Speculum examination –either dorsal position (Bivalve) or left lateral position (Sims).
- Rectal examination - differentiate between rectocele (finger goes thru it) from enterocele (finger goes high up) .

INVESTIGATIONS:
- MSU for analysis and culture.
- Renal ultrasound and IVU in cases of procidentia and severe cystocele to exclude hydroureter & hydronephrosis.
- Cystometry in cases of incontinence, to exclude urge incontinence</td><td>DDx:
• Anterior vaginal wall prolapse:
DDx: Congenital Gartner's cyst, inclusion dermoid cyst & urethral diverticulum.
• Uterine prolapse:
DDx: large cervical or endometrial polyp & chronic uterine inversion</td></tr>
<tr><td colspan="4">MANAGEMENT</td></tr>
<tr><td colspan="4">CONSERVATIVE TREATMENT: PESSARY</td></tr>
<tr><td>INDICATIONS</td><td>TYPES</td><td colspan="2">SIDE EFFECTS & THEIR MANAGEMENT</td></tr>
<tr><td>- Patient unfit for surgery .
- Patient refuses surgery .
- During pregnancy and after delivery .
- During waiting time for surgery.
- As a therapeutic test to confirm that surgery may help.</td><td>• Ring pessary – commonly used pessary.
• Shelf pessary – rarely used</td><td colspan="2">Vaginal infection and discharge, ulceration and bleeding.

Precautions:
Use silicon pessary.
Change the pessary yearly or earlier if infection or ulceration occurred.
Use of vaginal estrogen cream in menopausal patients</td></tr>
</table>

<table>
<tr><th colspan="4">SURGICAL TREATMENT</th></tr>
<tr><th>PRE-OPERATIVE ASSESSMENT</th><th colspan="3">TYPE OF SURGERY</th></tr>
<tr><td rowspan="4">Aims of surgery: Correct prolapse, maintain continence and preserve coital function.
Success of the surgery depends on:
1. Preoperative preparation of the patient such as reduce weight in obese, stop smoking and treatment of chronic cough. (Gynaecologist can't do his part unless the patient fulfills hers).
2. Postoperative care</td><td colspan="3">Depends on: 1. Type of prolapsed, 2. Age and parity of the patient</td></tr>
<tr><td>UTERINE</td><td>VAGINAL</td><td>LEFORT</td></tr>
<tr><td rowspan="3">i. Vaginal hysterectomy –the preferred operation in uterine prolapsed. For young patients who complete the family and in menopausal patients.

ii. Manchester (Fothergill) operation.
- Indicated in young patients who not complete the family.
- Consisted of :
1. Partial amputation of the cervix
2. Shortening of the transverse cervical ligaments and suturing them to the front of cervical stump.
3. Anterior and posterior repair.

iii. Sacrohysteropexy
In patients who complete the family and wish to conserve the uterus</td><td rowspan="3">i) Cystocele & Urethrocele: Anterior colporrhaphy.
ii) Rectocele: Posterior colpoperineorrhaphy.
iii) Enterocele: Resection of enterocele sac.
iv) Vault prolapsed: Abdominal sacrocolpopexy.</td><td rowspan="3">- Rarely indicated in elderly and frail patients who are unfit for vaginal hysterectomy or pelvic floor repair.
- Rectangular strips of vaginal epithelium are removed from the anterior and posterior vaginal walls in order to obtain a partial closure of the vagina.</td></tr>
<tr><td>POST-OPERATIVE CARE</td></tr>
<tr><td>Immediate postoperative care :
➢ Vaginal pack – remove within 24h.
➢ Foley's catheter - remove after 1- 2 days.
➢ Prophylactic antibiotics: Metronidazole and cephalosporin
Instructions after discharge - to minimize recurrence
➢ Avoiding intercourse for 6 wks.
➢ Gradual return to normal activities over 2 months.
➢ Avoiding smoking, obesity, constipation and lifting of heavy objects.
➢ Elective C.S. in the subsequent pregnancy.</td></tr>
<tr><td>RECURRENT PROLAPSE</td><td colspan="3" rowspan="2"></td></tr>
<tr><td>• Recurrence occur in about 20-25%
• Even with expert surgery and good postoperative care, recurrence can occur, esp in the presence of obesity, smoking and constipation.</td></tr>
</table>

GENITAL PROLAPSE: APPROACH

HISTORY

PROFILE : Name, Age, Gravida, Para, Occupation, Ethnic

C/C: LUMP / SOMETHING PROTRUDYING THRU INTROITUS / COMING DOWN BELOW

HPI Lump analysis: Duration. Present always/goes back in?

↑ by long standing, at end of the day.

↓ by lying dow

Impact on social & sexual life

Associated sx:

Back pain – uterine prolapsed

Urinary sx, can't empty bladder, must reduce digitally – Cystocele/urethrocele

Constip, Incomplete evacuation, must reduce digitally – Rectocele

Ulcer, blood stain, purulent vaginal discharge – Procidentia

Risk factors

Childbirth – Multiparity? Vaginal delivery? Long labor?

Menopause, pelvic surgery

↑ Intra-ab pressure – Obese, Chronic cough, constip, mass

Gynea Hx: Menopause, HRT, Pelvic surgeries

Past Hx: Chronic cough (COPD, Asthma, Pneumonia, CF), Constipation, Previous surgeries

Social Hx: Smoking

DDx:

Congenital Gatner's cyst

Inclusion dermoid cyst

Urethral diverticulum

Large cervical / endometrial polyp

Chronic uterine inversion

EXAM

Pelvic Exam

Inspection – Vulva with cough & straining – Demonstrate prolapse ± incontinence

Speculum

Rectal Exam – to differentiate rectocele (finger goes thru) & enterocele (finger goes high up)

INVESTIGATIONS

MSU – analysis & culture

Renal US & IVU – in procidentia & severe cystocele, to R/O hydroureter & hydronephrosis

Cystometry – to R/O urge incontinence

URINARY INCONTINENCE APPROACH

HISTORY

Age, Parity

Analysis of Incontinence: Duration, frequency, amount

- Precipitating factors
- 3Ps: Position, Protection, Problem
- Progression
- Urinary Diary

Association

- Irritative sx (Freq, Urgency, Nocturia) – urge
- Obstructive sx (Poor stream, Incomplete void, Straining) – overflow
- Recurrent UTI – urge

Past Obs Hx: Mode of Delivery. Instrumental?, Prolonged 2nd stage? Baby birth W8.

Past Gynea Hx: Sx of Prolapse, Pelvic surgery, Hx of malignancy, Hx of radiotherapy

PHYSICAL EXAM

General Look

Abdominal Exam

Respiratory Exam

Neurological exam – Mental status, Gait, Lower extremity, Perineal sensation & reflexes.

Pelvic Exam

- Pelvic floor muscle tone
- Stress test - >90% diagnostic for stress inc
- Cotton swab @ Q-tip test

INVESTIGATIONS

1. Urine analysis & culture
2. Urodynamic studies
    a. Non-invasive methods:
        i. **Uroflowmetry**
        ii. Volume-frequency chart

iii. Ultrasound: Residual Volume, Urethral cyst, Diverticulation of urethra
iv. EMG by surface electrodes

b. Invasive methods
   i. Cystometry
   ii. Urethral Pressure Profilometry
   iii. RV by Catheter
   iv. EMG by needle electrodes

3. Ultrasound
4. VCU
5. IVU. Indication: Hematuria, Neuropathic bladder, Uterovaginal fistula.
6. Cystourethroscopy. Indication: Reduced bladder capacity, Short hx (<2yrs) of urgency & frequency, Persistent UTI, High suspicion of tumors or stone.
7. MRI

UROFLOWMETRY

Normal flow curve: Bell shaped

Normal flow rate >15ml/sec

Normal voidal volume >150ml

CYSTOMETRY

Indications	Normal Values
1. Previous unsuccessful continency surgery 2. Mixed incontinence 3. Voiding disorder 4. Neuropathic bladder	Residual Volume >50ml 1st Desire to Void: 150-200ml Bladder Capacity: 400-600ml Detrusor Pressure (Filling phase): <15cmH20 Detrusor Pressure (Voiding phase): 40-70cmH20 No leakage when coughing

MANAGEMENT

A. CONSERVATIVE

Success rate 40-60%. Mainstay in Stress Inc.
Stop smoking, alcohol, caffeine. Kegel exercise.
Intravaginal device to support bladder neck & urethra.

B. MEDICAL

Mild-Moderate Stress Incontinence	Urge Incontinence
Alpha-Agonist Pseudoephedrine Phenylpropanolamine	**Anticholinergic Drugs** Oxybutynin chloride Tolterodine **TCA**: Imipramine

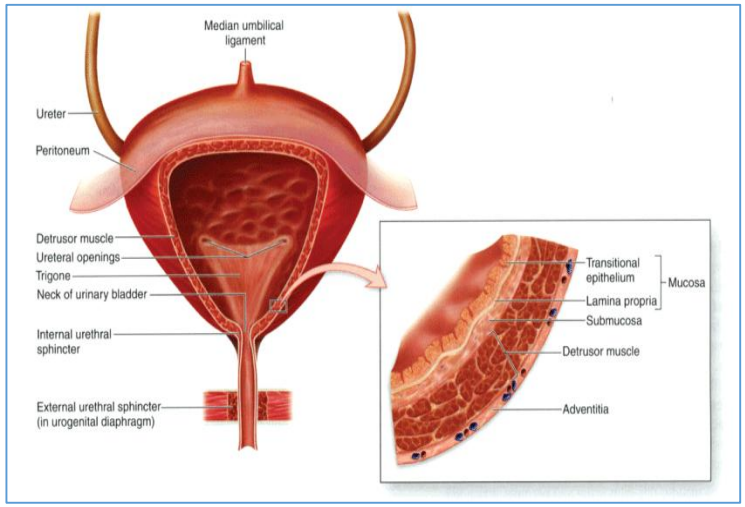
C. SURGICAL [Stress Incontinence]

Colposuspension	Tension-free Vaginal Tape (TVT)

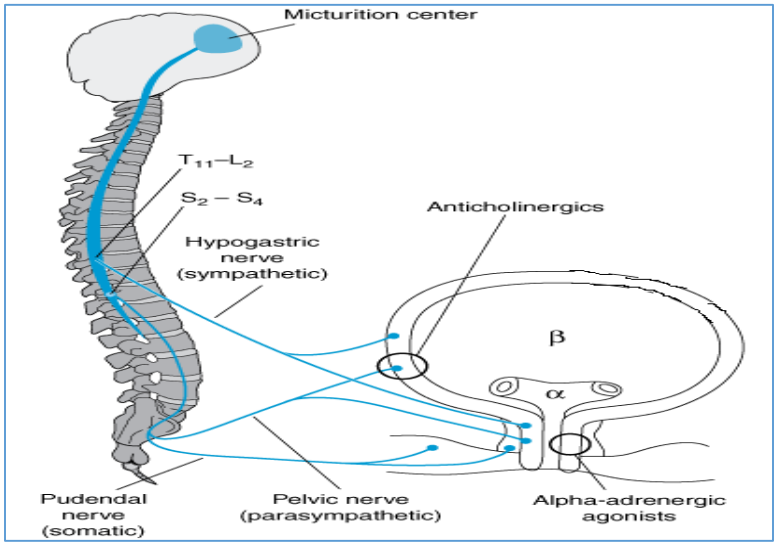
URINARY INCONTINENCE

Urinary incontinence, the involuntary leakage of urine, remains undetected and undertreated worldwide despite its substantial impact on affected individuals In a US survey, only 45% of women who reported urinary incontinence occurring at least once a week sought care for their incontinence symptoms The prevalence of incontinence in women is high In older women, the prevalence of urinary incontinence is 17-55%. For younger and middle-aged women, the prevalence is 12-42%	**Anatomy of the urinary system:** Normal Urethral Closure - Normal urethral closure is maintained by a combination of intrinsic & extrinsic factors: The extrinsic factors: The levator ani muscles, the endopelvic fascia, & their attachments to the pelvic sidewalls & the urethra The intrinsic factors includes: the striated muscle of the urethral wall vascular congestion of the submucosal venous plexus the smooth muscle of the urethral wall & associated blood vessels the epithelial coaptation of the folds of the urethral lining
The Bladder - The bladder is a low-pressure system that expands to accommodate increasing volumes of urine without an appreciable rise in pressure. - Micturition: During bladder filling, there is an accompanying increase in muscle fiber recruitment of the pelvic floor & urethra → increase in outlet resistance. The bladder muscle (the detrusor) should remain inactive during bladder filling, without involuntary contractions When the bladder has filled to a certain volume, fullness is registered by tension-stretch receptors, which signal the brain to initiate a micturition reflex → this reflex is permitted or not permitted by cortical control mechanisms, depending on the social circumstances & the state of the patient's nervous system.	**Innervations** - The lower urinary tract receives its innervation from three sources: Sympathetic nervous system Parasympathetic nervous system The neurons of the somatic nervous system (external urethral sphincter) The sympathetic nervous system: - Originates in the thoraco-lumbar spinal cord, principally T11 through L2-L3 - Acts on two types of receptors: Alpha-receptors → in the urethra & bladder neck → increases urethral tone & thus promotes closure Beta-receptors → in the bladder body → decreases tone in the bladder body The parasympathetic nervous system: Controls bladder motor function — bladder contraction & bladder emptying Originates in the sacral spinal cord, primarily in S2 to S4 The somatic nervous system

