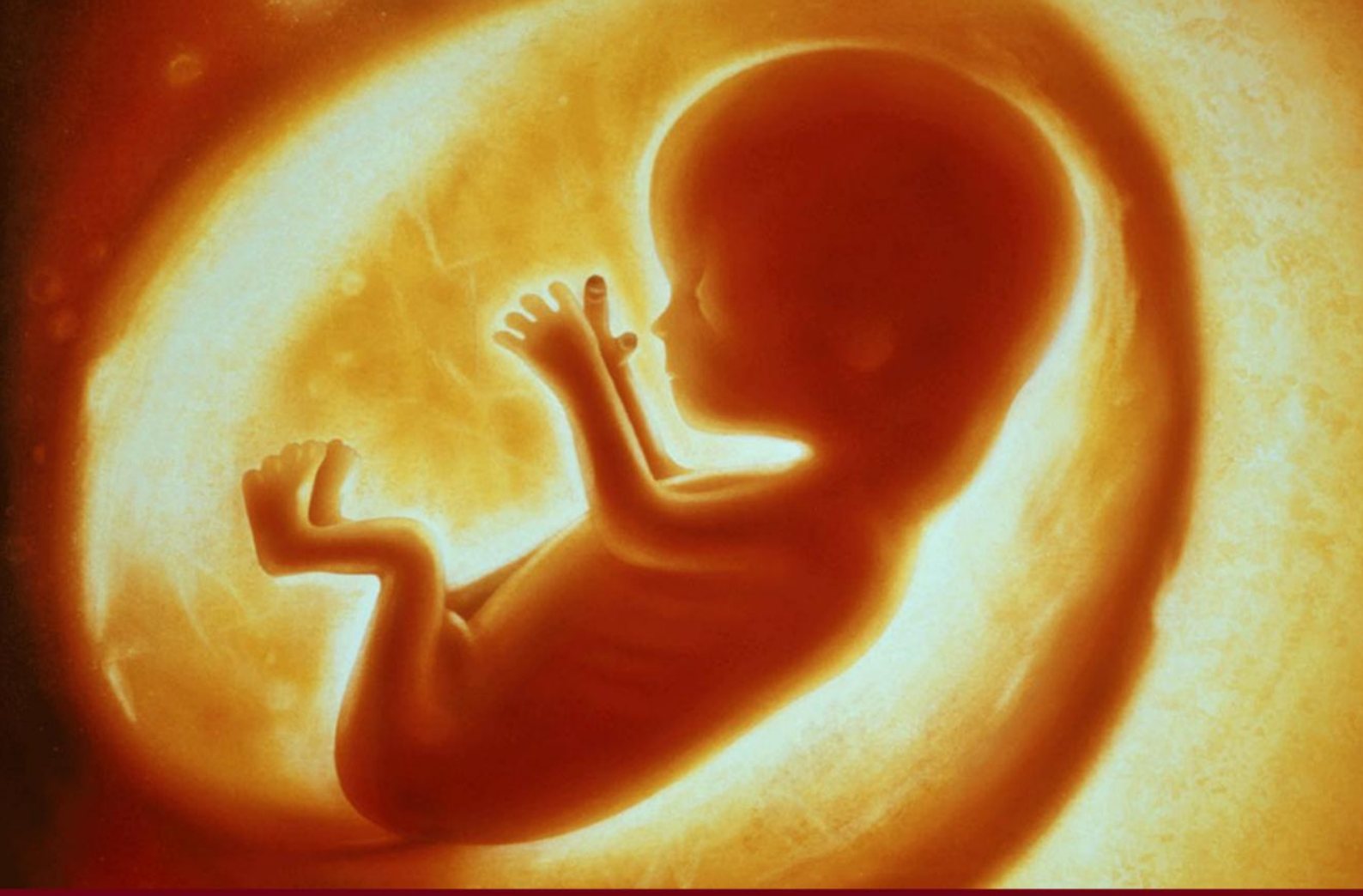


Obstetric and Gynecology

by: Ob/Gyn team 428

yusuf designs

بسم الله الرحمن الرحيم

تم بحمد الله ، بعد الجهد المتواصل . . . وعمل الفريق الدؤوب . . . والإصرار والعزيمة

كتابة هذه المذكرة وتنسيقها ومراجعتها .

حاولنا جاهدين على أن تكون شاملة ومرتبة ليسهل على الطالب فهمها وحفظها

وكانت مراجعنا كالآتي:

1-Broad review series Obstetrics and Gynecology 2nd edition
By: Elmar P. Sakala

2-Hacker and Moore's Essentials of Obstetrics and Gynecology 4th edition
By: N. F. Hacker, J. C. Gambone ,C. J. Hobel

3-Fundamentals of Obstetrics and Gynaecology, 8th edition
By: J.K. Oates, S. Abraham.

4-Doctors' lectures.

5- Lectures' and sessions' notes.

وبالنهاية نتمنى أن نكون قد وفقنا في عملنا ونلنا على استحسانكم ورضاكم

والتمسوا لنا العذر إن بدر منا أي تقصير

كل ما نريده. . . . دعوة في ظهر الغيب

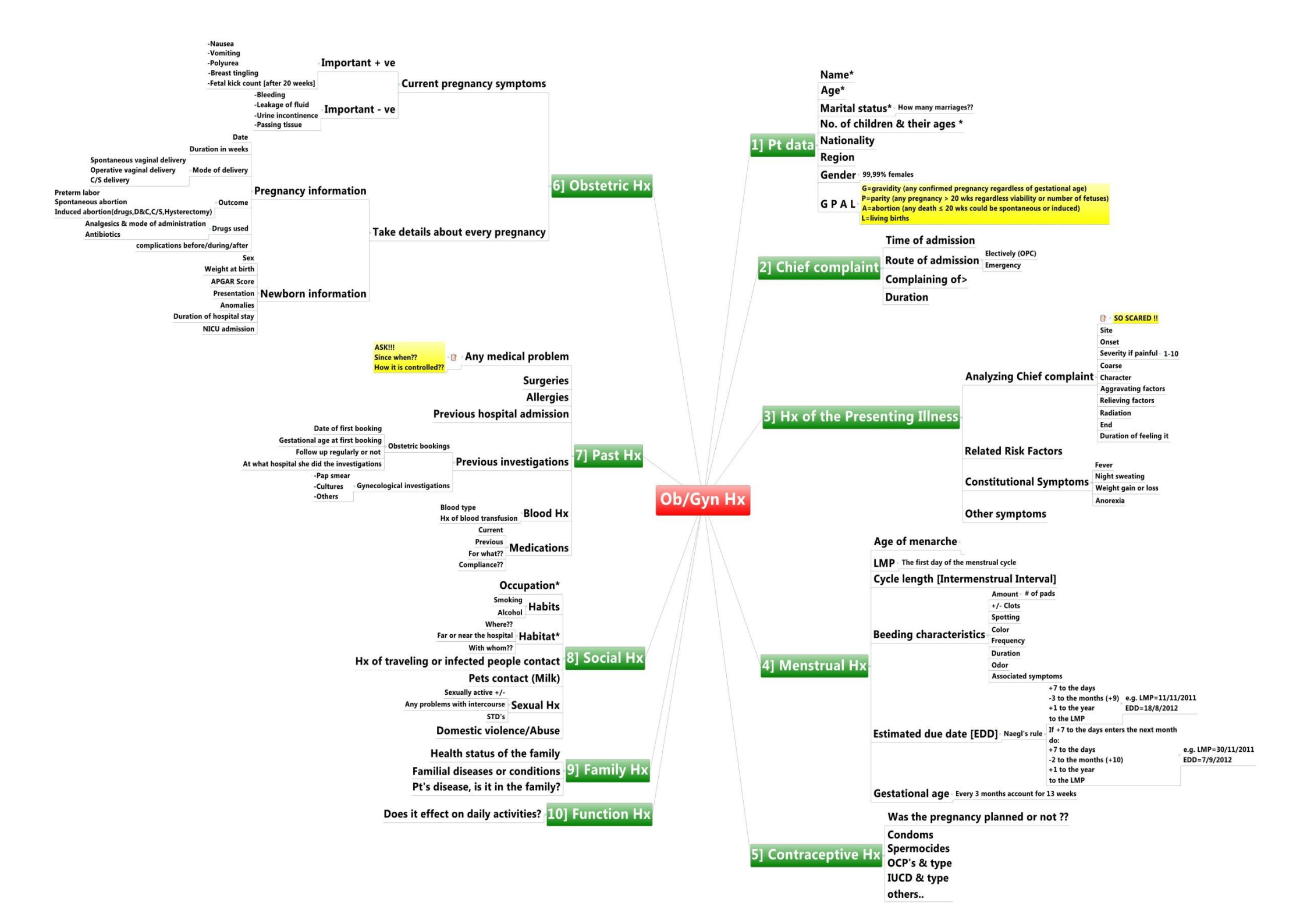
دراسة موفقة ونتائج مبشرة بإذن الله

*ملاحظة : الرجاء من الدفعات القادمة تطوير هذه المذكرة لكي تعم الفائدة.

Index

☒ **Part IV: Gynecology:**

☒ **Part V: Oncology:**


Ob/Gyn Hx
1] Pt data
Name*
Age*
Marital status*
How many marriages??
No. of children & their ages *
Nationality
Region
Gender
99,99% females
G P A L
G=gravidity (any confirmed pregnancy regardless of gestational age)
P=parity (any pregnancy > 20 wks regardless viability or number of fetuses)
A=abortion (any death ≤ 20 wks could be spontaneous or induced)
L=living births
2] Chief complaint
Time of admission
Route of admission
Electively (OPC)
Emergency
Complaining of>
Duration
3] Hx of the Presenting Illness
Analyzing Chief complaint
SO SCARED !!
Site
Onset
Severity if painful
1-10
Coarse
Character
Aggravating factors
Relieving factors
Radiation
End
Duration of feeling it
Related Risk Factors
Constitutional Symptoms
Fever
Night sweating
Weight gain or loss
Anorexia
Other symptoms
4] Menstrual Hx
Age of menarche
LMP
The first day of the menstrual cycle
Cycle length [Intermenstrual Interval]
Beeding characteristics
Amount
# of pads
+/- Clots
Spotting
Color
Frequency
Duration
Odor
Associated symptoms
Estimated due date [EDD]
Naegl's rule
+7 to the days
-3 to the months (+9)
+1 to the year
to the LMP
e.g. LMP=11/11/2011
EDD=18/8/2012
If +7 to the days enters the next month do:
+7 to the days
-2 to the months (+10)
+1 to the year
to the LMP
e.g. LMP=30/11/2011
EDD=7/9/2012
Gestational age
Every 3 months account for 13 weeks
5] Contraceptive Hx
Was the pregnancy planned or not ??
Condoms
Spermocides
OCP's & type
IUCD & type
others..
6] Obstetric Hx
Current pregnancy symptoms
Important + ve
-Nausea
-Vomiting
-Polyurea
-Breast tingling
-Fetal kick count [after 20 weeks]
Important - ve
-Bleeding
-Leakage of fluid
-Urine incontinence
-Passing tissue
Take details about every pregnancy
Pregnancy information
Date
Duration in weeks
Mode of delivery
Spontaneous vaginal delivery
Operative vaginal delivery
C/S delivery
Outcome
Preterm labor
Spontaneous abortion
Induced abortion(drugs,D&C,C/S,Hysterectomy)
Drugs used
Analgesics & mode of administration
Antibiotics
complications before/during/after
Newborn information
Sex
Weight at birth
APGAR Score
Presentation
Anomalies
Duration of hospital stay
NICU admission
7] Past Hx
Any medical problem
ASK!!!
Since when??
How it is controlled??
Surgeries
Allergies
Previous hospital admission
Previous investigations
Obstetric bookings
Date of first booking
Gestational age at first booking
Follow up regularly or not
At what hospital she did the investigations
Gynecological investigations
-Pap smear
-Cultures
-Others
Blood Hx
Blood type
Hx of blood transfusion
Medications
Current
Previous
For what??
Compliance??
8] Social Hx
Occupation*
Habits
Smoking
Alcohol
Habitat*
Where??
Far or near the hospital
With whom??
Hx of traveling or infected people contact
Pets contact (Milk)
Sexual Hx
Sexually active +/-
Any problems with intercourse
STD's
Domestic violence/Abuse
9] Family Hx
Health status of the family
Familial diseases or conditions
Pt's disease, is it in the family?
10] Function Hx
Does it effect on daily activities?

History Example

- Norah 36 years old , Saudi women , gravida 3 para 1 plus 1 at a gestational age of 38 weeks was admitted to antenatal care unit 2 days ago for Induction of labor because she has DM

- She had Booked for antenatal care at king Khalid university hospital at 15 weeks gestation and regularly attended the clinic. hemoglobin level at booking was 9.5 , her packed cell volume 25%, her platelets 320 X10^9 per liter , her blood group was AB positive , her blood pressure was 114/62 , her weight was 67.6 kg (BMI = 27.8) . Urinalysis was positive for sugar and negative for protein . Thyroid test was normal and rubella status was only positive for IGG antibody . Toxoplasmosis , HBV and other infection was negative and pap smear test was also negative . Ultrasound scan at booking revealed viable single active fetus with biparietal diameter of 48 mm (gestational age 15) , posterior upper placenta and normal amniotic fluid and no anomalies was detected .

- She was placed on IM insulin 3 doses daily , ferrous sulphate tablets 200mg three times daily, folic acid tablets 5mg daily and Ca^{++} tablet for the duration of pregnancy

- The women was referred to ophthalmologist and there was no abnormal detection

- In each antenatal care visit , fundal examination , urine dipstick , Blood pressure , weight , fetal heart and fetal movement has been done . Fundal examination was corresponding to gestational age , urine dipstick was negative to protein and glucose but sometimes positive to glucose , blood pressure in normal range , weight gain was normally increasing and the ultrasound reveal active viable fetus .

- At 34 weeks of gestation further investigation was done Biophysical profile was 8/8 and the CTG reveals 1 contraction /10 minute reactive and the heart rate 150 beat/minute and the ultrasound reveals single active viable fetus in longitudinal lie, cephalic presentation with normal breathing and normal amniotic fluid (AFI = 11.00cm) and the biparietal biometry was 86 mm which corresponding to 34 gestation age . Umbilical artery doppler reveals normal end diastolic waveform and Antibody screening coombs was negative

- At 35 weeks of gestational age , her doctor request stool culture because she has over abdominal distention and it was negative .

- The last antenatal care visit was in 5 days ago the weight was 78.6 and the blood pressure 115 /57
  ((it is important to mention the last investigation))

- The women denies bleeding , passing tissue , and leaking of fluid or urine during all time of pregnancy
- At the day of admission her doctor planned CBC , U & E , coagulation profile , LFT and cross matches

- Her HGB was 9.3 and platelet 320 X 10^9 , and the coagulation profile was normal (Pt : 13.4 Pt+INR = .99 APTT = 28.1)

- The last vaginal examination revealed The cervix was 2.5 cm length, and 2cm dilated. The fetal station was -3 And the fetal membranes were intact.

- **Obstetric Hx :**
    - 13 years ago , she delivered healthy normal boy in term without any complication before during or after delivery, his weight during delivery was 3.5 kg , the mode of delivery was vacuum and no I.C.U was needed . She got discharge from Military Hospital after 2 days of delivery
    - 2 years ago she had Induced abortion at 8 weeks of gestational age after heavy bleeding in KKUH

- **Menstrual Hx :**
    - age at menarche was 14 , LMP 14 -7 -1432 . The Menstrual cycle is regular about 6 days of menstruation every 28 days . The menstruation is mild (1 pad every 6 hour) which does not affect her daily activity and associated with mild pain and sometimes there is clotting and she denies intermenstrual bleeding

- **Oral Contraceptive Pill Hx :** the pregnancy was planned

- **Past Medical History :** she has DM 1 year and 6 months ago and she does not has any other chronic illnesses . Past surgery is negative . Past admission is negative . There was no history of allergy to drug , food or environment . There was no history of blood transfusion . There is no history of contact to infected person with varicella or other infection . There was no history of drinking unsterile milk . Past drug history before pregnancy include metformin and other hypoglycemic drug then she switched to insulin in current pregnancy but The DM was not controlled .

- **Social Hx :** she is housewife living in Riyadh near to hospital with her husband and she has one son from previous marriage and she is in second marriage 2 years ago . She never smoked or drank alcohol .

- **Family history :** Her father and her brother have DM

- **In summary :** Norah 36 years old with DM ,Saudi women , gravida 3 para 1 plus 1 at a gestational age of 38 weeks was admitted to antenatal care unit 2 days ago for Induction of labor .

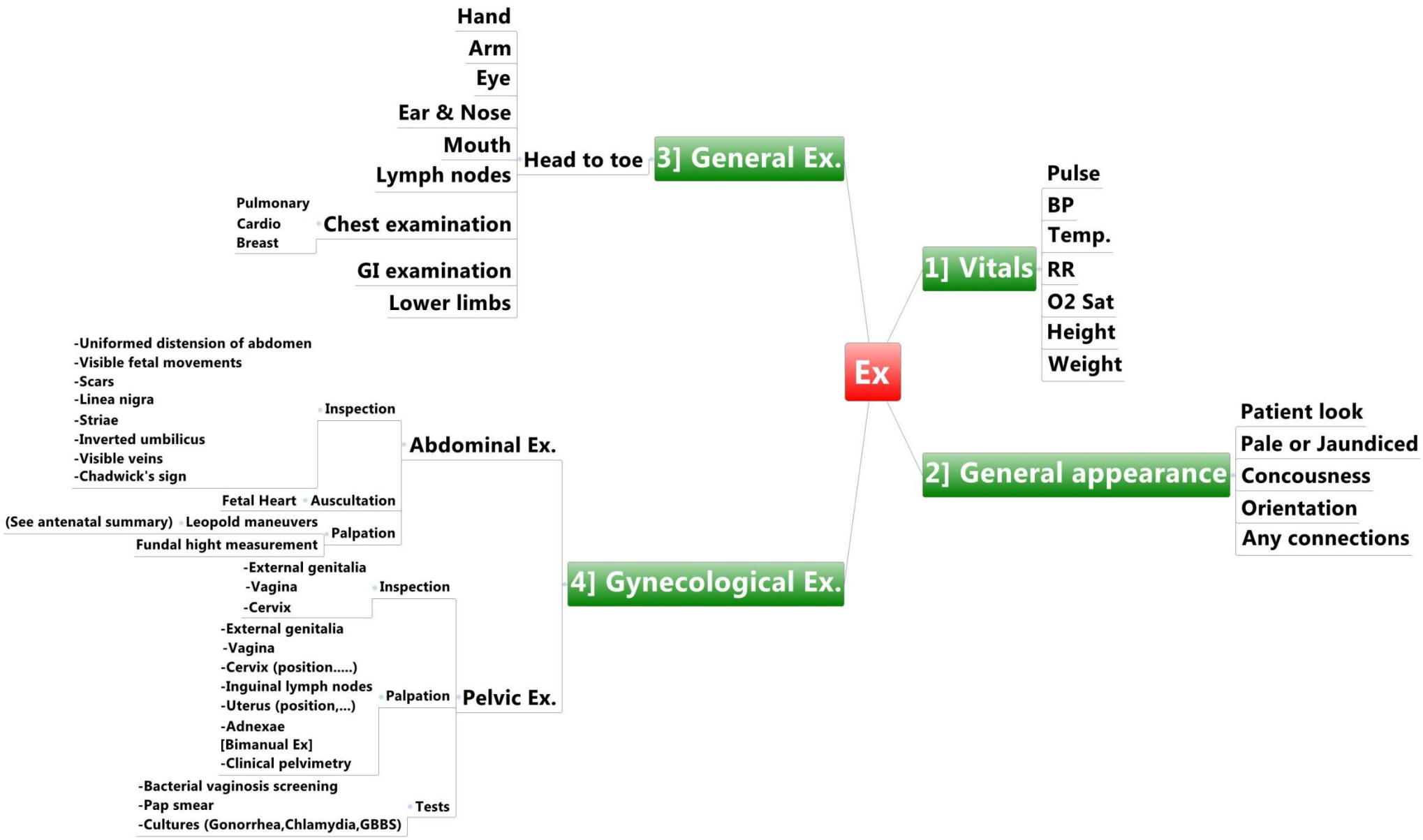
- **NB :** take the information from patient 7, computer(investigation of first booking) and from the file.

History taking for an infertile couples

[OSCE exam group B 428] [see infertility summary]

- **Personal & Social History**
    - Age – female partners.
    - Occupation especially the male – exposure to high temperature.
    - Chemical and radiation can affect sperm production.
    - History of travelling : Works away from home – affect frequency of sexual intercourse around the time of ovulation.
    - Ask about risk factors : Smoking – Alcohol and others .
    - Number of children.
- **Menstrual History**
    - Age of menarche and regularity of periods.
    - Irregular menstrual cycle, oligomenorrhea and amenorrhea are all suggestive of anovulation.
    - Amenorrhea – menopausal symptoms.
    - Weight loss or gain.
    - Symptoms of hyperprolactinaemia and hypothyroidism and endometriosis and PCOS .
- **Gynecological History**
    - Hx of PID.
    - Hx of Ovarian cancer.
    - Hx of Endometriosis.
    - Others.
- **Contraception**
    - Use of contraception pills and long acting progesterone followed by amenorrhea.
    - Use of long acting progesterone contraception followed by delay in the resumption of ovulation.
    - IUCD - ↑ risk of infection – young nulliparous leading to tubal disease.
- **Obstetric History**
    - Enquire about previous pregnancies, outcome.
    - Breast feeding and any sustained galactorrhoea.
    - Difficulties or treatment required prior to achieving a previous pregnancy.
- **Sexual History**
    - Frequency of sexual intercourse.
    - Ejaculatory dysfunction.
    - Protected or not protected.
    - Use of lubricant.
- **Family history :**
    - Diabetes, endometriosis, PCO.

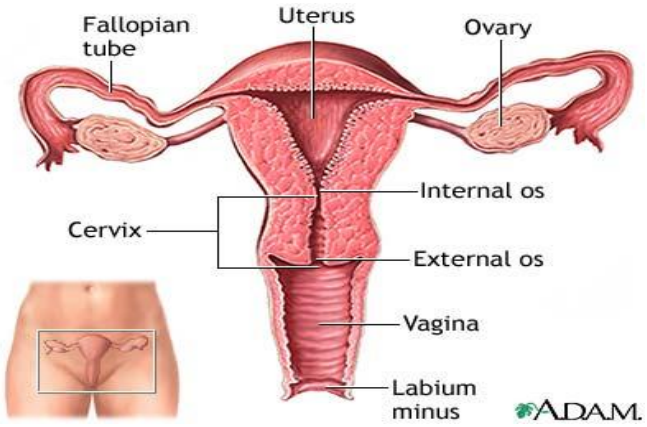
NB : the husband and wife both should come together in the infertility clinic


Ex
1] Vitals
Pulse
BP
Temp.
RR
O2 Sat
Height
Weight
2] General appearance
Patient look
Pale or Jaundiced
Concousness
Orientation
Any connections
3] General Ex.
Head to toe
Hand
Arm
Eye
Ear & Nose
Mouth
Lymph nodes
Chest examination
Pulmonary
Cardio
Breast
GI examination
Lower limbs
4] Gynecological Ex.
Abdominal Ex.
Inspection
-Uniformed distension of abdomen
-Visible fetal movements
-Scars
-Linea nigra
-Striae
-Inverted umbilicus
-Visible veins
-Chadwick's sign
Auscultation
Fetal Heart
Palpation
Leopold maneuvers
(See antenatal summary)
Fundal hight measurement
Pelvic Ex.
Inspection
-External genitalia
-Vagina
-Cervix
Palpation
-External genitalia
-Vagina
-Cervix (position.....)
-Inguinal lymph nodes
-Uterus (position,...)
-Adnexae
[Bimanual Ex]
-Clinical pelvimetry
Tests
-Bacterial vaginosis screening
-Pap smear
-Cultures (Gonorrhea,Chlamydia,GBBS)

The anatomy of the female reproductive tract

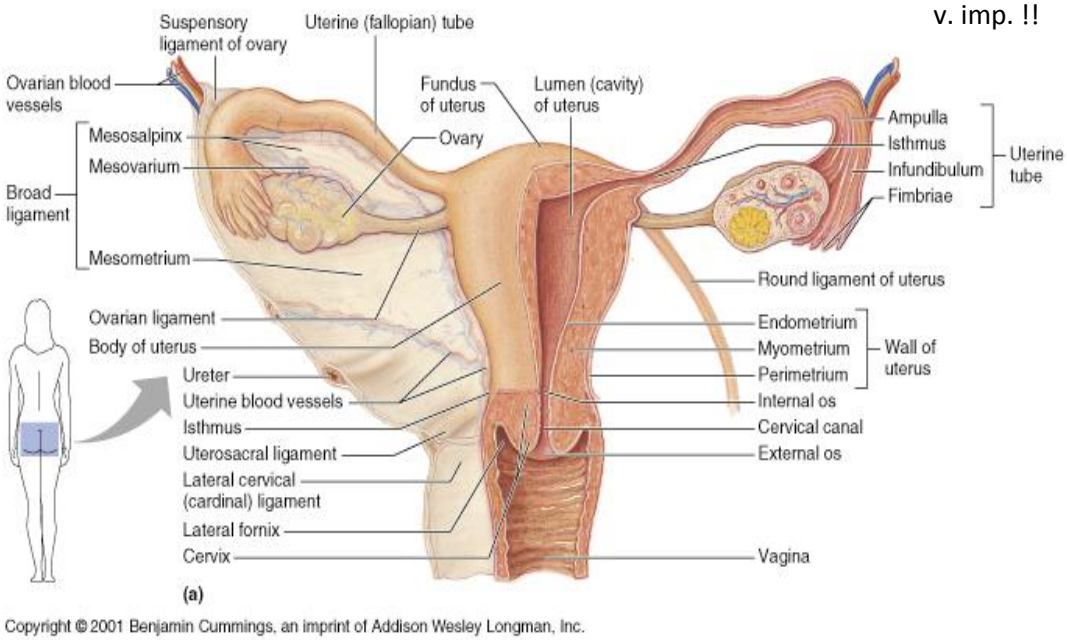
i. The uterus:

1. **Major structures:**
   a. Corpus.
   b. Isthmus.
   c. Cornu.
   d. Cervix.
2. **Layers of the corpus:**
   a. Mesometrium (serosa).
   b. Myometrium.
   c. Endometrium.
      1. zona functionalis.
      2. zona basalis.



3. **Ligaments:**
   a. **Cardinal:** containing u. blood supply and provide support.
   b. **Uterosacral:** provide support and neural innervation.
   c. Broad.
   d. Round.
   e. Uterovesical.
4. **Positions:** (anteverted: forward 50%, retroverted: backward 25%, midposition: 25%).
5. **Blood supply:** Uterine artery : branch of the hypogastric artery (internal iliac artery).
   Ovarian artery : Branch of the Aorta.
6. **Lymphatic drainage:** aortic, lumbar, and internal iliac lymph node.
7. **Innervation:** -Afferent pain fibers (T11-T12).
   -Sympathetic Inn.: hypogastric and ovarian plexus,
   -Parasympathetic Inn. (S2-S4).
8. **Functions:** (sperm transport, nourishment of fetus, location for the growing fetus, expulsion of the mature fetus).

v. imp. !!



ii. The fallopian tubes or oviduct:

1. **Segment:** (interstitium, isthmus, ampulla,, infundibulum) from medial To lateral.
2. **Layers:**
   a. Serosa.
   b. Loose adventitia: contain lymphatic and blood vessels.

c. Smooth muscle: outer longitudinal and inner circular,
d. Lamina propria.
e. Ciliated columnar epithelium.

3. **Blood supply: Uterine** and **Ovarian** arteries which anastomose in the mesosalpinx .
4. **Function:**
   a. Sperm migration: toward the ampulla.
   b. Ovum transport: toward the uterine .

iii. The Ovaries:

1. **Attachment: medially**: ovarian ligament, **laterally**: suspensory ligament**, inferiorly**: mesovarium .
2. **Blood supply:** ovarian arteries.
3. **Ovarian veins: right side** to the inferior vena cava , **left side** to the left renal vein.
4. **Lymphatic drainage:** pelvic and para aortic .
5. **Function:** house the oocytes, produce reproductive and sexual hormones .

iv. The vagina:

1. **Support:** lower third: pelvic and urogenital diaphragms and perineal body.
   Middle third : pelvic diaphragm and **cardinal** ligament.
   upper third : **cardinal** ligament and **uterosacral** ligament.
2. **Blood supply:** (hypogastric, uterine, middle rectal, Inferior vaginal (From internal pudendal) arteries).
3. **Innervation: sympathetic**: hypogastric plexus, **parasympathetic**: pelvic nerve S2-S4.
4. **Function:** female copulatory organ, distal outflow tract (for menstruation), birth canal.

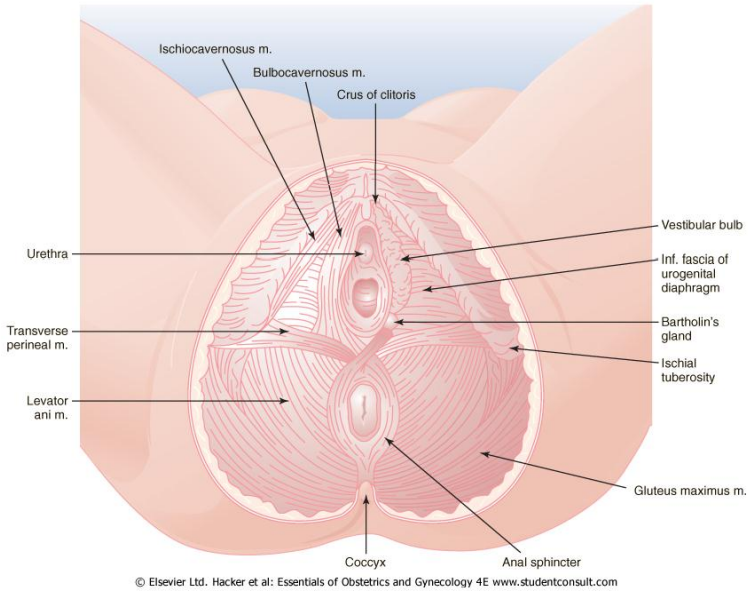
v. **The hymen:** perforated fold of mucosal-covered connective tissue between the distal vagina and the vestibule.

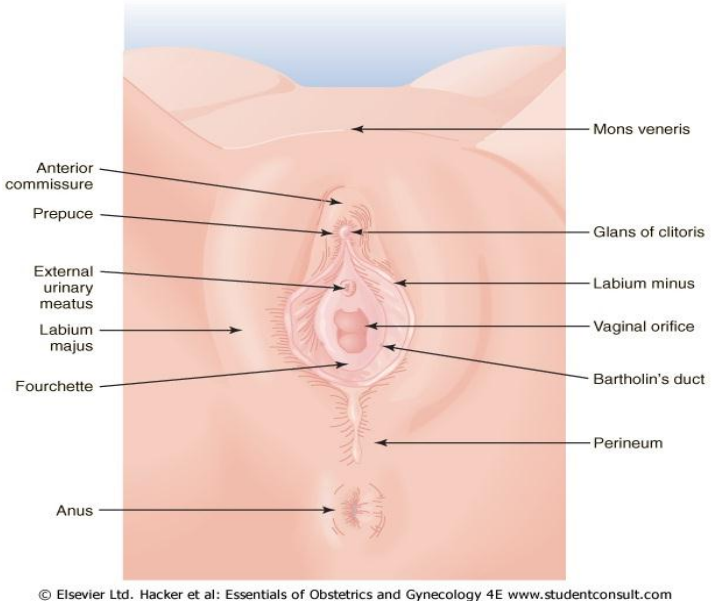
vi. **Structures within the urogenital triangle:** (the mons pubis, labia majora , labia minora, the clitoris, the urethra, skene's gland, bartholin's gland [lubrication]) .

Note: Vulva is innervated by [pig] : -Pudendal, ilioinguinal, and genitofemoral nerves.

vii. **Pelvic diaphragm**: extend from the pubic bone to the coccyx. the primary muscle is the **levator ani** which forms the floor of the pelvis and the roof the perineum.

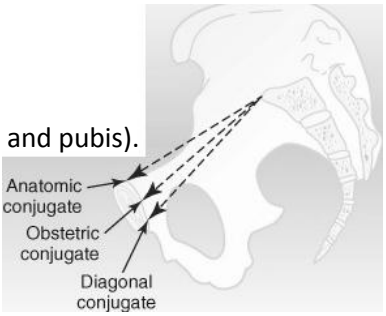
1. **Components of the levator ani**: pubococcygeus muscle (most significant), pubovaginalis muscle, puborectalis muscle, iliococcygeus muscle .
2. **Function:** flexing the coccyx, rising the anus, Constricting the rectum and the vagina.





Anatomy of the Bony Pelvis

-Bony pelvis is made up of 4 bones; -Sacrum
-Coccyx
-Two innominates (composed of the ilium, ischium, and pubis).



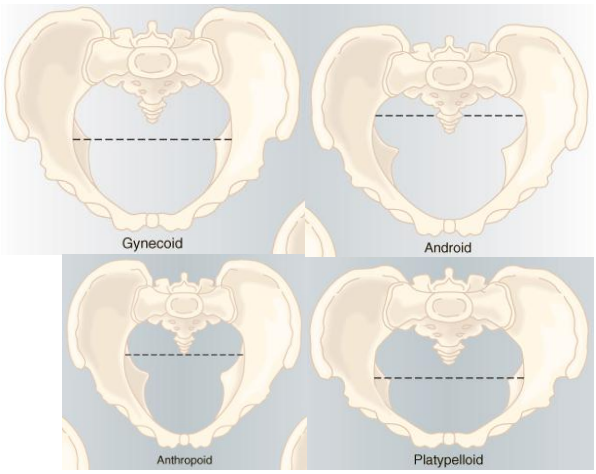
-Pelvic inlet diameters; -True conjugate (Anatomic conjugate) [11.5cm].
-Obstetric conjugate [11cm].
-Diagonal conjugate [12.75cm].

-**The sacrum:** consists of five fused vertebrae. The anterior surface is usually concave.
-The first sacral vertebra is called the **promontory**, which is used as a landmark of clinical pelvimetry.

-Clinical pelvimetry: -Pelvic inlet measured by diagonal conjugate.
-Midpelvis " " bi-ischial diameter.
-Pelvic outlet " " angle of the pubic arch.

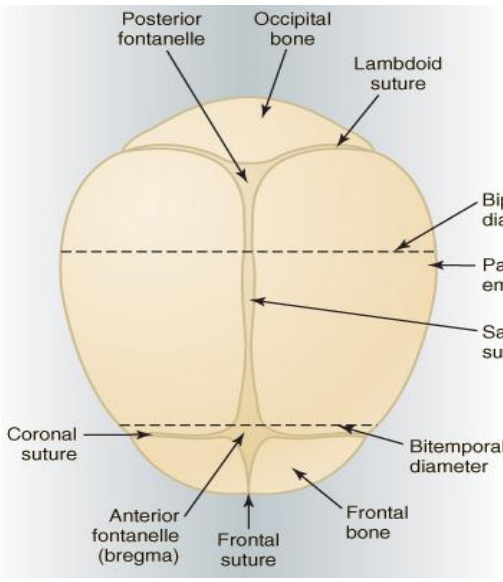
-Pelvic shapes: -Gynecoid, Android, Anthropoid, and Platypelloid.
-**(See table)**

Gynecoid	Android
-Found in 50% of women	-Found in 30% of women
-Cylindrical shape	-Android shape
-Round inlet	-Triangular inlet
-Transverse > AP diameter	-Transverse diam. close to sacrum
-Straight side wall	-Convergent side wall
-Ischial spines not prominent	-Ischial spines not prominent
-Large sacrospinous notch	-Long narrow sacrospinous notch
-Well curved sacrum	-Shallow sacral curve
-Wide subpubic arch	-Narrow subpubic arch

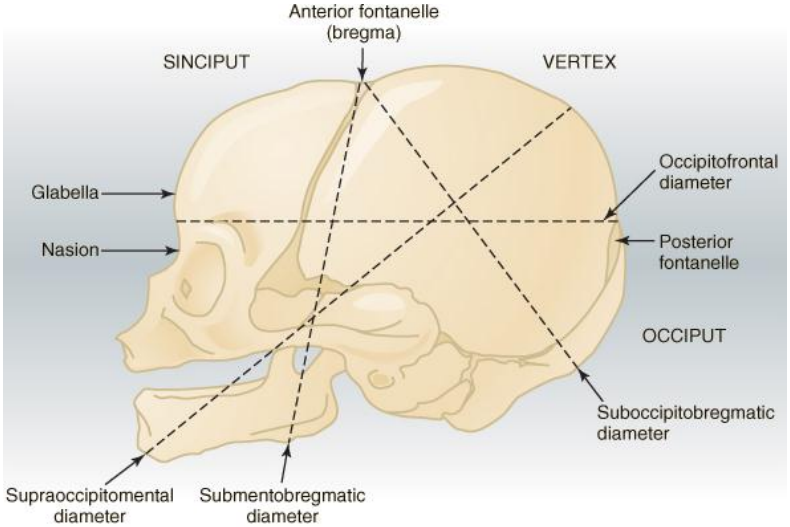


Fetal Skull

-Largest & least compressible part of the fetus & the most important part.
-The fetal skull consists of a base & a cranium (vault)
-The cranium consists of the occipital bone, parietal bones, temporal, and frontal bones.
-At birth, the cranial bones overlap under pressure & change in shape to conform maternal pelvis, a process known as **"molding"**
-Sutures: - 4 sutures **(See figure)**.
-Fontanelles:-Membrane-filled spaces. **(See figure).**
-They are helpful in diagnosing head position.
-Anterior Fontanelle closes at 6-8 weeks of life & Y shaped.
-Posterior Fontanelle closes at 18 months & diamond shape.



-Head diameters: a-Anteroposterior diameters;
-Supraoccipitomental (13.5 cm)
-Occipitofrontal (11 cm)
-Suboccipitobregmatic (9.5 cm)
-Submentobregmatic (9.5 cm)
b-Transverse diameters;
-Biparietal (9.5 cm)
the largest transverse diameter.
-Bitemporal (8 cm)
the shortest transverse diameter.



Labor

Definitions: -Labor; Painful, regular contractions more than 1 in 5 min. for 30-60 sec. with progressive cervical dilatation & effacement & fetal descent.

-False labor; Painless, irregular contractions without cervical dilatation & effacement (Braxton Hicks).