Normal voiding is accomplished by voluntary relaxation of the pelvic floor & urethra, accompanied by sustained contraction of the detrusor muscle, leading to complete bladder emptying



The somatic innervation of the pelvic floor, urethra, & external anal sphincter originates in the sacral spinal cord, primarily in S2 to S4



Classification:

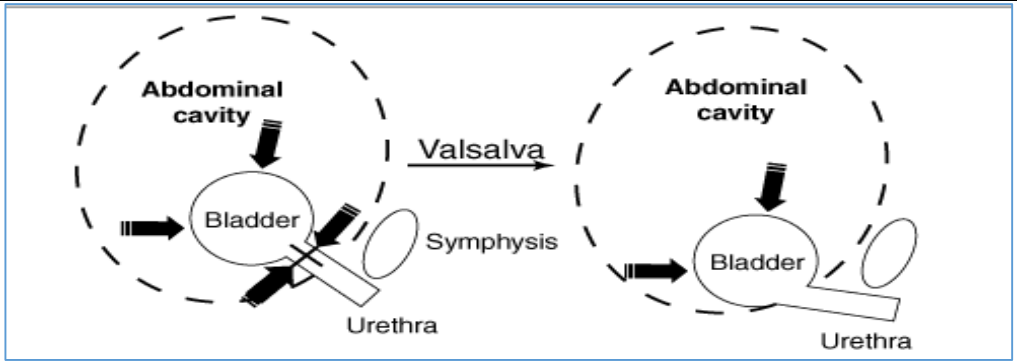
The four main types of urinary incontinence are urge, stress, mixed, and overflow incontinence

1. **Stress incontinence:**

The most common type

Patient have involuntary leakage of urine that occurs with sneezing, coughing, laughing, or anytime an increase in intraabdominal pressure exceeds urethral sphincter closure mechanisms

Stress incontinence may be provoked by minimal or no activity when there is severe sphincter dysfunction



2. Urge incontinence:

Patient typically have symptoms of involuntary leakage of urine accompanied by urgency.

Common triggers include running water, hand washing, and cold weather exposure.

Urgency incontinence is believed to be partly caused by detrusor overactivity.

3. Overflow incontinence:

Is involuntary, continuous, urinary leakage or dribbling and incomplete bladder emptying.

It is caused by impaired detrusor contractility or bladder outlet obstruction (rare in women)

If the bladder is over-distended:

An increases in intra-abdominal pressure can force urine past the urethral sphincter, causing stress incontinence

In some cases, bladder over-distention may provoke an uninhibited contraction of the detrusor muscle, leading to incontinence.

Symptom Comparison of Women with Stress or Urge Incontinence

Symptom	Urge Incontinence	Stress Incontinence
Urgency	Yes	No
Frequency with urgency	Yes	No
Urine leakage with increased intra-abdominal pressures	No	Yes
Ability to reach the toilet in time following an urge to void	Often no	Yes
Waking to void at night	Usually	Seldom

Causes:

Genitourinary

In older women, several physiologic changes occur in the lower urinary tract that can cause incontinence:

Involuntary detrusor contractions or overactivity

Decreased detrusor contractility

Low estrogen levels

Decrease in urethral closure pressure

Others:

Urogenital fistulas

Interstitial cystitis (painful bladder syndrome

Pelvic organ prolapse (e.g., cystocele)

Systemic conditions

Neurologic disorders: e.g. stroke, multiple sclerosis, Parkinson disease, disc herniation, spinal cord injury…

Diabetes mellitus: overflow incontinence and poor urinary stream can be present in patients with diabetic autonomic neuropathy

Cancers

Potentially reversible causes

- Medications (e.g., alpha blockers)
- Decreased mobility (e.g., post-surgery)
- Change in cognitive or mental status (e.g., sedation from medications)
- Stool impaction
- Alcohol and caffeine intake

EVALUATION

History:

- Patient's urinary symptoms (volume, onset of incontinence, timing, severity, hesitancy, precipitating triggers, nocturia, intermittent or slow stream, incomplete emptying, continuous urine leakage, and straining to void) Voiding (bladder) diaries are sometimes useful for assessing incontinence frequency, severity, and volume of urine loss during incontinent episodes

- Severity of symptoms & degree of bother and effect on quality of life
Urinary incontinence has profound effects on quality of life and is associated with depression and anxiety, work impairment, social isolation, and sexual dysfunction

- If there is indications to evaluate for underlying serious causes or potentially reversible conditions. Alarm symptoms on history include:
sudden onset of incontinence
the presence of abdominal or pelvic pain
Hematuria
changes in gait or new lower extremity weakness, cardiopulmonary or neurologic symptoms
mental status changes

- Other: drug history, constipation, caffeine intake …etc.

Physical examination

- All women presenting with incontinence need a pelvic examination. In addition, a comprehensive examination is often necessary to detect potentially reversible factors and underlying serious conditions

- The detailed pelvic examination in women includes:

- Inspect the vaginal mucosa for signs of atrophy (thinning, pallor, loss of rugae), and inflammation
- Palpate bimanually to evaluate for masses or tenderness.
- Assess for pelvic organ prolapse: hold the blade firmly against the posterior vaginal wall. Ask the woman to cough once, looking for urethral leakage &/or cystocele.
- Bladder stress test — is performed by asking the patient, with a full bladder, to stand, relax, and give a single vigorous cough

Investigations:

- Urine analysis

- **Post void residual volume (PVR)** — In general, a PVR of < 50 mL is considered adequate emptying, and a PVR > 200 mL is considered inadequate and suggestive of either detrusor weakness or bladder outlet obstruction

Urodynamic testing

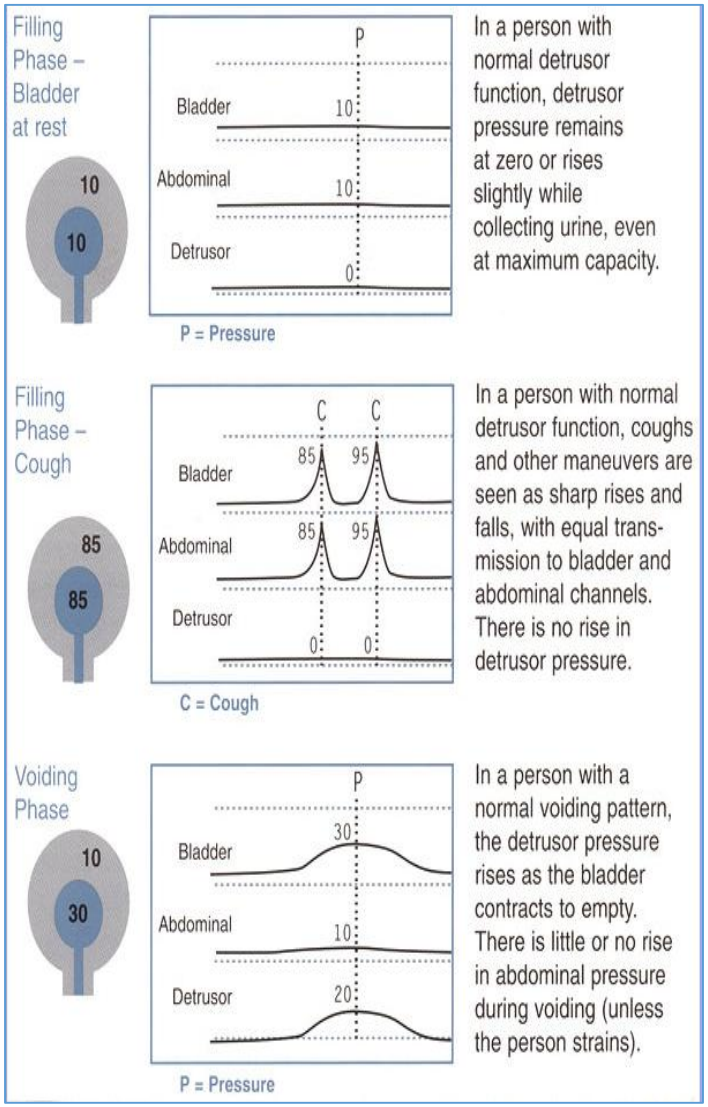
Urodynamics refers to a group of tests used to assess function of the urinary tract. Some specific types of urodynamic testing are:
Cystometry (or cystometrogram) evaluates bladder function by measuring pressure and volume of fluid in the bladder during filling, storage, and voiding.
Uroflowmetry measures the rate of urine flow.

Clinical evaluation with urodynamics may lead to a more accurate diagnosis of incontinence type

Normal observations and results — The normal bladder should not have involuntary phasic contractions during filling despite provocation. It should initially expand without resistance or increased intraluminal pressure. The urethral sphincter should relax and open when the patient wants to initiate voiding, accompanied by detrusor contractions. During voiding, detrusor contraction should be smooth and lead to a steady urine stream.

Technique

The patient begins by emptying her bladder as much as possible
A catheter is inserted into the bladder
Intravesical & rectal catheters are placed to measure detrusor and abdominal pressure
Water or normal saline is used to fill the bladder
the woman is asked to describe sensations during filling, including when the first feeling of bladder fullness occurs.
Provocative maneuvers, such as coughing, Valsalva, listening to running water are helpful for determining if they cause leakage and whether the leakage is related to uninhibited detrusor contractions or stress incontinence
Once the bladder is completely full, the woman is asked to begin voiding, and measurements are made of pressure, volume, and flow rate.



<table>
<tr>
<td>- Approximate of normal cystometric values for women are:
• Residual urine → < 50 mL
• First desire to void → occur between 150 & 250 mL
• Strong desire to void → doesn't occur until > 250 mL
• Bladder compliance → between 400 & 600 mL
• No uninhibited destructor contraction during filling, despite provocation
• No stress or urge incontinence
• Voiding occurs because of voluntarily initiated & sustained detrusor contraction
• Flow rate during voiding is > 15 mL / sec with detrusor pressure of < 50 cm H2O
Overactive bladder "Detrusor overactivity" can be diagnosed if there is urgency or leakage with a detrusor contraction that the patient cannot suppress
Stress urinary incontinence is characterized by leakage that occurs with an increase in abdominal pressure, such as coughing or Valsalva, without a rise in true detrusor pressure</td>
<td>Prevention
Behavioral and lifestyle changes: weight loss for obesity, smoking cessation, increasing physical activity/exercise, improving diet…etc.

Pelvic floor muscle exercises are effective in preventing and reversing some urinary incontinence in the first year after vaginal delivery or following pelvic surgery

Management of conditions associated with incontinence (e.g., diabetes, constipation…etc)

Specific medications and surgical procedures may adversely affect continence, and clinicians should include these risks in discussing treatment choice with patients</td>
</tr>
<tr>
<td>Treatment
1. Stress incontinence
Non-surgical Treatment
• Reduce factors that worsen the problem → obesity, smoking, medication, excessive fluid intake…etc
• Pelvic floor exercise & biofeedback
• Estrogen therapy (in postmenopausal women with urogenital atrophy)
• Electrical stimulation of pelvic floor muscle
Surgical Management
• Anterior vaginal colporrhaphy
• Retropubic bladder neck suspension operations
• Tension-free vaginal tape
• Sling operations
• Periurethral injections
2. Overflow incontinence:
Medical therapy to enhance bladder emptying provided there is no obstruction e.g. Bethanechol (used rarely). Treatment of the underlying cause of obstruction e.g. myomectomy or hysterectomy in the case of fibroid, removal of the urethral stricture, Intermittent self catheterization</td>
<td>3. Urge incontinence
Conservative measure:
Cut down volume of fluid consumed – should consume between 1 & 1.5 liters a day
Avoid caffeine based drinks

Bladder training: the patient is instructed to void on a timed schedule, starting with a relatively frequent interval

Medications:
Antimuscarinic drugs (e.g. tolterodine and oxybutynin)
Estrogen

Intra-vesical therapy (capsaicin, Botulinum toxin)

Sacral nerve root neuromodulation
Surgery (cystoplasty, urinary diversion) in refractory cases</td>
</tr>
</table>

VAGINAL DISCHARGE

Vaginal discharges are common and normal in all women, they have a lot of beneficial functions like :

1. Helps the vagina to stay healthy by regularly flushing them out.
2. Maintain the vaginal ph
3. Acts as a lubricant for sexual intercourse
4. Acts as a protective factor against infections.(because they are acidic)

It's the most common gynecological condition encountered by physicians in the clinic.

- Characteristic of normal vaginal discharge:

1. About 4 ml a day
2. White or transparent in color, becoming yellow on contacting with air.
3. Thick to thin in consistency.
4. Odorless

Components of normal vaginal discharge:

1. Desquamated epithelial cells.
2. Mucus from cervical glands(90% water)
3. Bacteria(lactobacilli)
4. Transudate from vaginal wall
5. Proteins ,polysaccharides ,amino acids, enzymes, Igs

- Any changes in color, amount, odor or consistency may indicate an underlying problem like an infections.
- It is not uncommon for the normal discharge to be dark, brown or discolored a day or two following the menstrual period.
- Vaginal discharge might be a sign of an infection if it:

1- causes itching
2- causes swelling
3- has a bad odor
4- is green, yellow or gray in color
5- looks foamy or like cottage cheese

Vaginites:

- significant inflammatory response in vaginal wall.
- Accompanied by high number of leukocytes in vaginal fluid. Found with candida and trichomonas infections.

Vaginosis:

- minimal inflammatory response with few leukocytes in vaginal wall.
- Associated with increase in bacterial concentrations

	normal	Cause for concern
Color	Clear or whitish discharge (may be yellowish when dried)	Yellow or greenish discharge, or discharge that suddenly changes color
Odor	Mild odor or odorless	A strong, foul, sometimes "fishy" odor, or a sudden change in odor
Texture	Can vary from "paste" like and somewhat sticky to clear and stretchy, depending on where you are in your cycle and whether you are aroused	Clumpy or lumpy discharge, with "cottage cheese" like texture
Volume	Can vary from very little to quite a lot (particularly when ovulating or aroused)	Sudden changes in volume, particularly if other symptoms are present

History

- Source of discharge (perineal discharge could be from vagina, cervix, urinary tract and rectum).
- Quantity
- Color
- Consistency
- Odor
- duration

Symptoms like:

- Itching
- Burning
- External disuria
- Dyspareunia
- Lower abdominal pain
- Fever and chills
- Nausea and vomiting

Also:

- Prior similar episodes
- Sexually transmitted infection
- Sexual activities
- Birth control method
- Last menstrual period
- Douching practice
- Antibiotic use
- General medical history

Physical examination

- Appearance of discharge
- Erythema and edema of vaginal mucosa

Investigations

- Nitrazine paper for PH
- Wet preparation: microscopic examination of discharge (clue cells of bacterial vaginosis)
- KOH preparation: dissolved cellular debris leaving pseudohyphae of candida
- Whiff test : fishy odor of bacterial vaginosis.
- culture

Causes of Changes in Vaginal Discharge

Non infective causes:

1.Physiological:

- Menstrual cycle
- Pregnancy
- Sexual excitement
- Emotional stress
- Nutritional status
- Medication

2. Cervical polyps and ectopy.
3. Foreign bodies (retained tampon)
4. Vuvular dermatitis
5. Erosive lichen planus (chronic condition affecting the mucosa)
6. Genital tract malignancy
7. fistula

Infective causes:

A : non-sexually transmitted infections

* bacterial vaginosis
* Candida vaginitis

B : sexually transmitted infections

*chalmydia trachomatis
*nisseria gonorreae
*trichomonas vaginitis
*syphills
*HSV
*HPV
*HIV

Bacterial Vaginosis

- Most common cause of vaginal discharge.
- Up to 50% are asymptomatic.
- Risk factors:
  It is seen in women without previous sexual experience. Many risk factors, however, are associated with sexual activity:

1.Oral sex
2.Douching(washing or cleaning out the vagina (birth canal) with water or other mixtures of fluids)
3. Black race
4. Cigarette smoking
5. Sex during menses
6. Intrauterine device
7. Early age of sexual partners
8. New or multiple sexual partners
9. Sexual activity with other women

How to diagnose bacterial vaginosis

- By amsel criteria:

1. Gray, homogenous discharge adherent to walls
2. Ph >4.5
3. Fishy odor with 10% KOH (+ve whiff test)
4. Clue cells on wet preparation

3 out of 4 at least must be present to confirm the diagnosis.

Nitrazine ph test

- Vaginal ph (3.5-4.5)
- Turns blue on alkaline media
- 93.3% sensitivity
- False positive (1-17%)
- Clue cell on wet prep:

saline wet preparation reveals clue cells.
Squamous cells covered with bacteria to the extent that cell borders are blurred and nuclei are not visible.

Whiff test(amine test)

- Addition of 10% KOH to a sample of vaginal discharge produces fishy odor (+Ve).
- It has positive predictive value of 90% and specificity of 70%.

Management

- The treatment and management of bacterial vaginosis found to be 90% effective in usage of combination of topical and systemic agents with anaerobic activity.

A. Topical (vaginal) preparations:

1-intravaginal 2% clindamycin cream is used at bed time for 7 days
2-intravaginal metronidozole is used once a day for 5 days.

B- oral regimens:

1.Metronidazole : either 500 mg twice daily for 7 days
or single dose of 2 gr.
2. Clindamycin : 300 mg twice daily for 7 days

No treatment is needed for sexual partner.

Candidal Vaginitis

- It's the second most common cause of vaginal discharge.
- The causative agents:

-candida albicans
-candida tropicalis
-candida glabrata.

Risk factors for candidal vaginitis

1. The presence of diabetes mellitus.(uncontrolled)
2. Chronic usage of antibiotics (decrease lactobacilli concentration)
3. Contraceptive practice.
4. Altered immune status.
5. Clothing and bathing suits.

Clinical presentation

- Symptoms of candidal vaginitis:

1. Vaginal itching and burning
2. Vulvar burning and dysuria
3. Dyspareunia

Patients with candidal vaginitis caused by c.tropicalis and c.glabrata may complain of atypical presentation

Diagnosis

- History
- Physical examination:

1-vulvar erythmea with satellite lesions (discrete pusulopapular lesions)
2-excoritation of the vulva.
3-whitish discharge varying from thin to crud consistency (cuttage cheese like)
C.Tropicalis and c.glabrata associated vaginal discharge maybe white-gray and thin.

Investigations

- Vaginal PH is usually normal.
- Wet amount microscopic examination:

Hyphae or psudohyphae with budding yeast in 50% to 70% of woman with yeast infection.

- Cultures are not necessary to make diagnosis except in some cases of recurrent infections.

Management

- Topical agents:

Available over the counter(sold directly to a consumer without a prescription) or by prescription.

- Oral agents:

Available by prescription only.
No treatment is needed for sexual partner
Topical creams

- Butaconazole
- Clotimazole
- Miconazole

these drugs used as a single dose, 3-day course, 7-day course.
Over the counter drugs should be used only by women who has been diagnosed with a yeast infection in the past and experiencing identical symptoms.
Oral agents

- Fluconazole:

For treatment of uncomplicated vaginal candidiasis.
Single dose (150 mg)

- Ketoconazole:

For treatment of chronic and recurrent candidiasis.
200 mg twice a day for 5 days.
It may cause hepatotoxisity.

Chronic recurrent yeast infections

- It occurs in 5% of woman caused by:

1. Failure to complete a full course of thereby
2. HIV infection
3. Chronic antibiotic usage
4. Infection with resistant organism like: c.tropicalis, c. glabrata
5. Sexual transmission from male partner
6. Allergic reaction to male partner's semen
7. Diabetes.

- If the discharge is caused by a sexually transmitted disease, sexual partner (or partners) must be treated as well, even if they have no symptoms.
- Failure of partners to accept treatment can cause the infection to keep coming back and may lead to pelvic inflammatory disease or infertility

The patient should seek medical help right away if:

- The discharge is associated with fever or pain in the pelvis or abdomen.
- If the patinet have been exposed to a sexual partner with gonorrhea, chlamydia, or other sexually transmitted disease.
- If the patient have increased thirst or appetite, unexplained weight loss, increased urinary frequency, or fatigue -- these may be signs of diabetes.

1. <u>To help prevent and treat vaginal discharge:</u>
2. Keeping genital area clean and dry.
3. Avoiding douche. While many women feel cleaner if they douche after menstruation or intercourse, it may actually worsen vaginal discharge because it removes healthy bacteria lining the vagina that are there to protect you from infection. It can also lead to infection in the uterus and fallopian tubes, and is never recommended.
4. Using an over-the-counter yeast infection treatment cream or vaginal suppository, if you know that you have a yeast infection.
5. Eating yogurt with live cultures or take Lactobacillus acidophilus tablets when you are on antibiotics to avoid a yeast infection.
6. Using condoms to avoid catching or spreading sexually transmitted diseases.
7. Avoid using feminine hygiene sprays, fragrances, or powders in the genital area.
8. Avoid wearing extremely tight-fitting pants or shorts, which may cause irritation.
9. Wearing cotton underwear or cotton-crotch pantyhose. Avoid underwear made of silk or nylon, because these materials are not very absorbent and restrict air flow. This can increase sweating in the genital area, which can cause irritation.
10. Using pads and not tampons.
11. Keeping blood sugar levels under good control if you have diabetes.

UTERINE FIBROID (LEIOMYOMA)

Benign tumor that arises from the smooth muscle cells of the myometrium , along with fibrous tissue.

Epidemiology:
-most common benign tumor of the uterus (45% of women by 5^{th} decade).
-a primary indication of 200,000 hysterectomies yearly in the US.
-has a very high potential to grow in size,but has a very low potential to become malignant (leiomyosarcoma) 1/1000

Pathogenesis is unknown,but there is theory :
1) Ovarian sex steroids (estrogen and progesteron)
-as studies show that leiomyomas very rarely develops before menarche or after menopause (except if there is exogenous hormonal intake), also dramatically increases in size during pregnancy
2) Increased levels of estrogen and progesterone receptors in fibroid
#Direct effect of each of them ?
-estrogen : stimulate the proliferation of smooth muscle cells
-progesterone : increases production of proteins that interfere with smooth muscle cell apoptosis .
3) Other factors like the growth factors which stimulate the production of fibronectin and collagen (main components of extracellular matrix).

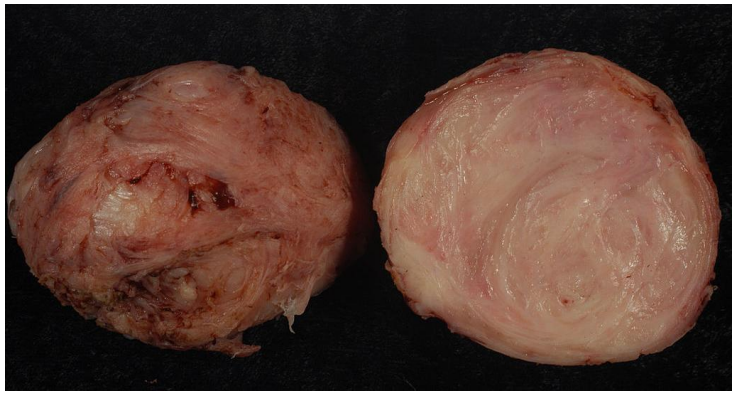
Factors affecting :

RISK FACTORS		PROTECTIVE FACTORS
1) age (during reproductive period)	4) family history	1) OCP
2) ethnicity (mostly in african americans)	5) increase bmi	2) depot medroxyprogesterone acetate
3) nulliparity	6) high consumption of red meat	3) high consumption of green vegetables , fruit & fish

Characteristics :

Spherical,well circumscribed white, firm lesions

Whorl appearance



Pseudocapsule (compressed smooth muscle cells in the tumor periphery)

Blood vessels transverse through pseudocapsule
→if blood supply become insufficient→
Degenerative changes
(most common is hyaline change :
> more severe > cystic degeneration, calcification or fatty changes)
During pregnancy 5-10% will have red (corneous) degeneration

Types, symptoms and signs of uterine fibroid :

Types	Symptoms (usually asymptomatic)	Signs
1) intramural : within the myometrium 95% (most commonly in the fundus)	-if large enough → can distort the shape of the uterus and cause prolonged, heavy periods, pain and pressure	-can be palpated abdominally if large -bimanual pelvic exam > firm , irregular , enlarged uterus with smooth rounded protrusions
2) subserosal : just below serosa	-urinary symptoms (press on urinary bladder) -backache (press on spinal nerve) -pressure sensation (press on rectum) -abdominal pain and constipation (press on bowel)	
3) submucosal : near the endometrium	-prolonged, heavy menstrual bleeding and are sometimes a problem for women attempting pregnancy	can be palpated abdominally if large

fibroids can extend through IVC reaching the heart (intravenous leiomyomatosis** → confined to vessels, whereas benign metastasising leiomyoma shows no relation to vascular channels)
→should not be confused with benign metastasising leiomyoma, in which it is associated with a benign smooth muscle tumor located in the parenchyma of a distant organ, such as lung
**signs : usually non-tender , usually at the midline ,and its consistency varies from rock hard (if calcified) to very soft in cystic degeneration

DDX:
1) ovarian neoplasm
2) tubo-ovarian mass
3) pelvic kidney
4) diverticulum
5) colon CA
6) adenomyosis
7) uterine sarcoma

Complications :

1) **Infertility**:

-Fibroids can narrow the isthmus portion of fallopian tube.

-Or it can interfere with implantation if it was submucosal .

2) **Complication during pregnancy** :

-Spontaneous miscarriage.

-Intrauterine growth restriction (IUGR).