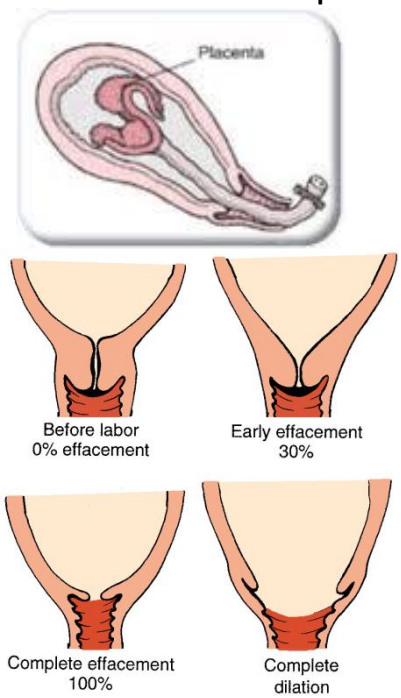
Signs that Labor is within a Few Weeks or Days:

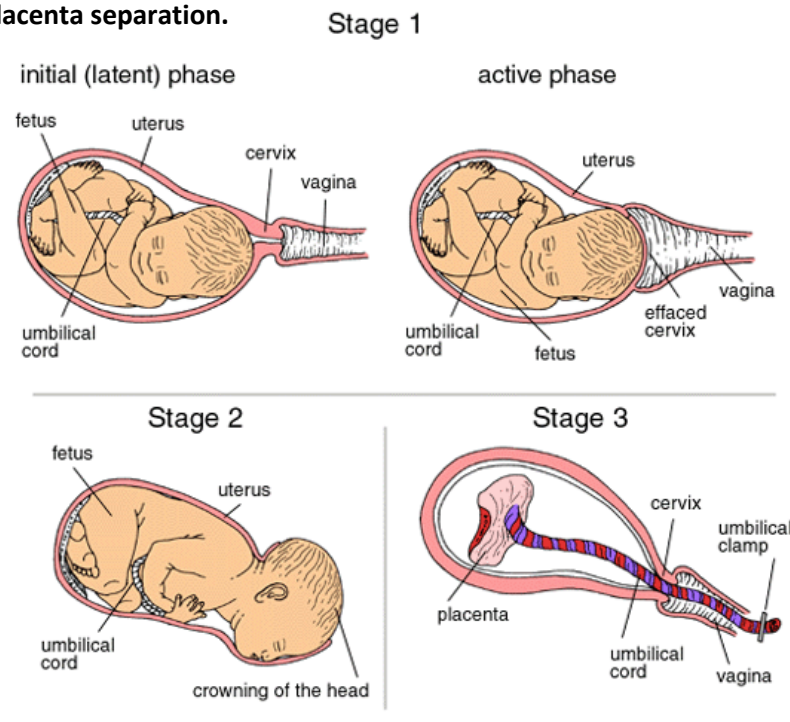
1. Lightening (the mother is relieved when the fetus is engaged).
2. Bloody show (Loss of mucus plug).
3. Rupture of membranes
4. Nesting (a burst of energy that a woman has while she is pregnant).
5. Effacement.
6. Dilation.
7. Consistent Contractions.

Stages of labor: (see table & figure).

	Stage I [Latent phase]	Stage I [active phase]	Stage II	Stage III
Begins	-Onset of regular UC	-Cervical dilation (≥3cm)	-Complete cervical dilation (10cm)	-Delivery of neonate
Ends	-Cervical dilation (≥3cm)	-Complete cervical dilation (10cm)	-Delivery of neonate	-Delivery of placenta
Purpose	-Coordination of UC -Cervical softening & effacement	-Active cervical dilation. -Beginning fetal decent. -Beginning cardinal movements of labor	-Completion of fetal decent -Completion of cardinal movements of labor	-Shearing of anchoring villi. -Delivery of placenta.
Normal Duration	-Multipara (<14 hrs.) -Primipara (<20 hrs.)	-Multipara (<4 hrs.) (Cervical dilation≥1.5 cm/hr.) -Primipara (<5 hrs.) (Cervical dilation≥1.2 cm/hr.)	-Multipara (<30 min.) -Primipara (<60 min.)	- <30 min.
Abnormality	-Prolongation disorder	-Prolongation disorder -Arrest of dilation disorders	-Arrest of descent disorders	-Prolongation disorder
Causes of disorders	-Irrational analgesic use -Hypo/hypertonic UC	-Inadequate bony pelvis -Abnormal fetal orientation or size. -Inadequate or ineffective UC.	-Similar to Stage I [active phase]	-Inadequate UC. -Abnormal placentation -Placenta ac/in/percreta.
Management	-Rest -Sedation	-IV Oxytocin -Cesarean delivery	-Forceps. -Vacuum extractor -Cesarean delivery	-Oxytocic agents. -Manual removal. -Uterine curettage. -Hysterectomy.

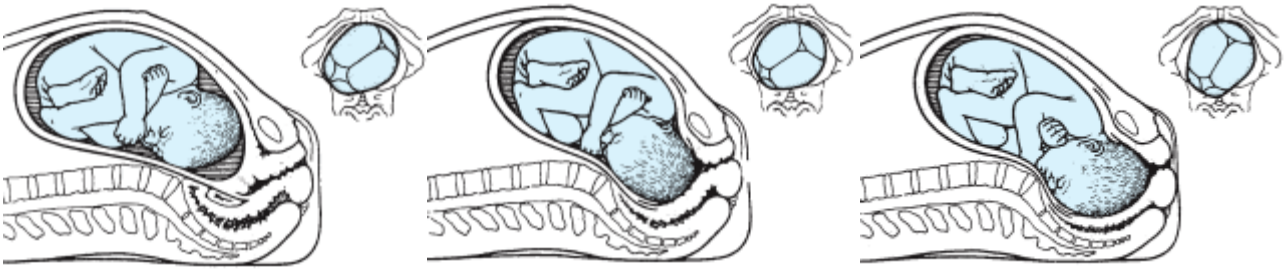
In stage III uterine contractions is responsible for placenta separation.



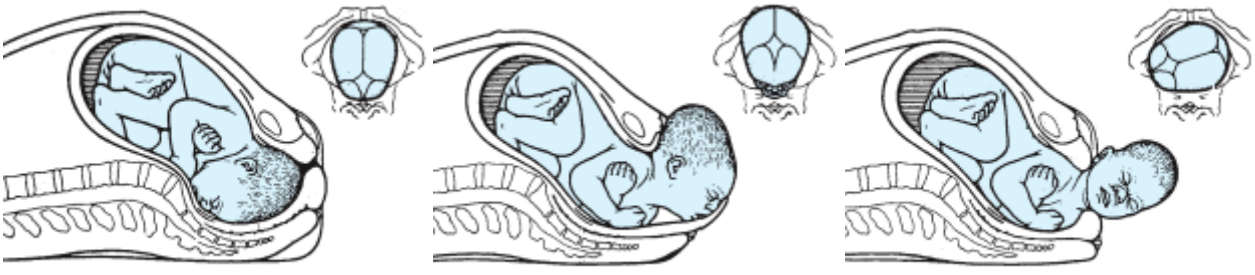


Mechanisms of labor (Cardinal movements of labor):- (see table & figure)

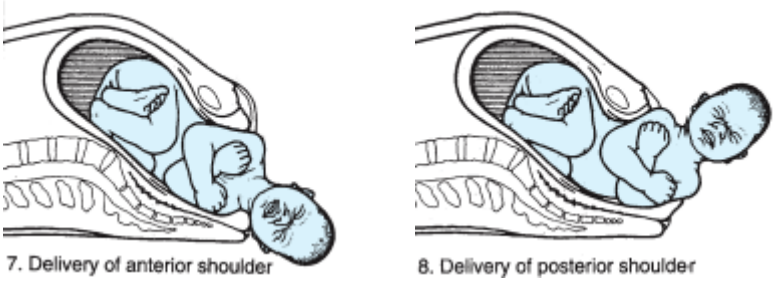
Movement	Definition	Purpose	Occurrence
Engagement	-Descent of BPD to below the plane of the pelvic inlet. -Head position is transverse to accommodate the widest diameter of pelvic inlet.	-Demonstrate adequacy of maternal bony pelvic inlet.	-Prior to labor in primigravidas -After labor onset in multiparas.
Descent	-Movement of fetal head down through the curve of birth canal.	-Most important component of labor.	-Begins gradually in latent phase. -Most rapid in late active phase and stage II.
Flexion	-Placement of fetal chin on thorax	-Allows narrowest AP diameter of fetal head (suboccipito-bregmatic) to present to the birth canal.	-Usually by beginning of the active phase.
Internal rotation	-Rotation of position of fetal head in the mid pelvis from transverse to AP	-Allows the widest diameter of fetal head to present to the widest diameter of mid pelvis.	-Usually by the end of the active phase
Extension	-Movement of fetal chin away from the thorax as the fetal head passes through the pelvic outlet.	-Directs the axis of the fetal head upward to the pelvic outlet.	-Begins with onset of stage II and ends with delivery of fetal head.
External rotation	-Rotation of fetal head outside the mother from AP to transverse after the head has been delivered.	-Allows the transverse diameter of fetal shoulders to present to the widest diameter of the mid pelvis.	-After the fetal head has been delivered but before the shoulders have been delivered.
Expulsion	-Delivery of fetal shoulders and body.	-Completes the birth process of the fetus.	-Begins with delivery of fetal shoulders & ends with delivery of the body.



1. Head floating, before engagement
2. Engagement; descent, flexion
3. Further descent, internal rotation



4. Complete rotation, beginning extension
5. Complete extension
6. Restitution (external rotation)



7. Delivery of anterior shoulder
8. Delivery of posterior shoulder

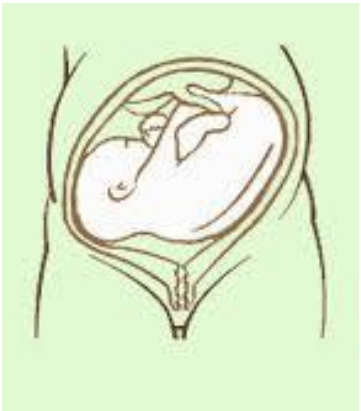
Utero Fetal Orientation: -(see table & figures)

Reference Category	Definition	Most common subcategory
Lie	-Relationship between long axis of fetus & long axis of mother	-Longitudinal lie (99%) (fetal body is vertical to the mother) -Transverse lie (<1%) (fetal body is horizontal to mother) -Oblique lie
Presentation	-Portion of fetus overlying pelvic inlet	-Cephalic (95%) (fetal head lies closest to pelvic inlet) -Breech (4%) (longitudinal lie with head in uterine fundus) -Compound (e.g. shoulder presentation) (>1 fetal part is presenting)
Position	-Relationship between a reference point on the presenting fetal part & maternal bony pelvis	Direct occiput anterior (OA) (most common if cephalic presented) Others; OA positions:(ROA,LOA) occiput transverse:(ROT,LOT) occiput posterior:(OP,ROP,LOP)
Attitude	-Degree of flexion or extension of fetal head	-Vertex (Complete flexion, chin against chest) -Military [Sinciput] (Partial flexion) -Brow (Partial extension) -Face (Complete extension)
Station	-The degree of descent of the presenting part through the birth canal, expressed in cm; the presenting part is above or below the maternal ischial spine (i.e. Station 0).	-Variable (depends on decent of presenting part throughout the stages of labor). -Prior to engagement the station will be -4, moving to +4 just prior to delivery of the head.
Synclitism	-Orientation of the fetal sagittal suture to the midline between maternal symphysis & sacral promontory.	-Synclitic (the sagittal suture is midway between the front & back of the pelvis) -Anterior or posterior asynclitism (when either the anterior or the posterior parietal bone precedes the sagittal suture)

Longitudinal lie
Vertex presentation

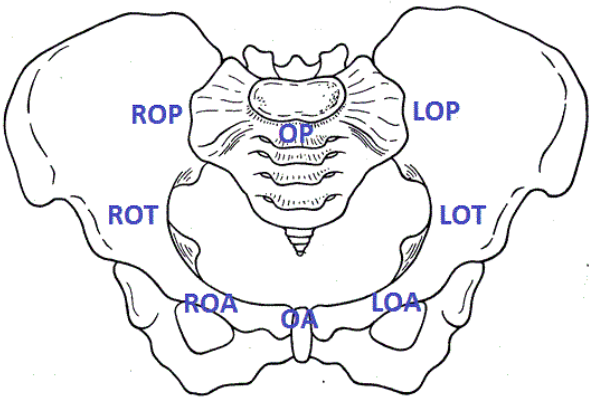
Longitudinal lie
Breech presentation

Transverse lie
shoulder presentation

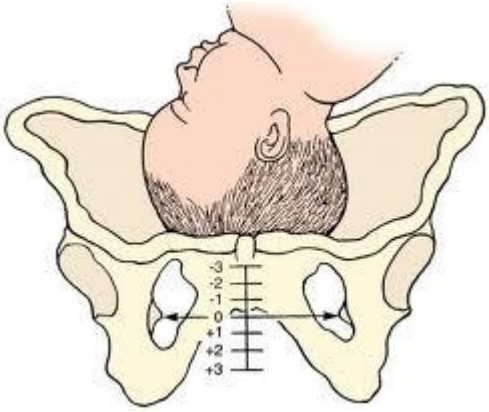


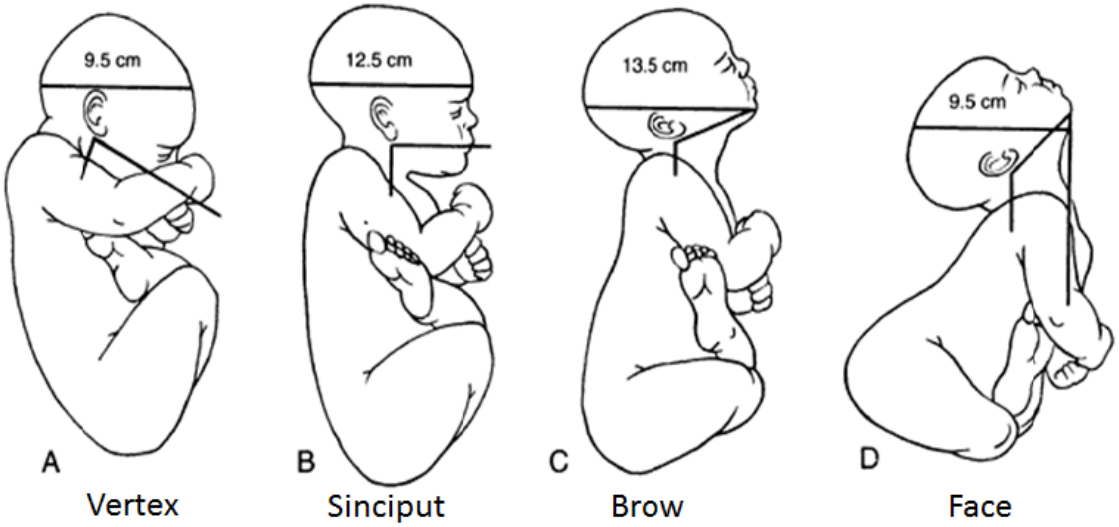
Vertex presentation

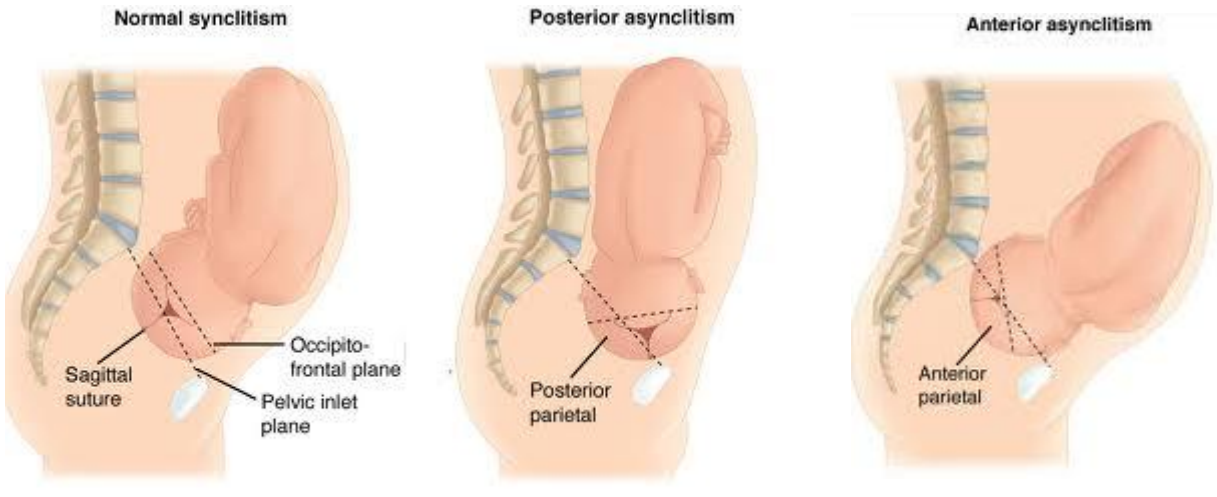
Breech presentation



Shoulder presentation







BPD=Biparietal diameter. , AP=Anterior-posterior. , UC=Uterine contractions.

Intrapartum Fetal Monitoring [Fetal Surveillance]

- **Monitoring methods:**

A. <u>Intermittent auscultation of Fetal heart rate (FHR);</u>

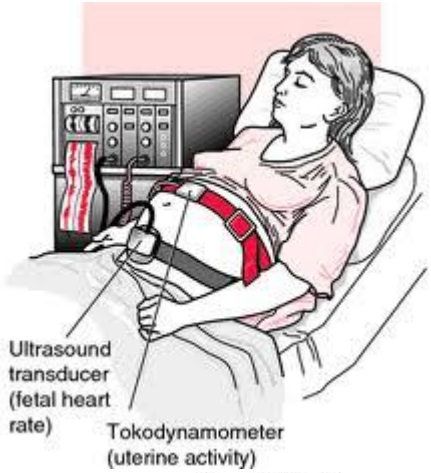
- **<u>Direct :</u>** By DeLee fetoscope or Doppler US device .
  - <u>Standard:</u> during a contraction and for 30 S. after that every 15 M. for the 1st stage → and every 5 months for the 2nd stage .
  - <u>Nonreassuring findings:</u> <100 beats baseline 30 second after contraction. OR >160 baseline, 30 second after contraction.

B. <u>Continuous electronic monitoring of Fetal heart rate (FHR) & Uterine contraction (UC);</u>

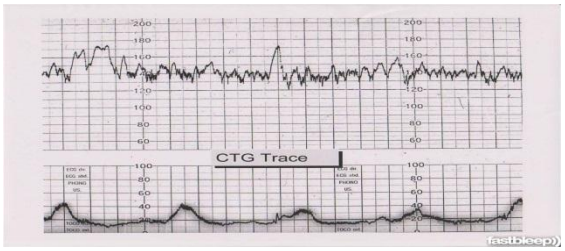
- **(See table)**

Modality	Device	What is recorded	Risks	Advantages	Disadvantages
External - indirect FHR monitoring	Ultrasound sonocardiogram attached by belt to maternal abdomen	Fetal cardiac motion frequency	None	-Noninvasive -Used in intact membranes	-Discomfort of belt. -Poor signal quality if obese abdomen
External- indirect UC monitoring	Spring-loaded tocodynamometer strain gauge attached by belt to maternal abdomen	Uterine wall tautness & relaxation indicating beginning & end of contraction	None	-Noninvasive -Used in intact membranes -Contraction frequency & duration are measured	-Discomfort of belt. -Poor signal quality if obese abdomen -Doesn't measure contraction intensity
Internal- direct FHR monitoring	Electrode attached to fetal scalp through a dilated cervix	Fetal cardiac electrical activity frequency	-Fetal scalp abscess -Inoculation of maternal genital tract infections	-No belt discomfort -High-quality signal regardless of obese abdomen	-Membrane must be ruptured -Cervix must be dilated
Internal- direct UC monitoring	Intrauterine pressure catheter through a dilated cervix	Changing intrauterine amniotic fluid hydrostatic pressure	-Low-lying placenta could be perforated -Inoculation of maternal genital tract infections	-No belt discomfort -Measures contraction frequency, duration, & intensity.	-Membrane must be ruptured -Cervix must be dilated

- Could be any combination of the above modalities.
- Modality selection depends on the mother's condition.
- Internal modalities are the best methods.
- Normal electronic fetal monitoring parameters are:

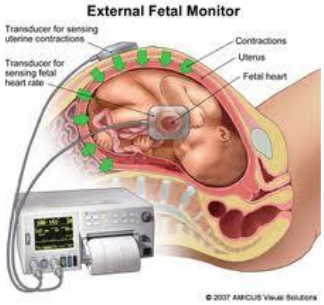


1. **Baseline HR;**
   - Normal rate (120—160 beats/min).
   - **Causes of baseline tachycardia:**

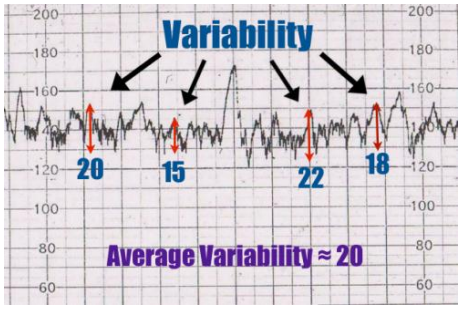


1. Hypoxemia
2. Fever
3. Prematurity
4. Fetal arrhythmia
5. Prolonged fetal movement
6. Maternal hyperthyroidism
7. Drugs (atropine , β-adrenergic agonists)

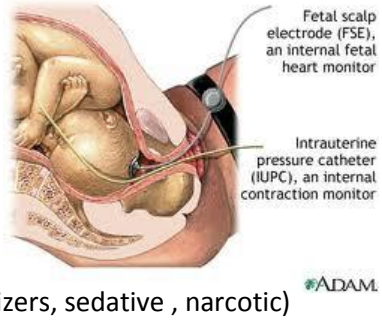
   - **Causes of baseline bradycardia:**
     1. Hypoxemia
     2. Fetal arrhythmia
     3. Drugs (local A, β-adrenergic blockers)



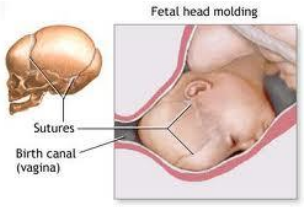
2. **Beat to beat variability;**

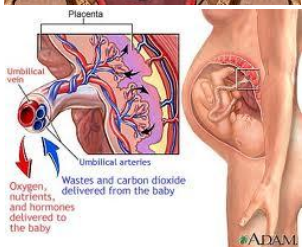


- Most important sign of fetal wellbeing.
- Normal Baseline variability (6—10 beats/min)
- Short term variability controlled by parasympathetic.
- Long term variability controlled by sympathetic.
- **Causes of increased variability:**
  1. Early hypoxemia
  2. Fetal movement
  3. Fetal arrhythmia
- **Causes of decreased variability:**
  1. Chronic hypoxemia
  2. Prematurity
  3. Fetal sleep
  4. Drugs (parasympatholytics, tranquilizers, sedative , narcotic)



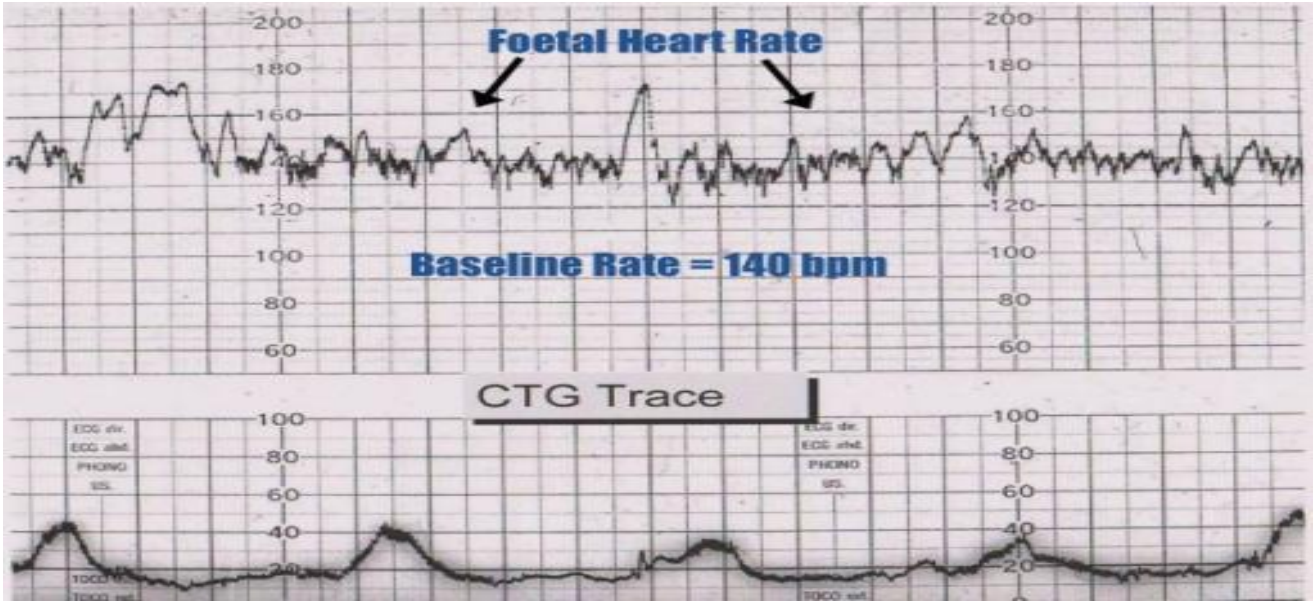
3. **Periodic changes; -** Changes in FHR in relation to contraction .

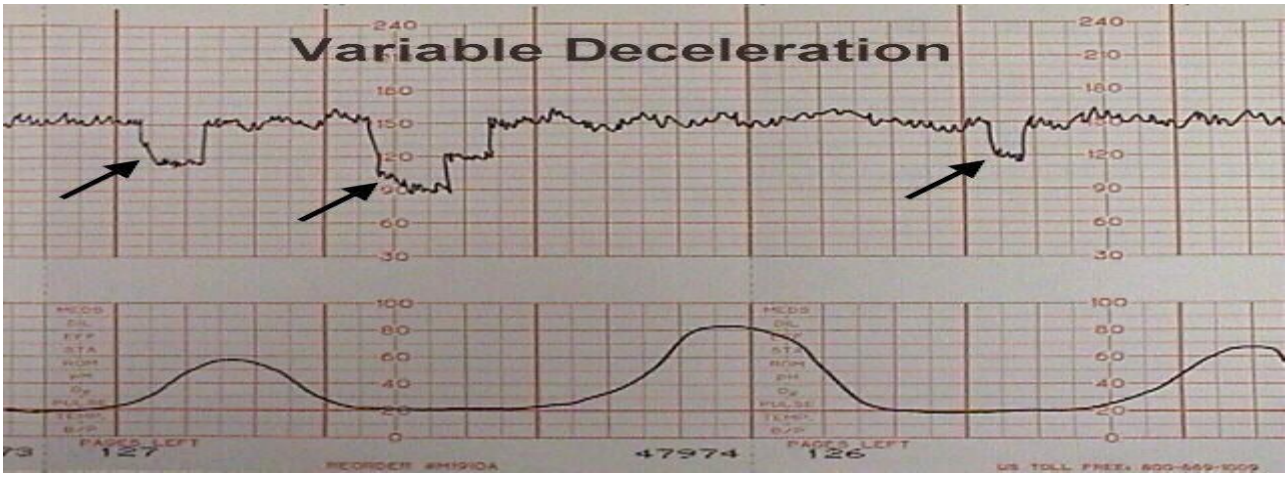


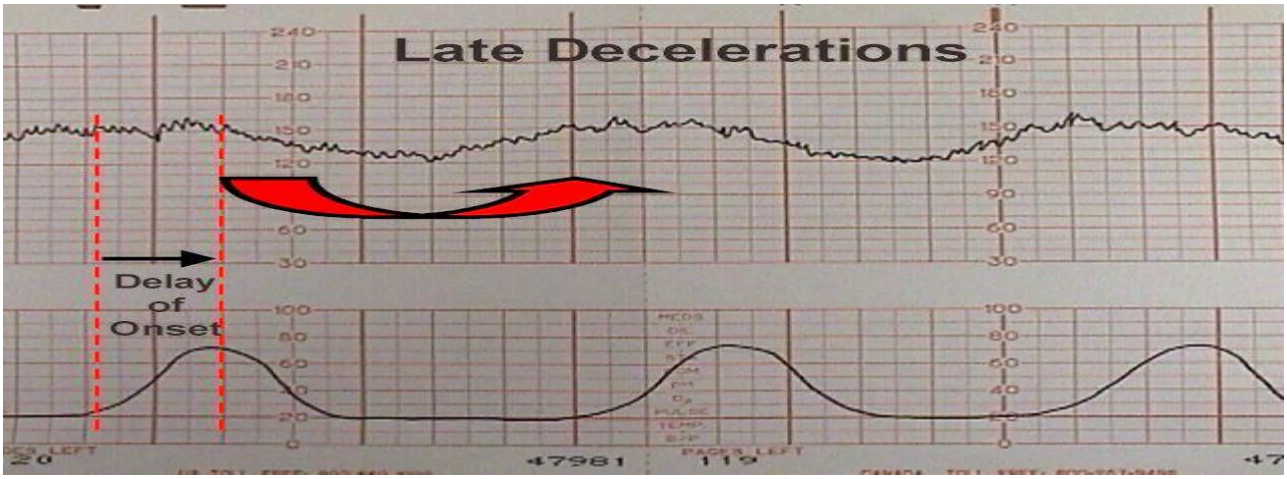


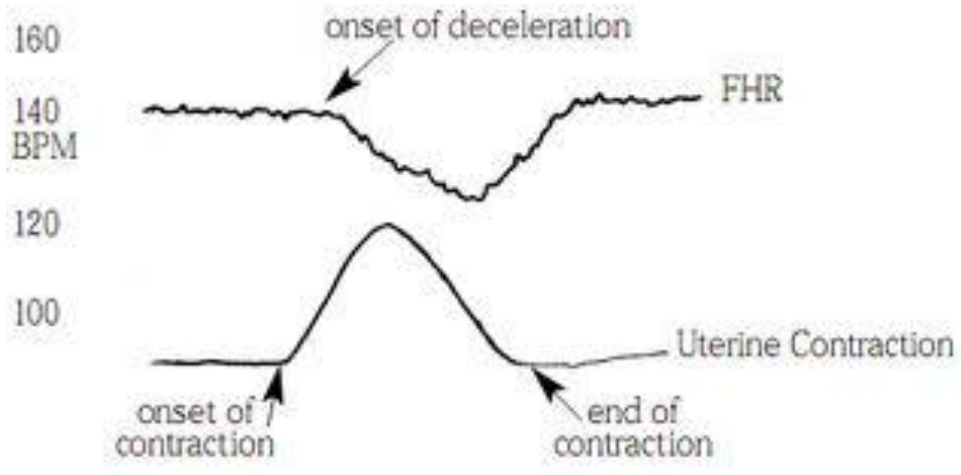
1. **<u>Acceleration</u>**
   - increase baseline ≥ 15 beat for ≥ 15 seconds.
   - Sympathetic >>> due to fetal movement or stimulation.
   - Always reassuring
2. **<u>Early deceleration</u>**
   - Drop below baseline appearing as mirror image of UC.
   - Parasympathetic (vagal) >>> due to fetal head compression.
   - Not important clinically.
3. **<u>Variable deceleration</u>**
   - Sudden drop below baseline with sudden return**.**
   - Parasympathetic (vagal) >>> due to umbilical cord compression.
   - <u>Mild to moderate</u>>>> not significant.
   - <u>Prolonged repetitive</u> <u>or deep</u> are not reassuring.
   - <u>Severe</u> >>> drop 60 beat for 60 s. >>>or 60 below the baseline.
4. **<u>Late deceleration :</u>**
   - Gradual drop below baseline with gradual return after a UC.
   - Parasympathetic (vagal)>>> due to uteroplacental insufficiency (<u>hypoxia</u>).
   - Myocardial depression>>> uteroplacental insufficiency (<u>acidosis</u>).

- **Sings of well-fetal being:**
  1. Normal baseline.
  2. Normal variability.
  3. No late deceleration.
  4. No sever variable deceleration.



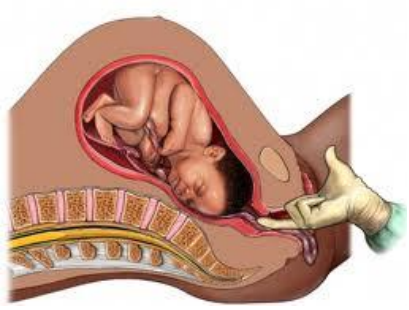






C. <u>Normal PH for scalp/cord blood gas >7.20.</u>

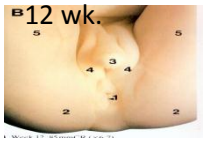
- **Intervention in non-reassuring FHR monitoring:**
  a. Decrease uterine activity >>> STOP oxytocin >> START tocolytics.
  b. Correct hypotension >>IV 500 ml isotonic without dextrose
  c. Change maternal position >>>> change to the left side
  d. Give high flow OXYGEN 8—10 L/min
  e. Do vaginal EX. >>>>role out umbilical cord prolapse
  f. Stimulate the fetus >>>look for acceleration.



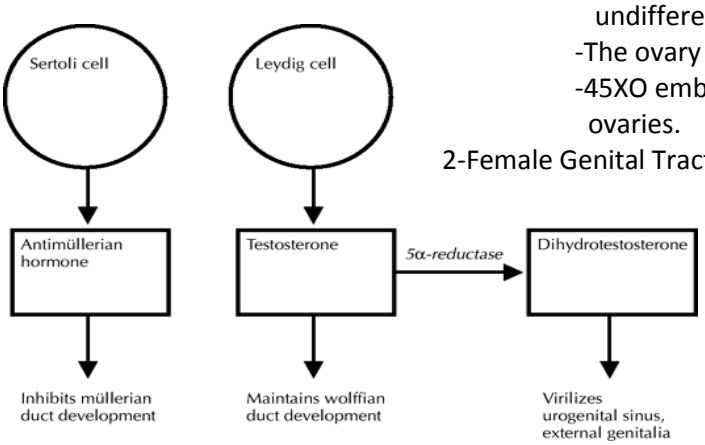
Extra:::
== How to read a CTG == [http://geekymedics.com/body-systems/og/how-to-read-a-ctg/]

Female genital tract embryology, malformation, and intersexuality

Embryology: -Sexual differentiation; -1st step is determining genetic sex (XX or XY).
-♀ sexual development does not depend on the presence of ovaries.
-♂ sexual development depend on the presence of functioning testes & responsive end organs.

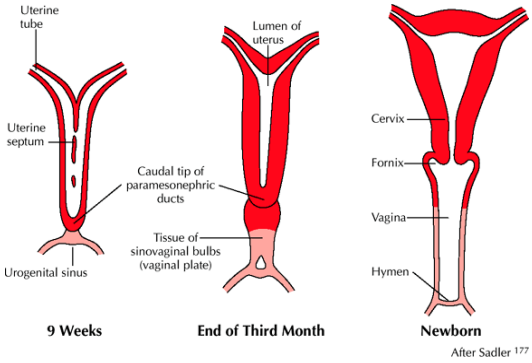


-External genitalia; -Prior to 7th week, ♂ & ♀ look the same externally.
-By 12 wk. gestation ♂ & ♀ genitalia can be differentiated.
-In the absence of androgens ⇨♀ external genitalia develop.
-The development of ♂ genitalia requires the action of androgens, specifically DHT.
-Internal genital organs; 1-Gonads; -Undifferentiated gonads begin to develop on the 5th wk.
-If the Y chromosome (testes-determining factor) is absent, the undifferentiated gonad will develops into an ovary.
-The ovary contains 2 million 1ry oocytes at birth.
-45XO embryo the ovaries develop but undergo atresia ⇨ streak ovaries.

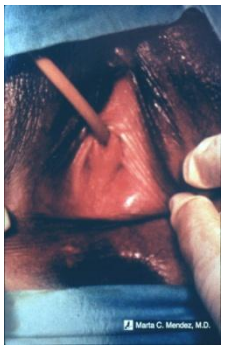


2-Female Genital Tract; -Fusion of the two paramesonephric ducts (mullerian ducts) ⇨fallopian tubes, uterus, and upper vagina.
-The male fetus testes secrete anti-mullerian hormone ⇨ regression of the mullerian ducts.
-The lower 1/3 ⇨ urogenital sinus.
-By the 20th week uterine mucosa is fully differentiated into the endometrium.
-Development of uterus, cervix and vagina is complete by the 22nd week.
-Gartner's duct cysts are remnants of wolffian ducts in ♀.

Malformation: -<u>Mullerian agenisis;</u> a-Mayer-Rokitansky-Kuster-Hauser syndrome;

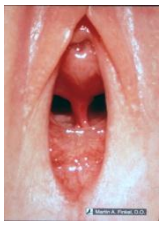


-Absence of the upper vagina, cervix & uterus(+/-uterine remnants).
-The ovaries & fallopian tubes are present.
-Normal 46XX ♀ with normal external genitalia.
-Pt. present with 1ry amenorrhea
-47% have associated urinary tract anomalies
-12% skeletal anomalies
-Rx ⇨psychological counseling
⇨surgical -Vaginoplasty.
-Excision of uterine remnant if present.
-Vaginal dilators.