-Uterine dyskinesia (loss of uterine contraction during labor) during labor (so we use caesarian section)

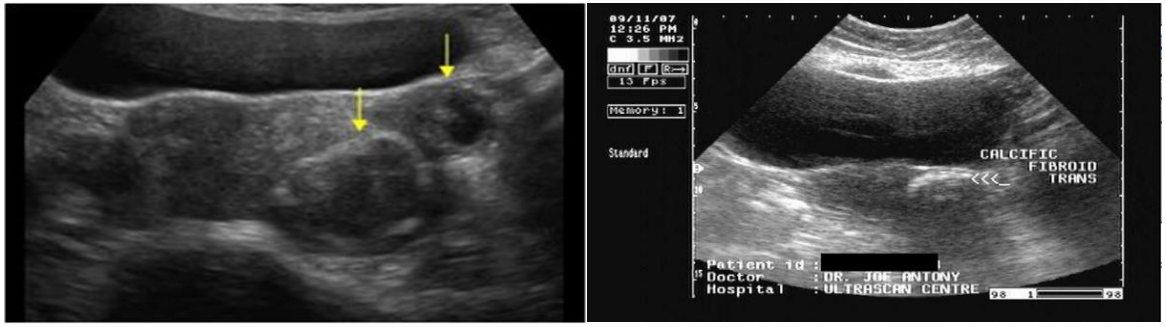
-Fibroids can obstruct the birth canal , also indication for caesarian section

3) **Postpartum hemorrhage**,occurs due to:

- Uterus become atonic after delivery

-Fibroid is too big so uterus can't contract so she will be more prone to bleeding

→ if we want to deliver a pregnant woman by c/s , and we found incidentally a uterine fibroid we shouldn't try to remove it as it may induce severe bleeding, that may end with the death of the mother. Diagnosis confirmed mostly by US, but we can use MRI or CT



Treatment :

-If it is asymptomatic >> not necessary to interfere

((except if it size is more than 12 week gestation sized uterus or if it is implicated as case of infertility))

** the 1st line of treatment is targeting the symptoms

(A) **Medical treatment** - hormonally (main aim is to manage menorrhagia) :

i) progestin only therapies (Medroxyprogesterone acetate , levonorgestrel).

ii) combined contraceptives (oral , vaginal , patches).

iii) GnRH agonist : block ovarian steroidogensis (decreases endometrial proliferation and volume of myometrium) but it decreases bone density so it should be used in combination with OCP

** also used to prepare for surgery .

((clinical trials using the selective antiprogesterone receptor antagonist (mefiprestone) to reduce the size of uterine myomas have shown reduction of 50% by volume over 3 months period))

(B) **Surgery**

i) Myomectomy : this is surgical procedure performed if the patient desires to maintain fertility . The uterus is incised and the myoma removed through either a laparoscopic or a laparatomic approach. If the myomectomy incision enterd the uterine cavity, delivery of any subsequent pregnancy should be by caesarian section because of increased risk of scar rupture.

ii) Embolization : this is invasive radiology procedure in which a catheter is placed into the vessels supplying the myoma, can cause ischemia and necrosis of myoma

iii) Hysterectomy : if the patient has completed her childbearing, definitive therapy is an abdominal or vaginal hysterectomy

TABLE 19-1

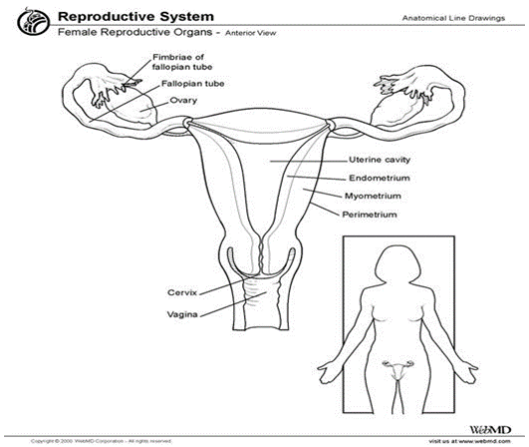
INTERVENTION FOR PATIENTS WITH LEIOMYOMAS NOT AMENABLE TO MEDICAL THERAPY*

Clinical Presentation	Nonmedical Options	Comments
Desired fertility	Myomectomy or uterine artery embolization (UAE)†	Usually used for a limited number of leiomyomas
Desired uterine preservation or poor surgical risk	Endometrial ablation or UAE	UAE only for a limited number of leiomyomas
No desired fertility or uterine preservation	Endometrial ablation or hysterectomy	Hysterectomy is definitive therapy
Rapidly growing uterus (double in size in 6 months)	Exploratory laparotomy, abdominal hysterectomy	More extensive surgery if malignancy discovered

*Generally, failed medical therapy or large (>12-14 weeks' gestational size) uterus.
†Pregnancies after UAE are at higher risk.

RECAPS ANATOMY ☺

- Inner layer (endometrium) : the most active layer and responds to cyclic ovarian hormone changes; is highly specialized and is essential to menstrual and reproductive function
- Middle layer (myometrium) makes up most of the uterine volume and is the muscular layer, composed primarily of smooth muscle cells
- Outer layer of the uterus (serosa or perimetrium) is a thin layer of tissue made of epithelial cells that envelop the uterus



ENDOMETRIAL CANCER

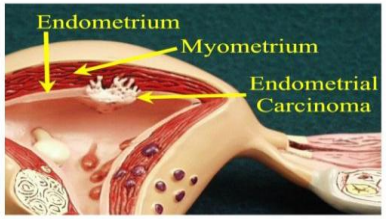
Malignant tumor of uterus according to their origin :
Endometrium (90%)

- glands : endometrial adenocarcinoma (commonest)
- stroma: endometrial stromal sarcoma
- glands + stroma : MMMT (uterine carcinosarcoma)

Myometrium (10%o=)

- leomyomsarcoma
- others

Endometrial carcinoma



Any condition where the endometrium is exposed to only estrogen, this will lead to endometrial hyperplasia.

- Simple without atypia - 1% risk for cancer
- Complex without atypia - 5% risk
- Simple with atypia - 8% risk (treated by hysterectomy)
- Complex with atypia - 30% risk (treated like cancer)

Protective factors:

 COCP (estrogen + Progesteron)

 Progesterone

 Smoking is a protective factor, but if the endometrial ca. occurs in a smoker pt. the mortality rate will be high!

Endometrial Adenocarcinoma

- malignant cancer of endometrial glands.
- 75% of cases : postmenopausal women
- 20% in premenopausal women
- less than 5% before 40 years old (genetic risk factor like LYNCH 1, 2 genes)
- Peak incidence is at age of 61 years old .

Aetiology:

- Excessive unopposed **estrogen** stimulation of the endometrium .
- Normally, the endometrium is exposed to estrogen in the 1st half of the cycle & progesterone in the 2nd half (protective). So in any condition where the endometrium is exposed only to estrogen, there'll be endometrial hyperplasia & carcinoma.

Risk factors:

- Obesity (peripheral conversion of androgens into estrogens)
- Nulliparity, Infertility,Anovulatory cycles(never get pregnant)
- Early menarche, late menopause (many periods in their life so high exposure to estrogen)
- PCOS
- Estrogen hormonal replacement therapy.
- Tamoxifen: estrogen receptor antagonist in breast tissue, but acts as partial agonist on the endometrium.
- Estogen-secreting ovarian & breast tumours
- Family hx of breast,ovary,colon& endometrial tumours (lynch syndrome or HNPCC-Hereditary nonpolyposis colorectal cancer)
- Endometrial polyps or other benign growths of the uterine lining (like fibroid) because they are estrogen secreting polyps
- Pelvic radiation therapy
- High intake of animal fat, alcohol &Lack of exercise
- DM, HTN (associated factors)

Note: 70-80 % of endometrial cases are diagnosed in early stages because the PMB will worry the pt. and they will seek the help quickly and diagnosed at early stage.

Protective : OCCP, Progesterone

Spread: - direct invasion
- lymphatic
- blood (very rare)

The subtypes of Endometrial Adenocarcinoma :

- Endometrioid(most common , 90% of cases)
- Clear cell (the most malignant)
- Papillary serous (with hereditary types) ** Clear cell & Papillary serous are represent only 10% of endometrial ca. , but they are responsible of >50% of endometrial ca. related death.
- Adenoacanthoma : endometrioid adenocarcinoma with (benign appearing) squamous differentiation(good prognosis)
- Adenosquamous carcinoma: contains both malignant glandular and malignant squamous components(poor prognosis). * the last 2 are the second most common after Endometrioid

Presentation:

• Post-menopausal bleeding (Most common presentation)

ENDOMETRIAL BIOBSY SHOULD BE DONE IN ALL PATIENTS WITH PMB

Note : the most common cause of PMB is atrophic endometrium , and only 10% of PMB cases are due to endometrial ca.

• Interenstrual bleeding or irregular periods in premenopausal women
• Heavy regular periods in premenopausal women
• Watery discharge / offensive
• Pain (late presentation with local invasion, compressing pelvic nerves).

Diagnosis

Always investigate PMB, continuous or irregular bleeding before assuming benign cause for the bleeding

1. Cervical smear
2. TVS : Trans-vaginal US (normally the endometrial thickness <4mm in postmenopausal, if >4mm take a biopsy to R/O Ca).
3. Endometrial biopsy :

- D&C
- hysteroscopy guided biopsy
- pt. is morbidly obese and can't tolerate anaesthesia ,we will do office (outpatient) maneuver by a pipelle cannula which will pass through the cervical canal to the uterine cavity and suck endometrial cells by the osmotic property

(the accuracy of this maneuver is about 80 % of D&C accuracy)

4. If confirmed, CBC,KFT,URINE, MRI, CXR, ECG

Prognosis

• Stage, Grade (high grade & stage - poor prognosis)
• Myometrium invasion (more than 50% invasion - poor prognosis)
• Age (old - poor prognosis)
• LN involvement (poor prognosis)
• Clear cell & Papillary serous types (poor prognosis)
• Recurrence usually within 2y (70%)
• Overall 5 year survival is 60%

assessment of these factors require laparotomy and histology (surgical pathological staging - after the surgery and sending the tumour to the pathology , unlike the ovarian ca. that need CT , MRRI)

Staging

1 : confined to body of uterus
1A if less than 50% of myometrium is involved
1B if more than 50% of myometrium is involved

PHYSICAL SIGNS Rarely suggest the diagnosis Uterine enlargement Palpable lymph node in the groin.supraclavicle Vaginal nodule	2 : involve cervix 3 : Extension into adnexa(ovaries & tubes), vagina or positive L.N 4 : Distant Metastasis

Grading:
A measure of differentiation, the extent to which cancer cells are similar in appearance and function to healthy cells of the same tissue type.
Grade 1 well differentiated
Grade 2 moderately differentiated
Grade 3 poorly differentiated

Treatment: The definitive treatment is surgery (Total Abdominal Hysterectomy & bilateral oophorosalpingectomy).
TAH is recommended over vaginal hysterectomy because it affords the opportunity to examine & obtain washings of the abdominal cavity to detect any further evidence of cancer.

- Stage I

Low Risk : TAH& BSO± LN dissection *Generally they must do MRI before starting the treatment , MRI detect the invasion of myometrium , if the invasion is less than 50% there is no need for LN dissection , if more than 50 % they do LN dissection . or they do TAH & BSO then send the specimen to pathology (Frozen Section Biopsy), if the invasion is less than 50% there is no need for LN dissection

High Risk : we add postoperative radiotherapy *Stage 2 or up * Grade 3 *Clear cell & Papillary serous types * Invasion more than 50%

stage 1, grade 1&2 there is no need for postoperative radiotherapy we just follow up the pt. for any new symptoms every 3 months in the first year, then every 6 months in the second year , then yearly for 5 years.

- Stage II : TAH, BSO + radiotherapy or radical hysterectomy due to cervical involvement and no need for LN dissection if involvement is negative.
- Stage III&IV: combination radio , hormonal & chemo (rarely surgery)

o External radiation
o Hormonal therapy (progesterone)
o Note: there is no role of Chemotherapy in uterine ca. unless there is distant mets.