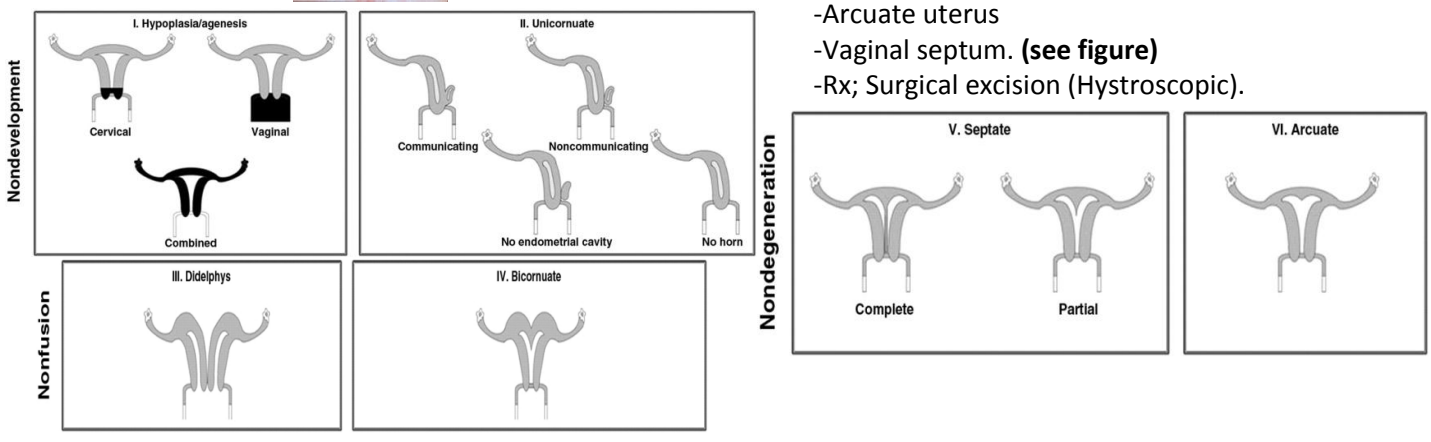


b-Unicornuate uterus-with no other horn.
-Almost all pt. have associated single kidney.
-Rx; No corrective surgery

-<u>Mullerian dysgenisis;</u> =Types: a-Incomplete development; -Non-communicating Unicornuate uterus.
-Communicating Unicornuate uterus.
-Rx; Surgical excision



b-Non-fusion; -Didelphus Uterus (duplication of uterus, cervix & vagina)
-Bicornuate uterus
-Rx; -Surgical correction (metroplasty*)
c-Non-degeneration; -Septate uterus (intrauterine septum)
-Arcuate uterus
-Vaginal septum. **(see figure)**
-Rx; Surgical excision (Hystroscopic).

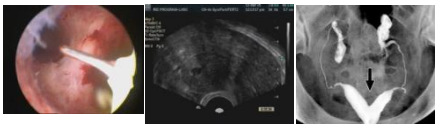


=Etiology: -All happen spontaneously, but maternal exposure to DES can cause it.

=Clinical presentation & Complications: **(see tables)**

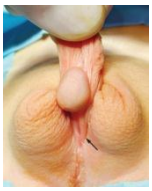
Clinical presentation
-1ry amenorrhea (if there is an obstruction).
-Difficulty in intercourse (vaginal septum)
-Dysmenorrhea or menorrhagia
-Abnormality detected on D&C
-Palpable mass
-EP or endometriosis in the rud. horn of the Unicornuate.
-Complications of pregnancy.

↑ Incidence of
-Infertility (4%)
-Recurrent abortions (6-10%).
-Fetal loss (RFL).
-Premature birth.
-Fetal malpresentation.
-C/S.
-Cervical incompetence.



=Dx; Hysteroscopy, U/S, HSG, laparoscopy, or laparatomy.

-External genitalia defects; -Ambiguous genitalia; -Hermaphrodites **(see intersexuality)**

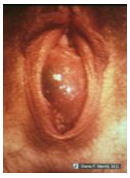


-Due to CAH.

-Defects of the clitoris; -Bifid clitoris.

-Hypertrophied clitoris.

-Androgen effect.



-Imperforated hymen; -1ry amenorrhea with cyclic abdominal pain

-Hematocolpus.

-Acute urinary retention.

-Rx; -Cruciate incision.

Intersexuality:-Sex chromosome abnormality: -Turner's syndrome;-Karyotyping (45XO)

-Gonadal dysgenisis.

-Streak ovaries.

-High FSH.

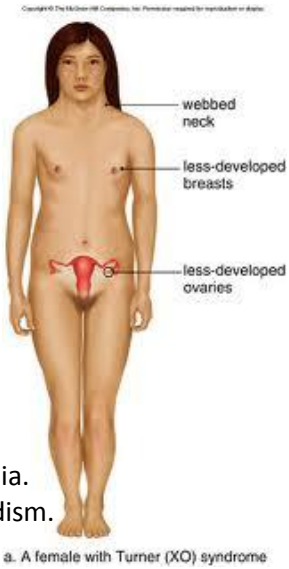
-Present uterus.

-No 2ry characteristics (breasts).

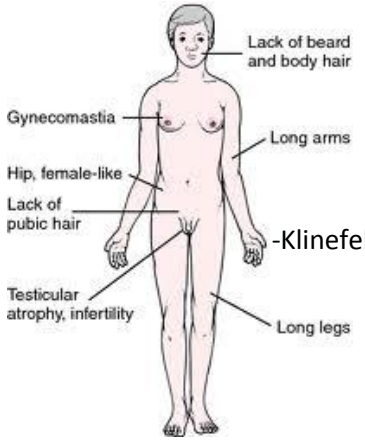
-Short stature.

-Webbed neck.

-Rx.; E therapy with cyclic P.



a. A female with Turner (XO) syndrome



-Klinefelter syndrome;-Karyotyping (47XXY)

- Normal male external genitalia.

-Present testes but hypogonadism.

-Azospermia (infertility).

-Gynecomastia.

-Tall stature.

-Musculanization of ♀ external genitalia [♀ Pseudohermaphrodite];- Karyotyping (46XX).

-Due to ↑ androgen in utero.

-CAH is the M.C. cause of ♀ intersex.

-Mother androgen ingestion [drugs].

-In CAH, ↓ 21-hydroxylase.

-Enlargement of the clitoris.

-Genital folds fusion (scrotum).

-Under-musculanization of ♂ external genitalia [♂ Pseudohermaphrodite];-

a-Anatomical testicular failure:- Karyotyping (46XY).

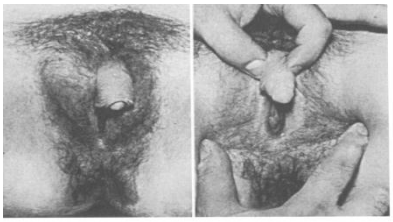
-Normal female

-Present uterus.

b-Enzymatic testicular failure: - Karyotyping (46XY).

-↓ Testosterone biosynthesis enzymes.

-Affected External genitalia.



c-End-organ insensitivity: - Karyotyping (46XY).

-↓ 5α reductase⇨↓DHT **(see figure above)**.

-Female external genitalia [Absent uterus].

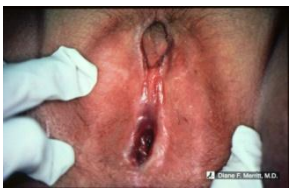
-At puberty ⇨ ↑ testosterone ⇨ virilization.

D-Androgen insensitivity: -Called Testicular feminization.

- Karyotyping (46XY).

- ↓ androgen receptors.

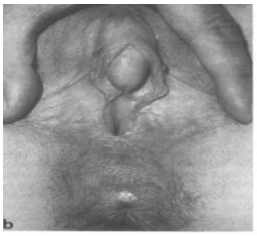
-Clinical features: **(see table)**



Clinical features
- ♀ external genitalia with blind vagina.
-Absent uterus and pubic hair.
-Breast development.
-Present with 1ry amenorrhea.
-Testes found in abdomen or inguinal canal.
-Normal ♂ Testosterone level.

-Rx;-Gonadectomy after puberty [5% malignancy].
-Estrogen replacement therapy.

-True hermaphrodites: -Testis & ovary both present, either joined [ovetestis] or separated.
-Varying degrees of sexual ambiguity.
-Karyotyping:- 46XX ⇨ most common
-46XY
-47XXY
-Dx.; -Requires laparatomy



Note: Generally Dx. is made by ; -History.
-Physical examination.
-Pelvic US.
-Karyotyping.
-Hormonal studies.
-Lab tests accordingly.

_____
DHT= di-hydro-testosterone
Pt.=Patient
Metroplasty= plastic surgery on the uterus
DES= Diethylstilbestrol.
♂= Male
♀= Female
HSG= Hystero-salpingio-graphy
RFL= recurrent fetal loss
CAH= Congenital adrenal hyperplasia
M.C.=Most common
Dx.= Diagnosis
Hx.=History
E= Estrogen
P= Progestin

Puberty

-Pubertal changes in both genders are mediated by sex steroid produced from gonads in response to hypothalamic pituitary gonadal axis (HPGA).

-The dominant sex steroids are: ovarian estrogens in females, testicular androgens in males.

-**Lower age limits** of puberty are 8 yrs. in females and 9 yrs. in males but the average is 11 yrs. Age of puberty may depend on **mean body weight** or **percentage of body fat (more fat = early menarche).** The puberty start by ↓GnRH sensitivity to estrogens→ rise GnRH level (occur during sleep) → rise FSH production (occur during sleep) → ovarian follicular maturation, production of sex hormones from gonads, and development of sexual characteristics .

-The age of menarche has decreased over the last 3-4 decades due to improved nutrition, general health & life style.

-Low level of gonadotropins in prepuberty because:

1- High sensitivity of gonadostat (hypothalamic-pituitary system regulating gonadotropin release) to –ve feedback of low level of estrogens.

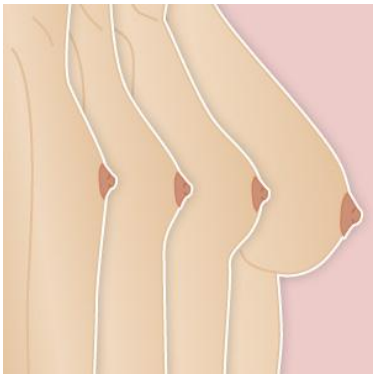
2- Intrinsic central nervous system inhibition of GnRH. (main inhibitor during 4yrs until prepuberty)

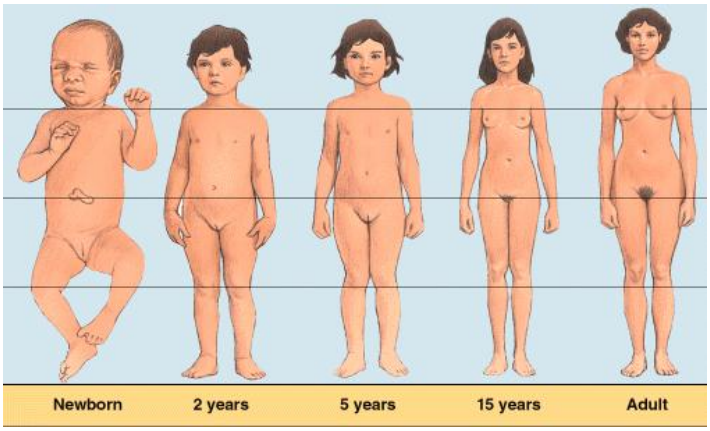
-Developmental endocrinologic changes:

	Sex steroid level	**Gonadostat function**	**Gonadotropin level**
Newborn	**Estrogens fall** in first of Delivery.	-ve feedback lost because low Estrogen level	Rapidly rise
Early childhood	**Estrogen at low basal Level** from ovarian follicles	Sensitivity increase Leading to suppression. With low estrogen.	Fall to basal level
Late childhood	**Estrogen at low basal level** from ovarian follicles	Sensitivity is high	Low level
prepuberty	**Adrenal androgens rise** (adrenarche)	adrenarche independent to gonadostat	Low level
Puberty	**Estrogen rise gradually** from ovarian follicles	Gonadostat sensitivity decline	Rise , from sleep-associated Increase of GnRH.

-Normal development of secondary sexual characteristic:

Age	**Pubertal change**
8-9 yrs	Slow growth rate
9-10 yrs	Telarche: breast budding
10-11 yrs	Adrenarche: pubic and auxiliary hair
11-12 yrs	Maximal growth rate
12-13 yrs	Menarche: onset of menses
13-14 yrs	Adult pubic hair
14-15 yrs	Ovulation



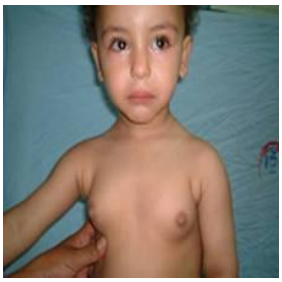


Note: anovulatory cycle occur in the first 6-18 months.

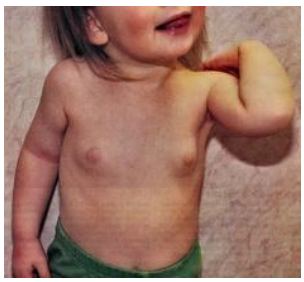
- and remember : if endometrium is not exposed to progesterone" --> irregular unpredictable menstrual flow.

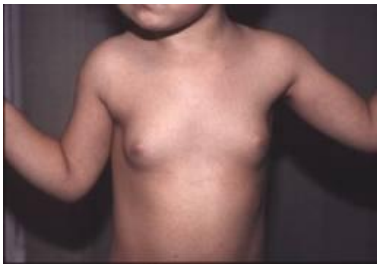
- Precocious puberty: when puberty occur before expected age [≤ 7 years]. F/M ratio is 5:1.

1**- Incomplete precocity**: -Only one sign of puberty is present (premature telarche, premature adrenarche, or premature pubarche), occur because of transient hormone elevation (estrogen or androgen), or end-organ sensitivity (breast or pubic region).



- Diagnosis: by exclusion
- Management: conservative.





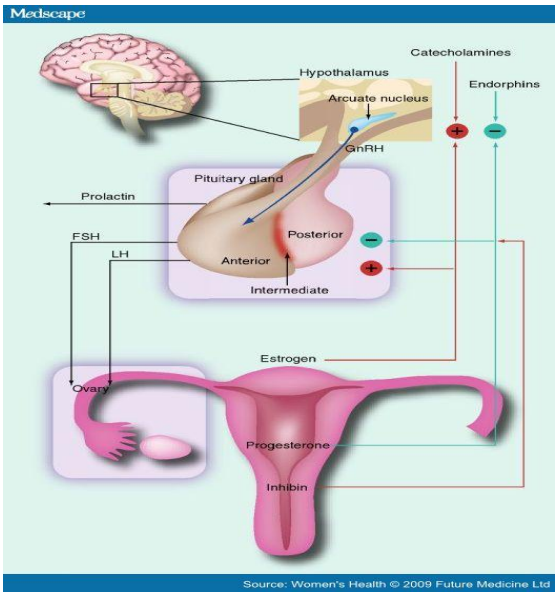
2- Complete precocity: all sing of puberty are present. The hazard is premature closure of long bone epiphysis resulting short stature.

***A*-Isosexual complete precocious puberty:** development of full complement of secondary sexual characteristics and increase sex steroids level, mediated by ↑level of estrogens.

1- GnRH dependent causes (true isosexual): involvement of HPO axis.

a- Idiopathic: 75% of cases, good prognosis with ttt.(**premature activation of HPO axis.),** in GnRH stimulation test which is administration of exogenous GnRH →LH will ↑as in mature girls. Treated by GnRH agonist(leuprolide acetate) to suppress ovarian estrogens.

b- CNS lesion: 10 % of cases, obstructive like (hydrocephalus), infective(TB, meningitis),tumor (glioma, neorofibromatosis). They interfere with hypothalamic GnRH release. Patients often show neurologic symptoms before precocious puberty. Treat the underlying cause.



2- GnRH-independent causes (pseudoisosexual): no HPO axis involvement.

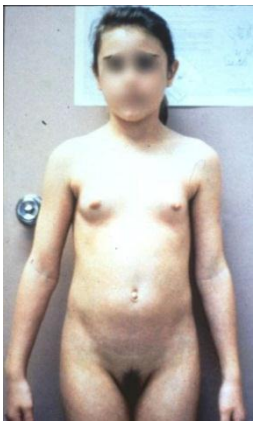
a- McCunn-Albright syndrome(most frequent 5%): characterized by polyostotic fibrous dysplasia, café au lait skin spots, bone defect that fracture easily, here ovarian follicles producing estrogens autonomously (independently)**. Aromatase enzyme inhibitor** is the treatment.

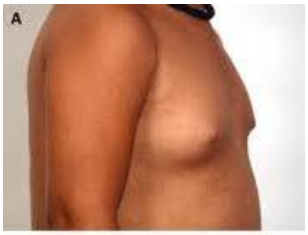
b- Prolonged hypothyroidism: TRH mimics GnRH to stimulate gonadotropin release. Treated by hormone replacement therapy.

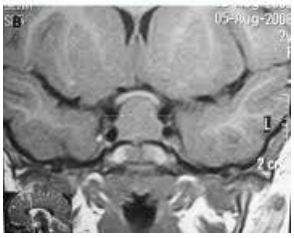
c- estrogens-producing ovarian tumors(rare): (granulose, fibrothecoma) ,here over production of estrogen by hormonally active tumor. Surgical removal of tumor.

d- Peutz-Jeghars syndrome(rare).

e- Exogenous(rare): combined OCP, estrogens creams.







***B*- Heterosexual Complete precocity:** Virilization changes(facial hair, clitromegaly, male body contours). Caused by increase circulating androgens, always pathological, most common cause congenital adrenal hyperplasia (CAH). Treated by corticosteroid replacement.

Note: Delayed puberty occurs if a girl ≥ 14 years and has no pubertal changes.

The physiology of the menstrual cycle

Menstrual cycle represent a complex interaction between the **hypothalamus, pituitary, ovaries and endometrium**, these cyclical changes in gonadotropins induce a functional as well as morphological changes in ovaries resulting in ovulation and corpus luteum formation and also at the endometrium level.

❖ **HYPOTHALAMUS.**

- Is controlling the pituitary gland through releasing hormones which **are peptide hormones**. so they are very important in the synthesis and release of the trophic hormones of the pituitary gland except prolactin hormone which is under **inhibitory effect,** dopamine effect.
- The gonadotropins which are **glycoproteins,** FSH and LH, all contain **alpha and beta** subunits alpha subunit is similar in all hormones but beta subunit is **specific** to individual hormone.
- Normal ovulatory cycle is divided into **proliferative or follicular** phase and **secretory or luteal** phase. Follicular phase starts with the onset of the menses till preovulatory LH surge and ovulation, luteal phase then starts till next menses .

❖ **OVARIAN CYCLE.**

- ESTROGENS, mainly **estradiol E2** ,is relatively low at the beginning of the follicular phase then increase rapidly reach a maximum one day before the LH surge, Just before ovulation there is a marked fall, during the luteal phase E2 rises to a maximum **5-7 days** after ovulation and return to base line shortly before menses.

- PROGESTERONE, is secreted in small amount in the follicular phase but start to increase just before ovulation also reach a maximum **5-7 days** after ovulation and return to baseline shortly before menstruation

❖ **FOLLICULAR DEVELOPMENT:**

- It starts since fetal development and known as the **primordial follicles .** Under the effect of FSH the adult ovary containing **the graafian follicles** which is an oocyte covered with granulosa cell and theca cell start to grow, usually many follicles are recruited every cycle and **only one** usually continue differentiation and Maturation and ultimately ovulate .
- The remaining follicles become atretic and the Follicular maturation depends on FSH and LH receptors ,the FSH receptors are found in **granulosa cell** where LH receptors are found in **theca cell**. Granulosa cell secretes **E2** and when increase will cause a **negative feedback** Effect on the hypothalamus and pituitary gland and will stimulate proliferation of the endometrium . The rest of the follicles will not find enough hormone to survive FSH will also enhance the induction of LH receptors on the granulosa cell. "FSH" causes the LH surge

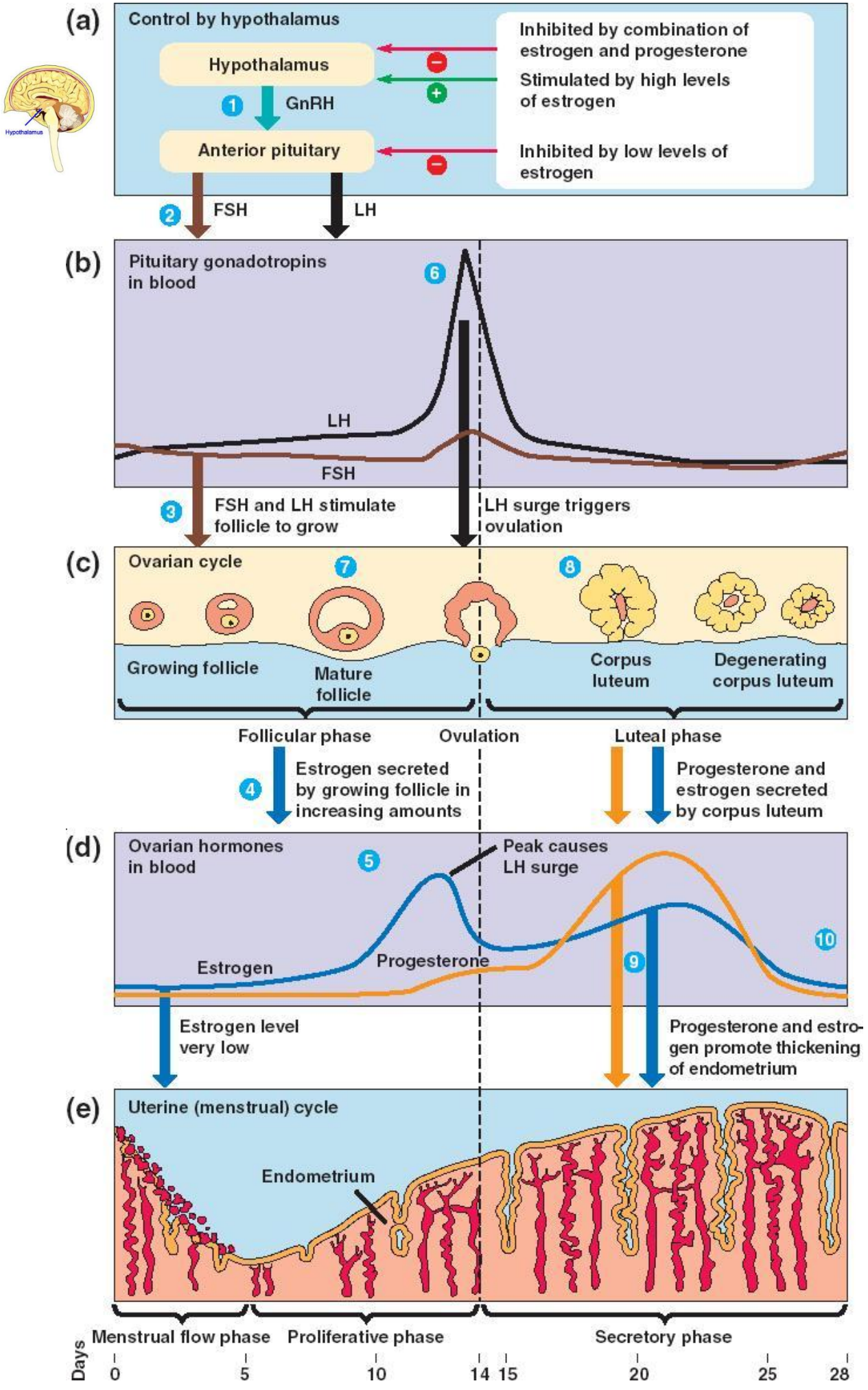
❖ **OVULATION:**

When the follicle mean diameter reach **18-24 mm** LH surge will initiate sequence of structural and biochemical changes resulting in ovulation .

❖ **CORPUS LUTEUM .**

After ovulation the granulosa cell of the ruptured follicle undergo lutenization these cell plus the surrounding theca cell Form the corpus luteum which produces copious amount of progesterone and little estrogen .The normal life span of the corpus luteum is **10 days** then it regress and replaced by an avascular scar called **corpus albicans**. Next menses will come **unless** pregnancy occurs.

Extra hormonal notes: -**Polypeptides=** GnRH, hPL**,** Prolactin
-**Glycoproteins=** LH, FSH, TSH , CRH & β-hCG.
-**Steroids=** Progesterone, Estrogens, Androgens, & Glucocorticoids.


(a)
Control by hypothalamus
Hypothalamus
Hypothalamus
1
GnRH
Anterior pituitary
Inhibited by combination of estrogen and progesterone
Stimulated by high levels of estrogen
Inhibited by low levels of estrogen
2
FSH
LH
(b)
Pituitary gonadotropins in blood
6
LH
FSH
3
FSH and LH stimulate follicle to grow
LH surge triggers ovulation
(c)
Ovarian cycle
7
8
Growing follicle
Mature follicle
Corpus luteum
Degenerating corpus luteum
Follicular phase
Ovulation
Luteal phase
4
Estrogen secreted by growing follicle in increasing amounts
Progesterone and estrogen secreted by corpus luteum
(d)
Ovarian hormones in blood
5
Peak causes LH surge
10
9
Estrogen
Progesterone
Estrogen level very low
Progesterone and estrogen promote thickening of endometrium
(e)
Uterine (menstrual) cycle
Endometrium
Menstrual flow phase
Proliferative phase
Secretory phase
Days
0
5
10
14
15
20
25
28

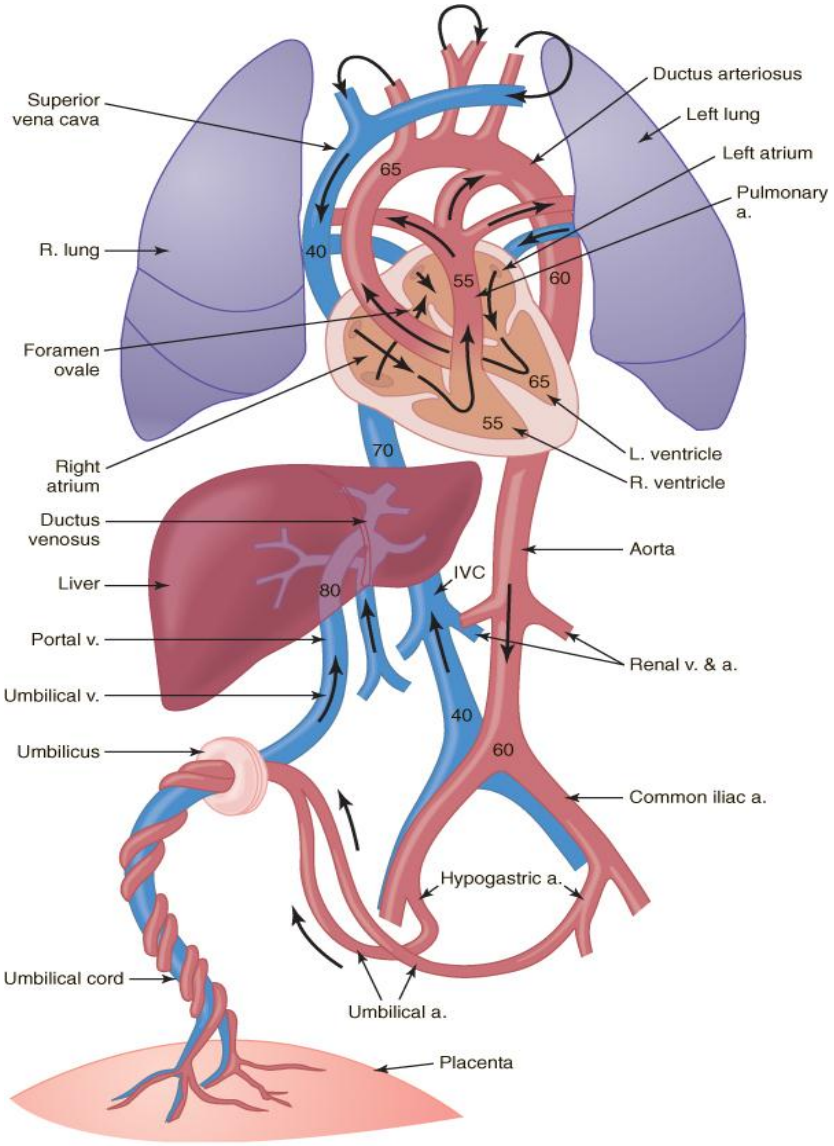
Fetal Circulation

In the fetus the Circulation is called in parallel system

- Right ventricle --> pulmonary artery --> ductus arteriousis --> descending aorta very small portion of the right ventricle goes to the pulmonary circulation
- Left ventricle --> aortic arch brain and upper body mainly highly oxygenated
- Fetal circulation characterized by channels (ductus arteriousis, venouses, foramen ovale) and preferential streaming as in likes brain more
- Umbilical vein carries oxygenated blood 80% from the placenta to the fetus
- Majority of This blood **bypass the liver via ductus venouses** and enters the inferior vena cava
- Portion of it enter liver microcirculation then hepatic vein then IVC
- IVC to right atrium has 70% saturation , 1/3rd of it goes through foramen ovale to left atrium mixes with pulmonary venous return then to ascending aorta with 65% saturation going to brain and upper body mainly
- Right ventricle outflow will have 55% saturation because the unsaturated blood from superior vena cava will mix with it this blood will go from right ventricle to ductus arteriousis mainly then descending aorta to the lower body
- Right ventricle --> pulmonary artery --> majority bypass lungs some go to lungs --> ductus arteriousis --> descending aorta then to the two umbilical arteries then placenta

After birth anatomical changes include

1) Elimination of placental circulation
2) Closure of ductus venouses and foramen ovale
3) "Gradual" closure of ductus arteriousis
4) Dilatation of pulmonary vessels and establishment of pulmonary circulation



Physiological changes in pregnancy

Progesterone has a relaxing effect on smooth m.

GI : -Appetite is unchanged.

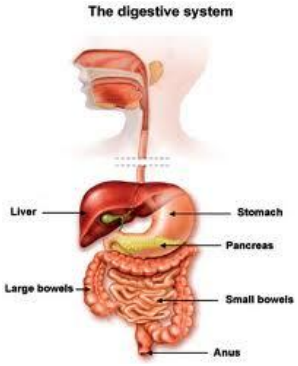
-Pica (dietary craving for nonnutritive substances) may happen.

-Mouth: -Excessive loss of saliva (Ptyalism).

-Gums become edematous and may bleed or result into gingivitis.

-Stomach: - ↓ Tone and motility from ↑ progesterone , ↓motilin.

- ↓ Tone of gastroesophagal sphincter from ↑ progesterone == Heartburn
- ↓ Acid secretion == ↓ Peptic ulcer disease
- ↑ Mucous secretion == ↓ Peptic ulcer disease
- ↑ Emptying half-time == ↑ Residual volume



-Small bowel: - ↓ Motility

- ↑ Iron absorption and others unchanged.

-Colon: - ↓ Motility from ↑ progesterone == Constipation.

- Water absorption increase up to 60%, and sodium absorption increase up to 45%.
- Hemorrhoids

-Gallbladder : - ↑ Emptying time

- ↑ Fasting & residual volume
- ↑ Biliary cholesterol saturation == Gallstone

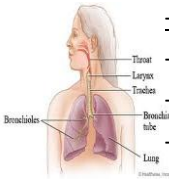
-Liver: - ↑ Alkaline phosphatase (from placenta not maternal liver production),Fibrinogen, Steroid-binding hormone , lipids(cholesterol),All gamma globulins

-↓ Albumin

- Nausea & Vomiting:- ↑ due to ↓ stomach tone and ↑ hCG

- Hyperemesis gravidarum frequently occurs in molar pregnancy

Respiratory: -Nose: Hyperemic ,edematous, stuffiness, epistaxis, polyps may develop



-Diaphragm is pushed up 4cm.

-All lung volumes are ↓ except Tidal volume is ↑ by 40%.

-All lung capacities are ↓ except Inspiratory capacity is ↑ and Vital capacity unchanged.

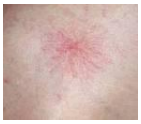
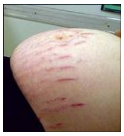
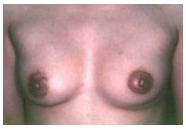
-RR remains unchanged== minute ventilation increase by 40%.

-"Hyperventilation due to progesterone " & "Dyspnea of pregnancy" may occur.

- O2 consumption increase by 20%.

-↓Pco2 & ↑Po2,pH .other arterial gasses aren't changed.

Skin: -Spider nevi , palmar erythema due to estrogen effect.

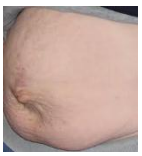
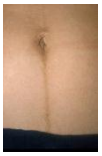




-Striae gravidum due to stretched skin

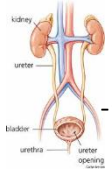
-Darkening nipples, areolae, umbilicus, perineum, linea nigra, malasma (cholasma) and pigmented nevi due to melanocyte stimulation.

-Hair loss.

-Separation of rectus m. (diastasis recti).

Urinary: -Kidneys, renal pelvis and ureters increase in size (Rt. more than Lt.)==Physiologic hydronephrosis. ==High risk of pyelonephritis if asymptomatic bacteriuria occurs.



-↑ Renal blood, GFR(50%), Creatinine clearance, Glycosuria(common) , Aminoaciduria and water-soluble vitamin excretion.

-↓Blood urea nitrogen, Serum Creatinine, Serum uric acid due to ↑ GFR.

- Proteinuria > 300 mg in 24 hrs. urine collection is abnormal.

CVS: -Heart lies more horizontally give the appearance of cardiomegaly.

-↑ CO,SV, HR and Lower extremity venous pressure in pregnancy.

-↓ PVR due to progesterone effect.

-CO is dependent on maternal position. Optimal CO when the mother in left lateral position and lowest when supine which may results into "supine hypotensive syndrome" .

-BP in normal pregnancy never higher than nonpregnant values. It is lowest when woman on her side and highest when seated.

- Normal changes **include** Systolic ejection murmur, exaggerated S1,S3 gallop, mammary soufflé.

- Changed ECG, Diastolic murmurs or changed S2 are never normal.

Hematologic: -↑ Plasma volume by 50%, RBC mass by 30%, WBCs(left shift),ESR and Coagulation factors (Fibrinogen, others) [Blood volume↑ by 50%] .

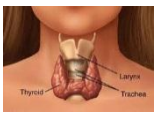
-↓Hb, Hematocrit and Platelets(but platelet count remain in the normal range, because it's just a slightly decreased).

-Physiologic anemia results from the disproportionate increase in the Plasma volume (50%) & RBC mass (30%)

-Pregnancy is a hypercoagulable state. Thromboembolism is the number one cause of maternal mortality. The risk of having Thromboembolism is higher during puerperium than pregnancy.

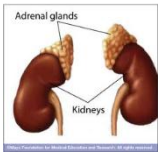
-The mean daily requirement of elemental iron for pregnant woman is 3.5 mg. Daily iron supplementation of 60 mg prevents development of iron deficiency anemia.

Endocrine: =Thyroid:- ↑ Size, Thyroid-binding globulin (TBG),Total and bound T3 and T4. Others unchanged including free T3,4.

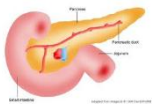


-Just Thyroid stimulating immunoglobulin, Thyroid releasing hormone(TRH) and Antithyroid drugs can cross the placenta.

=Adrenal: ↑ Total and free Cortisol, Aldosterone and Deoxy-corticosterone.



=Pancreas: -↑ Hypertrophy, Hyperplasia and Hypersecretion of Beta-cells.



-Maternal response to feeding are prolonged ↑ hyperglycemia, Hyperinsulinemia and Hypertriglyceridemia.

- Maternal response to fasting is accelerated starvation that could result in "Exaggerated Starvation Ketosis".

-Insulin & Glucagon can't cross the placenta BUT Glucose, Ketones, Amino acids, Free fatty acids can.

-Peripheral tissue resistance to insulin is **suggested** by :

1- Increased insulin response to glucose.

2- Decreased glucose uptake peripherally.

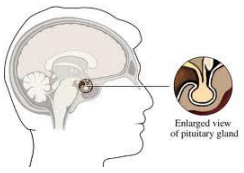
3- Decreased glucagon response.

-Insulin resistance in pregnancy **caused by**:

1- Human placental lactogen (HPL).

2- Placental insulinase.

3- Increased free cortisol and progesterone.

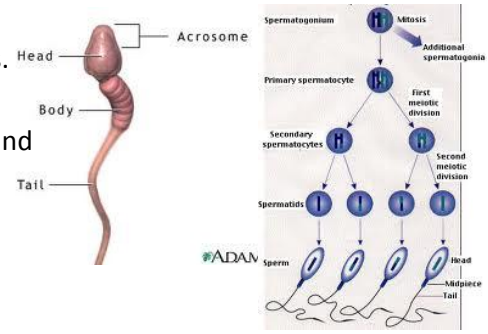


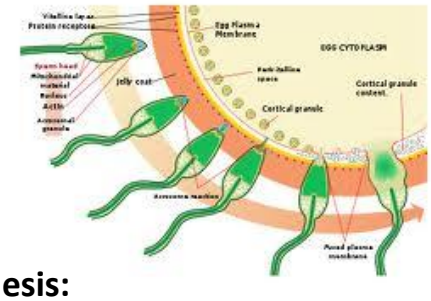
=Pituitary gland: -↑size by 100%.

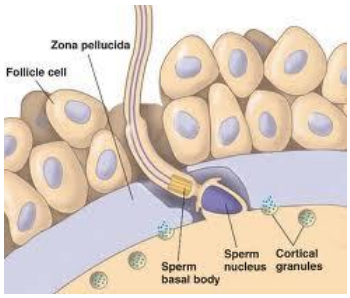
-Sheehan's syndrome due to severe PPH. Failure to lactate is characteristic to it.

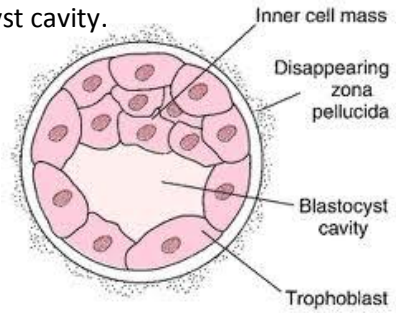
Reproductive biology

- **Spermatogenesis and fertilization**:
  - Spermatogenesis is in the seminiferous tubules of the testes
  - Diploid spermatogonia → mitosis → more spermatogonia → spermatocyte→ meiosis twice → 4 haploid spermatids → each becomes a sperm .
  - Sperm cells are stored in epididymis
  - Testes are also endocrine glands secreting testosterone by interstitial cells.
  - Spermatogenesis requires about 74 days.
  - Sperm composed of head (acrosome , nucleus), midpiece (mitochondria and single centriole) and a tail.
  - Average ejaculate from 2-5 ml of semen with around 300 million sperm
  - Within 5 min sperm reaches ovum in fallopian tubes.
  - Ova are usually fertilized within 12 hours of ovulation.
  - Acrosin helps in penetrating the zona , once the sperm nucleus incorporates to the ooplasm zona pellucida prevents any other sperm from going in, this process is called zona reaction.
  - Male pronucleus and female pronucleus form the diploid zygote! And determines the sex
  - Following fertilization cleavage occurs via mitotic division producing the morula , its outer cells secrete fluid making a cavity known as blastocyst cavity
  - Then a blastocyst is formed , it's made of trophoblasts , inner cell mass , blastocyst cavity.

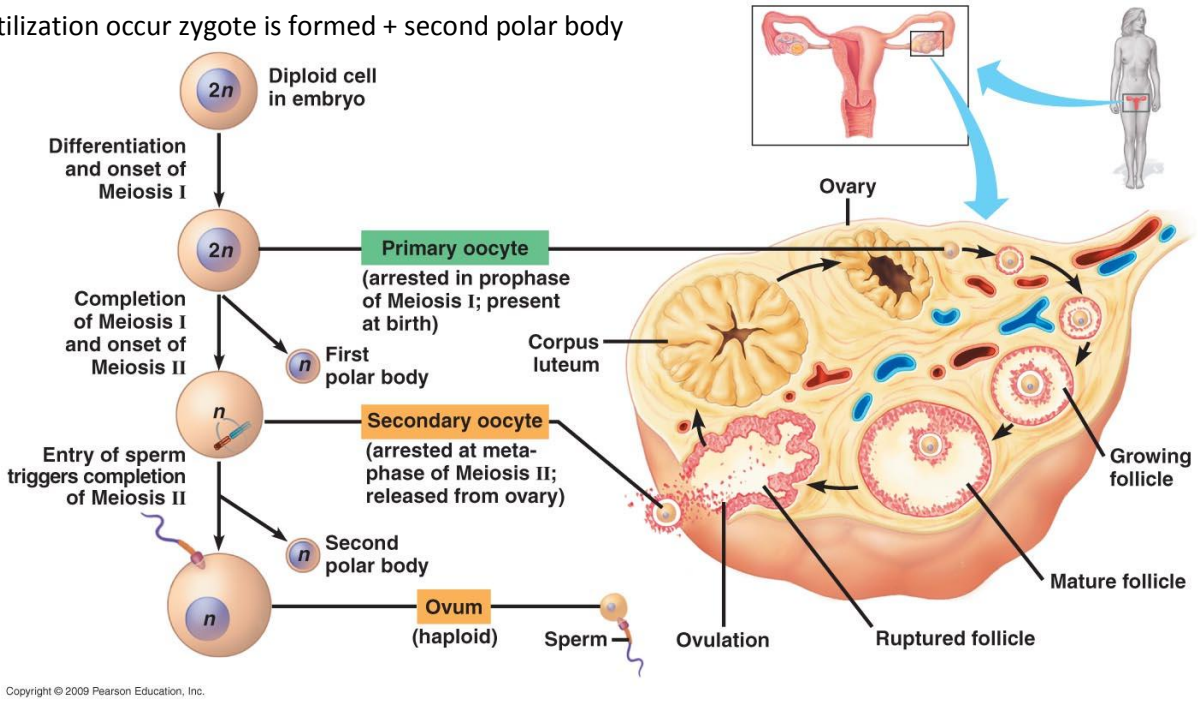




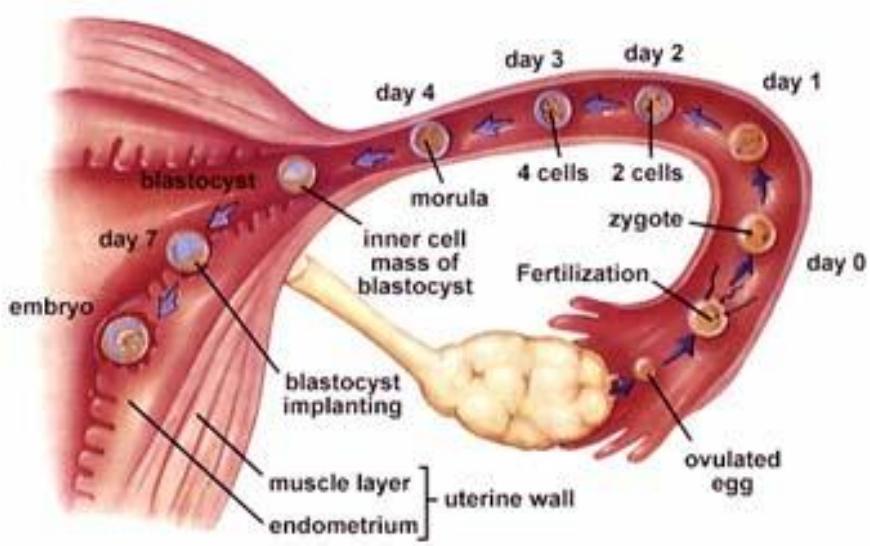


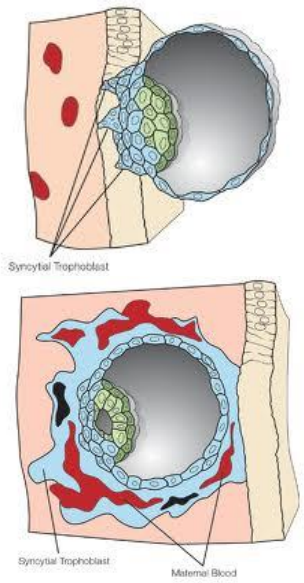


- **Oogenesis:**
  - Egg production prior to birth , by 20 weeks she will have 4 million
  - It happens by mechanism of oogonia mitosis then differentiate to primary oocyte
  - When born she will have 1 million left that started meiosis 1 then no further development till puberty
  - Each ovary at puberty contain 200 000 primary oocytes
  - At puberty each month primary oocyte completes meiosis 1 then forms secondary oocyte and first polar body
  - At ovulation, secondary oocyte stops at meiosis 2 until fertilization occurs
  - Fertilization occur zygote is formed + second polar body



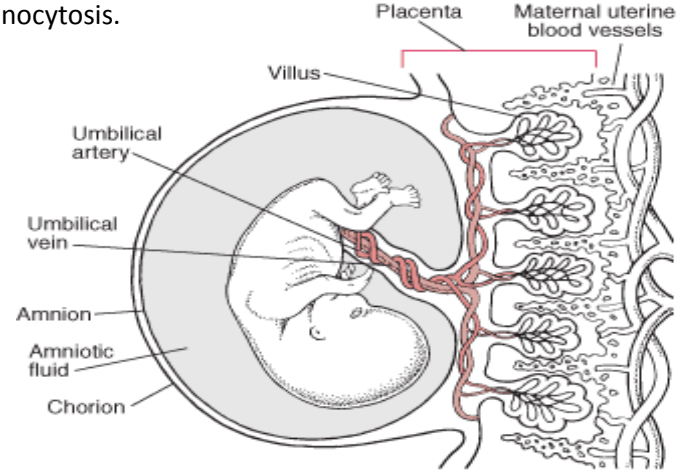
- **Implantation**:
  - Fertilized ovum reaches uterus 3 days after ovulation
  - Estrogen causes locking of egg in tubes
  - Progesterone E causes relaxation of isthmus of tube
  - Progesterone F stimulates tube motility
  - After reaching the uterus it goes through changes for an additional 2-3 days then it implants so in total around five to six days till it implants





- **Placenta :**
  - Fetal in origin.
  - The surface trophoblastic cells of the adhering blastocyst differentiate into → cytotrophoblast + syncytiotrophoblast
  - Syncytiotrophoblast secretes HCG which aids in maintaining the function of the corpus luteum to secrete estrogen and progesterone .
  - Further proliferation of trophoblast → finger Like projection (chorionic villi) →chorion frondosum → placenta
  - By day 17 fetal and maternal vessels are functional
  - By 70th day the placenta development is complete
  - Maternal blood flow through placenta increase from 300 ml/min at 20 weeks gestation to 600 ml /min at 40 weeks
  - Function of placenta as organ of respiration , nutrient transfer and excretion and hormonal synthesis (HCG +estrogen + progesterone +human placenta lactogen) .

- **Transfer of substances through the placenta :**
  - H_2O + O_2 + CO_2+Urea + Na + K + Vitamin A → Passive transport
  - Glucose + Polysaccharides + Protein + Fat + Vitamin B + Vitamin C + Iron + Phosphorus → active
  - IgG antibody → Passive + pinocytosis.



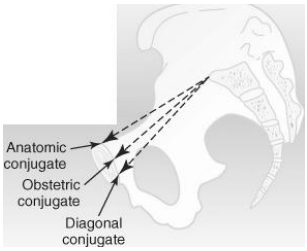
Antenatal care

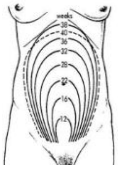
Aim: -To have a healthy infant by a healthy mother
-To prevent or manage conditions that cause maternal or fetal morbidities and mortality.

How to approach a pregnant lady came to you for her first visit at the Ante-natal care unit??

1-Taking a good Hx: -**See Hx**.

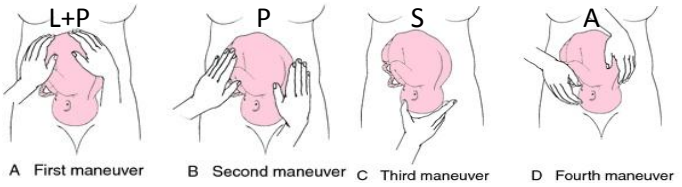
2-Physical Ex. (head to toe): -General physical Ex: -Baseline parameters (BP, Weight, PPW, and Height).
-Specific organ systems Ex.(Cardiac, Lung, Abdomen, Eye, Breast).
-Pelvic Ex: - Inspection & Palpation **see Ex .**
-BV screening by -Assessing vaginal pH(normal 4.5).
-Ex. vaginal discharge for clue cells.
-Pap smear
-Screening culture for Gonorrhea, Chlamydia, and GBBS.
-Clinical pelvimetry: -Pelvic inlet measured by diagonal conjugate.
-Midpelvis " " bi-ischial diameter.
-Pelvic outlet " " angle of the pubic arch.





-Fundal measurement, done if >20 weeks. **Number of cm.=Gestational age**.

3-Leopold's maneuvers: -1st to know the Lie & Presentation.
-2nd to know the Position.
-3rd to know the Station.
-4th to know the Attitude.



4-Dx of pregnancy: -Positive signs: -Fetal heart auscultation.
-Fetal movement recognized by external examiner.
-Radiographic imaging of fetus
-Sonogram imaging of fetus
-Probable signs: -Uterine enlargement
-Uterine softening (Hegar's sign)
-Uterine contractions
-Palpation of fetal parts
-Presumptive signs: -Amenorrhea
-Mastalgia and breast swelling
-Nausea and vomiting
-Skin changes, (Chadwick's sign, increased skin pigmentation, Abd. striae)

5-Antenatal laboratory tests:
-At first antenatal visit: -CBC for Anemia+ to know the base line of platelets .
-Blood type & Rh for risk of Rh isoimmunization.
-Atypical antibody screen for previous isoimmunization.
-Selected sickle cell test for sickle cell disease.
-Glucose Challenge Test (GCT) **if mother at high risk for GDM**. If not see below.
-Urine screen for Asymptomatic bacteriuria [midstream].
-Syphilis serology for infection.
-Rubella antibody titer for susceptibility to infection.
-HBsAg screen for active or carrier hepatitis.
-HIV screen for infection.
-Tuberculin skin test for TB infection.
-Toxoplasmosis antibody for infection.
-Pap smear for CIN.
-Later in pregnancy: -Maternal serum α-fetoprotein for NTD and Down syndrome (At 15-19 weeks).
-Triple marker screen for down syndrome (At 15-19 weeks).
-Glucose Challenge Test (GCT) for GDM (At 24-28 weeks).

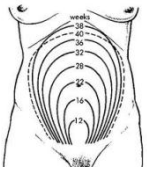
6-Antenatal follow up :
-Follow up should be done every 4wks if < 32wks, 2wks if 32-36wks , and 1wk > 36wks.

(What to do in each visit??):

a-Weight measurements: -Wt. gain should be < 4kg in total before the 20th week and < 0.5kg/week after that.
-The average total weight gain throughout pregnancy 11.5-13.5 kg.

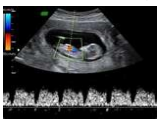
-Causes of failure to gain weight:-Dehydration(Severe hyper-emesis)
-Severe anorexia or bulimia
- Fetal death
-IUGR
-Oligohydramnios

-Causes of excessive weight gain: -Fluid retention (edema from PET)
-GDM (Macrosomia)



b-Urine glucose & protein measurements (dipstick).
c-BP (vitals).
d-Fundal measurement. If measurement is 3 cm more or less than expected, further evaluation is needed.
e-Fetal heart rate (normally 120-160 b/min) by fetal auscultation (special stethoscope or doppler).

Note: -Fetal heart activity can be seen by US from 6 weeks.



7-Patient education:-Exercise (not aggressive)
-Work (minimized)
-Sex (safe except in risk of incompetent cervix, preterm labor, PROM, Placenta previa)
-Danger signs: -Vaginal bleeding or fluid leakage(PROM).
- Abdominal pain(PET), cramping, decreased fetal movements or hyper-emesis.
-Swelling in face or fingers (PET)
-Cerebral disturbances (PET)
-Urinary complaint (cystitis, pyelonephritis)
-Chills or fever (infection)
-Seat belts wearing is recommended.

Antenatal diagnostic tests

Indications: - (see table)

Indications for antenatal diagnostic tests
=Hx of child with or family Hx of; -Birth defects -Mental retardation;-Fetal alcohol syndrome -Fragile X syndrome -Chromosome disorder -Known genetic disorder =Multiple fetal losses =Hx of unexplained neonatal death =Maternal factors; -Age > 35 years (trisomies 21,13,18) -Overt diabetes (CNS & cardiac anomalies) =Paternal factors; -Advanced age -Autosomal dominant mutations =Maternal & Paternal factors; -Balanced translocation -Aneuploidy -Mosaicism =Current pregnancy associated with; -Hx of teratogenic exposure (alcohol, drugs, radiation, chemicals, hyperthermia). -Abnormal Maternal serum α-fetoprotein screen (MSAFP) -Abnormal triple marker screen. -Abnormal fetus detected on ultrasound.

Tests: (see table)

Test	Timing	Test basis	Target	Risks
MSAFP	15-19 wks	Fetal AFP in maternal serum	-NTD -VWD -Trisomy 21	None
Triple marker screen	15-19 wks	-serum MSAFP -Serum estriol -Serum β-hCG	-NTD -VWD -Trisomy 21	None
Amniocentesis	≥ 15 wks	Desquamated fetal cells (amniocytes) in amniotic fluid are examined	-Cytogenetic disorders -Enzyme disorders	Spontaneous abortion(0.5% loss rate)
CVS	10-12 wks	Transvaginal or transabdominal aspiration of chorionic villus tissue	-Cytogenetic disorders -Enzyme disorders	-Fetal limb reduction defects if < 10 wks -Spontaneous abortion(0.7% loss rate)
PUBS	> 20 wks	Fetal blood from umbilical cord	Any blood test -Isoimmunization Dx & Rx -Karyotyping, -fetal infection -Blood genetic diseases -Fetal pH	-Spontaneous abortion (1-2% loss rate)
Sonography	18-20 wks	Assessment of fetal anatomy	gross structural anomalies	Non
Fetoscopy	15-20 wks	Transabdominal placement of fiber-optic scope into amniotic sac	-Visualize fetal anatomy -Biopsy fetal skin (Icthyosis Dx)	Spontaneous abortion (2-5% loss rate)

-MSAFP:

- AFP is synthesized by fetal yolk sac (GI tract + Liver)

-Test values **(see table)**

High MSAFP values(> 2.5 MoM)	Low MSAFP values
-Pregnancy dating errors (most frequent explanation) -Multiple fetuses -Placental bleeding -Open NTD -Ventral wall defects (gastroschisis, omphalocele) -Renal anomalies (polycystic ,absent kidney, congenital, nephrosis) -Fetal demise. -Sacrococcygeal teratoma.	-Pregnancy dating errors (most frequent explanation) -Trisomy 21

- Triple marker screen:

-Test values **(see table)**

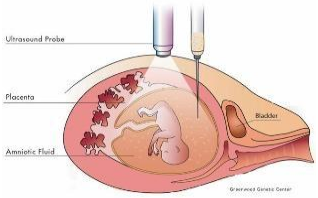
Triple markers	Trisomy 18	Trisomy 21
MSAFP	↓	↓
Estriol	↓	↓
β-hCG	↓	↑

-Amniocentesis:- Indications; -karyotyping

-RH-isimunisation

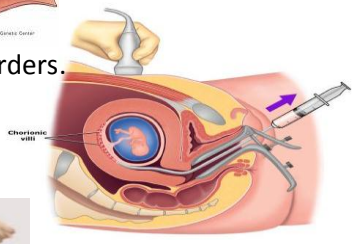
-Fetal lung maturity (L/S)

-Therapuetic in polyhydranios



-CVS: -It is the procedure of choice for first trimester prenatal diagnosis of genetic disorders.

-It is the earliest invasive test that can be used in pregnancy [10th week]



-Cordocentesis:-Indication; -Rapid karyotyping

-Diagnosis of inherited disorders

-Fetal HB assessment

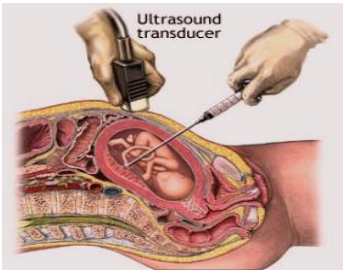
-Fetal plt level

-Fetal blood transfusion

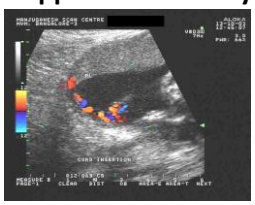
-Complication; -bleeding

-bradycardia

-infection



-Doppler velocimetry:-Used in the measurement of blood flow velocities in maternal & fetal vessels.

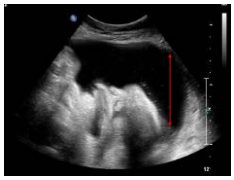


-Reflects fetoplacental circulation.

-Doppler studies is mostly valuable in IUGR.

-In IUGR absent or reversed EDF (end diastolic flow) associated with fetal hypoxia

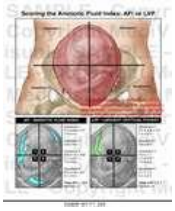
-Amniotic fluid index (AFI):- Definition; -The sum of the maximum vertical fluid pocket diameter in the 4 abdominal quadrants using US measurements.



- Values; -Normal 5-25cm.

-Oligohydramnios if < 5cm.

-Polyhydramnios if > 25cm.



Prevention of NTDs:-Give 0.4mg daily preconception for all women of reproductive age.

-Give 4mg daily 3 months preconception for high-risk women(DM, seizure disorder, Hx of NTD infant).

==========

Amniotic Fluid: -Sources: -Fetal urine output (main source after 1st trimester)

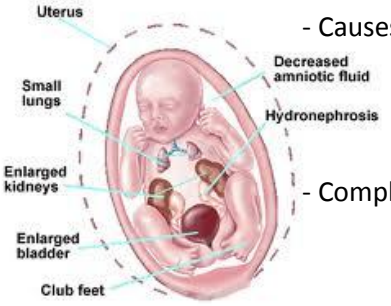
-Fetal lung fluid

-Placenta surfaces

-Fetal membranes

-Functions: -Protects the fetus from blunt trauma
-Provides space for fetal extremity & gross body movement.
-Provides space for chest wall breathing movements allowing lung development.
-Cushions the umbilical cord from compression.

-Oligohydramnios:- Definition; -either -inadequate volume of amniotic fluid (AFI< 5cm).
-a four-quadrant AFI < 5cm.



- Causes;-PROM
-Fetal urinary tract anomaly (renal agenisis, obstructive lesions)
-Maternal medications [ACE inhibitors(-pril) , Indomethasone (NSAID)]
-Placental insufficiency (Preeclampsia)
- Complications; -Musculoskeletal deformations (Clubfoot)
-Pulmonary hypoplasia
-Umbilical cord compression
-IUGR (in 60%)

-Polyhydramnios:-Definition; -either -excessive amniotic fluid (2L).
-a single amniotic fluid vertical pocket > 15cm.
-a four-quadrant AFI > 25cm.

-Classification;-Acute polyhydramnios: -Start before 24 weeks'
-Has ↑ incidence of fetal anomalies & perinatal loss.
-Chronic polyhydramnios: -Start after 30 weeks'
-No identifiable etiology.
-Perinatal outcome is good.
- Causes;-↓ fetal swallowing;-Anencephaly
-Spina bifida
-Arthrogryposis
Note:- fetus normally swallows up to 1.5 L/day.
-Fetal GI obstruction;-Doudenal atresia
-Tracheoesophageal fistula
-Twin-twin transfusion syndrome (recipient twin)
-Others with unknown mechanism; -Cystic adenomatoid malformation
-Maternal DM
-Fetal hydrops (immune, nonimmune).

Extra notes:

-Ultra sounds: 1- viability -2-gestational age + placenta localization +anomaly scan + cardiac evaluation +amniotic fluid.
-A nuchal translucency ultrasound measures the fluid at the back of a baby's neck **between 11 and 14 weeks** of pregnancy for Trisomy exclusion.
-At 13-14 weeks for anencephaly.
-At 20-24 weeks for anomaly scan.
-At 24 weeks for cardiac evaluation.

BV=Bacterial vaginosis , PPW=Pre-pregnancy weight
MSAFP = Maternal serum α-fetoprotein screen , CVS =Chorionic villus sampling.
PUBS =Percutsneous umbilical blood sampling , NTDs =Neural tube defects
VWD = *Von Willebrand disease* , AFP = α-fetoprotein

Antepartum Fetal Assessment

Note: -Antepartum fetal assessment is performed when the gestational age of fetal viability is reached (24th week)**.**

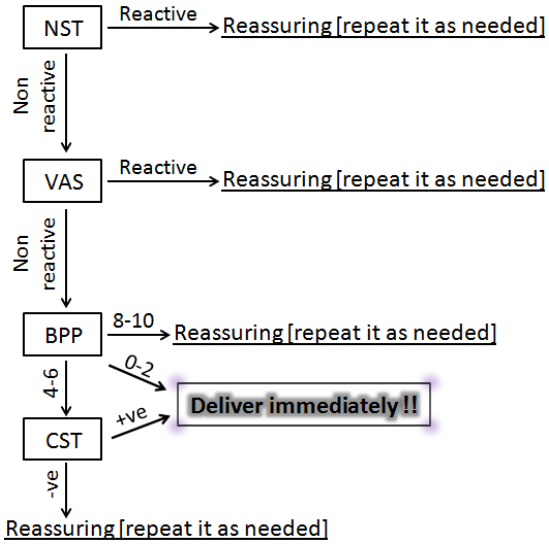
Purposes: -Identifying potential fetal compromise.
-Assessing fetal well-being in high-risk conditions **(See figure)**.

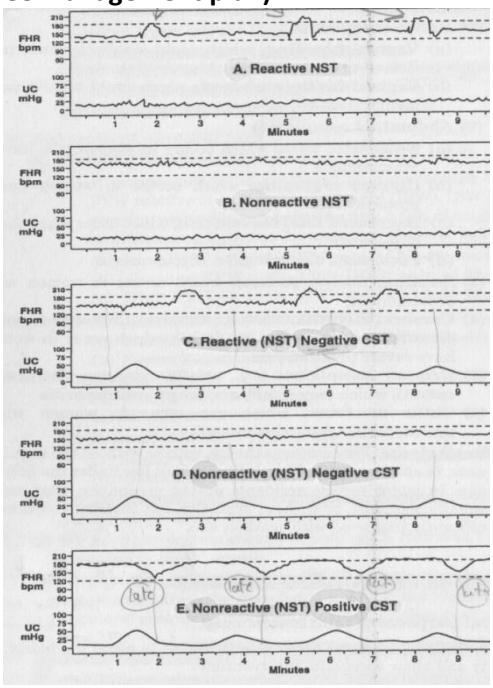
Maternal indications	Pregnancy related indications
-Type 1 DM	-Preeclampsia
-Hypertensive disorders	-↓ fetal movement
-Poorly controlled hypertension	-Oligohydramnios
-Cyanotic heart disease	-Polyhydramnios
-Chronic renal disease	-IUGR
-Hemoglobinopathies	-Postterm pregnancy
-SLE	-Isoimmunization
-Antiphospholipid syndrome	-Hx of unexplained fetal demise
	-Multiple gestation

Methods: a-Fetal kick count (FKC);

=Assumption: -Adequately oxygenated fetus moves its body & limbs.
-Compromised fetus fetal movements gradually diminish (early indication).
-Mother's perception of fetal movements is accurate.
=Method:-The mother is instructed to keep daily records of fetal movement.
-Abnormal count if -10 movements don't occur in 12 hours.
or -10 movements take twice time to occur as it did 1 week earlier.
=Interpretation: -Normal counts are highly reassuring **but** abnormal counts are poor predictors of fetal compromise.
=Recommendation: -It is just used as adjunctive fetal assessment method in normal patients.
-If abnormal counts occurred, do NST. **(See management plan)**.

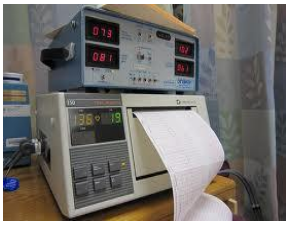
Note:-Management plan in suspicion of fetal compromise:- **(see algorithm)**





b-Nonstress test (NST); (see CTG)

=Definition: -Any electronic fetal monitor (EFM) strip with a FHR tracing.
=Assumption: -Adequately oxygenated fetus moves its body & limbs.
-Fetal movements are accompanied by FHR accelerations (after 30 weeks' gestation).
-Lack of FHR accelerations may occur with;-**(See table)**



Lack of FHR
-Fetal hypoxia
-<30 weeks' gestation age.
-Fetal sleeping.
-Fetal CNS anomalies.
-Maternal sedative or narcotic administration.

=Method:- Fetal movements & FHR are recorded (avoid supine hypotension).
-FHR accelerations are ↑ of **≥ 15** beats/min over baseline for **≥ 15** sec.
=Interpretation:-Reactive [reassuring];-If 2 accelerations noted in 20 min.
-Nonreactive [nonreassuring];-If either;- <2 accelerations in20 min.
- ↑ of **< 15** beats/min over baseline.
- ↑ for **< 15** sec.

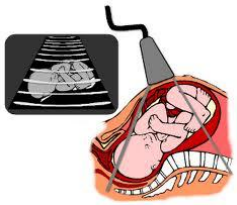
=Recommendation: **(see algorithm above)**

b- Vibroacoustic stimulation (VAS);

=Definition: -The application of a vibratory sound stimulus to the abdomen of a pregnant woman.
=Assumption: -A healthy fetus will accelerate its heart rate in response to vibratory sound stimulus.
=Interpretation:-Present FHR accelerations reliably predicts the absence of fetal metabolic acidemia.
=Recommendation: **(see algorithm above)**

c-Biophysical profile (BPP);

=Definition: -A scoring system that evaluates fetal well-being.
=Assumption: -A healthy fetus moves, thereby accelerating its heart rate.
-Adequate fetoplacental blood flow results in normal amniotic fluid volume.
-Compromised Fetus has the opposite: ↓ movements,↓ heart rate &↓ amniotic fluid volume.
=Method:- Types; -Complete BPP involves 5 components;



1- Fetal movement (body) [using US]
2- Fetal tone (extremity) [" "]
3- Fetal breathing [" "]
4- Amniotic fluid volume (pockets) [" "]
5- Fetal Heart Rate [using NST]

-Modified BPP involves 2 components; 1- Amniotic fluid volume (pockets) [using US]
2- Fetal Heart Rate [using NST]

=Note: - **NST** is the most predictive for immediate assessment of placental function.
- **Amniotic fluid volume** is the most predictive for long term assessment of placental function.

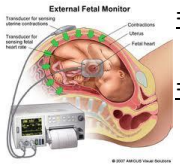
=Interpretation:- **(see tables)**

BPP parameter	Score = 2 points	Score = 0 points
-Fetal movement	≥ 3 discrete movements	≤ 2 discrete movements
-Fetal tone	1 extension / flexion	None
-Fetal breathing	1 episode of ≥ 30 sec	No breathing
-Amniotic fluid volume	≥ 1 × 1 cm pocket	Oligohydramnios
-Fetal Heart Rate	Reactive NST	Nonreactive NST

Total score	Test result	Perinatal mortality/1000 within 1 wk
0	+ve = fetal jeopardy	600
2	+ve = fetal jeopardy	120
4	Equivocal	90
6	Equivocal	50
8 or 10	-ve = fetal well-being	< 1

=Recommendation: **(see algorithm above)**

d-Contraction stress test (CST); (see above CTG)



=Definition: -Any electronic fetal monitor (EFM) strip with both a FHR tracing & UC. It can evaluate both NST & CST.
=Assumption: -UC diminish the flow of oxygenated intervillous blood to the fetus.
-Fetus with adequate metabolic reserve FHR remains stable even with transient O_2 deprivation in UC.
-A compromised fetus displays late decelerations (persisting after a UC).
=Method:-FHR & UC are recorded (avoid supine hypotension).
-If there are no spontaneous UC, it can be induced by ; -Nipple stimulation.
-IV Oxytocin infusion.
-#Goal is > **3** UCs in **3**0 min.

=Contraindication:-**(See table)**

Contraindications
-Hx of classic C/S.
-Hx of myomectomy entering uterine cavity.
-PROM.
-Incompetent cervix.
-Placenta previa.

=Interpretation:-Negative (-ve) CST; -No late decelerations.
-Highly predictive of fetal well-being (adequate placental O_2).
-Positive (+ve) CST; -Repetitive late decelerations with **3** consecutive UC in **10** min.
-Highly predictive of fetal compromise[fetal metabolic acidemia].
-Equivocal (worrying) CST; -Nonrepetitive late decelerations.
-Indicates incipient fetal compromise
-It is called Reactive-negative CST if there is negative CST with reactive NST.
-Also called Nonreactive-negative CST if there is negative CST with nonreactive NST.

False positivity if used alone:- FKC = 80% , - NST = 80% , - BPP = 30% , -CST = 50%

FKC=Fetal kick count, FHR=Fetal heart rate., UC=Uterine contractions., US=Ultra sound., EFM=Electronic fetal monitor

Neonatology

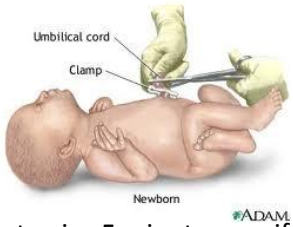
❖ **The neonates:**

A) Prematurity leading cause of poor neonatal outcome

B) Cortisol levels increase in fetus days before labor inducing conversion of t4 to t3 thyroid hormones , at birth there is a surge of TSH followed by hyperthyroid neonatal state necessary to maintain its body temp

C) Fetal breathing is rarely observed once labor is established associated with pulmonary fluid decrease

D) Labor stimulates catecholamines release which mobilizes glucose and leads to fluid absorption and helps in onset of respiration



❖ **Facilitating the neonatal adaptation:**

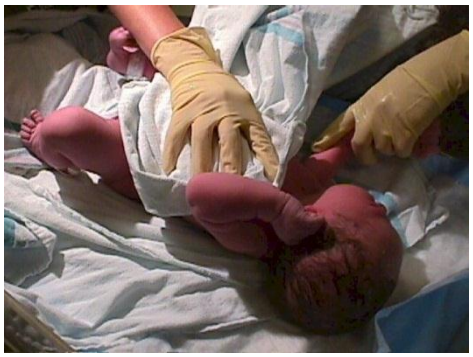
A) Clear airway by suction of oropharynx (meconium), never apply suction to nose may initiate bradycardia!!!!

B) Dry the baby , stimulates respiration

C) Clamp cord , umbilical arteries close seconds after birth , but veins 5 minutes , so if you don't clamp within 20-30 seconds after delivery baby will have increased amount of blood leading to jaundice and tachypnea.

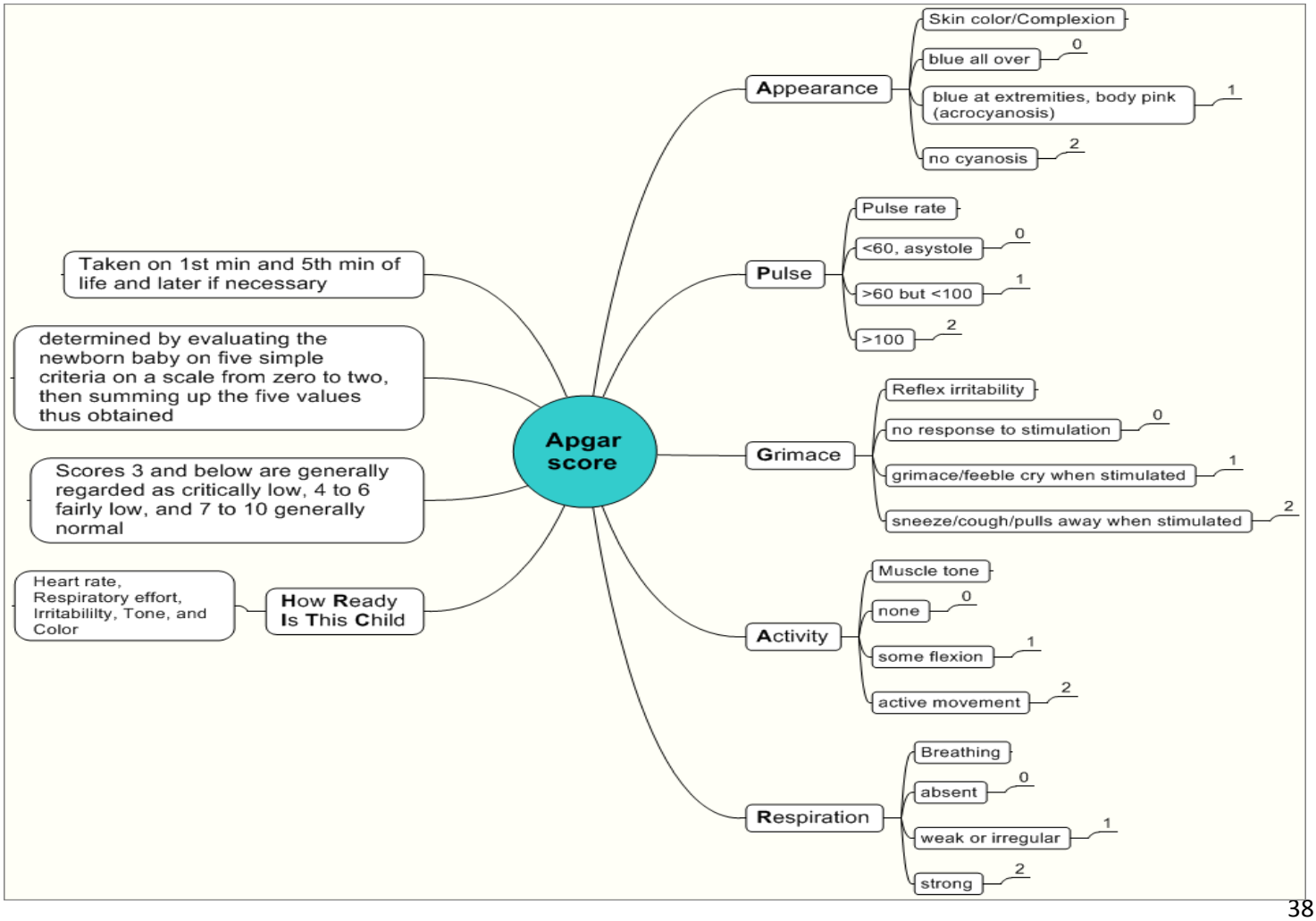
D) Onset of respiration around 30 seconds or earlier after delivery

E) Correct surfactant deficiency in premature infant by tracheal injections of surfactant to prevent respiratory distress syndrome.



❖ **Apgar score :**

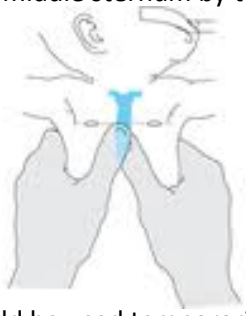
- Assesses the new born soon after birth at 1 minute and 5 minutes.
- Normal at one minute score is 7 at five minutes score is 9-10.
- **It is based on the following :**
  1) Heart rate.
  2) Respiratory rate.
  3) Color.
  4) Muscle tone.
  5) Reflex irritability.



❖ **Asphyxiated baby:**

- **Remember your** ;
  - **A=** Airways.
  - **B**= Breathing.
  - **C=** Circulation.
  - **D=** Drugs.

- Provide warmth, position head & clear airway as necessary, dry, and stimulate the baby to breathe **[A]**.
- You have **30 seconds** to try **[A]** before moving to **[B]**.
- Initiate breathing by endotracheal tube with 100% oxygen , normally heart rate goes back to normal and increases after correction of apnea **[B]**.
- You have **30 seconds** to try **[B]** before moving to **[C]**.
- If cardiac performance is still poor best way is to massage middle sternum by two fingers twice per second for three times then give 2-3 seconds for ventilation **[C]**.
- You have **30 seconds** to try **[C]** before moving to **[D]**.
- If massage still didn't work give epinephrine **[D]**.



❖ **Acidosis correction**

- Umbilical artery catheter, and infuse sodium bicarbonate.

❖ **Anemia correction:**

- Immediate blood transfusion , solution of 5% albumin could be used temporarily.

❖ **Narcotic depression:**

- Use naloxone intramuscularly.

❖ **Hypoglycemia:**

- Infuse glucose , but its contraindicated in case of asphyxiated babies because it will lead to anaerobic metabolism and lactic acidosis.

✓ **Low birth weight and IUGR have risk for cerebral palsy.**

Multiple gestation

Definition: is a pregnancy with 2 or more fetuses or embryos existing simultaneously.

- Mean age for delivery of twins is approximately 36 weeks , triplets 33 weeks , quads 29 weeks.

Incidence:

- Twins are twice as common in women over 35 years of age
- Incidence of multiple gestation with using clomiphene was 6%
- Incidence of multiple gestation with using gonadotropin therapy was 30%

Etiology and classification of twinning:

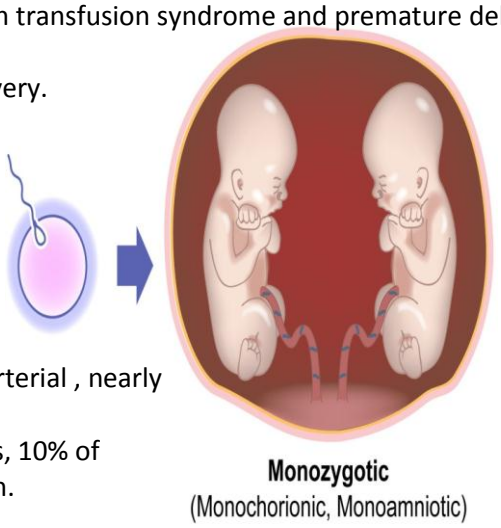
1. Splitting of the embryo monozygotic
2. Fertilization of two or more eggs dizygotic , each has own amnion chorion and placenta.