Endometrial sarcoma (rare) o Endometrial stromal sarcoma o Uterine carcinosarcoma (MMMT) – malignant mixed Mullerian Tumor • More in **black** • History of previous pelvic irradiation • Present with **bleeding**&**pain** • **Poor prognosis** with about 2y survival • Treated with hormonal therapy	**Leiomyosarcoma** • Mostly arise from normal myometrium • 5-10% may arise from transformation of fibroid (0.2%) ▪ Peak incidence is 45-55y (10 years older than fibroid) • Present with abnormal **bleeding**, pelvic **pain** & **wt loss** • Diagnosis: 80% is made after **hysterectomy** (histological exam), by US resemble fibroid. • Should be suspected in rapidly enlarging fibroids or keeps enlarging after menopause • Management (surgery): TAH,BSO, washing & full staging

FIBROID

benign monoclonal tumor of myometrium smooth muscle and fibers, termed as , leiomyoma, leiomyomata, myoma and fibromyoma

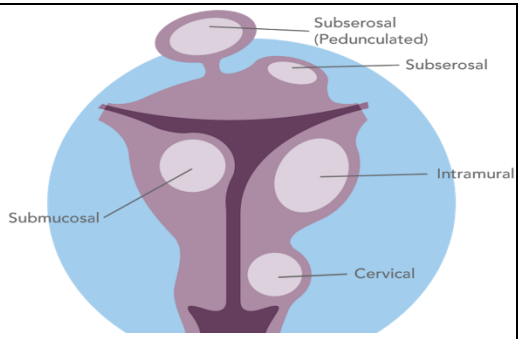
- Most common neoplasm of uterus
- 30% to 50% of women in reproductive age
- Mostly in women over 30

Pathogenesis - unknown

- Estrogen dependent (so estrogen stimulate proliferation , progesterone interfere with apoptosis)
- Enlarge dramatically during pregnancy, shrink after menopause
- Usually spherical well circumscribed , white , does not have a true capsule
- whorled appearance on cut section .
- Compression of smooth muscle on the tumors periphery ((pseudocapsule)) ; few blood vessels will traverse if insufficientnDegenerative changes : most common is hyaline change
- > more severe > cystic degeneration , calcification or fatty changes .
- Rarely before menarche or after menopause

Classification

According to growth location ：



- Myomas on the body & fundus （ 97%）
- Myomas on the cervix （3%）
- ((more than 95% in fundus))

According to the relation to uterine muscle ：

- Submucous leiomyomas
- Intramural leiomyomas Subserosal leiomyomas
- Myomas always arise within myometrium , but some migrate toward serosal surface (subserosal), or toward endometrium (submucosal)

1. Submucosal uterine fibroids

- These fibroids develop just under the lining of the uterine cavity.
- These neoplasms often protrude into the uterine cavity , even the cervical os .
- These are the fibroids that have the most effect on heavy menstrual bleeding and the ones that can cause problems with infertility and miscarriage
- If aborting through the cervical os __ asspciated with heavy bleeding , and crampy abdominal pain

• Has higher expression of estrogen receptors Risk factor • Increasing age (within reproductive years). • Ethnicity (more in black) • Nulliparity • Family Hx • Data suggest : • Higher risk with high BMI • OCP , depot medroxyprogesterone acetate injection & high consmuption of green vegetables , fruit & fish may reduce risk	2. Intramural uterine fibroids • The most common type of fibroid (>95%) • These develop within the uterine wall • if it was small it will be asymptomatic .. • If it large enough it may be palpable and expand making the uterus feel larger than normal 3. Subserosal uterine fibroids • These fibroids originate from the serosal surface of the uterus • Parasitic fibroid - fibroid gets detached from uterus and attaches to a vascular organ (omentum or bowel). • Torsion of the pedicle
Secondary changes Benign degeneration: • Atrophic .. Menopause • Hyaline degeneration (muscle and fibers are replaced by hyaline tissue) with further reduction in blood supply • Cystic degeneration - central necrosis • Calcification (particularly after menopause) • Red degeneration Malignant Transformation • Sarcomatous change **Red Degeneration** Occasionally seen as a complication of pregnancy Red degeneration follows an acute disruption of the blood supply to the fibroid during active growth (infarction), classically during the mid-second trimester of pregnancy. cut surface resembles raw meat.	Symptoms : Related to size , site and number of tumors • Mostly asymptomatic (incidentally found) • Generally not painful (severe pain may develop with red degeneration- during pregnancy) (if incarcerated painful +- dyspareunia) , dysmenorrhea . • The symptoms tend to decrease at the time of menopause • Intermenstrual bleeding is in submucosal type if ulcerating the endometrium. • Abnormal uterine bleeding Mechanism : un-proper response to hormonal fluctuations (metro) , interfere with clotting cascades (meno) • Distorting the endometrial cavity by increasing the endometrial surface area. • Intramural fibroid prevents adequate contraction and retraction of uterus • Distorted endometrium respond abnormally to normal hormones fluctuation • Excessive bleeding may lead to anemia , dyspnea , CHF • pelvic pressure and pain - Pressure on ureter … hydronephrosis - Pressure on bladder … urinary symptoms - Pressure on rectum … constipation • problems related to pregnancy : 40 % of fibroids enlarge during pregnancy most of the growth occurs in 1st trimester and they seldom interfere with the course of pregnancy

Sarcomatous Change

- Rare : <0.1%
- More common at 40～ 50 years old
- Usually occur in intramural fibroids
- Raise suspicion when:
- grow quickly
- vaginal bleeding
- Older age
- With necrosis
- Remember that malignant tumors do not generally arise from benign tumors

It has a very high potential to grow in size , but it has a very low potential to transfer into malignant tumor (leiomyosarcoma) 1/1000

Management : target the symptoms & patient desire
Medical
Surgical

1. Progestin

Heavy or prolonged menstruation ---

- progestin only therapies (oral , injection , levonorgestrel-releasing intra uterine device)
- combination hormonal contraceptive methods (OCP , vaginal rings or patches) .
- It will also reduce the dysmenorrhea

2. GnRH agonist

- help reducing the proliferation and the size
- Preparation for surgery
- Short term or intermittent use only

Spontaneous miscarriage, IUGR, Uterine dyskinesia > CS, obstruct the birth canal, Postpartum haemorrhage, 5-10% undergo painful red degeneration during pregnancy
Distort the endometrium so Increased chances of abortion is seen with submucous fibroid due to improper implantation

- Infertility - Compression on fallopian tube & Growth factors secreted by sub-mucosal fibroid can interfere with implantation

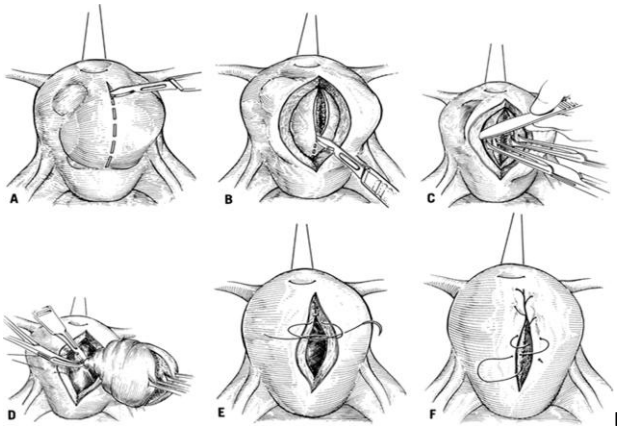
mass effect : Pelvic pressure, Bloating , Feeling of heaviness
Signs

- Palpable non tender(unless degenerating) mass (>12 week G.size –confined to pelvic)
- Bi-manual : firm regular enlarge uterus with smooth rounded or bosselated protrusion (subserosal , intramural)
- Vary in consistency, Mostly med line mass, Move with the cervix

Investigations
US is preferred -Hypoechoic mostly If calcified or Hyperechoic, MRI could be used,
Surgical : depend on the location
So we do an MRI to localize and estimate the volume :

- Submucosal – hysteroscopical
- Subserosal ,Pedunculated , & some intra-mural : removed laparoscopically
- Large ones : laparotomy

1. Myomectomy : If endometrial cavity is entered during myomectomy, so do c-section for future deliveries

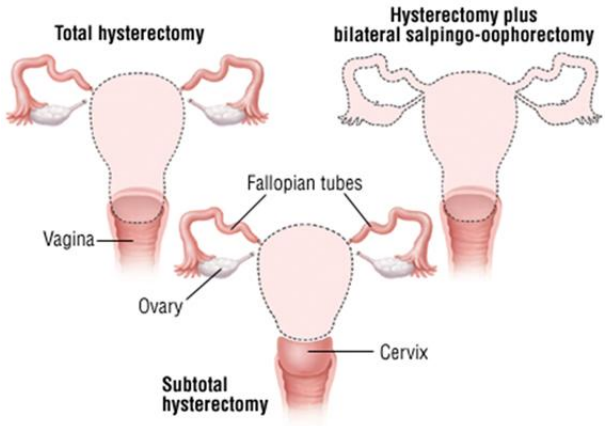


myomectomy

- Long term use affect bone density which may be overcome by adding low dose of hormonal agents
- Very expensive

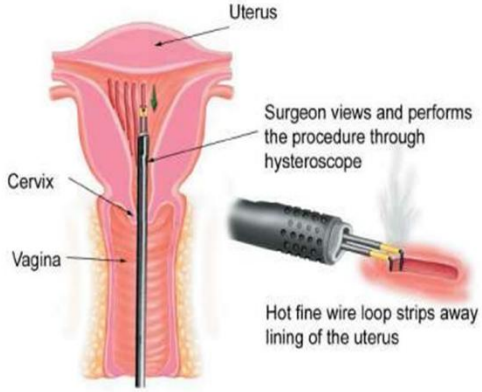
3. Selective antiprogesterone receptor antagonist
- Mifeprestone (RU486) to reduce the size of the tumor
- No effect on bone density

Note : new drug called Esmya "ulipristal acitate" is introduced for the treatment of fibroid but not approved yet.



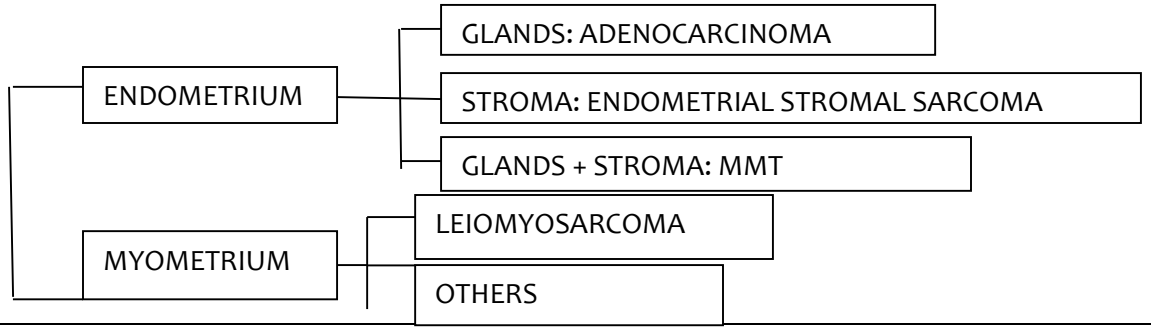
2. Endometrial ablation

For Excessive bleeding and the patient want Uterine preservation without fertility
Placenta accrete may occur if the patient becomes pregnant after endometrial ablation, so birth control is recommended



3. Uterine artery embolism
- Few , small/ moderate size neoplasms
- uterine preservation without fertility
- Shrinkage up to 40-60% of the size , reduce bleeding
- Pregnancy still possible but of higher risk
- No need for General Anesthesia

4. Hysterectomy
- Provide definitive Tt
- According to size :
- Large bulky uterus ----laparotomy
- Smaller ----- vaginal hysterectomy or laparoscopic hysterectomy
- ** usually ovarian preservation is encourged unless the women >60 yrs or has a risk for ovarian cancer
- ** uterine fibroid is the leading cause for hysterectomy in US
- hysterectomy may be total (removing the body, funus , and cervix of the uterus; often called "complete")
- partial (removal of the uterine body while leaving the cervix intact; also called "supracervical")



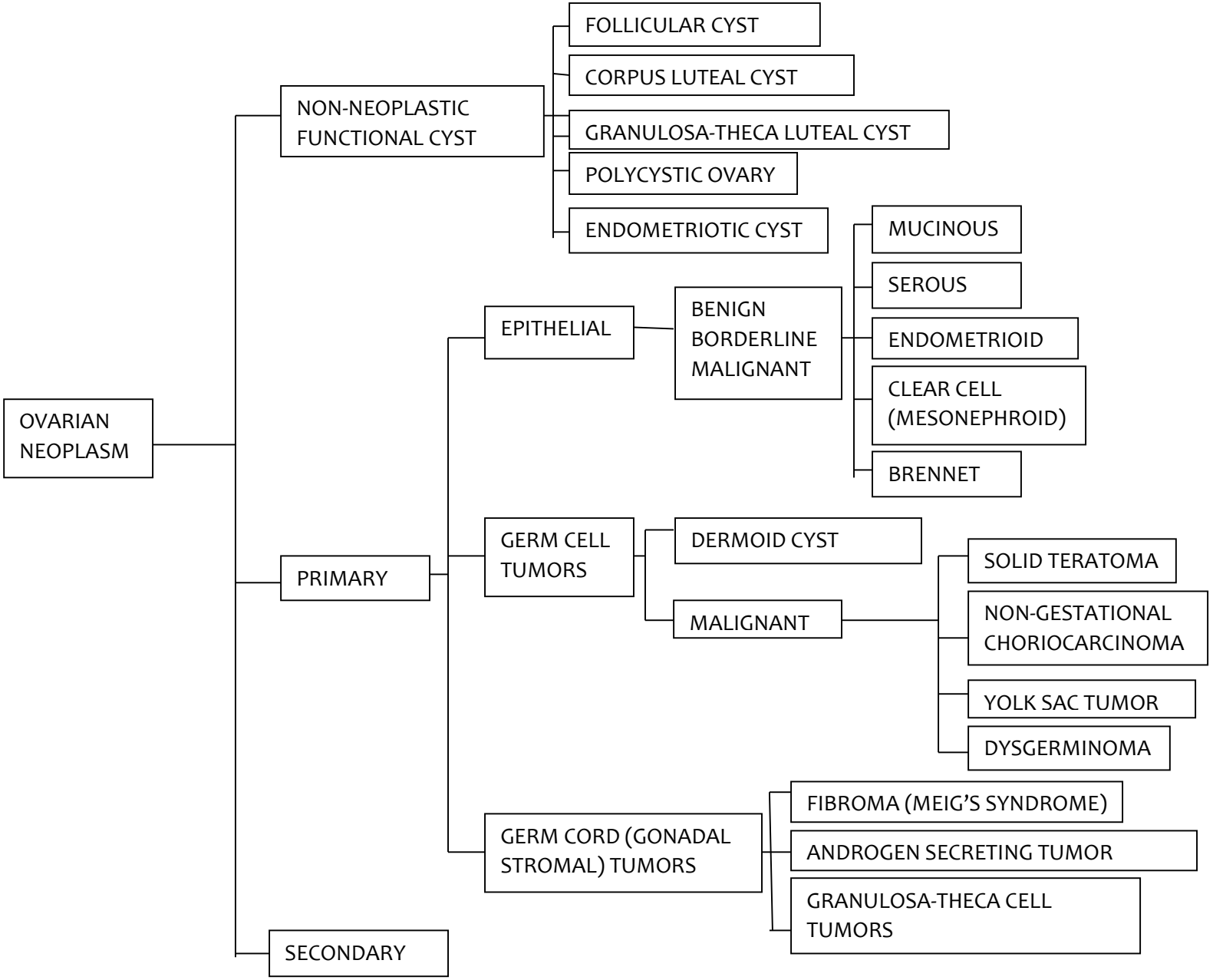
ENDOMETRIAL CARCINOMA			
INTRODUCTION	ETIOLOGY	CLINICAL PRESENTATION	PHYSICAL FINDINGS
● Peak incidence is at age of 61 years ● 75% occur in postmenopausal women ● Only 5% occur before age of 40 ● There is marked geographical and racial variation in the incidence	Excessive unopposed estrogen stimulation of the endometrium **Increase** Obesity, Nulliparity, Late menopause, PCO, Estogen-secreting ovarian tumors, Unopposed estrogen therapy, Family history of breast, ovary, colon, endometrial tumors, DM **Decrease** OCCP, Progesterone	● PMB ● Intermenstrual bleeding/irregular periods ● Heavy regular periods ● Watery discharge/offensive ● Pain ENDOMETRIAL BIOPSY SHOULD BE DONE IN ALL PATIENTS WITH PMB	● Rarely suggest the diagnosis ● Uterine enlargement ● Palpable lymph node in the groin. Supraclavicle. ● Vaginal nodule
PATHOLOGY	DIAGNOSIS	PROGNOSIS	TREATMENT
● Growth is usually adenocarcinoma ● Adeno-acanthoma/ adenosquamous tumors ● Serous papillary/ clear cell ● Grade 1 → grade 3 ● Spread: Direct invasion, Lymphatic, Blood	● Always investigate PMB, continuous or irregular bleeding before assuming benign cause for the bleeding ● Cervical smear ● TVS ● Endometrial biopsy ● Hysteroscopy +curettage ● If confirmed, CBC,KFT,URINE, MRI.CXR	● Stage ● Grade ● Myometrial invasion ● Age ● Tumor size Assessment of these factors require laparotomy and histology (surgical pathological staging)	● Low risk stage I: TAH, BSO ● High risk: postoperative radiotherapy ● Stage II: TAH,BSO+ radiotherapy/radical hysterectomy ● Stage III /IV: individualized .rarely surgery usually chemo, radiotherapy and hormonal ● Follow up

			• Recurrence usually within 2 years (70%) • Overall 5 year survival is 60%

FIGO STAGING

Stage 1	Confined to the body of uterus
Stage 2	Involvement of cervix
Stage 3	Extension into adnexae,vagina or positive L.N
Stage4	Distant mets

ENDOMETRIAL SARCOMA	LEIOMYOSARCOMA
• Endometrial stromal sarcoma • Malignant mixed mullerian tumors (carcinosarcoma) • More in black. Previous pelvic irradiation • Present with bleeding and pain • Poor prognosis	• 5-10% May arise from transformation of fibromyoma (0.2%) • Mostly arise from normal myometrium • Peak incidence is 10 years older than fibromyoma • Present with abnormal bleeding and pelvic pain and wt loss • Should be suspected in rapidly enlarging fibroids • In 80%,diagnosis is made after hysterectomy • Ideally should be treated by TAH, BSO, washing and full staging • Adjuvant radiotherapy or chemotherapy?


OVARIAN NEOPLASM
NON-NEOPLASTIC FUNCTIONAL CYST
FOLLICULAR CYST
CORPUS LUTEAL CYST
GRANULOSA-THECA LUTEAL CYST
POLYCYSTIC OVARY
ENDOMETRIOTIC CYST
PRIMARY
EPITHELIAL
BENIGN BORDERLINE MALIGNANT
MUCINOUS
SEROUS
ENDOMETRIOID
CLEAR CELL (MESONEPHROID)
BRENNET
GERM CELL TUMORS
DERMOID CYST
MALIGNANT
SOLID TERATOMA
NON-GESTATIONAL CHORIOCARCINOMA
YOLK SAC TUMOR
DYSGERMINOMA
GERM CORD (GONADAL STROMAL) TUMORS
FIBROMA (MEIG'S SYNDROME)
ANDROGEN SECRETING TUMOR
GRANULOSA-THECA CELL TUMORS
SECONDARY

OVARIAN NEOPLASM

NON-NEOPLASTIC FUNCTIONAL CYST

FOLLICULAR CYST	CORPUS LUTEAL CYST	GRANULOSA-THECA LUTEIN CYST	POLYCYSTIC OVARY
- Usually less than 5 cm - Benign and asymptomatic - Thin wall, contain clear fluid - Rescan in 4 weeks - If enlarge or symptomatic, consider surgery	- Excessive bleeding into corpus luteum - Cyst filled with blood - Delayed period + pain - Usually the following period is heavy	- in molar pregnancy or part of hyperstimulation syndrome - Due to excessive gonadotrophin	ENDOMETRIOTIC CYST

PRIMARY OVARIAN TUMORS

A] EPITHELIAL

- Benign
- Borderline:

- Epithelial tumors with no invasion of basement membrane
- 15% of epithelial tumors, mostly serous and stage 1 (70-85%).
- 10 year survival is 95%. Late recurrence.
- Extensive histological sectioning is essential to exclude invasion.

- Malignant

MUCINOUS	SEROUS	ENDOMETRIOD	BRENNER	CLEAR CELL
- Large tumors. Multilocular filled with mucin - If ruptured → pseudomyxoma peritonei	- Most common - Contain clear fluid - Often bilateral. Around age of menopause - Malignant type is the commonest ovarian cancer	- Few cases arise in endometriosis - 30% coexist with primary endometrial cancer	- Usually benign.occur in reproductive life - May be associated with endometrial hyperplasia - May coexist with mucinous cystadenoma	@MESONEPHROID - Associated with endometriosis in 25% - Worst prognosis

<table>
<tr><td colspan="5">OVARIAN NEOPLASM</td></tr>
<tr><td colspan="5">PRIMARY OVARIAN TUMORS</td></tr>
<tr><td colspan="5">B] GERM CELL TUMORS</td></tr>
<tr><td>BENIGN</td><td colspan="4">MALIGNANT</td></tr>
<tr><td>DERMOID CYST @ BENIGN CYSTIC TERATOMA</td><td colspan="4">Rare. 3% of ovarian cancers</td></tr>
<tr><td rowspan="2">- 25% of all ovarian neoplasm
- Contain tissue derived from two or more germ cell layers
- Unilocular cyst. May contain teeth, bone , cartilage, nerves, hair, thyroid,.. Tissues
- Almost always benign. Malignant changes may occur in any component
- Occur at any age. Peak is 20-30 years.
- Bilateral in 20%</td><td>SOLID TERATOMA</td><td>NON-GESTATIONAL CHORIOCARCINOMA</td><td>YOLK SAC @ ENDODERMAL SINUS</td><td>DYSGERMINOMA</td></tr>
<tr><td>Peak incidence in second decade</td><td>- Secrete HCG
- May be component of solid teratoma</td><td>- Highly malignant. Affect young age
- Partly solid. Secrete alpha feto-protein</td><td>- Most common. Highly malignant
- Usually spread by lymphatics
- Very radiosensitive
- Occur in young women. May arise in gonadal dysgenesis</td></tr>
<tr><td colspan="5">C] GERM CORD @ GONADAL STROMAL @ SEX CORD TUMORS</td></tr>
<tr><td>GRANULOSA THECA CELL TUMOR</td><td colspan="2">ANDROGEN-SECRETING TUMORS</td><td colspan="2">FIBROMA</td></tr>
<tr><td>- Moderate to large size
- Solid, as enlarge may have cystic spaces
- Yellow tinge on cut surface
- Thecoma is benign, but granulosa is malignant
- Occur at any age .50% postmenopausal
- Secrete estrogen
- Usually stage 1. Late recurrence</td><td colspan="2">- Androblastoma
- Sertoli-leydig
- Gynandroblastoma
Cause virilization</td><td colspan="2">- Solid tumor
- May be associated with meigs' syndrome
- Tend to have long pedicle</td></tr>
<tr><td colspan="5">SECONDARY @ METASTATIC OVARIAN TUMORS</td></tr>
<tr><td colspan="5">● Always bilateral. From mucin secreting tumors, stomach and colon (Krukenberg tumors)
● May be secondary to breast</td></tr>
</table>

MALIGNANT EPITHELIAL OVARIAN TUMORS			
INTRODUCTION	ETIOLOGY	PRESENTATION	PHYSICAL FINDINGS
- Wide variety of tumors - 25% of female genital tract tumors - In U.K, the most common pelvic cancer - Worst prognosis of all female genital tract cancers - Life time risk is 1% - Spread by local spread, lymphatic and rarely by blood	Risk Factors: Nulliparity, Family history, Fertility drugs Protective Factors: Number of pregnancies, OCCP, Tubal ligation.	● Silent disease – 75% present at advanced stage ● Symptoms of abdominal involvement ● Symptoms of distant metastases ● General malaise, weight loss ● Hormonal production	Benign: - Usually mobile. unless large or complicated - Dermoid cyst anterior to bladder • Malignant: - Bilateral - Ascites - Hard deposit in pelvis - Leg edema - Signs of bowel obstruction of ureteric obstr

COMPLICATION	OVARIAN TUMOR IN PREGNANCY	INVESTIGATION
• Torsion - common with dermoid/fibroma - Severe abdominal pain/vomitting • Rupture • Haemorrhage • Impaction • infection	• Found incidentally • Corpus luteal/dermoid • 2% are malignant • If discover early and persist , surgery around 16 weeks • If complicated, operate immediately	• Uss /CT scan • Tumor markers(ca125,CEA, HCG,alpha FP • Urea and electrolyte • LFT • Chest X ray • Ascitic tap • Calculate **risk malignancy index.**

FIGO STAGING

Stage 1	Growth limited to one or both ovaries
Stage 2	Growth limited to one or both ovaries with pelvic extension
Stage 3	Tumor involving one/both ovaries with peritoneal implants outside pelvis/positive retroperitoneal or inguinal nodes.
Stage 4	Growth involving one or both ovaries with distant mets.

RISK MALIGNANCY INDEX

- CA 125 estimation
- Menopausal status

  pre menopausal score = 1

  post menopausal score= 3
- Ultrasound score

Multi locular, solid areas, bilateral, ascitis, intra ab mets.

if 0 or 1 score = 1

if 2-5 score= 3

RMI = CA125 x M x U

MALIGNANT EPITHELIAL OVARIAN TUMORS			
MANAGEMENT			
SURGICAL			
PRIMARY	INTERVAL DEBULKING	SECOND LOOK SURGERY	PALLIATIVE SURGERY
- Primary cytoreduction - TAH, BSO, OMETECTOMY, WASHINGS, BOWEL SURGERY - Optimal debulking: less than 2 cm residual tumors - Staging once histology is available - If confined to ovary and young age, conservative surgery	- Alternative to primary surgery medically unfit large ascitis severe malnutrition - 3 cycles of chemotherapy –surgery – 3 more cycles of chemotherapy - Aim : to improve patient condition less extensive surgery to achieve optimal debulking - May improve survival	- Assess response to chemotherapy - Plan future management - Only in research context.	- Removal of intestinal obstruction - Survival is very poor - Quality of life considerations

CHEMOTHERAPY	FOLLOW UP	SCREENING
Indication – stage 1c and above • Platinium based - Taxol - 6 cycles at 3 weekly intervals - Monitoring: examination CA125 FBC, U&E	How aggressive?. Three monthly for one year then six monthly then yearly History, examination and CA125 Imaging if recurrence is suspected clinically or by CA125	Life time risk is 1% 5% of tumors are genetic History of breast cancer increases risk by factor of 2 History of ca ovary increases the risk by factor of 3 One first degree relative affected: risk 2.7% 2 first degree relatives affected : risk is 13% ● If BRCA1 mutation carrier : risk is 50% ● Problems : - no pre-cancerous stage - unknown natural course ● TVS AND CA125 ON YEARLY BASIS ● ONGOING STUDY TO EVALUATE THIS.

PREINVASIVE CERVICAL DISEASE

DYSPLASIA

Def: Lesion in which part of the epithelium is replaced by cells showing varying degree of atypia.