In monozygotic twins account 1/3 of all twins(identical)

- The earlier the embryo splits the more separate the membranes and placenta will be;
  - 0-72 hours diamniotic (2 sacs with a septum) dichorionic(two placentas)
  - 4-8 days diamniotic, monochorionic (one sac, no septum), this is most common monozygotic 69% of them.
  - 9-12 days monochorionic monoamniotic.

-Monozygotic are more likely to have congenital anomalies, twin-to-twin transfusion syndrome and premature delivery

- Ultrasound very helpful
- Confident diagnosis of zygosity is after detailed examination after delivery.



Monozygotic
(Monochorionic, Monoamniotic)

Dizygotic twins (fraternal)

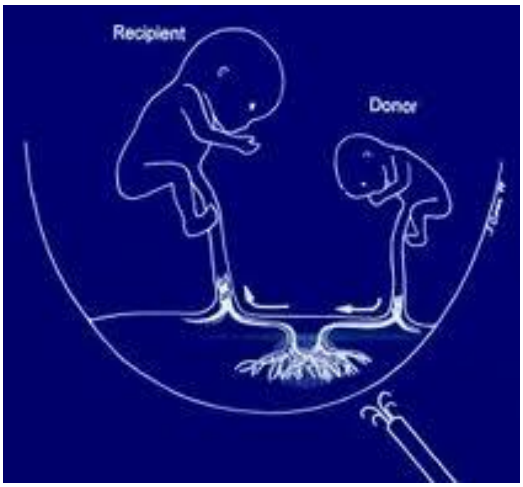
Two eggs and represent 2/3 of all twins.

Abnormalities in twinning:

- Conjoined twins are due to incomplete cleavage of embryo, thoracopagus most common type →must be monoamniotic.
- Interplacental vascular anastomosis , most commonly arterial-arterial , nearly always in monozygotic
- Twin-twin transfusion syndrome [TTTS], typically arterial venous, 10% of monozygotic twins, donor arterial goes to venous recipient twin.
- Umbilical cord abnormalities.
- Retained dead fetus syndrome, if less than 12 weeks the fetus will be resorbed, if more fetus will shrink and becomes dehydrated and flattened [**fetus papyraceus].**

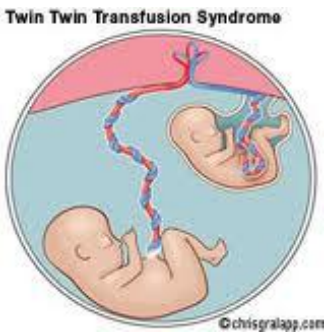
In twin-to-twin transfusion syndrome

- Complications of donor fetus hypovolemia hypotension IUGR oligohydroamnios anemia.
- Complications of recipient twin are hypervolemia, hydramnios, hypertension, polycythemia, cardiomegaly, congestive heart failure, thrombosis, and edema.



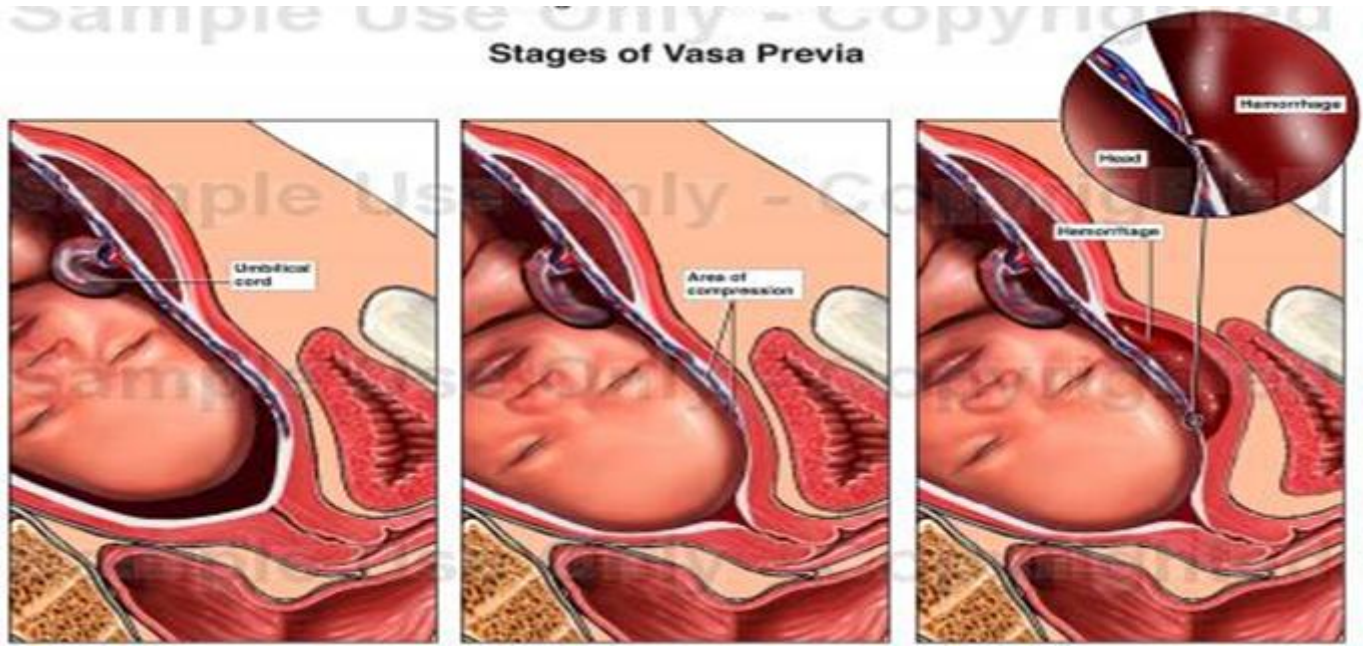
Diagnosis is by

1. Excessive fetal movement
2. Excessive weight gain
3. Excessive fundal growth
4. And definite by sonographic two separate fetuses and heart activities



Fetal malformation: three times higher in multiple pregnancy than in single, and highest with monozygotic who share one placenta (later cleavage, the higher risk).and lowest with dizygotic.

Umbilical cord abnormalities: two-vessel cord, velamenayous insertion, Vasa previa.
- Monozygotic has the highest risk, and dizygotic is the lowest.



Cord entanglement → risk is only with monoamniotic who must be monozygotic and monochorionic could lead to death.

Maternal risks:
1- Nutritional anemia (iron deficiency, folate deficiency), occur twice in multiple pregnancy **(MP)**.
2- Pregnancy induced hyper tension→occur three times more in MP.
3- Preterm labor: in 50% of MP.
4- Postpartum uterine atony with hemorrhage.
5- C/S performed in 50% of MP.
6- IUGR: in 50% of twins.
7- Malpresentation: occur ten times more.

Presentations:
- **Vertex-vertex** (cephalic-cephalic) most commonly 50% of time.
  Vertex-breech (cephalic-breech) 30%.
- **Breech-vertex** (breech-cephalic) 10%.
- **Breech-breech.**
- **Single or double transverse**.

Antepartum management:
First and second trimester
Cervical assessment every 2 weeks to check cervix, Incompetent cervix risk, and cerclage suture maybe used
Women with twins can gain up to 20.5 kgs!!

Intrapartum management:
- If the first baby is vertex (cephalic) deliver them **vaginally**.
- If the first baby is not vertex (cephalic) deliver them by **C/S**.
- If they were monochorionic monoamniotic deliver them by **C/S**.
- **E**xternal **f**etal **m**onitoring of heart rates of both fetuses should be performed.

Postpartum care:
- Give IV oxytocin as soon as the placenta(s) is (are) delivered to prevent uterine atony PPH.

Abortion

- **Abortion** is defined as the termination of pregnancy prior to viability (Viability ≥ 500 g or ≥ 24 weeks).
- Is the commonest gynecological & obstetric disorder
- The most common cause of 1st & 2nd trimester bleeding is abortion.
- About 15% of clinically recognized pregnancies end in abortion
- Most abortions occur between 8 and 12 weeks (first trimester) of pregnancy.
- **Etiology** :

1. Implantation : in 20% of miscarriages the trophoblast has failed to implant adequately
2. Chromosomal abnormality is the **most common** etiology (trisomy 16 is the most of them other example is monosomy)
3. Blighted ovum **(discussed later)**
4. Maternal cause (SLE, septic ,DM, thyroid disorder Maternal shock , hypoxia , smoking ,uterine abnormality , uterine fibroid, cervical incompetence , genital tract infection or psychological factor) → increase in 2nd trimester .
5. Multiple pregnancy

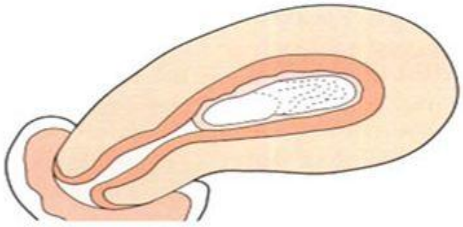
- **Types : (see table)**

1. Missed abortion.
2. Threatened abortion.
3. Inevitable abortion.
4. Incomplete abortion.
5. Complete abortion.
6. Septic abortion.
7. Recurrent abortion.

Type of abortion	Viable Embryo?	Patient bleeding?	Patient cramping?	Open cervical internal os?	Tissue passed?	Patient febrile?	Rx mainly
Missed abortion	No	No	No	No	No	No	Suction D&C
Threatened abortion	Yes	Yes (minimal)	Yes (mild)	No	No	No	Observe
Inevitable Abortion	Yes/No	Yes (profuse)	Yes (severe)	Yes	No	No	ER Suction D&C
Incomplete Abortion	--	Yes (profuse)	Yes	Yes	Yes	No	ER Suction D&C
Completed abortion	--	Yes (minimal)	Yes (mild)	Yes	Yes	No	-R/O EP -Observe
Septic abortion	--	Yes/No	Yes/No	Yes	Yes	Yes	-Antibiotics -Gentle Suction D&C

☒ Missed abortions :

- Most of missed abortions are diagnosed accidentally during routine U/S in early pregnancy.
- In some cases there may be a past history of :
  1- Regression of early symptoms of pregnancy.
  2- Stop of fetal movements after 20 weeks gestation.
  3- Threatened abortion or bleeding during pregnancy.
- **Ex** : The uterus may be small for date
- **U/S**: no evidence of heart activity.

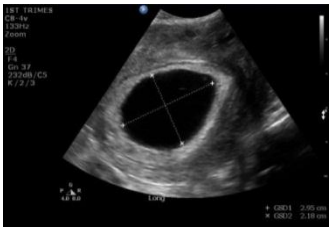

Missed miscarriage

- **Management** :
  1. CBC (Platelets count+ hemoglobin), blood grouping, blood cross match.
  2. Coagulation profile → to exclude DIC
  3. **NB :** DIC does not occur before 5 weeks of missed abortion or IUFD and if occurred will be of mild grade
  4. Options of treatment (let the patient choose)
     A. **Conservative** treatment: → if left alone spontaneous expulsion will occur +monitoring of DIC
     B. **Surgical** evacuation of the uterus; by D & C:Indicated in **1st trimester** missed abortion
     C. **Medical** termination of pregnancy**:** by Misoprostol (PGE1)

  - **Cytotec (misoprostol):**
    - Indicated in **1st & 2nd trimesters** missed abortions.
    - Cytotec vaginal (is the best) or oral tab
    - Subsequent surgical evacuation is needed in cases of retained products of conception (RPOC)

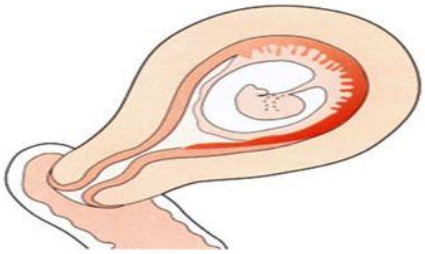
  5. Post-abortion management **(discussed later)**

☒ Anembryonic pregnancy [Blighted ovum]



- It is due to an early death and resorption of the embryo with the persistence of the placental tissue.
- Diagnosed by U/S which shows empty gestational sac.
- Management as missed abortion.

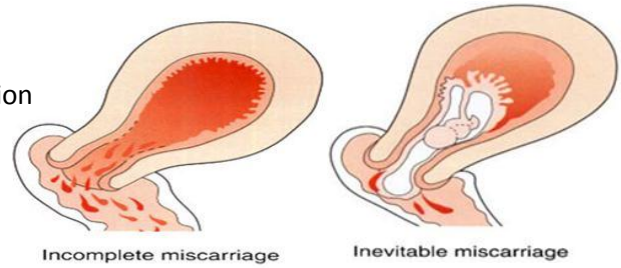
☒ Threatened abortion



Threatened miscarriage

- **Hx:** Mild vaginal bleeding with No abdominal pain or just mild abdominal pain**.**
- **Ex :** Good general condition + The cervix is **closed** + The uterus is usually correct size for date
- **U/S** which is essential for the diagnosis. Showed the presence of fetal heart activity.
- **Management :**
  - Reassurance if fetal heart activity is present (> 90% of cases will be progressed satisfactorily)
  - Non-sensitized rhesus-negative women should receive anti-D immunoglobulin if threatened miscarriage occurs after at least 12 weeks of pregnancy.

☒ Inevitable and incomplete abortions



Incomplete miscarriage Inevitable miscarriage

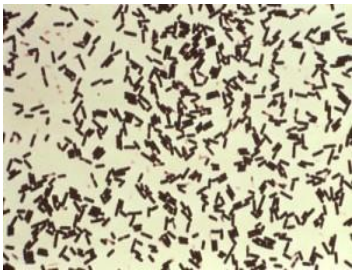
- **Hx :**
  - Heavy vaginal bleeding with strong uterine contraction
  - with **no** passage of products conception (inevitable)
  - with passage of products conception (incomplete)
- **Ex :**
  1. Poor general condition
  2. The cervix is dilating and products of conception maybe passing through the os
  3. The uterus may be the correct size for date (inevitable abortion) or small for date (incomplete abortion)
- U/S → Fetal heart activity may or may not present in inevitable abortion or in incomplete abortion.
- **Management :** 1- CBC , blood grouping , blood cross match
  2- Resuscitation →large IV line, fluids & blood transfusion.
  3- Ergometrine 0.5 mg IM + Oxytocin infusion (20-40 units in 500 cc saline)
  4- Evacuation & curettage
  4. Post-abortion management **(discussed later)**.

Complete abortion

- **Hx :**
  1. Heavy vaginal bleeding →which has been stopped.
  2. Lower abdominal pain which follows the bleeding →which has been stopped.
- **Ex:** The cervix closes gradually.
- **U/S :** (transvaginal) showed empty uterine cavity
- **Management :**
  1. Ruling out ectopic pregnancy via transvaginal sonography & ensuring no trophoblastic remain by serial β-hCG titers.
  2. Cautious observation.
  3. If there is RPOC evacuation & curettage is needed.
  4. Post-abortion management **(discussed later)**.

Septic abortion

- It is an incomplete abortion which complicated by infection of the uterine contents.
- This may be due to criminal interference [illegal abortion].
- **Hx :**



1. Include the features of incomplete abortion: severe vaginal bleeding with passage of product of conception, with or without history of evacuation.
2. Features of pelvic infection: pyrexia , tachycardia , general malaise , lower abdominal pain, pelvic tenderness & purulent vaginal discharge.

- Bacteriology : The commonest organisms are :
    1. Gram -ve : E.coli , streptococcus & staphylococcus
    2. Anaerobics : Bacteroids

: Rarely Cl. tetani , which is potentially lethal if not treated adequately

- **Types :**

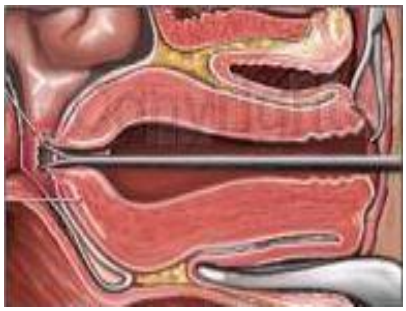
1. **Mild** → the infection is confined to decidua : 80%
2. **Moderate** →the infection extended to myometrium15%
3. **Severe** →the infection extended to pelvis + Endotoxic shock + DIC 5%

- **Management** :

1. CBC , blood grouping , blood cross match
2. Cervical swabs (not vaginal) for culture and Antibiotic sensitivity
3. Coagulation profile , serum electrolytes & blood culture if pyrexia > 38.5
4. Antibiotics : I.V + Metronidazole I.V
5. Surgical evacuation of uterus → usually 12 hours after antibiotic therapy (until a reasonable tissue levels of antibiotics have been achieved)
6. Post-abortion management **(discussed later)**

Complications of abortion

1. **Hemorrhage**
2. **Complication related to surgical evacuation: E&C and D&C**.
    - Uterine perforation (see the Pic) **-** which may lead to rupture uterus in the subsequent pregnancy.
    - Cervical tear & excessive cervical dilatation **–** which may lead to cervical incompetence.
    - Infection **–** which may lead to infertility
    - Excessive curettage **–** which may lead to Asherman's syndrome



3. **Rh- isoimmunization** → if the anti –D is not given or if the dose is inadequate.
4. **Maternal depression after fetal lost**

Post-abortion management

In cases of incomplete, inevitable, complete, missed & septic abortions

1. Support: from the husband, family& obstetric staff
2. Anti D – to all Rh –ve, non-immunized patients, whose husbands are Rh +ve
3. Counseling & explanation:
    - **Contraception** : <u>Should start immediately after abortion</u> if the patient choose to wait , because <u>ovulation can occur 14 days after abortion</u> and so pregnancy can occur before the expected next period .
    - Best to wait for 3 months before trying again. This time allow to regulate cycles and to know the LMP and to give folic acid .

Recurrent abortion

- Is defined as ≥ 3 consecutive spontaneous abortions.
- **Types :**

1. **Primary** : All pregnancies have ended in loss
2. **Secondary**: One pregnancy or more has proceeded to viability (>24 weeks gestation) with all others ending in loss.

- Occurs in about 1% of women of reproductive age .
- Idiopathic recurrent abortion, **in about 50%,** in which no cause can be found .
- **The known causes : see the table**
- **Management :**

▪ in idiopathic recurrent abortion :

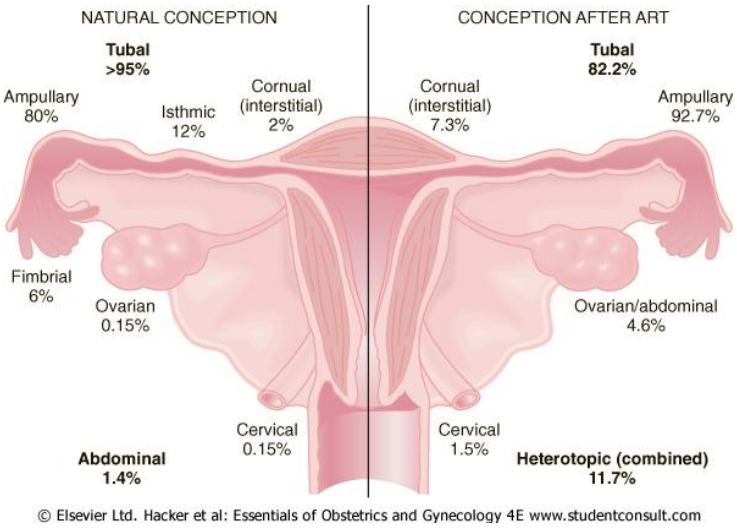
1. **Support** : from husband, family & obstetric staff.
2. **Advice *:*** stop smoking & alcohol intake, decrease physical activity
3. **Drug therapy**
   - Progesterone & hCG: start from the luteal phase & up to 12 weeks.
   - Low dose aspirin start from the diagnosis of pregnancy & up to 37 weeks
   - LMWH start from the diagnosis of fetal heart activity & up to 37 weeks

▪ In the presence of a cause treatment is directed **to control the cause .**

Causes of recurrent abortion	
Fetal	chromosomal abnormalities & structural abnormalities
Anatomical disorders	Cervical incompetence submucosal fibroids, uterine anomalies & Asherman's syndrome
Medical disorders	diabetes , thyroid disorders , PCOS , SLE, antiphosphlipid syndrome , Thrombophilia & Rh – isoimmunization
Infections	CMV & Bacterial vaginosis

Ectopic Pregnancy [EP]

- **Ectopic pregnancy :** Implantation outside uterine cavity
- It is the most common **cause** of pregnancy-related death in the first trimester.
- The vast majority of ectopic pregnancies implant in the Fallopian tube (oviduct) [98%]
    - The ampullary section (80%)→ wide tube→the rupture occur more late than other
    - The isthmus (12%) →narrow tube →the rupture is more common than others.
- **Incidence:** 1 in 100 of all pregnancies and increase to 1 in 30 in high risk population arising in the west in parallel with increased number of cases of chlamydia infection.



- **Risk Factor for Ectopic Pregnancy & DDx : (see table)**

Risk factors for EP
-Hx of PID and chlamydia infection [×8 times]
-Hx of EP [×2 times]
-Hx of tubal ligation
-Hx of tubal surgery
-IUD
- Multiple sexual partners
-Prolonged infertility
-Diethylstilbestrol (DES) exposure in utero.
-Smoking.

Reproductive DDx	Nonreproductive DDx
-PID (Salpingitis)	-Acute appendicitis
-Ovarian torsion	-Pyelonephritis
-Abortion	-Pancreatitis
-Molar pregnancy	-Diverticulitis
-Rupture of corpus luteum or follicular cyst	-Ulcerative colitis
-Delay menstrual cycle	-Perforation of peptic ulcer
-Fibroid red degeneration	

- In case of intrauterine device when the pregnancy occurs → the likely is Ectopic.
- In rare cases of ectopic pregnancy, there may be two fertilized eggs, one outside the uterus and the other inside. This is called a **heterotopic pregnancy**.
- Higher Mortality at →the isthmus or within interstitial cornual part → as there is increased vascularity → hemorrhage.
- **Classic symptom with unruptured ectopic pregnancy:**
    1. Amenorrhea (6 weeks)
    2. Abdominal pain (constant and cramp like)
    3. Abnormal vagina bleeding.
- **Classic signs: -**Adnexal or cervical motion tenderness and Adnexal mass.
- 25% of cases presents without any vaginal bleeding
- Ruptured ectopic pregnancy associated with abdominal guarding and rigidity, shoulder pain and fainting attacks and shock (pallor, weak rapid pulse , falling blood pressure)
- **Diagnosis :**
    1. Hx + Ex
    2. Pregnancy test → positive → do the following
    3. Transvaginal U/S →positive (shows ectopic pregnancy) → the diagnosis is confirmed
    4. β-HCG→ more than 1500 mIU/ml+ empty uterus → suggestive ectopic →repeat βHCG after 48 hours → double β-HCG → normal pregnancy → if the rise less than 66% [ectopic pregnancy has a prolonged doubling time]→ suggestive of EP.

5. progesterone levels usually lower in EP→A value of 25ng/mL or more is 98% of the time associated with a normal Intrauterine pregnancy, while < 5ng/mL identifies a nonviable pregnancy, regardless of location
6. Laparoscopy can used for :
   - identifying an unruptured tubal pregnancy
   - exclude salpingitis
   - exclude small ovarian cyst

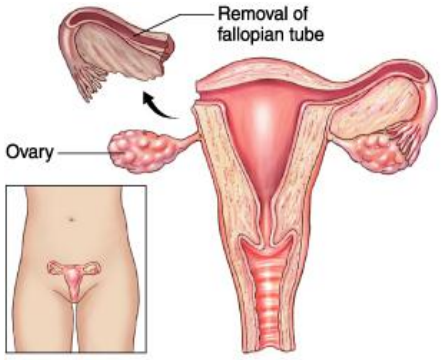
Sonography Mode	Gestational age	Discriminatory threshold in Dx EP
Transvaginal	35 days	1500 mIU/ml
Transabdominal	42 days	6500 mIU/ml

- Treatment :

1. Nonsurgical treatment

a. Expectant management : serial Beta HCG in case of falling HCG until it's reach 0 or 25 as many Ectopic pregnancy will resolve spontaneously

b. Methotrexate + folic acid **(see table below for contraindications)**



2. Surgical option

a. Laparotomy with

b. Laparoscopy with Salpingotomy or salpingectomy

-Laparotomy is the method of choice in case of hemodynamically unstable patient or rupture of the ectopic

-Laparoscopy is the method of choice in a stable patient

-Laparoscopic salpingectomy is the treatment of choice if the fallopian tube is extensively diseased or damaged as there is high risk of recurrent EP

-Laparoscopic salpingotomy is therapeutic option in an attempt to maintain **fertility**.

3. Combined medical-surgical treatment: Laparoscopic salpingotomy + local methotrexate injection.

- **Criteria for using methotrexate:** a. β-HCG titer < 6000
  b. Unruptured EP
  c. Ectopic mass of < 3 cm

Methotrexate C/I
HCG ≥ 6000
Ectopic adnexal mass ≥ 3 cm
Ruptured EP
Viable fetus
Hemodynamically unstable or Hemoperitoneum
Have signs of bone marrow depression
Liver dysfunction
Renal dysfunction
Evidenced by leucopenia
Thrombocytopenia.
Heterotopic pregnancies
Active pulmonary disease
Active peptic ulcer or colitis
Breastfeeding
Immunodeficiency / active infection

- **Contraindication of methotrexate : (see table)**
- **Treatment effects & Side effects of methotrexate :**
  1. Destroys growing tissue by antagonizing folic acid.
  2. Abdominal pain (2/3 of patient)
  3. Rising of HCG during first 3 days of treatment
  4. Vaginal bleeding
- **NB :** Reassure the patient about the pain

- **Signs of Treatment failure and tubal rupture:**

1. Significantly worsening abdominal pain
2. Hemodynamic instability
3. Level of HCG do not decline by at least 15% between Day 4 & 7 post treatment
4. Rising or plateauing HCG level after first week of treatment

- **Follow up :**

1. Repeat HCG on Day 5 post injection if <15 % decrease→consider repeat dose
2. If β-HCG >15 % decrease → recheck weekly until <25 ul/l
3. No test in the first 3 day → normally the HCG will rise in the first 3 days.

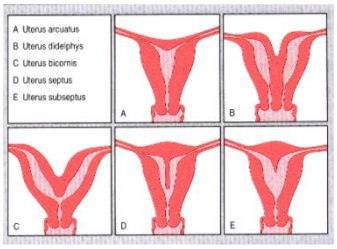
- **Additional notes :**

- ❖ In any lower abdominal pain we should exclude ectopic pregnancy
- ❖ Any women come to ER we should do pregnancy test
- ❖ Folic acid supply with methotrexate treatment
- ❖ We can see the fetus when beta HCG reach 1500 ul/l
- ❖ Anti D is given if the patient is Rh -ve.

Second Trimester Loss

Causes:: A-Maternal causes::

1-Uterine anatomic defects;-Mullerian duct fusion anomalies **(see Female genital tract malformation summary)**.



-Uterine septum **(see Female genital tract malformation summary)**.

-Uterine duplication **(see Female genital tract malformation summary)**.

-Uterine fibroids; -Only the submucosal ones.

-They limit the vascular supply for placentation & they contribute to atypical hormone responsiveness of the overlying endometrium

-Dx; -HSG, Hysteroscopy

-Rx; - Hysteroscopic resection.

2-Incompetent cervix;-Definition;-Passive, painless midtrimester cervical dilation due to structural weakness results in membrane prolapse & rupture leading to expulsion of an immature fetus.

-Etiology;-Congenital;-Diethylstilbestrol (DES) exposure.

-Spontaneous.

-Traumatic;-Mechanical cervical dilation.

-Second trimester abortion.

-Cervical amputation, conization, or laceration.

-Dx;-History;-The most accurate diagnosis.

-Hx of painless midtrimester cervical dilation resulting in expulsion of a nonviable fetus.

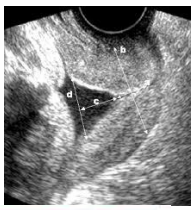
-Hx of any congenital or traumatic causes of an incompetent cervix.

-Pelvic examination;-For-Vaginal septae

-Cervical anomalies.

-Abnormal uterine configuration.

-Cervical length, thickness, and dilation assessment.



-Vaginal ultrasound;-Start serial weekly US examinations during pregnancy for high-risk women.

-Cervical funneling is an indication of present incompetent cervix.

-Nonpregnant workup;-There should be no internal os resistance to an 8-mm Hegar dilator.

-Retraction test with an inflated Foley balloon or balloon hysterography.

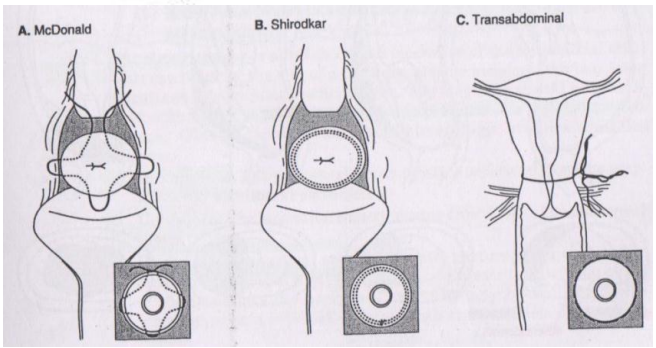
-Rx;- Cervical cerclage;-<u>Definition;</u> The placement of a circumferential cervical suture that externally supports the internal os.

-<u>Timing;</u> -scheduled between 10-12 weeks' gestation.

-<u>Methods;</u>-Shirodkar method:-Performed vaginally

-Cerclage not removed.

-Scheduled C/S is performed.



-McDonald method: -Performed vaginally

-Cerclage removed at 36 weeks.

-Delivered vaginally.

-Transabdominal method: -Performed by laparatomy

-Used if the cervix is too short.

-Cerclage not removed.

-Scheduled C/S is performed.

-<u>Complications;</u>-Infection.

-Bleeding.

-PROM.

-Preterm labor

Causes:: B-Fetal causes::

1-Diseases intrinsic to the fetus;-Chromosomal anomalies (monosomy X, trisomy 21)

-Anatomic anomalies (cardiac, osteo-chondro-dysplasias)

-Nonimmune hydrops (tumor, α-thalassemia, tachyarrhythmia)

2-Diseases mediated through the placenta;-Immune hydrops (immunization)

-Nonimmune hydrops (fetomaternal hemorrhage)

-Maternal infections (syphilis, parvovirus B19)

-Maternal systemic conditions (type I DM, anticardiolipin antibody).

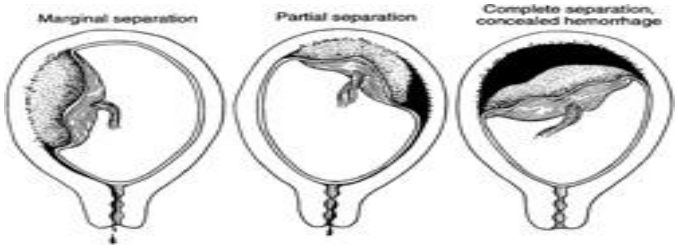
Antepartum Hemorrhage (APH)

- **Antepartum Hemorrhage**: is bleeding from the vagina during pregnancy from the 24th week (sometimes defined as from the 20th week) gestational age to term.
- Death from hemorrhage still remains a **leading** cause of maternal mortality.
- **Differential diagnosis of APH :**

1. Bloody show (sign of normal delivery)
2. Placenta previa
3. Placental abruption
4. Vasa previa (commonly presented in delivery)
5. Uterine rupture
6. Cervical bleeding (cervicitis, cervical neoplasm, cervical polyp)
7. Vaginal bleeding (trauma – neoplasm)
8. Coagulation Disorder
9. Others (GI bleed - hemorrhoids, inflammatory bowel disease , Urinary tract bleed - urinary tract infection)

Abruptio Placenta

- **Placenta Abruption:** the premature separation of the normally implanted placenta after 20 weeks of gestational age.
- It is the **common pathological** cause of APH and it is happened in 1-2 % of all pregnancy.
- Hemorrhage into the decidua basalis → decidua splits → decidural hematoma → separation, compression, destruction of the placenta adjacent to it.
- **Types of placental abruption :**



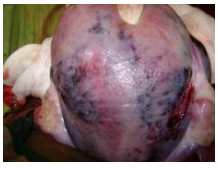
1. external hemorrhage
2. concealed hemorrhage
3. Total
4. Partial
- **Risk factors: (see table)**

Abruptio placenta	Placenta previa	Vasa previa	Uterine rupture
-Hx of Abruptio placenta -↑ age -Multiparity -Multiple gestation -Chronic Hypertension - Preeclampsia -Trauma -Polyhydramnios with rapid decompression. -Smoking -Cocaine use -PROM -Short umbilical cord -Folate deficiency -Uterine fibroid	-Hx of Placenta previa -↑ age -Multiparity -Multiple gestation -Hx of C/S -Smoking	-Hx of Vasa previa -Velamentous cord insertion -Bi-lobed placenta - Succenturiate (Accessory) lobe. -Low-lying placenta -Multiple gestation - IVF Pregnancies	-Hx of Uterine rupture -Hx of C/S (classic) [MOST COMMON] - Previous uterine surgery - Grandmultiparity - Excessive uterine stimulation [Oxytocin] -Marked uterine distension. -Non-vertex fetal presentation -External/internal cephalic version -Trauma -Shoulder dystocia -Forceps delivery

- The most common presentation is **pain** which can vary from mild cramping to severe pain.
- Sometimes it is associated with bleeding and sometimes not → concealed bleeding
- **Examination:** A **firm, tender uterus** and a possible sudden increase in fundal height on exam.
- The amount of external bleeding may not accurately reflect the amount of blood loss.
- Ultrasound only shows **25% of abruptions**.

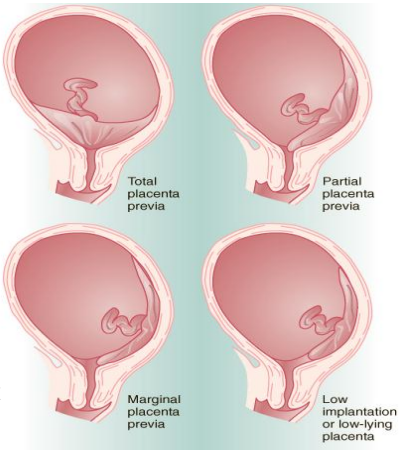
	Abruptio placenta	Placenta previa	Vasa previa	Uterine rupture
Painful bleeding?	Yes	No	No	Yes

- **Complication of placenta abruption in severe type :**
- Shock
- DIC (damaged vessels → releasing of thromboplastins → widespread intravascular coagulation → microthrombi → dissolve by plasmin → releasing of fibrin degradation product → with all the result is consuming of coagulation → DIC)
- Renal failure (due to shock or microthrombi has escaped and deposited in the renal vessels)



- Fetal death
- Couvelaire Uterus (Black colored uterus, due to bleeding inside it's myometrium)

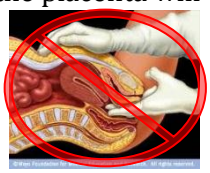
Placenta previa



- **Placenta Previa**: a placenta implanted in the lower segment of the uterus.
- Has major effect on the mother not the fetus.
- **Types of placenta previa** :

1. **Total placenta previa:** The internal cervical os is covered completely by placenta.
2. **Partial placenta previa:** The internal os is partially covered by placenta.
3. **Marginal placenta previa:** The edge of the placenta is at the margin of the internal os.
4. **Low-lying placenta:** The placenta is implanted in the lower uterine segment such that the placenta edge actually does not reach the internal os but is in close proximity to it.

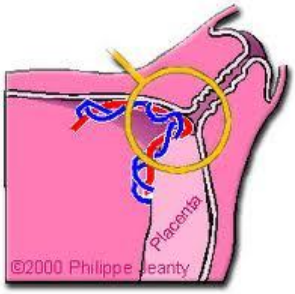
- Bleeding in case of placenta previa results from small disruptions in the placental attachment during normal development and thinning of the lower uterine segment and it usually ceases spontaneously.
- Most presentation of placenta previa is **painless bleeding** or bleeding without contraction. This bleeding often starts mildly and may increase as the area of placental separation increases. Abdominal examination usually finds **the uterus is non-tender and relaxed .**(**Avoid digital cervical examination**)
- In more than 90 % of women diagnosed with placenta previa in the second trimester, the placenta will correct itself by the end of the pregnancy.