Cervical Intraepithelial Neoplasia (CIN) System

Intraepithelial dysplastic atypia occurring within the metaplastic epithelium of the transformation zone.

Mild dysplasia	Moder8 dysplasia	Severe dysplasia	CIN	Cancer
CIN 1	CIN 2	CIN 3		

BETHESDA SYSTEM

ACSUS	LSIL	HSIL	CANCER

OSCJ – Original squamocolumnar junction.
NSCJ – New squamocolumnar junction
TZ – Transfomation zone – area between OSCJ & NSCJ.

OVERVIEW

Cervical neoplasia originates within TZ.
Low risk HPV (types 6 & 11) are associated with low-grade cervical lesions (condyloma acuminata and CIN1)
High risk HPV (type 16, 18, 31, 33 or 35) associated with high-grade cervical lesion (CIN2,3 and Cancer).
HPV type 16 is the type universally detected with the greatest frequency in high grade lesion & cervical ca. 50% SCC, 30% adenoca, & >80% preinvasive lesions.
AT least 35% pt with CIN3 will dev invasive ca within 10yrs , whereas lower grades may spontaneously regress.

RISK FACTORS

- Persistent HPV infxn of high risk types.
- Young age at first coitus
- Multiple sexual partners. Sex partner with multiple sex partners.
- Young age at first pregnancy.
- Multiparity.
- Low socioeco status
- Smoking & OCP
- Genital warts
- Exogenous / endogenous immunosuppresion

SCREENING

PAP SMEAR

Both the end cervical canal and the ectocervix should be sampled when taking the Pap smear
• The false negative rate for Pap smear for high grade lesions is 20%
• New automated liquid based slide preparation systems to decrease the false negative rate

COLPOSCOPY

Stereoscopic binocular microscope of low magnification, usually 10x to 40x.

Indications for colposcopy:
- Abnormal cervical smear
- Abnormal findings on adjunctive screening tests (HPV testing and cervicography)
-If the cervix is clinically abnormal or suspicious on naked eye exam.
- Unexplained IMB or PCB
- Persistent vaginal discharge
- Personal history of in utero DES exposure, vulvar or vaginal neoplasia.

Before Colposcopy

Complete hx and general exam.
• A clinical and speculum examination of the cervix, vagina and vulva.
• A 3% to 5%acetic acid solution is liberally applied to the cervix using soaked swap
- The abnormal findings are acetowhite epithelium & abnormal vascular patterns (mosaicism and punctuation)
• Lugol's iodine application to the cervix is called shiller's test
-Normal ectocervix and vaginal squamous epithelium contains glycogen and stains mahogany-brown
• Normal columnar and squamous metaplasia and neoplastic epithelium do not contain glycogen, and appear mustard yellow
• Satisfactory Colposcopic Examination: If the new SCJ & entire TZ are seen.

EVALUATION FOR ABNORMAL PAP SMEAR
• Any patient with a grossly abnormal cervix should have a punch biopsy performed regardless of the results of Pap smear • Patients with ASCUS found in their smear may have a repeat smear in 6 months or HPV testing • About 6-10% of patients with an ASCUS smear will have high-grade CIN on colposcopy, 90% of these can be detected by HPV testing for high-risk types • The colposcopic hallmark of CIN is an area of sharply delineated acetowhite epitheilum, or/and abnormal vascular pattern: punctuation and mosaicism • Micro invasive carcinoma: extremely irregular puncate and mosaic patterns are found. • If colposcopic examination is satisfactory, punch biopsy from the suspicious area with end cervical curettage specimen. • Diagnostic cone biopsy of the cervix is indicated if: - colposcopic examination is unsatisfactory - Endocervical curettings show a high-grade lesion - Pap smear shows a high-grade lesion that is not confirmed on punch biopsy - Pap smear indicates Aden carcinoma in situ - Microinvasion is present on punch biopsy
TREATMENT OF ABNORMAL INTRAEPITHELIAL NEOPLASIA CIN
• Low Grade Lesions (CIN1) - Repeat smear in 6 month interval until normal then back to the normal screening program. • High grade lesions (CIN 2,3): 1. Loop Excision of The Transformation Zone (LLETZ), relatively cheap, it can be performed on an outpatient basis under local anesthesia, and tissue is obtained for histologic evaluation. 2. LASER, destruction of the TZ by CO_2 laser, ablation can be performed as an outpatient procedure with local anaesthesia, expensive. 3. Cryosurgery, relatively painless outpatient procedure without anaesthesia, cheap, high failure rate for large lesions, copious vaginal discharge for several weeks. 4. Electrocoagulation, Requires general anesthesia, cervical stenosis may occur, success rates up to 97%. 5. Cervical conization: (cold knife or laser) -mainly diagnostic but it may be used for treatment, cure rates are as high as with hysterectomy for high grade lesions. -Major complications: Bleeding, infection, cervical stenosis and incompetence. • Simple hysterectomy is rarely necessary, it may be applicable when sterilization is desired in a patient with CIN III or when there is concomitant uterine or adnexal disease.

CERVICAL CANCER			
INTRODUCTION	SYMPTOMS	FINDINGS	INVESTIGATIONS
Worldwide, cervical ca is the most common cause of cancer death in women. Mean age for cervical ca is 51.4yrs, with the number of pt evenly divided between age groups 30-39 and 60-69. Most common type is SCC (80%), adenocarcinoma & adenoquamous account for 20-25%, others are rare. PATTERN OF SPREAD 1. Direct invasion into cervical stroma, corpus, vagina and parametrium. 2. Lympathic permeation & mets. 3. Hematogenous dissemination.	Abnormal vaginal bleeding is the most common presenting sx. Postcoital bleeding in sexually active women, IMB, PMB. Asymptomatic until quite advanced in women who are not sexually active. Persistent vaginal discharge, pelvic pain, leg swelling & urinary frequency are usually seen in advanced disease. Vesico-vaginal & rectovaginal sx.	Usually normal general exam. In advanced disease, enlarged inguinal or supraclavicular LN, edema of the legs, ascites, pleural effusion, hepatomegaly. Pap smear may be normal in up to 50% of cases (false negative) Pelvic exam in early disease may be normal, esp if lesion is endocervical. Visible disease may be ulcerative, exophytc or necrotic.	CBC, LFT, KFT CXR, Pelvi-abdominal CT Bx of lesion Cystoscopy & proctoscopy for clinical staging. PET scan to delineate the extent of disease at the primary site and in LN.

TREATMENT	
Stage IA	IA1 – Total abdominal / vaginal hysterectomy. - Cone bx alone may suffice if pt wants to preserve fertility, as long as cone margins free from disease & endocervical curetting -ve. IA2 – Modified radical hysterectomy and pelvic LN dissection. - If wants childbearing, large cone biopsy or radical trachelectomy & pelvic LN dissection are offered.
Stage IB	a) Radical Hysterectomy & Bilateral pelvic lymphadenectomy – Removes uterus, adjacent portions of vagina, cardinal ligaments, uterosacral ligaments & bladder pillars. Spares ovaries, can surgically stage, prevent chronic radiation. Most common complication: Bladder dysfunction, 1-2% permanent. Most serious complication: ureteric fistula or stricture 1-2%. Lower limb lymphoedema 15-20%. b) Radiation Therapy – Begins by external radiation to shrink central tumor & cavitary lesion. Also done postop for pt with LN mets, or inadequate surgical margins. Addition of chemo to radiotherapy improves survival.
Stage II	IIA with minimal involvement of vaginal fornix - Radical surgery or chemo radiation. IIA – IVA – Pelvic chemo is the rx of choice.
Stage IVB	Palliative radio or chemotherapy.
Recurrent or Mets	Chemotherapy – Limited effectiveness. Most active drug is cisplastin. Pelvic exenteration – Removal of pelvic viscera (uterus, tubes, ovaries, bladder, rectum) - For pt who have central recurrence following irradiation. Radiotherapy if initial disease treated by surgery only.

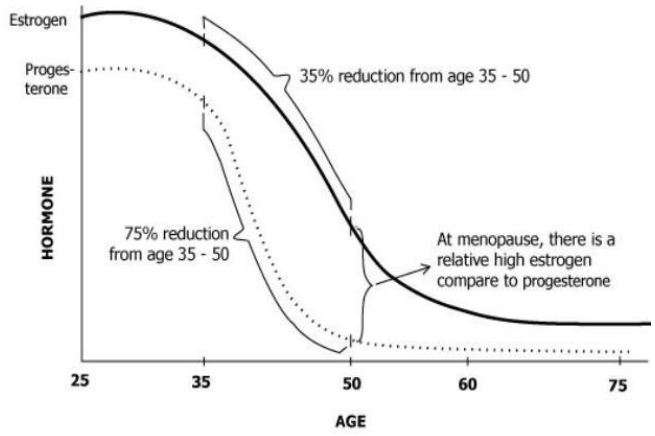
PROGNOSIS
Directly related to clinical staging. With higher stage, nodal mets escalate, & 5yr survival diminishes.

COMPLICATIONS OF RADIOTHERAPY	
ACUTE	CHRONIC
• Acute cystitis – Hematuria, urgency, frequency. • Proctosigmoiditis – Tenesmus, diarrhea, passage of blood & mucus in stool. • Enteritis – Nausea, vomit, diarrhea, colicky ab pain. • BM depression.	• Radiation enteropathy: Proctosigmoiditis (pelvic pain, tenesmus, diarrhea, rectal bleeding), ulceration (rectal bleeding & tenesmus), rectovaginal fistula (stool thru vagina), Rectum or sigmoid stenosis (progressive large bowel obstr), Small bowel injury (cramping ab pain, vomit, diarrhea) • Vaginal vault necrosis - Severe pain in vaginal vault & profuse discharge. • Urologic injury: Hemorrhagic cystitis, Vesicovaginal & Ureterovaginal fistula (constant urine leakage), Ureteric stenosis (hydronephrosis)

CLIMACTERIC (MENOPAUSE)

Permanent cessation of menstruation at the end of reproductive life due to loss of ovarian follicular activity
-determined retrospectively after a woman has experienced 12 months of amenorrhea without any other obvious pathological or physiological cause

How menopause occurs?
As a result of complete, or near complete, ovarian follicular depletion, with resulting
1- hypoestrogenemia
2- high FSH concentrations
-Physiologically, menopause occurs when the ovaries are totally depleted of eggs and no amount of stimulation from the regulating hormones can force them to work.
→ With aging, we have decrease in number of ovarian follicles and ovulation
Ovulation fails, no corpus luteum forms and no progesterone is secreted by the ovary.
As a result, there is a fall in Inhibin production by the ovaries and a resultant increase in FSH levels.
Later, graffian follicle fails to develop, estrogenic activity decreases and endometrial atrophy leading to amenorrhea.
This insufficient estrogen levels causing the usual menopausal symptoms and changes.



Changes of estrogen and progesterone level

Physiological Menopause	Pathological Menopause
Normal decline in ovarian function due to ageing begins in most women between ages 45 and 55 on average 51 and result in infrequent ovulation, decreased menstrual function and eventually cessation of menstruation.	Gradual or abrupt cessation of menstruation before 40 years occur idiopathically in about 5% of women in USA.

Phases of menopause :

<table>
<tr><th colspan="3">Perimenopause</th></tr>
<tr><td>The initial stage of the menopausal transition is referred to as the “early transition”
Women experience a change in intermenstrual interval. It may increase to 40-50 days.
Change in bleeding pattern
Accompanied by hormonal fluctuations and a variety of symptoms
Early follicular phase FSH levels are high but variable
The more irregular cycles are accompanied by more dramatic fluctuations in FSH and estradiol</td><td>With time, more dramatic menstrual cycle changes with skipped cycles, episodes of amenorrhea, and an increasing frequency of anovulatory cycles.
This stage is referred to as the “late transition” and typically lasts for 1-3 years before final menstrual period
Of course not all women will follow a typical bleeding pattern</td><td>Random serum sample may demonstrate high FSH and low estradiol concentrations consistent with menopause
FSH >25 IU/L is characteristic of the late menopausal transition, but not routinely recommended since it is variable
Some women do experience heavy or prolonged bleeding (due to anovulatory cycles and prolonged exposure to unopposed estrogen,obesity and uterine fibroids)</td></tr>
<tr><td>Menopause
It is the end of menstruation. The age of menopause ranges between 45 – 55 years, average being 51 years.</td><td colspan="2">Postmenopause : It is the time after which a women has experienced 12 consecutive month of amenorrhea
> early up to 5 years
> late after 5 years till death</td></tr>
<tr><td colspan="3">In menopause and postmenopause : The increase in serum FSH becomes sustained near the final menstrual period, then increases over several years to levels in the 70 - 100 IU/L range, followed by a decline with increasing age</td></tr>
</table>

Types of menopause :