- The diagnosis can be **confirmed** by U/S.
- **Risk factors of placenta previa : (see table above)**
- Placenta previa may be associated with placenta accreta, placenta increta or percreta.
- Placenta previa increases the risk of puerperal sepsis and postpartum hemorrhage because the lower segment to which the placenta was attached **contracts less** well post-delivery (less muscle in the lower segment)
- **DDx :** Abruptio Placentae, Premature Rupture of Membranes , Vaginitis , Cervicitis

Vasa previa

- **Vasa previa:** "fetal vessels crossing or running in close proximity to the inner cervical os.
- The classic triad of the vasa previa is: membrane rupture, painless vaginal bleeding and fetal bradycardia.
- This is rarely confirmed before delivery
- the bleeding is from **fetal** circulation→ high fetal mortality rate (50 -95%)



- **Causes :**
    1. Bi-lobed placenta
    2. Velamentous insertion of the umbilical cord
    3. Succenturiate (Accessory) lobe
- **Risk factors : (see table above)**

Uterine rupture

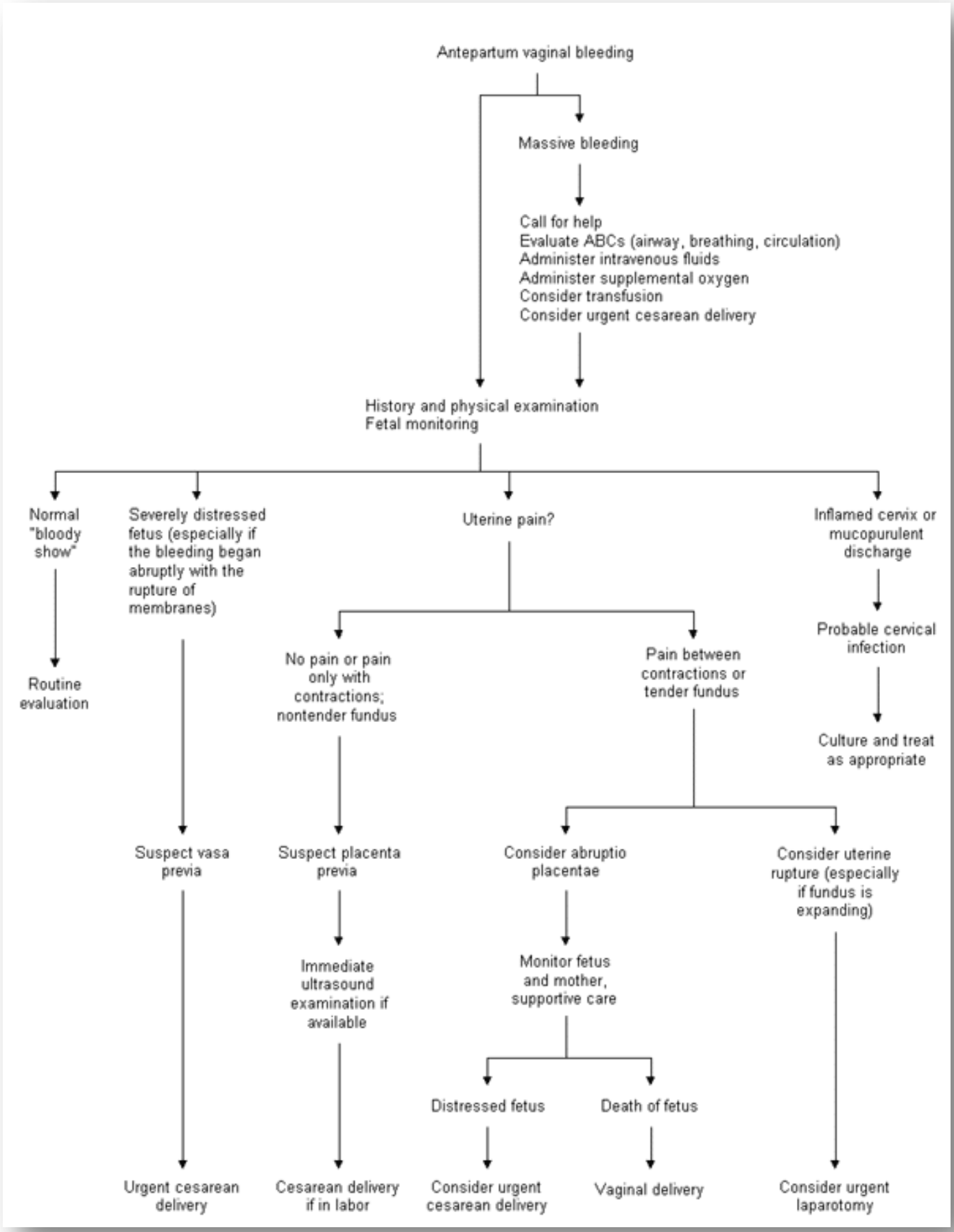
- **Uterine rupture**: Separation of the muscular wall of the uterus.
- A uterine rupture typically occurs during early labor but may already develop during late pregnancy.
- **Classic presentation includes :**
    1. vaginal bleeding
    2. abdominal pain and tenderness
    3. cessation of contractions
    4. absence/ deterioration of fetal heart rate
    5. easily palpable fetal parts
    6. profound maternal tachycardia and hypotension
- **Risk factors and causes : (see table above)**

- **General complications of third trimester bleeding:**
    - Hemorrhagic shock
    - DIC
    - Postpartum hemorrhage
    - Renal failure(ischemic necrosis of the kidney)
    - Sheehan syndrome (ischemic necrosis of anterior pituitary

Management of APH:

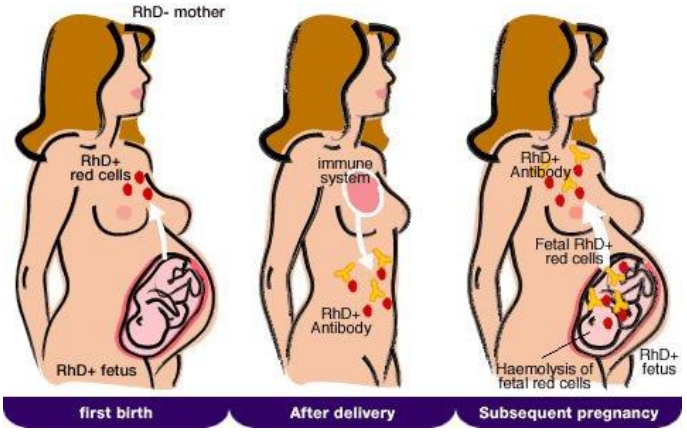
1- Admission
2- Hx (IMP : ask about **pain , amount of bleeding , duration of bleeding**)
3- Ex (without vaginal examination until we exclude placenta previa)
4- Blood withdrawal for Hemoglobin level to asses anemia (more hemorrhage more anemia) and for cross match , blood grouping and coagulation profile)
5- Restoring the blood lost (fresh blood or crystalloid or colloid fluid)
6- If coagulation defect or DIC → fresh blood, frozen blood plasma, fibrinogen, blood platelet +Heparin +Anti-fibrinolysis
7- US → to assess fetal heart , placenta localization , amniotic fluid index , fetal presentation and lie
8- CTG → to assess fetal distress → if there is fetal distress → immediate delivery
9- Kleihauer-Betke test (see below)
10- Apt test (see below)
11- The mother may be given Anti-Rh if she is Rh negative and the father Rh positive
12- **Placenta previa :**
    - Expectant management for stable women and stable fetus and less than 36 weeks → home management (limitation of activity + no coitus)
    - If the bleeding tends to recur → the patient remains in hospital.
    - In case of unstable women or unstable fetus→ ER C.S
    - Mode of Delivery is only by **Caesarean section**
    - Occasionally Caesarean hysterectomy necessary (in case of placenta accrete increta or percreta).
    - In placenta accrete If it is important to save the woman's uterus (for future pregnancies) then resection around the placenta may be successful.
13- **placenta abruption:**
    - Type of delivery: vaginal delivery or CS and in case of fetal distress the CS is preferred.
    - In case of severe bleeding + family size is completed → hysterectomy.
14- **vasa previa :**
    - it is Emergency condition → C.S
15- **Uterine rupture :**
    - Emergency C.S
    - Uterine Rupture following obstructed labor or trauma generally requires hysterectomy.
    - Uterine Rupture of caesarean scar generally can be sutured.

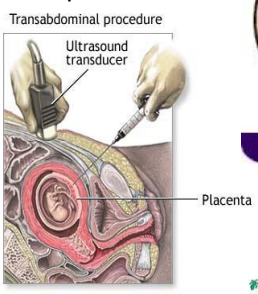
- **Additional notes :**
    - **Apt test:** clinician to determine whether the blood originates from the infant or from the mother. (vasa previa originate from fetus)
    - **Kleihauer-Betke Test :**
        - Is a blood test used to measure the amount of fetal hemoglobin transferred from a fetus to the mother's bloodstream.
        - Indicated to all APH and after delivery in women with – RH.
        - Used to determine the required dose of Rh immune globulin.


Antepartum vaginal bleeding
Massive bleeding
Call for help
Evaluate ABCs (airway, breathing, circulation)
Administer intravenous fluids
Administer supplemental oxygen
Consider transfusion
Consider urgent cesarean delivery
History and physical examination
Fetal monitoring
Normal "bloody show"
Routine evaluation
Severely distressed fetus (especially if the bleeding began abruptly with the rupture of membranes)
Suspect vasa previa
Urgent cesarean delivery
Uterine pain?
No pain or pain only with contractions; nontender fundus
Suspect placenta previa
Immediate ultrasound examination if available
Cesarean delivery if in labor
Pain between contractions or tender fundus
Consider abruptio placentae
Monitor fetus and mother, supportive care
Distressed fetus
Consider urgent cesarean delivery
Death of fetus
Vaginal delivery
Consider uterine rupture (especially if fundus is expanding)
Consider urgent laparotomy
Inflamed cervix or mucopurulent discharge
Probable cervical infection
Culture and treat as appropriate

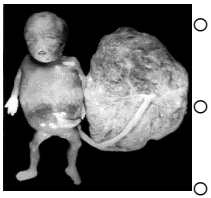
Rhesus iso-immunisation

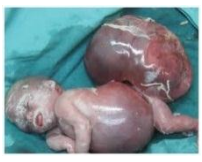
- Is one of the causes of hemolytic disease of the newborn (HDN).
- It occur in Rh negative women where the fetus's father is Rh positive, leading to a Rh+ pregnancy
- During birth, the mother may be exposed to the infant's blood, and this causes the development of antibodies (IGG which can cross the placenta) which may affect the health of subsequent Rh+ pregnancies (the first baby can't be affected because IGG takes time to be produced)
- In mild cases→ the fetus may have mild anemia with reticulocytosis (increase in immature red blood cells).
- In moderate or severe cases →the fetus may have a more marked anemia and erythroblastosis (nucleated red blood cells or erythroblastosis fetalis).
- In very severe cases→ HDN, hydrops fetalis, or stillbirth
- The risk and severity of sensitization response increases with each subsequent pregnancy involving a fetus with Rh-positive blood→ second pregnancy with an Rh-positive fetus often produces a mildly anemic infant
- **Causes :**
    1. Spontaneous or induced abortion
    2. Trauma
    3. Invasive obstetric procedures as amniocentesis, Chorionic villous sampling or ectopic pregnancy
    4. Normal delivery
    5. Rh-negative female receives an Rh-positive blood transfusion.
    6. APH
    7. Threatened miscarriage
    8. Toxemia
    9. Hypertension
    10. External cephalic version





- **Prevention :**
    - All rhesus-negative unsensitized women who have any risk factor are given anti-D IgG.
    - All rhesus-negative unsensitized women are given anti-D IgG at 28 weeks gestation and repeated at 34 weeks gestation
    - All rhesus-negative unsensitized women after delivery are given anti-D Igg within 72 hours
    - If the mother has been sensitized previously, as determined by elevated level of maternal Rh antibodies, administration of Rh IgG has no value
    - Atypical antibodies (antibodies against fetal RBC) should be screened by AAT or coombs test: if negative>>no fetal risk. If positive<1:8 it's insignificant and HDN is negligible. If positive>1:8 it's significant and fetal evaluation for anemia should be carried out
- **Antenatal care :**
    1. Blood group screening at first visit
    2. In case of women with rhesus- negative → maternal Rh antibody titers → negative → repeat at 28 and 36 weeks
    3. In case of positive antibody



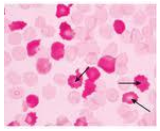


- Serial Ultrasound to detect hydrops fetalis (skin edema, ascites, pleural or pericardial effusions, cardiomegaly and an edematous placenta **)**
- Serial Doppler examinations of middle cerebral artery and umbilical artery for detection increased blood flow velocities (sign of anemia)
- Quantitative analysis of maternal anti-RhD antibodies - an increasing level is a sign of fetal Rh disease
- amniocentesis for the level of bilirubin to assess the severity of hemolysis→ Amniotic fluid bilirubin concentration can be quantified by spectrophotometry by assessing the change in optical density at 450nm.

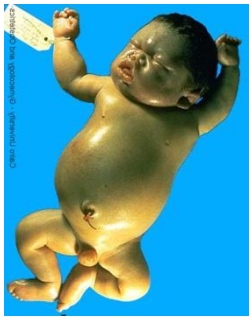
- Severe may be associated with hydrops fetalis(ZONE III) → blood transfusion into fetal umbilical vein until 32 weeks then immediate delivery

- Moderate to mild(ZONE II)→ delivery at 35 weeks to term
- Mild or nil (ZONE I)→ delivery at term

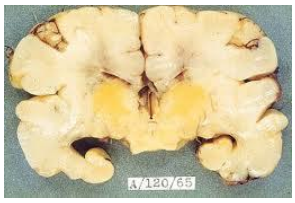
- **Prenatal emergency care :**
  1. Determination of Rh blood type is required in every pregnant female
  2. Kleihauer-Betke test used When a high clinical suspicion of large fetomaternal hemorrhage
     - for measurement of fetal red blood cells in maternal blood.
     - for determining if additional amounts of Rh IgG should be administered
     - In case of very small amounts of fetomaternal hemorrhage→ Rosette test.



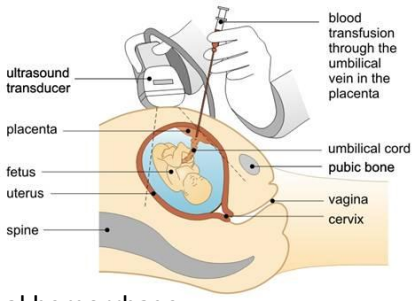
  3. maternal Rh antibody titers (indirect coombs test) can be helpful for future follow-up care of pregnant females (to know if the patient sensitized or not)
- **Postnatal emergency care:**
  1. examine blood from the umbilical cord of the infant for ABO blood group and Rh type, measure hematocrit and hemoglobin levels, perform a serum bilirubin analysis, obtain a blood smear(shows increased reticulocytes or Erythroblasts) and perform a direct Coombs test
  2. Elevated serum bilirubin measurements, low hematocrit, and elevated reticulocyte count from the neonate can help determine if an early exchange transfusion is necessary.
  3. An emergent exchange transfusion, preferably performed is required in infants born with erythroblastosis fetalis, hydrops fetalis, or kernicterus
  4. Phototherapy for neonatal jaundice in mild disease



- **Complication of Hemolytic disease of the newborn :**
  1. Hydrops fetalis **(see figure)**
  2. Stillbirth
  3. Hepatosplenomegaly
  4. Kernicterus **(see brain figure)**
  5. Severe anemia



- **NB:** Fetal heart rate changes have been noted with severe anemia. A sinusoidal pattern with the loss of normal baseline variability of the CTG is highly suggestive of severe anemia
- **COMPLICATIONS FOLLOWING INTRAUTERINE TRANSFUSION:**
  1. Premature labor
  2. Pre-labor ruptured membrane
  3. Fetal hemorrhage
  4. Fetal bradycardia
  5. Failure to obtain a sample
  6. Increase in maternal isoimmunization by inducing feto-maternal hemorrhage



ABO hemolytic disease of the newborn

- ABO blood group isoimmunization may occur when mother's blood group is O and baby's either group A or B.
- Anti-A and anti-B antibodies are present in the maternal circulation naturally, and hence do not require prior sensitization in order to be produced
- This means that ABO incompatibility may occur in a first pregnancy.
- anti-A or anti-B antibodies pass to the fetal circulation, causing fetal hemolysis and anemia
- most of cases ABO incompatibility causes mild hemolytic disease of the baby→ no screening for ABO hemolytic disease
- Diagnosis is usually made by investigation of a newborn baby who has developed jaundice during the first day of life
- **Treatment:** Neonatal jaundice caused by ABO HDN is usually successfully treated with phototherapy and in case of severe hemolytic anemia → the management as Rhesus isoimmunization.

Postpartum Hemorrhage [PPH]

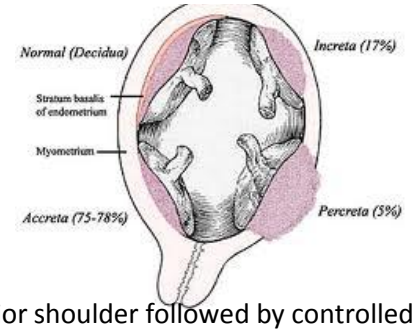
- **Primary postpartum hemorrhage:** is the loss of > 500 ml of blood following vaginal delivery, or > 1000 ml of blood following cesarean section or 1500 ml of blood following C/S hysterectomy within 24 hours and If less than 500ml and causing hypovolemic shock. Can occur before, during, or after placenta delivery.
- **Secondary postpartum hemorrhage:** It is a blood loss of a volume greater than expected after 24 hours within the first 6 weeks of delivery [mainly due to trauma].

- Secondary PPH is more likely due to infection and retained placental tissue.
- Hemorrhage is still one of the leading causes of maternal mortality all over the world.
- Incidence of primary PPH is 10% of all delivery.
- **Causes & Risk factors of PPH** :

Causes [4T]	**Tone (Uterine atone) 70-80%**	**Trauma (Lacerations) 20%**	**Tissue (Retained tissue) 10%**	**Thrombin (DIC) 1%**
Risk factors	**Overdistended uterus;** -Multiple gestations -Polyhydramnios -Fetal macrosomia **Myometrial dysfunction;** -Prolonged labor -Oxytocin augmentation of labor -Grand multiparity (≥ 5 times) -Chorioamnionitis -Uterine Leiomyomas **Pharmacologic;** -Magnesium sulfate Rx of PET -Halogenated anesthetics	-Uncontrolled vaginal delivery -Forceps delivery -Ventouse delivery -Internal podalic version (abnormal lie)	-Accessory placental lobes -Abnormal trophoblastic invasion; •Placenta accreta (80%) •Placenta increta (15%) •Placenta percreta (5%)	**Vascular endothelium injury;** -Hypovolemic shock -Severe PET -Septicemia **Release of thrombogenic factors;** -Abruptio placenta -Amniotic fluid embolus -Prolonged IUFD

- **Other Causes & Risk factors of PPH** :

Other Causes	**Uterine inversion**	**PPH as a Sequences of APH**
Risk factors	-Hx of uterine inversion -Fundal placental implantation -Partial placenta accreta -Congenital or myometrial weakness	-Abruptio placenta **[see summary]** -Placenta previa **[see summary]** -Vasa previa **[see summary]** -Uterine rupture **[see summary]**



- **Prevention of PPH by :**

1. Active management of third stage of labor : Syntometrine at delivery of anterior shoulder followed by controlled cord traction will reduce the incidence of PPH
2. Correcting anemia prior to delivery
3. Episiotomies only if necessary

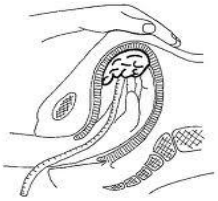
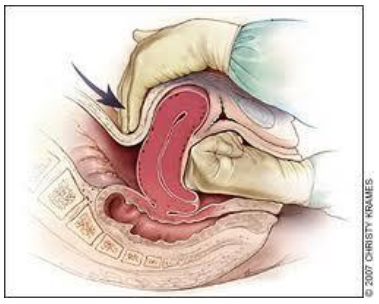


Figure 106
ARTIFICIAL REMOVAL OF THE PLACENTA
Left hand on the fundus, fingers of the right hand inserted between the placenta and uterine wall, removal with the cubital border of the hand

- **Management :**

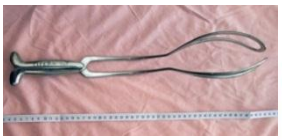
1. Call for help→ midwifery , anesthetist and hematologist
2. O2 and IV access must be secured with a large bore cannula
3. Blood is withdrawn for hemoglobin, cross matching, coagulation profile and blood grouping.
4. Infusion of crystalloid or colloid to maintain the pressure
5. If coagulation defect happened→start Fresh frozen plasma, cryoprecipitate, and platelet transfusion.
6. Examine genital tract, inspect placenta, observe clotting and manual exploration of the uterus to rule out birth canal injury and retained placenta→ if birth canal injury present →Suturing under anesthesia with good exploration to the whole birth canal
7. If uterine inversion → gently push the uterus back into position.
8. Uterine massage (see figure)
9. Ergometrine ,Oxytocin ,prostaglandins
10. Uterine packing
11. Embolization of the pelvic vasculature
12. Laparotomy : Ligation of uterine then → internal iliac artery
13. Hysterectomy.

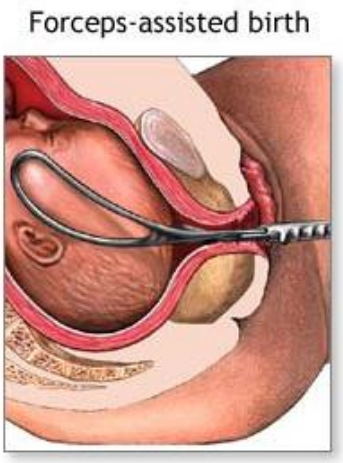


Operative delivery

A- Forceps delivery:

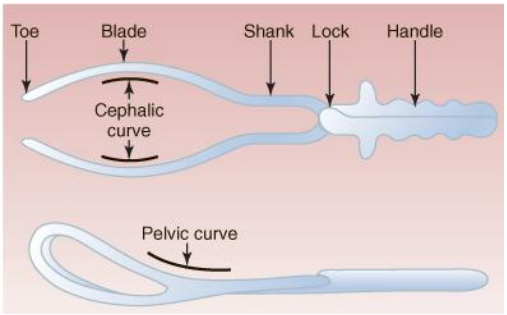
- Two matched metal branches are maneuvered in appropriate relationship with fetal head, then articulate.
- I**ndications**:
  - **MATERNAL**
    - Exhaustion
    - Prolonged second stage,
    - Cardiac / pulmonary disease, diabetes.
    - Toxemia of pregnancy (preeclampsia, eclampsia).
    - Previous C/S or weak abdominal wall.
  - **FETAL**
    - Failure of the fetal head to rotate
    - Fetal distress
    - Control of the fetal head in vaginal breech delivery.
    - Large sized fetal head.





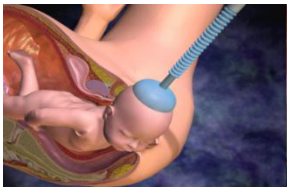
- **Classifications:**
  - Based on the station of the leading bony part of the fetal skull when the procedure begun.
  - Generally the higher the station the higher the maternal and fetal risk.
  - **1.** Outlet forceps: ⇨ Scalp visible at the vulva without separating the labia (reach pelvic floor).
  - **2.** Low forceps: vertex at +2 station.
  - **3.** Mid forceps: head is below 0 station (engaged) but has not reached +2 station.

- **Prerequisites: [**ABCDEFGHIJ**]**
  - A→Anesthesia.
  - B →Bladder should be empty.
  - C→ Cervix should be fully dilated and effaced /membrane should be ruptured.
  - E→Equipment →know your forceps.
  - F→ phantom application.
    - Lt. Blade, Lt. Hand, maternal Lt Side pencil grip & vertical insertion with Rt. thumb directing blade.
    - Rt. blade , Rt. hand, maternal Rt. side pencil grip & vertical insertion with Lt. thumb directing blade
  - G→Gentle traction.
  - H→ Hand elevated, Head is engaged, and Head in vertex or face presentation (also breech).
  - I→Incision (consider episiotomy).
  - J→ withdraw forceps if Jaw is reachable.
- **Complications:**
  - Maternal laceration: (**Perineal tears) [see episiotomy summary**]
  - Bleeding from laceration.
  - Trauma to urethra and bladder.
  - Fetal face laceration.
  - Fetal skull fracture.
  - Permanent nerve damage → facial nerve palsy.



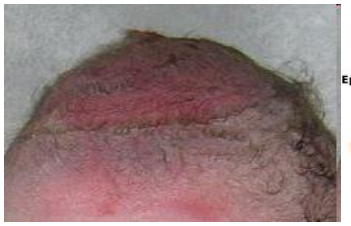
B- Vacuum extractor delivery:

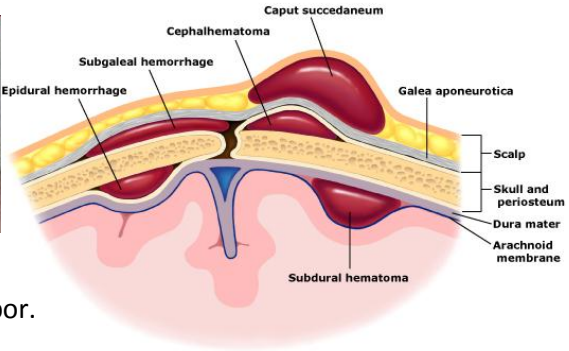
- **Held on the fetal head by negative pressure→ augment maternal expulsive effort.**
- **Indication:**
  - **MATERNAL**
    - Exhaustion
    - Prolonged second stage
    - Cardiac / pulmonary disease.



- **FETAL**
  - Failure of the fetal head to rotate, and increase flexion of deflexed head.
  - Fetal distress in the second stage
  - Control bleeding by traction on head in placenta previa.

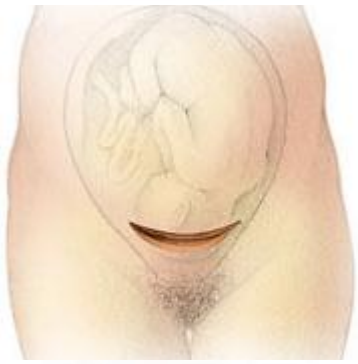
- **Prerequisites**: [Same as forceps but] :
  - F→Fontanelle ⇨ position the cup over the posterior fontanelle
  - H→Halt [stop] ⇨ if no progress with three tractions aided contractions.
- Vacuum is the instrument of choice, and less traumatic to the mother & fetus than forceps
- It is contraindicated in face presentation, breech presentation, and preterm labor, and moderate or severe cephalopelvic disproportion.
- **Complications:**
  - Maternal laceration.
  - Fetal scalp injury.
  - Cephalohematoma.
  - Intracranial hemorrhage.
  - Sublgaleal hematoma.
  - Retinal hemorrhage.





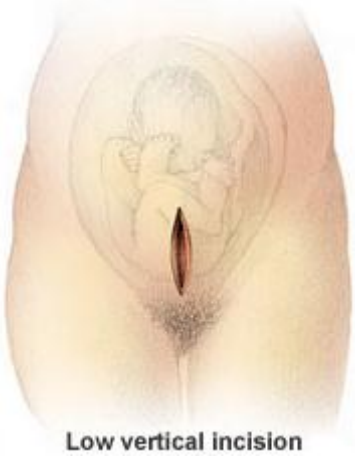
Note: Never use two instrumental modalities if one has failed to assist labor.

C- Cesarean section [C/S]:



- **Types of c/s:**
  1- Low segment transverse: **incision of choice**.
  [In non-contractile portion of lower uterine segment]
  2- Low vertical:
  [In contractile portion of lower segment]
  3- Classical:
  [In contractile fundal portion]

- **Indications of elective C/S (scheduled C/S):**
  1-Fetal macrosomia.
  2-Repeated C/S.
  3-Uterine surgeries (myomectomy)
  4-Malpresentation like: breech, transverse lie.
  5-Multiple pregnancy.
  6-Placenta Previa.
  7-Infections like: HIV, active genital herpes.
  8-Cervical cancer.
  9- Full-thickness non-transverse incision through myometrium (classical incision) →absolute indication.
  10- Severe IUGR.


Low vertical incision

- **Indications of emergency C/S:**
  1-Maternal distress.
  2-Fetal distress.
  3-Uterine rupture.
  4-Vasa previa.
  5-Severe PET
  6-Severe Abruptio placenta
  7-Cord prolapse
  8-Abstructed labor (failed instrumental deliveries)
  9-Brow, posterior-mental & shoulder presentation.


Classical incision

- **Indications of classical CS:**
  1. Transverse lie (with SROM).
  2. Structural abnormality that makes lower segment approach difficult (Fibroids)
  3. Anterior Placenta Previa.
  4. Cervical cancer.

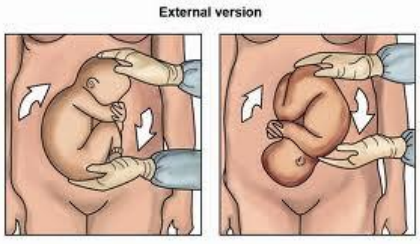
	Lower segment transverse	Low vertical segment ***	Classical
Advantage	↓blood loss ↓adhesion VBAC is ok	↓blood loss ↓adhesion VBAC is ok	Any fetus can be delivered Regardless of orientation In uterus or GA
Disadvantage	Risk of bladder injury Lower uterine segment must Be formed. Deliver longitudinal only	Risk of bladder injury Lower uterine segment must Be formed. Deliver longitudinal only May extend to upper segment	↑blood loss ↑adhesion VBAC is not safe Risk of uterine rupture.

VBAC= vaginal birth after cesarean. GA= gestational age.

*** Low vertical segment: if limited to the lower segment, it will have all LST advantages, but if extend to the fundal part; it will have all Classical disadvantages.

- **Complication of C/S:**
  1. Bleeding & the need for blood transfusion
  2. Hysterectomy
  3. Complications of anesthesia
  4. Damage to the bladder, ureter, colon, retained placental tissue.
  5. Fetal injury
  6. Infection.

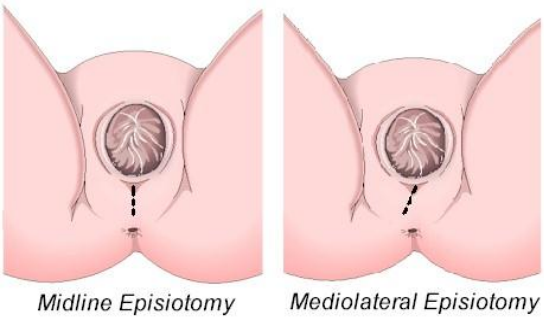
- **Vaginal delivery after previuos C/S:**
  - Pt. must agree to the procedure.
  - That C/S should be a low transverse uterine incision.
  - Non recurrent cause of the previous C/S,
  - No current macrosomia, malposition, multiple gestation, breech.
- **External cephalic version [ECV]:**
  - It is uterine manipulation to change breech presentation to cephalic presentation.
  - Success rate is **65%**.
  - **The optimal GA time is 37 weeks** [because if anything went wrong → immediate delivery].
  - **Methodology:**
    a. US examination to confirm: fetal orientation, placental implantation, amniotic fluid volume, and to roll out anomalies.
    b. Confirm fetal-wellbeing by NST.
    c. Administration of SQ to relax myometrium.
    d. Monitor FHR while the head is moved downward.
    e. Discontinue if → excessive discomfort, persistent fetal bradycardia.
    f. Administration of RH immune if mother is negative.

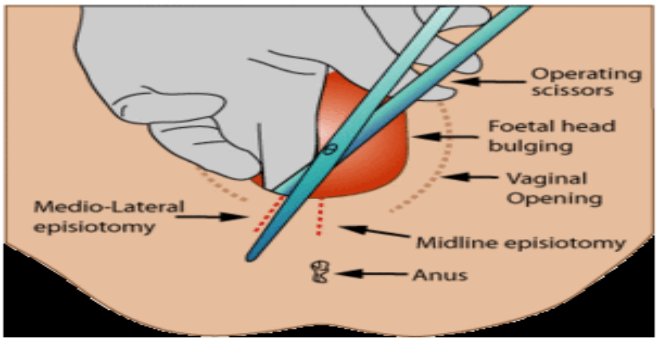


  - **Contraindications:** uteroplacental insufficiency, hypertension, intrauterine growth restriction, oligohydramnios, Hx of uterine surgery, multiple pregnancy, placenta previa, or contracted pelvis.
  - **Complication**s:
    a. Fetomaternal hemorrhage.
    b. Labor.
    c. Fetal bradycardia.

Episiotomy

- It is a surgically planned incision on the perineum and the posterior vaginal wall during second stage of labor.
    1- Midline
    2- Mediolateral (oblique technique)

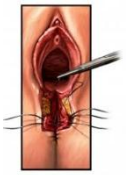




- Is performed under local anesthetic (pudendal anesthesia), and is sutured closed after delivery.
- Episiotomy is done as prophylaxis against soft-tissue-trauma or vaginal tears.
- Vaginal tears can occur during childbirth, most often at the vaginal opening as the baby's head passes through, especially if the baby descends quickly.
- In the oblique technique, the perineal body is avoided, cutting only the vagina epithelium, skin and muscles
- **Indications:**
    1- There is a serious risk to the mother of second or third degree tearing
    2- The baby is very large
    3- When perineal muscles are excessively rigid
    4- When instrumental delivery is indicated
    5- Prolonged late decelerations or fetal bradycardia during active pushing
    6- shoulder dystocia →more room to perform maneuvers to free shoulders from the pelvis

Lateral incision	Midline
More safe → most common used	Less safe (**may involve anal sphincter**)
Harder to repair	Easier to repair
Have increased bleeding	Less blood loss
It may produce more scarring	Less scarring
More pain	Less pain
Less cosmotic	More cosmotic

- **Complications:**
    - Bleeding
    - Infection
    - **Swelling**
    - wound closure Defects.
    - **Local pain**
    - Short-term possibility of sexual dysfunction.
    - Incontinence
- **Perineal tears :**
    - **First degree tear:** laceration is limited to superficial perineal skin or vaginal mucosa **[skin]**.
    - **Second degree tear:** laceration extends beyond fourchette, perineal skin and vaginal mucosa to perineal muscles and fascia, but not the anal sphincter **[muscles]**.
    - **Third degree tear:** perineal skin, vaginal mucosa, muscles, and anal sphincter are torn **[anal sphincter]**.
    - **Fourth degree tear:** perineal skin, vaginal mucosa, muscles, anal sphincter, and rectal mucosa are torn **[rectum]**.
- Perineal tears usually follow forceps delivery in primigravida, big baby, occipitoposterior position.



- Pain after episiotomy due to : **very Important**
    - Tightening of the suture (Rx : observation –ice bag – paracetamol)
    - Hematoma [Rx: if small, observation is enough. If big, evacuation and I.V fluid is needed (make sure after delivery that she can pass stool and urine because big hematoma may cause obstruction)]

Preterm labor

Definition: the onset of labor after 20 weeks & before 37 completed weeks of gestation.

Early symptoms: -Menstrual-like cramps.
-New low back pain.
-Pelvic pressure.
-Change in vaginal discharge.

Risk factors: - **see table**.
-Most are unknown.

Socioeconomic factors	Lifestyle factors	Medical Hx	Obstetric Hx
-Low income	-Smoking	-Hx of Preterm labor	-Multiple pregnancy
-Low educational level	-Heavy physical activities (lifting heavy stuff)	-Hx of PROM	-Short cervix
-Maternal age extremes (<16 years;>40 years)	- Trauma	-Defects within membranes	-Dilated cervix
	-Anxiety	-Uterine anomalies	-Effaced cervix
	-Poor nutrition	-Bacterial vaginosis	-(incompetent cervix)
	-Sexual intercourse (late wks)	-2nd trimester abortion	-Polyhydramnios
		-DES exposure	-Hypertension
		-Renal disease	-Hemorrhage
		-Surgery	-Bacteriuria
		-Sepsis	-Amniocentesis
			-Low placental insertion

Dx: Criteria for preterm labor (<u>all must be present</u>): 1- Gestation age between 20 and 37 weeks.
2- ≥ **3** contractions lasted ≥**3**0 seconds in **3**0 minutes.
3- Cervix is dilated ≥2 cm. or effaced by 80%.

Complications: - Preterm uterine contractions can predispose to **PPROM** from the serial damage to membranes.
- Prematurity, which is the leading cause of infant mortality.
-See **PPROM** for other complications.

How to manage a mother with preterm contractions???

1-Checking the preterm labor criteria (Gestation age, contractions and Cervix).
2-Ruiling out PPROM by speculum examination.
3-Adequate hydration and bed rest.
4-Cervical culture to rule out bacterial vaginosis

If confirmed Dx and still contracting??

5-Ruling out contraindication(C/I) to tocolysis. **see table**.

Fetal C/I	Maternal C/I	Placental/Membrane C/I
-IUFD	<u>Advanced</u> cervical dilatation	<u>S</u>PROM
-Fetal distress	<u>Severe</u> PET or Eclsmpsia	<u>Severe</u> Abruptio placenta
-<u>Severe</u> IUGR	<u>Uncontrolled</u> DM	<u>Unstable</u> placenta previa
-Anomalies <u>incompatible with life</u>	<u>Uncontrolled</u> hyperthyroidism	Chrioamnionitis

6-Giving tocolytics (if no C/I) such as:
-Ritodrine or Trebutaline (β-agonists to relax smooth m.)(Delay delivery 1-2 days)
<u>-Side effects:</u> hypokalemia, cardiac arrhythmia, hyperglycemia.
-Magnesium Sulfate (Compete for Ca ions in depolarization)(Delay delivery 1-2 days)
<u>-Side effects:</u> m. weakness, respiratory depression, pulmonary edema.
-Indomethacin (PG synthase inhibitors) (Most effective)
<u>-Side effects:</u> PDA closure in uterus, Oligohydramnios, NEC.
-Nifedipine (Ca channel blocker) (Delays delivery 1-2 days)
<u>-Side effects:</u> tachycardia, hypotension, myocardial depression.

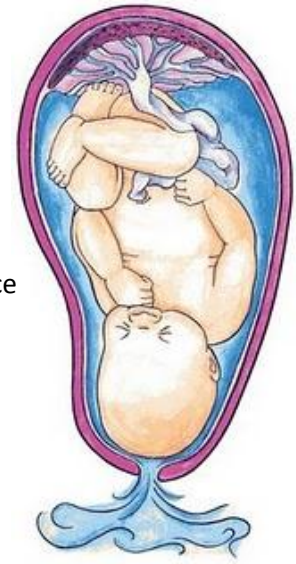
7- Maternal and fetal monitoring for well-being.
8-Giving Corticosteroids (Betamethasone and Dexamethasone) to induce pulmonary surfactant production.
It can be given at 24-34 weeks with great effect.

Prevention: -Life style change (see risk factors)
-Early detection of preterm labor by: -Pt. education about it's symptoms
-Pt. education for self-palpation of contractions
-Weekly digital cervical examination.
-Electronic US monitoring

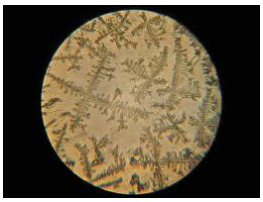
PDA: patent ductus arteriosus. , NEC: necrotizing enterocolitis.

PROM

- **Premature rupture of membrane (PROM):** rupture of the membrane prior to onset of labor.
- **Preterm premature rupture of membrane (PPROM)**: rupture of the membrane prior to 37 weeks gestation completed (24-37 weeks)
- Normal Rupture of membrane occur when cervical dilation 8 cm
- When cervical dilation is 4 cm we will rupture the membrane because it contain high prostaglandin→cause dilation of the cervix (artificial rupture of the membrane)
- PROM occurs in approximately 10%-15% of pregnancies→ 2/3 associated with term and 1/3 associated with preterm
- Most patients (90%) enter spontaneous labor within 24 hours when they experience ROM at term
- In case of preterm rupture, most of baby is born within 7 days of the rupture.



- **Patients with PROM may present with :**
    1. Gush of fluid then leakage of fluid (main presentation)
    2. Vaginal discharge
    3. Vaginal bleeding (it mix with fluid)
    4. Pelvic pressure
    5. Urine incontinence (actually it is leakage of fluid but the women may get mistaken)
    6. No uterine contraction (if there is contraction and cervical change → labor)
- **Rupture of membrane diagnosed by :**
    1. **Pooling of fluid** in the vagina or leakage of fluid from the cervix
    2. Leakage of fluid by cough or by fundal pressure
    3. **fering pattern** of the dried fluid under microscopic examination (see figure)
    4. **Nitrazine paper** → turn blue
    5. **AmniSure**



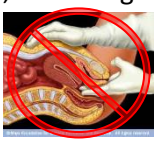
- Ultrasonographic examination may then show absence of or very low amounts of amniotic fluid
- Blood contamination of the Nitrazine paper and ferning of cervical mucus may produce **false-positive results.**

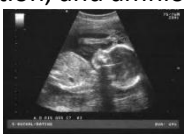
Maternal complication	Fetal complication
-chorioamnionitis -sepsis -Long bed rest → DVT -Placenta abruption	-Prematurity →(Respiratory distress syndrome (RDS) ,Brochopulmonary dysplasia (BPD) intraventricular hemorrhage (IVH) , Necrotizing enterocolitis (NEC) , retinopathy of prematurity (ROP) ,Patent ductus arteriosus (PDA), hypothermia , hypocalcemia and others. -Infection (congenital pneumonia,meningitis) -Cord prolapse -Malpresentation -Deformation

- **The risk factor the same as the risk of preterm labor; (see table)**

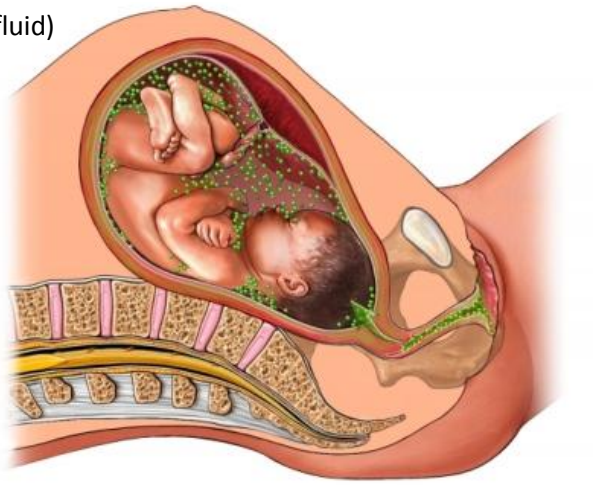
Socioeconomic factors	Lifestyle factors	Medical Hx	Obstetric Hx
-Low income -Low educational level -Maternal age extremes (<16 years;>40 years)	-Smoking -Heavy physical activities (lifting heavy stuff) - Trauma -Anxiety -Poor nutrition -Sexual intercourse (late wks)	-Hx of Preterm labor -Hx of PROM -Defects within membranes -Uterine anomalies -Bacterial vaginosis -2nd trimester abortion -DES exposure -Renal disease -Surgery -Sepsis	-Multiple pregnancy -Short cervix -Dilated cervix -Effaced cervix -(incompetent cervix) -Polyhydramnios -Hypertension -Hemorrhage -Bacteriuria -Amniocentesis -Low placental insertion

- **Management of prom :**
    1. Hx+Ex → confirmed PROM
    2. Cervical cultures before prophylactic antibiotic
    3. Look for the signs of chorioamnionitis (fetal tachycardia , maternal fever or tachycardia , abdominal tenderness high wbc and CRP) → immediate delivery
    4. Ultrasonographic documentation of gestational age, fetal weight, fetal presentation, and amniotic fluid index should be established
    5. **Digital cervical examinations should be avoided.**

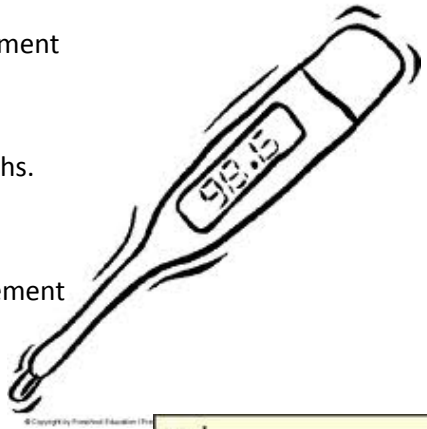




    6. Prophylactic antibiotic
    7. Maternal Corticosteroids for Fetal lung maturity between 24 and 34 weeks. Betamethasone IM twice and 24hours apart. Corticosteroids also decrease the risk of interventricular hemorrhage
    8. Informed consent should be obtained for expectant management versus delivery (If labor doesn't start after 48 hours the patient can go home and return to hospital if she develops a fever
    9. PROM at term:
        a. Awaiting the onset of spontaneous labor for 12-24h
        b. Termination of pregnancy after 24 hours Expectant management
    10. PROM before term:
        a. Indications for termination of pregnancy
            i. Evidence of fetal pulmonary maturation (L/S ration more than 2→ documented by either amniocentesis or collection of vaginal fluid)
            ii. Chorioamnionitis
            iii. Fetal distress
            iv. Advanced labor
            v. Placenta abruption

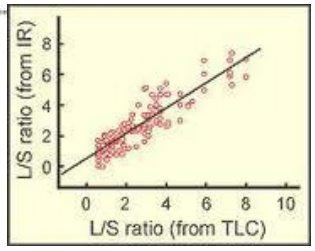


    11. Previable (<24wk): The Goal is Maternal Safety since neonatal survival chances are low and there are two options to be offered to the patient:
        a. Induction of labor (IOL)
        b. Home management
    12. The main indication for ***tocolytics*** is to gain time to:
        a. Give the possibility of using intra-muscular steroid treatment for 24 hours
        b. Transfer the patient to a tertiary intensive care unit
    13. Health Teaching for women with PPROM in case of home management
        a. Take temperature Report if more than 38^0C
        b. Remain on modified bed rest
        c. Insert nothing in the vagina, No sexual activity, No tub baths.
        d. Assess for uterine contraction & fetal movement
        e. Watch for foul-smelling vaginal discharge
        f. Wipe front to back after urinating or having a bowel movement
        g. Take antibiotics if prescribed



Additional notes:-

- Tests of fetal pulmonary maturity; -Mechanism;-
    - Measures phospholipid surfactant secreted into amniotic fluid from fetal lung [Type II pneumocyte].
    - Lecithin increases dramatically between 34-36 weeks gestation
    - So lecithin: sphingomyelin (L/S) ratio ≥ 2 indicates lung maturity.
    - Corticosteroids are given to promote lung maturity in 24-34 weeks' gestation



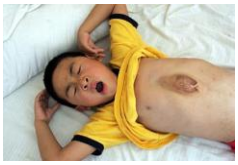
Perinatal Infections

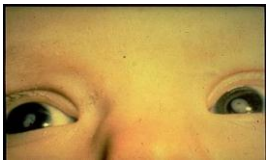
- **Infections that affects the fetus:**
  - **(See table)**

T	O	R	C	H	S
Toxoplasmosis	Others: Listeria Parvovirus Gonorrhea	Rubella	CMV Coxa virus Chlamydia	HSV HIV HBV	Syphilis Streptococcus
Gonorrhea + Chlamydia don't cross placenta, but others do.					

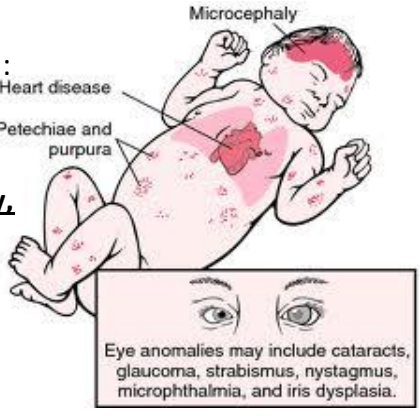
- **Mechanism of perinatal infections**
  1. Congenital : In utero by trans placental
  2. Perinatal : During labor and delivery by exposure to genital secretions and blood
  3. Neonatal : After birth by direct contact, breast feeding or nosocomial exposure

- ***General Principles of perinatal infections***
  - All viruses and most bacteria can pass through the placenta
  - The fetus does not make IGM until beyond 20 weeks gestation
  - Maternal IgG usually pass through placenta
  - IGM does not pass through placenta
  - Evidence of infection does not imply fetal damage
  - Teratogenic effect mainly in the first and early second trimester
  - All infections can cause **abortion, IUGR, premature labor, severe neonatal sepsis, or long term carrier states**.
  - Absence of fetal IGM at birth does not mean that infection did not occur unless the baby is 1 year old





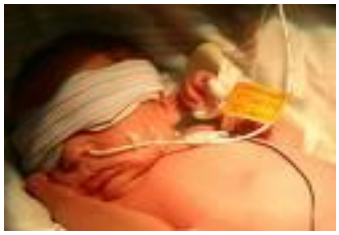
- **<u>Rubella</u>** : (**<u>very Important</u>**)
  - Spread by respiratory droplets
  - **Maternal Symptoms of Infection** (50% asymptomatic): Maculopapular rash , Fever , Lymphadenopathy ,Fatigue and Sore throat
  - Mainly first trimester infection can lead to congenital Rubella :
    <u>Deafness, cardiac abnormality (peripheral pulmonary stenosis, pulmonary valvular stenosis, patent ductus arteriosus, ventricular septal defect) cataract, microcephaly, mental retardation and thyroid disorders.</u>



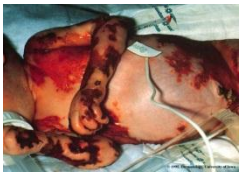
  - **Diagnosis by :**
    1- Serological test for the neonate: +ve IgM in umb. Cord, +ve IgG in blood after 5 months
    2- Serological test for the mother: IgM begins with the rash and disappear in 4-8 weeks; IgG begins with the rash and lasts for lifetime
  - **Treatment:** The only effective way to prevent Congenital Rubella is to terminate the pregnancy**.**

- **Prevention :**
  - Live-attenuated virus vaccine as part of MMR given in childhood
  - Screening of pregnant women to determine immune status → if the women non-immune → avoid exposure until delivery
  - Vaccination after delivery for non-immune women and the vaccine is live attenuated so, **3 months contraception** is advised after vaccination**.**

Congenital age(weeks)	Fetus infected %
<11	90
11-12	30
13-14	20
15-16	10
>16	5



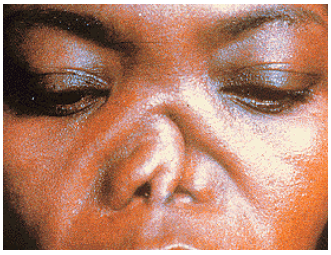
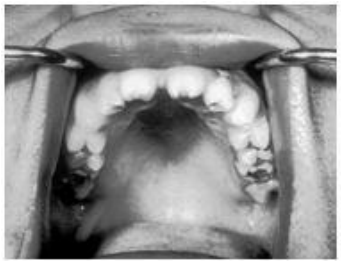
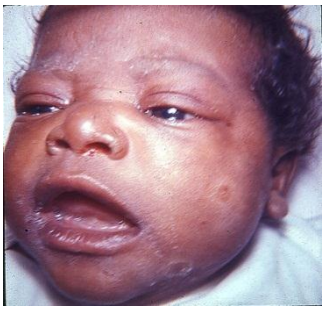
- **Varicella-Zoster Virus (Chicken Pox) :**
  - 90% of pregnant women already immune, therefore primary infection is rare during pregnancy
  - Infection before 20 weeks can lead to abortion, limb hypoplasia, skin scarring, IUGR, neurological abnormality and hydrops fetalis
  - up to 3% chance of transmission to the fetus, recognized congenital varicella syndrome in first 20 weeks
  - Infection late in gestation or immediately following birth is referred to as "neonatal varicella"
  - Zoster immunoglobulin should be given to susceptible pregnant women who had contact with suspected cases of varicella.
  - Zoster immunoglobulin should also be given to infants whose mothers develop varicella during the last 7 days of pregnancy or the first 14 days after delivery.



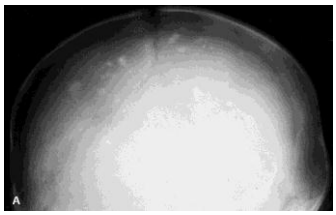
- **Genital Herpes Simplex Virus** :
  - Transmit Throw placenta (rare) or birth canal (more common)
  - If the mother had her first attack (primary) during pregnancy the risk is higher than recurrent attack (secondary).
  - Primary Herpes infection in the late third trimester is far more dangerous than earlier infection
  - If lesion are present, **cesarean section** is the optimal mode of delivery
  - Infection can cause neonatal viral sepsis, herpetic lesions on skin, eyes,
  - pneumonia, herpes encephalitis which can lead to neurological abnormality and death.
  - Infected infants should be treated with I.V. acyclovir

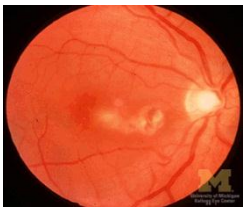
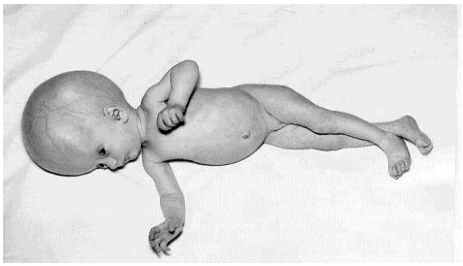
- **Syphilis(Treponema pallidum) :**
  - The longer syphilis exists in a women before pregnancy less likely the fetus will be affected
  - Spread by intercourse and oral
  - Transmit throw placenta
  - Can lead to abortion, still birth, or **congenital syphilis (maculopapular rash, hepato-spleenomegaly, lymphadenopathy, jaundice, 8th nerve deafness, saber shins (malformation of the tibia), Hutchinson's teeth, saddle nose)**
  - **Screening :** in the first antenatal care visit → VDRL or the rapid plasma regain test RPR
  - **Diagnosis :** IGM antitreponemal antibodies, and darkfield microscopy of lesions

- **Treatment:** Penicillin
- Latent Syphilis may not transmit the disease.

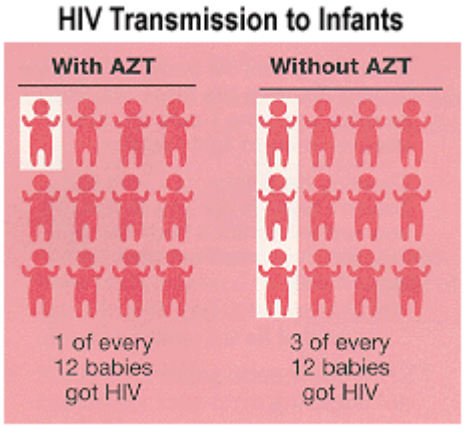




- **Toxoplasmosis [Toxoplasma gondii] :**
  - Host = cat
  - Risk factors: Consumption raw/undercooked meat, contact cat faeces, contaminated water
  - Risk of fetal infection (through vertical transmission[placenta])
    -In 1st trimester is 15%(perinatal death rate is 75%),
    -In 2nd trimester is 25%, and
    -In 3rd trimester is 65% (all are mild or subclinical)
  - 25% OF infected infants are symptomatic and 75% are asymptomatic.
  - Overt inf. Result in mononucleosis-like syndrome
  - Can lead to classic triad:- **hydrocephaly, intracranial calcifications , chorioretinitis,**
    other:-**Hepatosplenomegaly and CNS involvement (mental retardation and seizers).**
  - If IGM titer is rising, spiramycin or pyrimethamine and sulphonamide are the treatment.

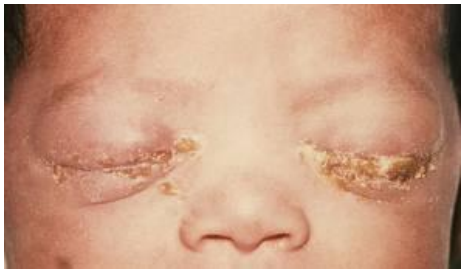


- **Human immunodeficiency virus**
  - 30% of infants born to HIV infected mothers will become infected with HIV
  - Vertical transmission is 13-30% and the rest is through the birth canal
  - HIV testing: ELISA AND WESTERN BLOT
  - There is no increased risk of preterm labor, low birth weight, or congenital malformations
  - **Prevention by : very important**
    - Cesarean section lower the transmission rate by two third in patients with no therapy
    - AZT (Zidovudine) that decrease the viral load during ante partum, intrapartum, and neonatal period can reduce the risk of fetal infection by two thirds in mildly symptomatic ladies (Newborn is given I.V AZT)
    - Avoidance of breast feeding reduce the risk of transmission by half

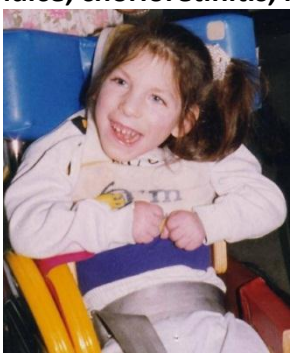


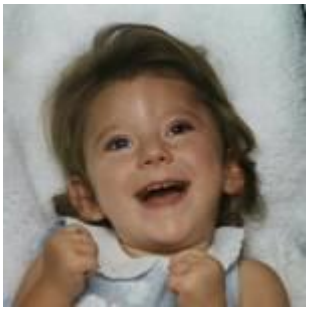
- **Chlamydia trachomatis** :
  - Infection is through the birth canal.
  - 40% of infants will develop conjunctivitis, 10% will develop pneumonia
  - Treatment is by erythromycin or azithromycin
- **Hepatitis B**
  - Transmission is vertical specially in the third trimester in acute infection
  - 75% OF PATIENTS ARE ASYMPTOMATIC
  - Fetal infection uncommon but most of them occur in 3rd trimester
  - HBsAg + anti-Hbe + → low infectivity 10-25% risk
  - HBeAg + HBsAg + →indicate high infectivity 90% risk
  - The baby should be given Hepatitis B immunoglobulin at birth and an active immunization and repeated at 3 and 6 months.
  - Cesarean section or breast feeding is unlikely to alter the incidence of neonatal infection
- **Group B Streptococci (GBBS)** :
  - 35% of ladies carry GBS in vagina
  - Transmission rate of GBBS to neonate at delivery is 50%
  - Attack rate of GBBS sepsis to neonate is 1%
  - Infection through birth canal
  - It is associated with **PROM**, preterm delivery, and intrapartum and puerperal fever
  - Can lead to neonatal meningitis, pneumonia, sepsis
  - Intrapartum prophylaxis is indicated for carriers
  - Diagnosis: culture or rapid assays which have low sensitivity and high specificity
  - Prevention of GBBS sepsis: intrapartum prophylaxis antibiotics
- **Listeria monocytogenes :**
  - Rare bacterial infection by food
  - It can cross the placenta leading to amnionitis ,preterm labor, abortion, still birth, jaundice, conjunctivitis, meningoencephalitis
  - Treatment by amoxil or erythromycin
- **Gonorrhea** :
  - Infection **through birth canal**.
  - Can lead to conjunctivitis, arthritis, meningitis.
  - Treatment by Penicillin and probenicid, or erythromycin.



- **Parvovirus B19 :**
  - Causes **<u>erythema infection</u>**, fifth disease or slapped cheek syndrome.
  - Vertical transmission can lead to hydrops fetalis, hemolytic anemia, myocarditis, abortion, death

- **Cytomegalovirus**:
  - Affected infants have **30% mortality, they may develop mononucleosis-like syndrome, mental retardation, hearing loss, cerebral calcifications, hepato-spleenomegaly, thrombocytopenia, jaundice, chorioretinitis, interstitial pneumonitis.**





	TOXOPLASMOSIS	RUBELLA	CMV	HSV	SYPHILLS	HEP B	HIV	GBBS
Origin involved	Parasite(toxoplasma gondii)	Virus RNA	Virus DNA	Virus DNA	Treponema pallidum	Virus DNA	Virus RNA	bacteria
Mode of trans.	Cat litter, raw meat, raw goat milk	Air droplets	Body fluids	Mucocutanous contact	Mucocutanous contact	Body fluids	Body fluids	genital tract colonization, feces
Seropositivity in general population	10-40%	85-90%	50%	50%	N/A	1%chronic carriers	0.1%	35% GENITAL COLONIZATION
Treatment in preg.	Pyrimethamine and sulfadiazine	None	Gancyclovir	acyclovir	Benzathine, penicillin	Hep B immune globulin	Zidovudine(AZT)	Penicillin
Routine screen.?	No	Yes	No	No	Yes	Yes	Yes	No
Residual status	Lifelong immunity	Lifelong immunity	Lifelong latency	Lifelong latency	Lifelong latency(if not treated but it's curable)	Lifelong immunity(with carrier status)	Lifelong inf.	Lifelong colonization
Transplacental inf. risk	Yes	Yes	Yes	Yes	Yes	Rare	Yes	Rare
Delivery(inf. risk)	No	No	Rare	Yes	No	Yes	Yes	Yes
Recommended delivery route	Vaginal	Vaginal	Vaginal	C/S if lesions at delivery and active	Vaginal	Vaginal	Vaginal	Vaginal
Maternal and infant immunization?	No	Yes(active ones) MMR	No	No	No	Yes(both active and passive)	No	No

Intrauterine Growth Restriction [IGUR]

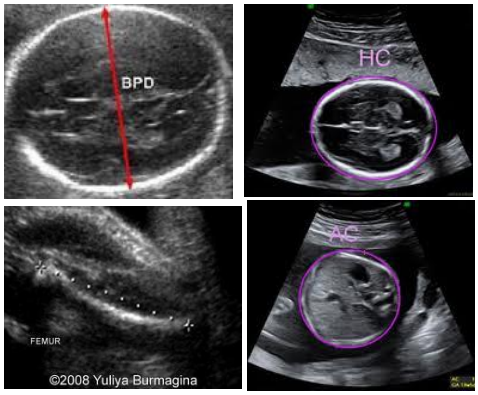
-Definition: when birth weight of newborn is below 10th percentile for a given gestational age.

-**Etiology:** Categorized into <u>three</u> types: Maternal, Placental, Fetal.

1. Maternal: poor nutrition, smoking, drug abuse, alcoholism, chronic renal disease, cyanotic heart disease, pulmonary insufficiency, antiphospholipid syndrome, hereditary thrombophlebitis.
2. Placental: inadequate substrate transfer because of placental insufficiently, condition lead to this state like: essential hypertension, chronic renal disease, and pregnancy induced hypertension.
3. Fetal: intrauterine infection (toxoplasmosis, rubella, TORCH, …etc.) congenital anomalies.

-**Diagnosis: Fundal height**, and assessment of **sonographic parameters** which are:

Sonographic parameters
-Bi-parietal diameter (BPD)
-Head circumference (HC)
-Abdominal circumference (AC)
-Head-to-abdominal circumference
-Femur length
-Femoral length-to-abdominal circumference
-Calculated fetal weight
-Amniotic fluid volume
-Umbilical and uterine artery Doppler



- Note: <u>Femur length</u> is most accurate to estimate gestational age, and abdominal circumference is most effective parameter to predict decrease fetal weight because its reduce in both symmetrical and asymmetrical IUGR.

- head circumference is greater than abdominal until 34 weeks the ratio point approach 1, after 34 weeks abdominal circumference will be greater than head.
- Doppler-derived umbilical artery systolic-to-diastolic ratios are abnormal in IUGR fetuses.

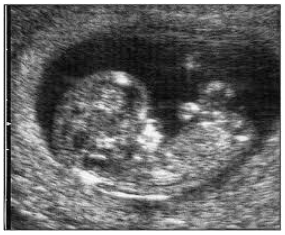
<u>Types:</u>

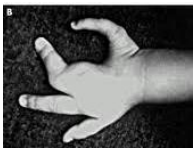
A-Symmetrical IUGR: BPD, HC, AC, FL all decrease. That's why head-to-abdominal circumference maybe normal but absolute growth rate will decrease.

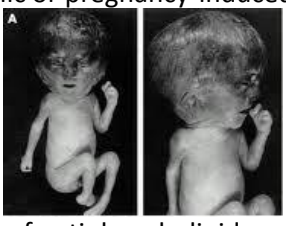
1- Develop in early pregnancy (first or early second trimester).
2- Associate with intrauterine infection and congenital anomaly (fetal causes).
3- Amniotic fluid volume is normal.

B-Asymmetrical: only AC decreases but BPD, HC, and Fl all are normal→ head -to-abdominal circumference will ↑.

1- Develop late in pregnancy (late second or third trimester).
2- Associate with placental causes like hypertension (chronic or pregnancy-induced), and maternal causes such as: poor nutrition, smoking.
3- Amniotic fluid volume will decrease.







-**Management**:

1- Pre-pregnancy: improve nutrition, stop smoking, in case of antiphospholipid syndrome with prior IUGR infant → low-dose aspirin.
2-Antepartume: the goal is to expedite delivery before fetal compromise occur, if US parameter strongly suggest IUGR→ delivery is indicated at GA 34 weeks or more, or any reasonable GA if lungs are mature.
3- Labor and delivery: monitor during labor for high risk patient to detect fetal distress early, after birth→monitor blood glucose is important because hypoglycemia is common, RDS is common with distress because fetal acidosis reduce surfactant production.

Intra-uterine Fetal Demise [IUFD]

-Definition: death after 20 weeks but before onset of labor.

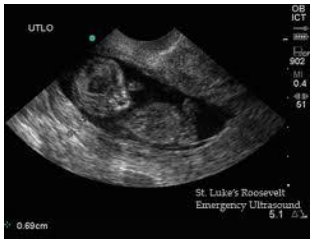
-**Conformation**: initially, absence of uterine growth or absence of fetal movement.

-Confirmatory by physician:

- Absence of fetal cardiac activity detected by:
    1. Auscultation: by Fetoscopy or Doppler stethoscope.
    2. Electronic fetal monitoring (EFM): external sonogram or fetal scalp electrode.
    3. C-US visualization (real time US): 100% accurate and method of choice.
- Radiographic finding: rarely used today: Roberts sign, Spalding's sing, Angulation of spine.
- Absence of fetal motion by US.

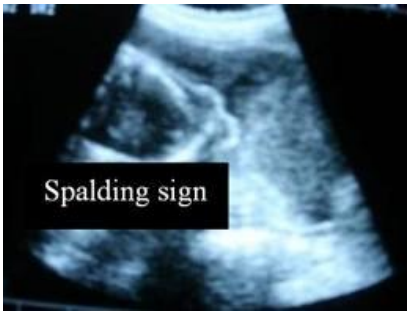
-Determining how long the fetus has been dead→ *<u>important to assess risk of DIC.</u>*

- Changes noted **immediately** after FD : absent cardiac activity, and fetal movement.
- Changes noted **after** few days: --Radiographic findings .**see above**.
- Amniocentesis: reveals port wine-colored amniotic fluid.
- US findings: generalized edema.



-**Causes**:

- **Idiopathic:** most common (50% of cases).
- **Placenta or cord problem:** abruptio placenta, fetomaternal hemorrhage (fetus will bleed into maternal circulation and become hypovolemic.
- **Fetal causes:**
    a. Abnormal karyotype (trisomy is most common).
    b. Anatomic anomalies.
    c. Infection: in first trimester → lead to demise or teratogenic effect.



- **Maternal causes:**
    - **Systemic diseases**
        a. <u>Anticardiolipin antibody-lupus anticoagulant-antiphospholipid syndrome</u> (autoimmune condition, in which maternal antibody attack fetoplacental unite.
            i. History of venous thrombosis, and RFL.
            ii. Diagnosis confirmed with prolonged partial thromboplastin time (PTT) or ↑ level of anticardiolipin antibody, lupus, and antiphospholipid antibody.
            iii. Treatment: low dose of one or more of: aspirin, heparin, and prednisone.
        b. <u>Type I DM:</u> fetal because of vascular disease, poor control, macrosomia, polyhydramnios.
    - **Maternal trauma:** cause hemorrhage → vasoconstriction, diminish uteroplacental blood flow result in fetal death
        a. Penetrating trauma (gun shot, stab wound) →direct injury to fetus.
        b. Blunt trauma.
    - **Maternal isoimmunization.**

✓ - It is important to determine the cause.

- Management: **<u>1- Cervical examination:</u>** is cervix is favorable for delivery or not?

- If favorable → induce labor, AROM and IV oxytocin used to induce labor.
- If not favorable: IOL **[see IOL summary]**.

<u>2- D&E:</u> if → 1- gestational age less than 20 weeks.
2- Autopsy is not necessary.

Notes:

-DIC: is most serious complication, triggered by release of tissue thromboplastin from fetus and placenta into maternal circulation (hypofibrinogenemia). DIC is rare until 4 weeks after fetal demise.

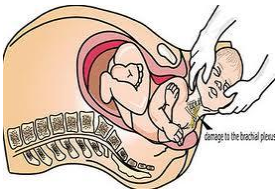
-Delivery should be attempted if: a- 4-5 weeks passed since fetal demise.
b- Serum fibrinogen level is <200mg/ml.
c- Platelet count decrease <100,000/ml.

Post-term Pregnancy

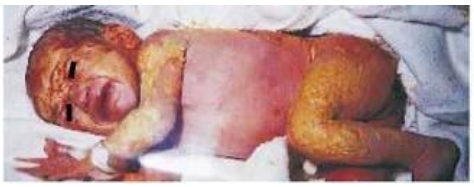
Definition: pregnancy that persisted more than 42 weeks from the onset of the LMP.

Epidemiology: -12% of all pregnancies.
-High incidence in Anencephalic fetuses, Placental sulfate deficiency and extrauterine pregnancy.

Complication:-Perinatal mortality is 3 times higher.
-Fetal post-maturity (dys-maturity) syndrome.
-Macrosomia (>4kg) results in:-Abnormal labor
-Shoulder dystocia
-Birth trauma
-Post-partum hemorrhage (atony).
-Increased incidence of C/S



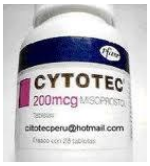
Fetal post-maturity syndrome: -Definition: placental insufficiency with impaired O2 diffusion and nutrients transfer due to aging & infarction of the placenta.
-Characteristics: -Loss of subcutaneous fat.
-Long fingernails.
-Dry and peeling skin
-Abundant hair



Etiology: -Unknown in most instances
-Lack of fetal labor-initiating factor from fetal adrenal gland (seen in Anencephalic fetus).
-Placental sulfate deficiency
-Extrauterine pregnancy
-Paternal genes

Management: -Labor usually should be induced.
-If the cervix is unripe: -Twice-weekly NST, AFI & Biophysical profile (BPP).
-Delivery is indicated if there is spontaneous deceleration in NST or any indication of Oligohydramnios (AFI ≤ 5).
-C/S is indicated for fetal distress.

Induction of labor (IOL)



Definition: -Process of artificially initiating labor.

Methods: A-Inducing cervical ripping; -Pharmacological: -PgE1 (Cytotec) [intravaginal tablets]
-PgE2 (Cervidil) [vaginally inserted].
-Osmotic dilators (laminaria or tent).

-Mechanical: -Intrauterine placement of Catheters.
-Artificial rupture of membrane (Amniotomy), it can also increase uterine activities.

B-Inducing uterine contraction; -IV Oxytocin-[the only one used]
-$T_{1/2}$= 1-5 min.
- Oxytocin infusions shouldn't exceed 72 hours.
- Oxytocin complications. **(See table)**

Oxytocin complications
-Hyperstimulation -> fetal distress.
-Rupture of uterus.
-Antidiuretic effect.
-Severe water intoxication (-/+ convulsions, coma)
-Uterine muscle fatigue.
-Post delivery uterine atony -> PPH.

Prerequisites: -Continuous electronic monitoring of fetal heart rate & uterine activity during IOL.
-Patient's blood typed & screened for antibodies.
-Blood specimen should be held in case of cross-matching needed.

Indications & contraindications: - (see tables)

Maternal indication	Fetoplacental indications
-Prolonged pregnancy	-Abnormal fetal testing
-Preeclampsia	-Rh incompatibility
-Diabetes mellitus	-Fetal abnormality
-Heart disease	-PROM
-IUGR	-Chorioamnionitis

Maternal contraindication	Fetoplacental contraindications
Absolute:	**Absolute**:
-Contracted pelvis	-Preterm fetus with immature lungs.
Relative:	-Acute fetal distress
-Hx of uterine surgery	-Abnormal presentation.
-Hx of Classic C/S	
-Hx of complete transection (myomecteomy,reconstruction)	
-Overdistended uterus	

Bishop score: -a score is given after pelvic examination to assess the likelihood of a successful IOL.
-Evaluates; a-Status of the cervix [CPDEكبدي]; -Consistency
-Position
-Dilatation
-Effacement (cervical length)
b-Station of fetal head.
-Score; - If 9-13 good likelihood of vaginal delivery.
- If < 5 poor likelihood success.

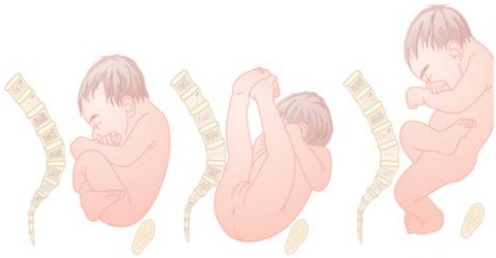
Abnormal Fetal Presentations

A. Breech presentation: occurs when the fetal buttocks or lower extremities present into the maternal pelvis. **The incidence is 4% of all deliveries.** The percentage of breech decreases with advancing gestational age from 25% of births prior to 28 weeks gestation to 1-3% of births at term.

- **Predisposing factors:** [abnormal fetal or uterine size]
  prematurity (major cause)**,** uterine anomalies (e.g., bicornuate uterus), fetal abnormalities, multiple gestation, placenta previa, hydramnios, contracted maternal pelvis, and pelvic tumors that obstruct the birth canal.

- **Diagnosis:** often made by **Leopold examination**.

- **Types of breech:**
  1. **Frank 65%:** hips flexed - knees extended**,**
  2. **Complete 25%:** hip flexed - knees flexed.
  3. **Incomplete or footling 10%:** one or both hips extended.

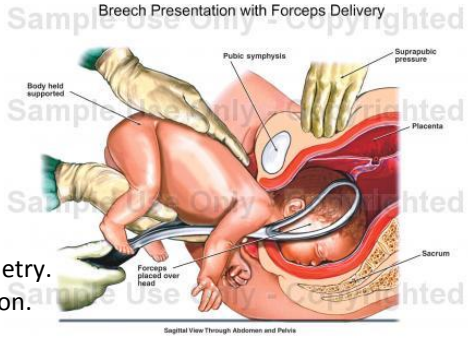


Complete **Frank** **Footling**

- **Pregnancy Management:**
  1. **Exclude fetal and uterine anomalies.**
  2. **External cephalic version (ECV): -**after **36 to 37 weeks** b/c there is a tendency to revert back to breach**.**

-(See operative delivery for more details)

- **Labor Management:**
  1. **Cesarean section:** The standard of care now.
  2. **Assisted Breech Delivery**: Criteria for vaginal delivery:
     - Fetus must be in a **frank or complete** breech presentation.
     - Gestational age **> 36 weeks**.
     - Fetal weight should be **2500-3800 g**.
     - Fetal head must be **flexed.**
     - **Maternal pelvis** must be adequately **large,** as assessed by x-ray pelvimetry.
     - There must be **no** other maternal or fetal **indication** for cesarean section.
     - **Anesthesiologist** must be in attendance.
     - Obstetrician must **be experienced.**
     - **Assistant** must be scrubbed and prepared to guide the fetal head into the pelvis.



- **Note:** Factors that contribute to increased perinatal morbidity and mortality include **lethal congenital anomalies, prematurity, birth trauma, and asphyxia.**

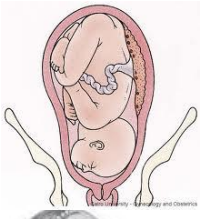
B. Face presentation: occurs when the fetal head is hyperextended such that the fetal face, between the chin and orbits, is the presenting part.

Predisposing factors:
Extreme prematurity, high maternal parity, and congenital anomalies such as fetal goiter. In the majority, however, no etiologic factor is evident.

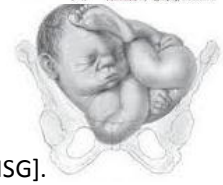
Types of face presentation: (mento-anterior 60%, mento-transverse 15%, mento-posterior 25%).

Labor management: mento-anterior (vaginal delivery), **mento-posterior** and persistent **mento-transverse** must be delivered by **cesarean section.**

C. Brow presentation: occurs when the presenting part of the fetus is between the facial orbits and anterior fontanelle. As a result of extension of the fetal head midway between flexion (vertex presentation) and hyperextension (face presentation)**;** the presenting diameter is the **supraoccipito-mental diameter,** delivered by **cesarean section**.



D. Compound presentation: -more than one part is presented. E.g. shoulder presentation.
-Deliver by C/S



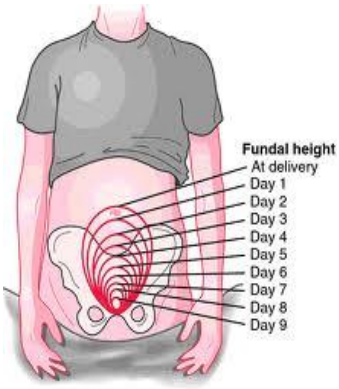
****Note:** recurrent abnormal presentation may indicate uterine anomaly so do hysterosalpingogram [HSG].

The Puerperium & Puerperal Infections

Definition:-The time immediately after the delivery of a baby (In the past it's the first 6 weeks but now it varies).

In puerperium: -Pelvic organs return to the non-gravid state,
-Metabolic changes of pregnancy are reversed and lactation is established.
-Puerperal Infections.

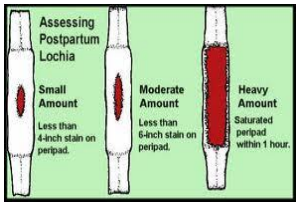
Puerperal care objectives: -To monitor the physiological changes of the puerperium.
-To diagnose and treat any postnatal complications.
-To establish infant feeding.
-To give the mother emotional support.
-To advise about contraception.



Main Puerperal changes:

A-Uterus; =Corpus -Uterine involution starts immediately after delivery this process in aided by Oxytocin.
-Oxytocin produced in response to nipple stimulation from the baby suckling.
-Immediately post-delivery it weighs 1kg and the fundus is at the level of umbilicus.
-By 10-14 days the fundus is usually behind the symphysis pubis.
-Delay in involution due to: -Infection
-Placenta problems

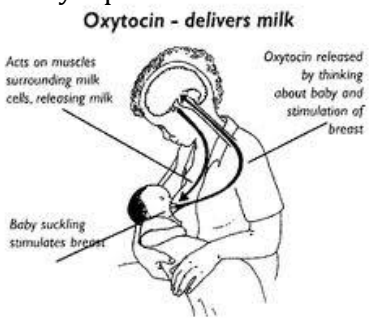
=Endometrium: -Superficial layer of decidua becomes necrotic & is sloughed off as lochia.
-Lochia = Blood + leucocytes.