Premature menopause (<40 y/o)	-Women in this age group with a change in intermenstrual interval and menopausal symptoms should not be diagnosed with either the menopausal transition or menopause. -Think of primary ovarian insufficiency (premature ovarian failure). -Other causes include abnormal karyotypes involving X chromosome, the carrier state of fragile X syndrome, galactosemia, and autoimmune disorders that may cause failure of a number of other endocrine organs.
Early menopause (40-45 y/o)	The menopausal age is directly associated with smoking and genetic disposition. -Smoking induces premature menopause. -There is also a tendency for women who never had children and for those with more regular cycles to have an earlier age of menopause -If a family history of early menopause (constitutional) -If type 1 DM
Delayed menopause (> 60 y/o)	-Due to good health and better nutrition. -Also seen in women with uterine fibroids, women with high risk of endometrial cancer
Surgical menopause (any age before natural menopause occurs)	- Due to both ovaries are surgically removed . - Symptoms are generally more intense than when menopause occurs naturally. -Induced menopause due to abrupt cutoff ovarian hormones, causes the sudden onset of hot flashes and other menopausal symptoms such as dry vagina and a decline in sex drive
Medical meopause	- permanent damage to both ovaries as in chemotherapy or radiotherapy - temporary as in GnRH treatment in endometriosis

SPECIAL SITUATIONS ☺

-Women with underlying menstrual cycle disorders, such as (PCOS) or hypothalamic amenorrhea

-**Women taking oral contraceptives**, difficult to determine if menopause has occurred or not

→stop the pills and measure serum FSH 2-4 weeks later, level ≥ 25 IU/L likely entered the menopausal transition

-**Post-hysterectomy or endometrial ablation**

→Look for menopausal symptoms and biochemical data

- FSH ≥25 IU/L menopausal transition
- FSH 70-100 IU/L postmenopause

DDX :

1)Hyperthyroidism should always be considered since present with: irregular menses, sweats, mood changes

2)Pregnancy

3) Hyper prolactinemia

Clinical manifestations and symptoms (due to estrogen loss) :

Short term (0-5 years)	hot flushes ,insomnia , labile mood , anxiety , loss of concentration, poor memory, joint aches , dry itchy skin , hair changes , decreased sexual desire
	HOT FLUSHES **-pathophysiology :** low estrogen levels → positive feedback stimulation on hypothalamus → stimulates GnRH release → affects adjacent thermoregulatory centers in hypothalamus → downshift of the set-point of this centre such that there is a frequent central misapprehension that body temperature is too high→ cutaneous vasodilatation and heat loss -Typically begin as sudden sensation of heat centered in upper chest and face then becomes generalized, associated with sweating dizziness and palpitation. -Usually occur several times per day, more frequently at night and it leads to night sweats , and as a consequence the woman experience sleep disturbance, fatigue and irritability and can affect mood , concentration and libido -Typically they start to occur a year or two before the menopause, peaking in frequency and intensity in the first year after menopause and on average lasting for up to 5 years. However, they may continue for 20 or more years
	SLEEP DISTURBANCES A distressing feature of hot flashes is that they are often associated with arousal from sleep. In addition, primary sleep disorders are common in this population, even in the absence of hot flashes.
	BREAST PAIN Common in the early menopausal transition, but begin to diminish in the late menopausal transition
	MENSTRUAL MIGRAINES Cluster around the onset of each menstrual period. In many women, these headaches worsen in frequency and intensity during the menopausal transition.
	SKIN CHANGES The collagen content of the skin and bones is reduced by estrogen deficiency. Decreased cutaneous collagen may lead to increased aging and wrinkling of the skin. The collagen changes may be minimized with estrogen.
	JOINT PAIN The prevalence is not known, but some women experience diffuse joint pain during the menopausal transition and postmenopausal period
	BALANCE May be due to central effect of estrogen deficiency.Problems with balance may play a major role in the incidence of forearm fractures in women. The incidence of Colles' fractures increases markedly in women at age 50 but remains stable in men up to the age of 80. A mechanism other than osteoporosis must be invoked to explain this observation because osteoporosis occurs gradually.
Intermediate (3-10 years)	(physical changes) : urogenital atrophy , vaginal dryness and soreness , stress (urinary)incontinence, and recurrent UTI, skin collagen loss , urogenital prolapse , dyspareunia .

	GENITOURINARY PROBLEMS/EFFECTS ON VAGINA (Normally vagina is sensitive to estrogen and it responds by producing thick, moist epithelium with acidic secretion (PH about 4), in case of estrogen deficiency : there will be thin dry epithelium with alkaline PH) -Increase in pH and vaginal atrophy → impaired protection against vaginal, UTI -Vaginal atrophy → loss of the normal architecture within the vaginal epithelium, reducing its secretions and elasticity → more prone to trauma, dryness, spontaneous bleeding and infection -Clinically vaginal atrophy (atrophic vaginitis) manifests as vaginal dryness , itching , dyspareunia, vaginal pain , discharge (on exam, the vagina typically appears pale, with lack of the normal rugae and often has visible blood vessels or petechial hemorrhages) -Distal urethra and trigone of the bladder (similar embryological origin as the lower vagina) so low estrogen production after the menopause → atrophy of the superficial and intermediate layers of the urethral epithelium → subsequent atrophic urethritis, diminished urethral mucosal seal, loss of compliance, and irritation; these changes predispose to both stress and urge urinary incontinence, urinary frequency and dysuria , in the absence of proven infection , sometimes referred to as the "urethral syndrome " , this responds well to local estrogen administrations . -Hormonal therapy markedly improves atrophic vaginitis and urethral symptoms, but cannot prevent adequately or treat urinary incontinence . -Loss of estrogen → pelvic floor dysfunction → weakening of tissues and ligaments which may already be damaged by childbirth→ increased incidence of genital prolapse and stress urinary incontinence -Estrogen deficiency → decrease in blood flow to the vagina and vulva → decreased vaginal lubrication and sexual dysfunction in menopausal women -This neuropathy appears to be completely reversible with estrogen replacement therapy - The cervix also can become atrophied and flush with the top of the vaginal vault. - The elasticity of the vaginal wall may decrease and the entire vagina can become shorter or narrower.
Long term (>10 years)	(diseases) : osteoporosis ,dementia (of Alzheimer 's type),cardiovascular disease and colon cancer (unclear relationship)
	OSTEOPOROSIS (skeletal disorder characterized by compromised bone strength predisposing to an increased risk of fracture) -Estrogen acts as an antiresorptive agent on trabecular bone and the fall in its level will result in decreased bone density → increased risk of osteoporotic fracture -Early clinical sign is loss of height greater than 1.5 inches due to vertebral compression (nontraumatic) fractures which may be accomplished by acute or chronic back pain. - Other effect : hip fracture and distal radius fracture from minor or moderate trauma . - Walking and weight bearing exercises help to increase bone mineral mass and reduce the risk for fracture causing fall - FRAX model screens postmenopausal women for risk factors , one of them age of menopause (premature menopause

	being particularly high risk) →Those deemed as increased risk undergo DEXA scan and those with low bone density offered preventative treatments . - For prevention : bisphosphonates are the principle class of drug used. Alternatives include strontium and raloxifen (a SERM). However,all these can have significant side effects and should only be prescribed to women over 60 who are at high risk of osteoporosis. -Para –thyroid hormone is reserved for women with a very high risk
	CARDIOVASCULAR DISEASE - Early menopause without additional estrogen is associated with 2-4 fold increased risk in CHD, as estrogen has a protective influence against CHD . - Menopause is associated with a number of metabolic changes, such as a rise in total and LDL cholesterol and a fall in HDL cholesterol .These changes are reversed by estrogen. - Estrogen also has a direct effect on the vessel wall , and loss of estrogen is associated with vasoconstriction and atherogenesis , so estrogen administration will stimulate vasodilatation via nitric oxide

Treatment

[HORMONE REPLACEMENT THERAPY]

for troublesome menopausal symptoms and simply acts by replacing the hormones that are normally produced by the human ovary at physiological levels

**The MHRA recommend taking 'the minimum effective dose' of HRT for the 'shortest duration' without defining any specific length of time, and others said 5 years.

** Once stabilized on treatment, women should be reviewed every six months or so. Their individual risk of VTE, stroke and breast cancer should be appraised regularly and balanced against the benefits they are gaining from the treatment.

<table>
<tr><td>TYPE OF HRT</td><td colspan="2">1)ESTROGEN
-Non-oral routes avoid the first-pass effect → reducing the impact on various metabolic parameters, such as the haemostatic and coagulation system, so it's a better option in women with a personal or relevant family history of venous thrombosis or known liver abnormalities
-Subcutaneous implants tend to be reserved for women who do not respond to standard levels of oestrogen.
→They tend to be used more in younger women who have had a hysterectomy and their ovaries removed.
-Implants also allow the addition of testosterone (if there is decreased libido)</td><td>2)PROGESTOGENS
(synthetic progesterones) are added to mimic the normal menstrual cycle and reduce the risk of endometrial hyperplasia and cancer associated with the prolonged use of unopposed oestrogen.
-can either be given cyclically, mimicking the natural 28-day cycle and resulting in a regular withdrawal bleed or continuously to prevent any bleeding, so-called 'no bleed' treatment</td><td>3) COC
4) TESTOSTERONE (together with estrogens)</td></tr>
<tr><td>BENEFITS</td><td colspan="2">Estrogen :
-relieving hot flushes with improvement usually noted within 4–6 weeks.
-symptoms of vaginal and urogenital atrophy respond well to systemic or vaginal oestrogens. Vaginal or topical oestrogen preparations do not have any significant systemic activity so can usually be given safely in women in whom HRT is otherwise contraindicated.</td><td colspan="2">-prevention of postmenpausal bone loss and osteoporotic fractures at the spine and hip.
→ not first-line treatment for osteoporosis prevention but most appropriate treatment for osteoporosis prevention in women with premature ovarian failure under the age of 50 and for women in whom the standard osteoporosis treatments are not tolerated or are unsuitable.</td></tr>
<tr><td>INDICATION</td><td colspan="4">Postmenopausal hormone therapy is currently recommended for the short-term management of moderate to severe vasomotor flushes. Also in the abscence of contraindications , it's used in urogenital atrophy.
Long-term use for prevention of diseases like cardiovascular diseases and osteoporosis is no longer recommended.</td></tr>
<tr><td>RISKS</td><td>BREAST CANCER
-small risk in healthy postmenopausal women in their late 40s and 50s</td><td>ENDOMETRIAL CANCER
-increase risk in unopposed oestrogen replacement therapy (so all non-</td><td>OVARIAN CANCER
-suggest a small increase in risk with very long term (>10 years) treatment.
-this increase does not seem</td><td>VENOUS THROMBOEMBOLISM
-two-fold increase, with the highest risk occurring in the first year of use.
-increased in women who smoke, obese, have an underlying thrombophili(eg: Factor V Leiden), have previously suffered a VTE.</td></tr>
</table>

	in the first few years -if take oestrogen alone, that risk is probably even lower -multifactorial disease and overall other personal risk factors, such as family history, are likely to be more important predictive factors	hysterctomized women should also receive progestogen. -usually given cyclically to mimic the natural menstrual cycle. → gives a monthly withdrawal bleed, but once a woman is clearly postmenopausal, she should be switched to a continuous combined (no bleed) regimen	apparent with combined therapy.	-Transdermal HRT has less impact on haemostatic mechanisms and appears to be associated with a lower risk of VTE even in women with a thrombophilia
COMPLICATION	**ABSOLUTE:** -Suspected pregnancy -Breast cancer -Endometrial cancer -Active liver disease -Uncontrolled Hypertension -Known VTE -Known Thrombophilia -Otosclerosis	**RELATIVE :** Uninvestigated abnormal bleeding -Large uterine fibroids -Past history of benign breast disease -Past history of VTE -Chronis stable liver disease -Migraine with aura	**SIDE EFFECTS** ESTROGEN -fluid retention -nausea -headaches -breast enlargement -leg cramps -dyspepsia	PROGESTERONE -fluid retention -breast tenderness -headaches -acne -mood swings -depression -irritability -bloating -constipation -increased appetite

EXTRA INFO ☺

To give more benefit and reduce risk of HRT, a new generation of selective tissue receptor modulators are being developed which will have selective action against oestrogen, progesterone and testosterone receptors. Currently, raloxifene is the only SERM commercially available. It acts by locking into the oestrogen receptors, but as it does so its side chain deactivates one of the activation functions of the receptor. This specific action only occurs in certain tissues, such as the breast and endometrium. In other tissues, such as the skeleton,the side arm does not deactivate the receptor and raloxifene behaves like an oestrogen.

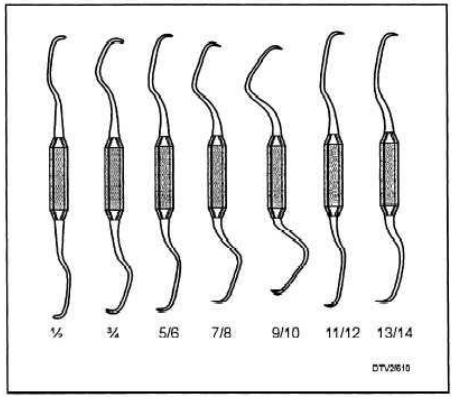
INSTRUMENTS

1) Sims Speculum.

Uses: for visualising fistulae (abnormal holes or connections) and prolapse (protrusion) of the rectum orbladder into the vagina

2) Bivalve speculum

Uses: Pelvic exams. To take Pap smear. To take cone & punch biopsy.



3) Curette

Dilation & curettage. Evacuation & curettage.