Color	Timing
-Lochia rubra (red)	Immediately after delivery
-Lochia serosa (pinkish-yellow)	During the 1st week
-Lochia alba (white)	During the 2nd week

-Basalis layer of decidua is the source of new endometrial regeneration.

B-Breast:-Estrogen & Progesterone antagonize Prolactin effect in the production of milk during pregnancy.
-Mechanisms of lactation; -Prolactin release acts on the glandular cells of the breast to secrete milk.
-Prolactin secretion occurs in response to nipple stimulation.
-Oxytocin release from post. Pituitary acts on the myoepithelial cells of the breast to induce the milk ejection reflex.



C-Fluid balance changes; -More than 10 L of fluid is lost during the Puerperium.
-2 L in the first week & 1.5 L lost during each of the next 5 weeks

Management of normal puerperium

1. Education
   - Early mobilization to prevent DVT
   - Breast feeding as soon as possible to prevent breast engorgement and to give immune to baby because in first days the breast milk contain clostrume which give high immunity to the baby
   - Diet
   - Contraception
   - Education about symptoms of puerperium (**heavy diuresis in first 2 days**)
   - Postnatal visit in 6 weeks after delivery.
2. Vital signs (HR, BP, Temp, RR) monitoring
3. Abdominal examination for Uterus contraction
4. Vaginal examination looking for bleeding (PPH)

5. Look for hemorrhoid
6. Episiotomy management (is there any pain or hematoma)
7. Hematocrit to see any active bleeding or anemia
8. Rh- antibody for o negative women
9. Breast examination to see if there is any inflammation +lower limb examination for the detection of signs of DVT every day.
10. The mother should be encouraged to pass urine and make sure there is no obstruction or hematoma.

Complications of Puerperium:

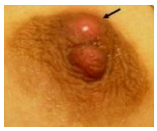
1-PPH (postpartum hemorrhage) [see summary]

2-Thromboembolism; -30 min after delivery → fibrinolytics will ↑ → ↑ chance for thrombolic diseases.

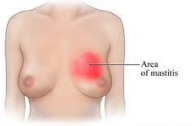
3- Pelvic Infections;-Predisposing Factors; -Hx of prolonged rupture of membranes.
-Protracted labor with multiple vaginal examinations
-Retained products of conception

-Most common organism is β hemolytic strept. E. Coli may also be responsible.
-Clinical features; -Pyrexia, Offensive lochia (smelly), Lower abdominal discomfort, Tender uterus and cervical excitation tenderness.
-Diagnosis; -Clinically, Swabs (from cervix, HVS, Urethra), and Blood culture.
-Treatment; -Evacuate the uterine cavity under anesthesia.
-IV broad spectrum antibiotics (Cephalosporin and Metronidazole).
-If untreated may progress to peritonitis, septicemia or bacteremic shock.

4- Breast infections; -Mastitis; a-Acute inflammatory mastitis.



b-Infective mastitis



c-Breast abscess; -Develops usually after the 14th day.
-Red, painful, fluctuant swelling, fever and ill looking.
-Antibiotics, incision and drainage under general anesthesia

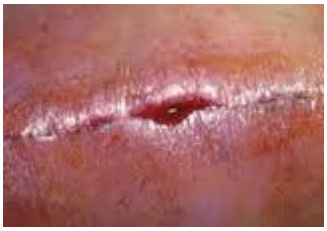
5- Bowel complications; -Constipation & Hemorrhoids are common.

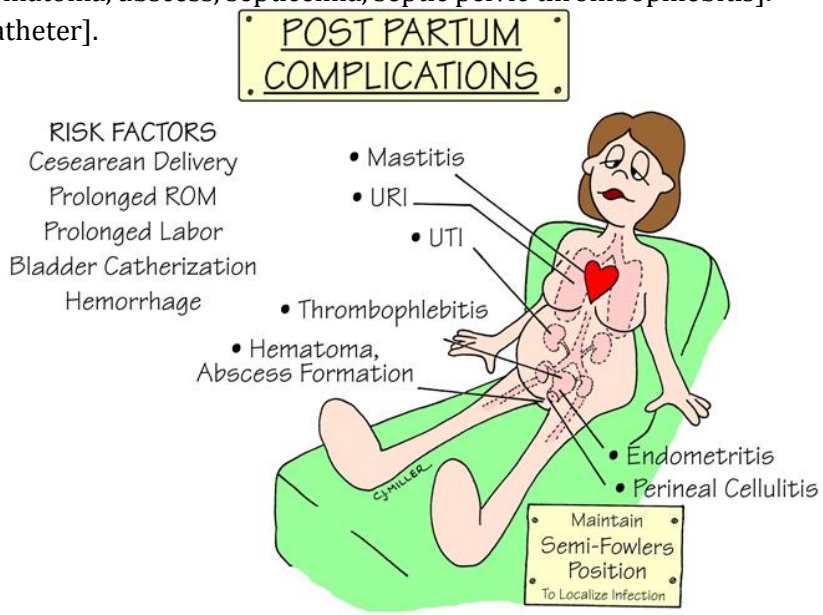
6-Urinary complications; -UTI
-Incontinence

7-Mental disorders; -Postpartum blues (80%) [Care of self & infant maintained]
-Postpartum depression (20%) [Care of self & infant neglected]
-Postpartum psychosis (0,1%) [May express ideation to harm self and/or infant].

If puerperal pyrexia happened, please check:

-Chest [upper respiratory tract infection, atelectasis from general anesthesia]
-Breast [mastitis].
-Pelvic organs [endometritis, hematoma, abscess, septicemia, septic pelvic thrombophlebitis].
-Urinary tract [infection from catheter].
-Surgical wounds [infection].
-Legs [DVT].





Hypertensive Disease in pregnancy

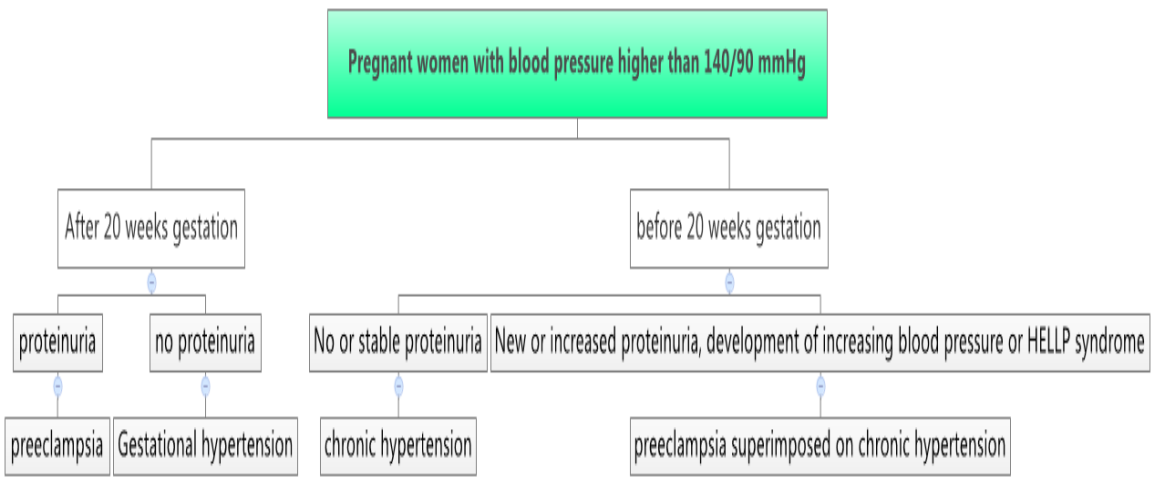
- **Classification**

1. **Gestational Hypertension**: hypertension that arises after 20 weeks gestation without any features of pre-eclampsia and resolves within 3 months [12weeks] after delivery.
2. **Pre-eclampsia**: multisystem disorder and the **hypertension** is the first manifestation followed by **proteinuria** or edema **after 20 weeks** gestation and resolves within 3 months of delivery.

   - Clinical diagnosis of pre-eclampsia:
   - **Mild pre-eclampsia:**
      1. **BP** of **≥ 140/90** mm Hg or :
         increase in **diastolic** BP of **≥ 15 mmHg** or increase in systolic BP ≥ 30 mm Hg.
      2. **Proteinuria:** 1-2+on dipstick or ≥ 300 mg/24 hours urine collection.
      3. **Edema** of the face or upper extremities.
   - **Severe preeclampsia:**
      1. **BP of ≥ 160/110** mm Hg.
      2. **Proteinuria** of **≥ 3-4+** on dipstick or **≥ 5 g /24 hours** urine collection.
      3. **Headache:** from cerebral edema
      4. **Visual disturbances**: from decreased cerebral perfusion of the occipital cortex.
      5. **Epigastric pain** from hepatocellular necrosis, edema, and ischemia stretching **glisson's capsule**.
      6. **Pulmonary edema** and **cyanosis.**
      7. **Oliguria** less than 500ml per 24 hour.
      8. **Thrombocytopenia:** ≤100,000 from vasospasm induced macroangiopathic hemolysis.
      9. **HELLP syndrome:** characterized by **H**emolysis, **E**levated **L**iver enzyme, **L**ow **P**latelet.
   - **Other symptoms:**
      1. Neurological problem → convulsions (eclampsia), hyperreflexia with clonus, severe headache with hyperreflexia, persistent visual disturbance (scotomata)
      2. Hematological disturbance → DIC
      3. Fetal growth restriction [IUGR]

- **Chronic hypertension**: blood pressure ≥ 140 /90 mm Hg prior to conception or in the first half of pregnancy.
   - Diagnosis : BP measurement of ≥ 140/90 mm Hg on two occasions + Before 20 weeks of gestation OR Persisting beyond 12 weeks postpartum.
   - Treatment of chronic hypertension has no benefits in preventing preeclampsia.
   - Antihypertensive treatment is needed to prevent maternal end-organ damage.

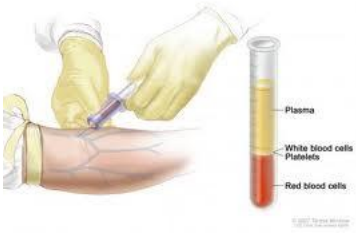
- **Pre-eclampsia superimposed on chronic hypertension:** the patient is known to have hypertension and is aggravated by pregnancy and usually carries a **worse prognosis**, it is suspected by new develop of proteinuria or sudden significant increases in B/P or proteinuria after the 20 weeks of pregnancy
- If BP arise earlier after 14 weeks and we should suspect hydatidiform mole or multiple pregnancy.

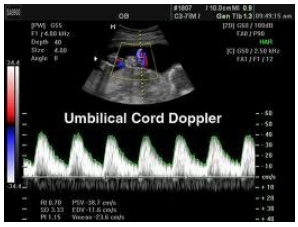


- ## Pre-eclampsia:
- **Etiology:** It is the disease of theories. (**Incidence:** 5-8%)
- **Pathophysiology :** late in the first trimester the secondary invasion of maternal spiral arteries by trophoblasts is impaired →impairment of placenta function → damaged endothelial cells will secret vasoconstrictors (e.g. thromboxane) exceeding the vasodilators (e.g. prostacyclin) → generalized vasoconstriction and decreased in aldosterone secretion = maternal hypertension + reduction in placenta perfusion + reduced maternal plasma volume → if vasospasm persist → trophoblastic injury → trophoblastic fragment carried to the lung where they are destroyed releasing thromboplastins → thromboplastins cause intravascular coagulation and deposition of fibrin in the glomeruli of the kidneys (glomerular endotheliosis) which reduce GFR and indirectly increase vasoconstriction → in severe cases fibrin deposits occur in vessels of the central nervous system leading to convulsions .
- **Increased capillary permeability:** results in:
    a. **Hemoconcentration:** from decreased intravascular volume (↑blood urea nitrogen (BUN), ↑ creatinine, ↑uric acid, ↑ hemoglobin, ↑ hematocrit)
    b. **Edema**.
    c. **Excessive weight gain:** form fluid retention.

Risk factors of preeclampsia	Complications of preeclampsia
-**Nulliparity** (most common). -**Extremes of age** (<20-year, >34-year). -**Chronic hypertension**. -**Multiple pregnancy**. -**Diabetes mellitus**. -Chronic renal disease. -Hydatiform mole. -Small vessel disease: SLE, DM type 1. -Obesity. -Black race. -Family history. -Past history. -Antiphospholipid syndrome.	-Placenta abruption. -Acute renal failure. -Intracranial hemorrhage. -DIC. -Retinal detachment. -IUGR. -Prematurity. -Placental insufficiency. -Liver hematoma.

- **Tests and fetal surveillance (gestational hypertension or preeclampsia) to assess the severity (imp) :**
    - **CBC** for platelet.
    - **Urinalysis.**
    - **Electrolytes.**
    - **Coagulation profile.**
    - **Liver function test** (ALT, AST).
    - **24-hour urine collection** for creatinine clearance and protein excretion.
    - **Uric acid.**
    - Serum creatinine.
    - Lactate and lactate dehydrogenase.
    - Indirect bilirubin → to detect hemolysis
    - Serum lipids
    - Fetal surveillance: fetal movement count, CTG, serial ultrasound to detect IUGR, and Doppler umbilical blood flow.



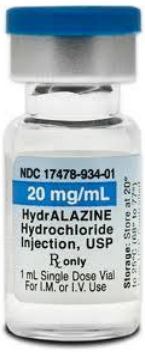


- ## Management (see summary next page)
- There is no cure except to **terminate the pregnancy** and treatment is buy time so that fetus becomes more mature in the uterus +monitoring + antihypertensive to prevent end organ failure.

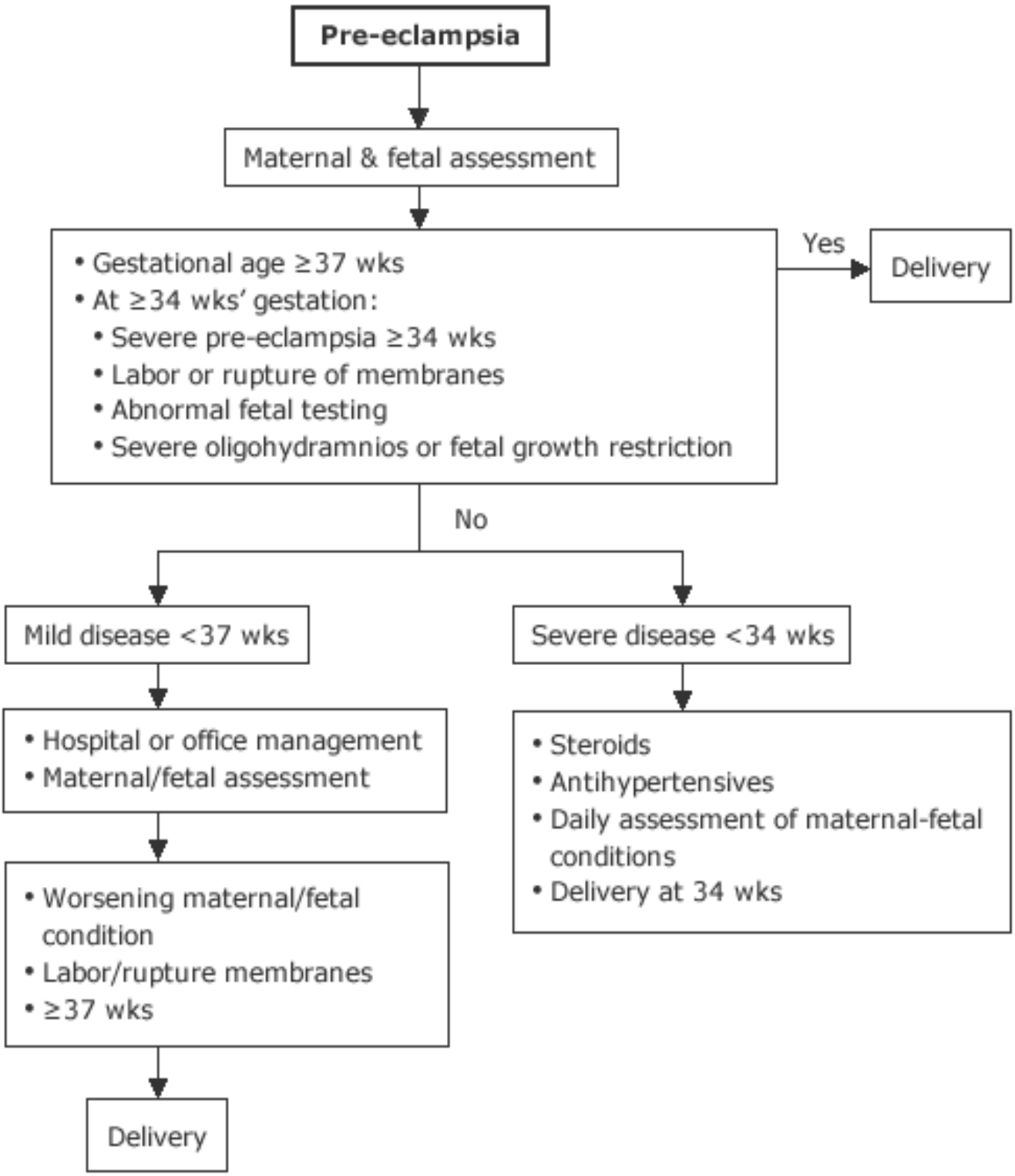
1. **Aggressive inpatient management (immediate delivery):**
   - **A. Mild or severe preeclampsia:** ≥ 37 weeks' gestation.
   - **B. Severe preeclampsia: when associated with:**
     - **i. Maternal jeopardy:**
       - a. Persistent severe headache or visual changes.
       - b. Persistent severe epigastric pain, nausea, or vomiting
       - c. Thormbocypenia or DIC**.**
       - d. Pulmonary edema.
       - e. Suspected placental abruption.
     - **ii. Fetal jeopardy:**
       - a. Repetitive late decelerations.
       - b. Repetitive BPP ≤ 4.
       - c. Severe intrauterine growth restriction.
       - d. Oligohydramnios
   - C. **Superimposed preeclampsia:** at any gestational age.
   - D. **Eclampsia or HELLP syndrome:** at any gestational age.

     Eclampsia:
     - Eclampsia = preeclampsia + convulsion
     - Incidence; 25% in antepartum period 50% during labor and 25% occur postpartum period.
2. **Conservative inpatient management: (antihypertensive +steroid for lung maturity + monitoring)**
   - **a. Mild preeclampsia**: < 37 weeks' gestation.
   - **b. Severe preeclampsia:**
     1. Gestational age: **> 26 weeks**.
     2. BP persistently **≥ 160/110** mm Hg
     3. **Absence** of maternal or fetal jeopardy.

- **Antihypertensive therapy :**
  - Should be initiated when diastolic B/P is more than **105 mm Hg** to prevent **CNS hemorrhage.**
  - **Hydralazine and labetalol** are used to control severe hypertension.
  - **Nifedipine** calcium ion influx inhibitor blocker is another option.
  - Alpha **methyldopa** is safely used in chronic hypertension.
  - **AVOID:**
    1. **ACEI and ARBs**: because of teratogenicity.
    2. **Atenolol**: has been associated with IUGR.
    3. **Diuretics**: exacerbate intravascular fluid depletion.
    4. **Labetalol** in women with asthma or congestive heart failure.



- **Management of eclampsia :**
  - **Prevention:** In patients with preeclampsia generally involves the use of magnesium sulphate as an agent to prevent convulsions, and thus preventing eclampsia.
  - Prevent the women injuring her self during convulsion.
  - Give oxygen mask.
  - Insert IV line for blood test and drug and fluid administration.
  - Foley catheter for input and output charting.
  - The best and safest drug for controlling convulsion is **magnesium sulfate.**
  - After stabilization delivery is considered either by induction of labor or by caesarian section.
  - Prophylaxis against convulsion is continued **after** delivery.


Pre-eclampsia
Maternal & fetal assessment
• Gestational age ≥37 wks
• At ≥34 wks' gestation:
• Severe pre-eclampsia ≥34 wks
• Labor or rupture of membranes
• Abnormal fetal testing
• Severe oligohydramnios or fetal growth restriction
Yes
Delivery
No
Mild disease <37 wks
Severe disease <34 wks
• Hospital or office management
• Maternal/fetal assessment
• Steroids
• Antihypertensives
• Daily assessment of maternal-fetal conditions
• Delivery at 34 wks
• Worsening maternal/fetal condition
• Labor/rupture membranes
• ≥37 wks
Delivery

GDM & DM

- **Gestational Diabetes**: is a condition in which women without previously diagnosed diabetes exhibit high blood glucose levels during pregnancy → especially during third trimester due to increase insulin resistance and usually comes normal after delivery.
- **DM during pregnancy:** is a condition in which women previously diagnosed diabetes.
- **Risk factors for GDM :** (see table)

Risk factors
-Hx of GDM -Family Hx of GDM -Hx of any GDM related maternal or fetal complication -Obesity -Prediabetes -Age (> 25 years)

Rule of 15s
-15% of gravidas have abnormal GCT -15% of gravidas GCT +ve have abnormal GTT -15% of GDM patients require insulin -15% of GDM patients have macrosomia

	Gestational diabetes	Type 1 diabetes	Type 2 diabetes
Common name	Pregnancy-induced	Juvenile-onset	Adult-onset
When diagnosed	During pregnancy (usually last half)	Onset prior to pregnancy	Onset prior to pregnancy
Mechanism	Insulin resistance	Pancreatic islet-cell destruction	Insulin resistance
Plasma insulin levels	High	Low	High
Therapeutic modalities	Diet (15% need insulin)	Insulin Diet Exercise	Insulin Diet Exercise
How diagnosed	Screening: GCT Diagnosis: GTT	Unable to achieve nonpregnant euglycemia without insulin	Unable to achieve nonpregnant euglycemia without insulin
How assess success	Home blood glucose monitoring (Blood sugar series)	Home blood glucose monitoring (Blood sugar series)	Home blood glucose monitoring (Blood sugar series)
Blood glucose target values	FBS < 90 mg/dl 1-hour PP < 140 2-hour PP < 120	FBS < 90 mg/dl 1-hour PP < 140 2-hour PP < 120	FBS < 90 mg/dl 1-hour PP < 140 2-hour PP < 120
↑ risk of fetal anomalies	None (because occurs after embryogenesis finished)	Possible	Possible
Fetal growth abnormalities	Macrosomia [No IUGR)	Macrosomia IUGR (if small vessel disease)	Macrosomia IUGR (if small vessel disease)
Neonatal hazards	Hyperbilirubinemia Polycythemia Hypoglycemia Hypocalcaemia RDS (↓surfactant)	Hyperbilirubinemia Polycythemia Hypoglycemia Hypocalcaemia RDS (↓surfactant)	Hyperbilirubinemia Polycythemia Hypoglycemia Hypocalcaemia RDS (↓surfactant)

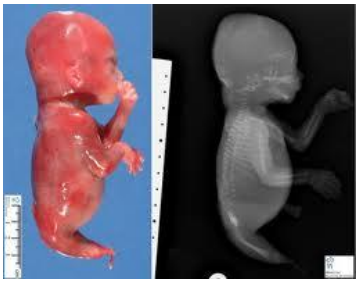
- **Maternal & Fetal complication in case of DM & GDM: (see tables)**

Maternal complication in case of GDM
-Increase risk of preeclampsia -Increase rate of instrumental delivery -Increase C/S rate -Vaginal Laceration -Bleeding from laceration -Polyhydramnios -Increase the risk of having DM in the future

Maternal complication in case of DM
-Increase risk of miscarriage -Increase risk of preeclampsia -Increase risk of infection eg vaginal candidiasis, UTI, endometrial or wound infection -Increase C/S rate -Hypoglycemia due to high insulin intake -Laceration due macrosomia -Bleeding from laceration (PPH) -Retinopathy -Nephropathy -Diabetic ketoacidosis -Polyhydramnios

Fetal complication
-Increase risk of congenital abnormalities: =sacral agenesis =congenital heart disease =neural tube defects -Increase risk of sudden unexplained intrauterine fetal death -Macrosomia → Shoulder dystocia Clavicle fracture -Hypoglycemia -Polycythemia -Hypocalcaemia -Hypomagnesaemia -Respiratory distress syndrome -Jaundice

- Congenital complication common in DM because in case of GDM the glucose intolerance happened usually in 3rd trimester and the organogenesis happened in the first trimester.
- **Congenital complication**: sacral agenesis (**most specific)**, congenital heart disease (**most common**), neural tube defects.



- **EFFECT OF PREGNANCY ON DIABETES :**
  - Increase requirement for insulin doses
  - Nephropathy
  - Retinopathy
  - Diabetic ketoacidosis
  - Difficult to control glucose level in pregnancy
- The cause of delayed Lung maturity→ fetal insulin antagonize the effect of cortisone on surfactant
- **Fetal hyperinsulinaemia** → cause macrosomia
- Increase susceptible of diabetic ketoacidosis in pregnancy because of :
  - Increase excretion of bicarbonate will decrease buffering of ketoacide
  - Decreased sensitivity of insulin due to antagonizing effect by cortisone, estrogen, progesterone HPL, and degradation of insulin by placental insulinase.
- **Screening test** :

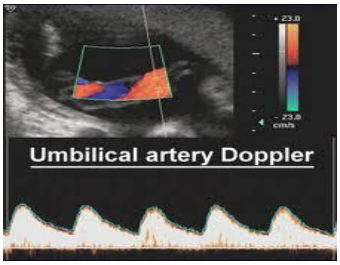
  - Random glucose test at first booking.
  - Glucose challenge test around 24–28 weeks' gestation (GCT is positive when it is ≥ 140 mg/dl and need GTT to confirm Dx, Unless it is ≥ 200 mg/dl GDM is suggested)
- **Diagnostic test** :
  - Oral glucose tolerance test (OGTT)[GTT] → indicated in high risk women :
    - Unexplained IUFD
    - History of congenital anomalies
    - Maternal weight > 90 KG
    - Previous big babies
    - Positive challenge test
    - Glycosuria in 2 times.

GTT (100g 3-hour)	Values
Fasting	≥ 95 mg/dl
1-hour	≥ 180 mg/dl
2-hour	≥ 155 mg/dl
3-hour	≥ 140 mg/dl
If 2 or more values are met = GDM	

- **Management:**

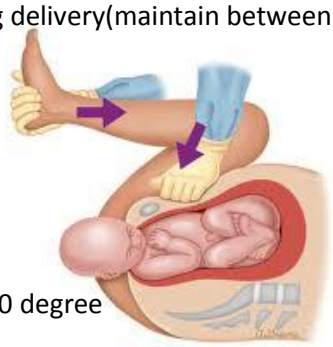
=Antepartum management:

  - Preconception counseling.
  - Screening of DM
  - Food plan: 50% carbohydrate 20% protein 20% fat, fiber.
  - If diet or exercise are inadequate to control glucose levels(in case of GDM)→ insulin therapy may become necessary → Combined short acting & intermediate acting insulin
  - Doses rise progressively with advancing pregnancy
  - Oral hypoglycemic **never** to be used.
  - Regular capillary glucose series (for monitoring and for choosing the type of management diet or insulin)
  - Target values are FBS < 90 mg/dl, 1-hour PP < 140, 2-hour PP < 120
  - HbA1C reflect average plasma glucose
  - Ophthalmologic referral & Rx of retinopathy
  - NST twice-weekly from 26 weeks' gestation in DM if risk factors present, if not start 32 weeks' gestation.
  - Detailed U/S screening for congenital malformations [anencephaly (13-14weeks), structural anomalies (18-22weeks)]
  - Serial U/S biometry to detect macrosomia, hydramnios, IUGR.
  - Umbilical Artery Doppler in Pt with IUGR.
  - Fetal echocardiography (22-24 weeks)
  - Triple markers for NTD [16-18week].

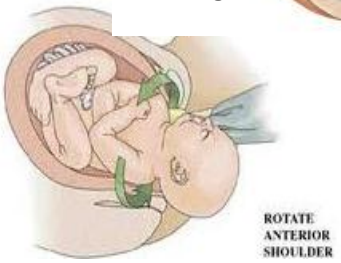


=Intrapartum management:

- In normal pregnancy, wait until 40 weeks.
- If the pregnancy can't be controlled and a complication happened (retinopathy) → immediate admission.
- Induction of labor IOL+ Insulin infusion +hourly checking of blood sugar during delivery(maintain between 80-100mg/dl)
- Close fetal heart monitoring.
- In risk of shoulder dystocia do :
  - McRoberts maneuver (sharp flexion of maternal thighs)
  - Woods maneuver (rotation of the anterior shoulder 180 degree)
  - Suprapubic pressure (to disimpact anterior shoulder)
  - Delivery of the posterior arm and rotation of the anterior shoulder 180 degree



- C/S is indicated if the fetal estimated weight is > 4 kg.
- Patient with diet management → admission after 40 weeks
- Patient with insulin management→ admission after 38 weeks



=Postpartum management:

- Regular screening for type 2 diabetes is advised in case of GDM +diet +exercise.
  (FBS should be < 126mg/dl, 2-hr GCT <200 mg/dl)
- Insulin requirement fall rapidly after delivery and the management as before pregnancy
- Dextrose for infant after delivery (due to fetal hyperinsulinemia)
- Breastfeeding (the breastfeeding helps the mother to reduce weight and back to normal glucose level faster).

- **MCQ** : in case of FBG = 12 mmol/L the management is immediate delivery (ask the doctor about it)

Anemia in pregnancy

Definition: a condition in which the oxygen-carrying capacity of the blood is decreased. Practically when **< 10 g/dl**.
Normal value in pregnancy is 10-12 g/dl , and 13-15 g/dl in non-pregnant state.

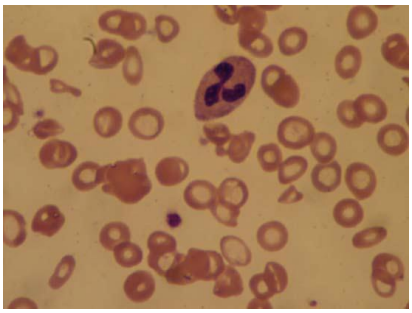
The cause: is the **hemodilution of pregnancy**: the plasma volume increases 50%, and the RBC increases 30% (the cause of iron deficiency), which cause 15% dilution (the cause for physiologic anemia).

Risk factors:

Iron deficiency (akon)	Folate deficiency (cosa)	Sickle cell disease	thalassemia
Obstetric factors: • frequent pregnancies. • Multiple gestation.	Obstetric factors: • frequent pregnancies. • Multiple gestation.	• African descent.	Ethnicity: • Mediterranean. • Africa. • Southeast asia.
Nutritional factors: • Poor dietary habits. • Pica.	Seizure medication: • Phenobarbital. • Phenytoin.		
Chronic bleeding: • Epistaxis. • Rectum. • Vaginal.	Antibiotic medications: • Pyrimethamine. • Trimethoprimsulfamethoxazol.		
adolescence	Chronic hemolytic anemia: • Sickle cell disease • Heritary spherocytosis		

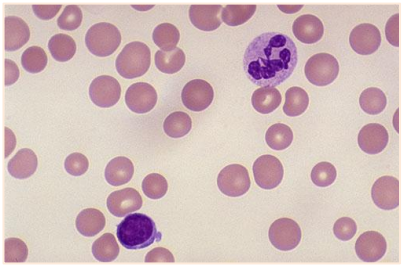
Classification of anemias:

1. **Iron deficiency anemia:**
   - **A. Incidence**: the **most common anemia** in pregnancy(90% of the cases)
   - **B. Pathophysiology**: iron located in the hemoglobin molecule, with increase requirements of iron with pregnancy as result of RBC production the anemia may occur only after the iron stores in bone marrow are completely depleted.
   - **C. Diagnosis:**
     - **a. RBC indices include :**
       1. ↓ MCV and MCHC.
       2. ↑ RBC distribution width (RDW).
     - **b. Serum iron studies:**
       1. ↑ Total iron-binding capacity.
       2. ↓ Serum iron and ferritin.
     - c. **Bone marrow aspiration:** seldom done in pregnancy.



   - **D. Fetal/neonatal complications:** do **NOT** occur, the iron is transported actively to the fetus.
   - **E. Maternal symptoms:** tiredness and weakness.
   - **F. Management:**
     - a. Diet rich in iron.
     - b. **Ferrous iron salts**: 3 times daily for 3-6 months.
     - c. **Parenteral iron:** for nonfunctional GI or noncompliance, there is no difference in the rapidity of bone marrow response whether given orally or parenterally.
   - **G. Prevention:** iron daily supplement on tablet.

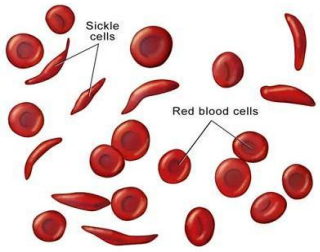
2. **Folate deficiency anemia:**
   - **A. Incidence:** the **second most common** (0.5%-15%)
   - **B. Pathophysiology:** the folate is required for carbon transfer during building of the hemoglobin skeletons. Normal body stores last for **90 day**. When it is deficient there is decreased production of the hemoglobin production. It is found in **green, leafy vegetables.**
   - **C. Diagnosis:**
     - a. **RBC indices** include: - Macrocytic RBC.
       - ↑ MCV and MCHC.
     - b. **RBC folate levels** are low
     - c. **Peripheral smear findings:** hypersegmented neutrophils.



D. **Fetal complication**: **low birth weight** and **NTDs** (when deficiency is during early embryogenesis)
E. **Maternal symptoms:** tiredness and weakness
F. **Management:**
    a. **Folic acid:** 1 mg daily.
    b. Iron supplements.
G. **Prevention:**
    1. **Normal requirements:** 0.4 mg of folic acid.
    2. **In high risk women** (DM, antiepileptic medication, pervious NTD): **4 mg/day** for 3 month before the pregnancy and during early embryogenesis.

3. Sickle cell anemia:

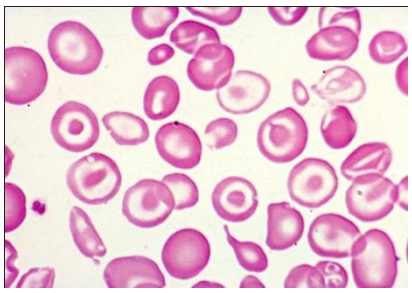
A. **Incidence:** mostly in blacks.
B. **Pathophysiology:** SCD is an autosomal recessive disease, with **abnormal hemoglobin S** molecule with a valine substituted for glutamic acid on β-chains:
    1. **Heterozygous (SA):** (<40% hemoglobin S) called **sickle cell trait**.
    2. **Homozygous (SS):** (> 40% hemoglobin S) called **sickle cell disease**.



C. **With reduction of oxygen tension:** RBCs with **more** than 50% hemoglobin S undergoes intravascular sickling which leads to:
    a. **Chronic hemolytic anemia:** from shortened survival of RBCs.
    b. **Sickle cell crisis:** capillary occlusion causes ischemic pain in the joint and long bone and can be precipitated by dehydration, acidosis, or infection.
D. Diagnosis:
    a. Hemoglobin electrophoresis: differentiate between homozygous and heterozygous states.
E. Fetal/neonatal complications:
    a. **Sickle cell trait:** no complications.
    b. **Sickle cell disease:** spontaneous abortions, preterm delivery, IUGR, and stillbirths.
F. Maternal complications
    a. **Sickle cell trait:** increased UTIs and asymptomatic bacteriuria.
    b. sickle cell disease:
        Increased morbidity: anemia, sickle cell crisis, congestive heart failure, PET and infections.
        Increased mortality: 1% maternal death.
G. Management:
    a. **Adequate oxygenation:** to avoid hypoxia and acidosis.
    b. **Narcotics:** in case of sickle cell crisis.
    c. **RBC transfusions**: (to keep hemoglobin S < 50%)
    d. Folate supplement.
    e. **Serial ultrasound:** for assessment of fetal growth.
    f. Serial antepartum fetal testing.

4. Thalassemia:

A. **incidence:** -Genetic disorder found in individual from Africa,
    -Southeast Asia and the Mediterranean.



B. **Pathophysiology:** impaired production of one or more of the peptide chains of the globins:
    a. **α-thalassemia:** impaired production of alpha peptide chains.
    b. **β-thalassemia**: impaired production of beta peptide chains.
C. **Diagnosis:** hemoglobin electrophoresis.
D. Fetal /neonatal complications:
    1. **α-thalassemia major** (4 gene deletion): result in **hemoglobin Bart disease**: causes non-immune hydrops and stillbirth or early neonatal death.
    2. **α-thalassemia minor** (2 gene deletion): mild microcytic, hypochromic anemia.

3. **β-thalassemia minor and major:** result in health appearing neonates who become anemic as **hemoglobin F** levels decrease.
    a. **With β-thalassemia minor:** have a mild anemia.
    b. **With β-thalassemia major:** Called (Cooley's anemia), children become severely anemic, show failure to thrive, and may die during childhood.

E. maternal complications:

1. **α - and β- thalassemia minor**: tolerate pregnancy well and my develop mild anemia.
2. **β-thalassemia major:** those who survive childhood are usually **sterile** and pregnant women with this condition are **rare.**

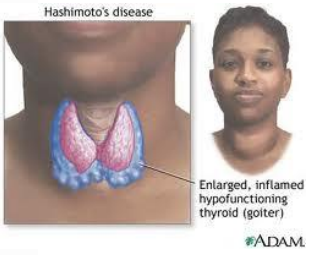
F. Management:

1. **Observation** in thalassemia **minor** states.
2. **RBC transfusion** in β-thalassemia major.
3. **Folate supplement.**
4. **Serial ultrasound:** for assessment of fetal growth.
5. **Serial antepartum fetal testing.**

Thyroid diseases in pregnancy

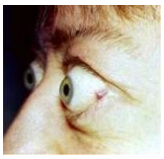
❖ ***Hypothyroidism***

- Epidemiology:
  - ○ Prevalence: 1%
  - ○ Occurs mainly as primary thyroid defect**.**
  - ○ Infertility is a result of overt hypothyroidism thus myxedema is rare in pregnancy.
  - ○ If untreated>>miscarriages, stillbirth, preterm delivery, and preeclampsia.
  - ○ If treated no impact on pregnancy outcome.
- Etiology:
  - **1.** Autoimmune (most common): like hashimoto thyroiditis and idiopathic myxedema**.**
  - **2.** Iatrogenic: radioactive iodine-131 and subtotal thyroidectomy**.**
  - **3.** Drug-induced: iodide deficiency, ↑ lithium, or antithyroid drugs**.**
- Clinical findings and diagnosis:
  - ○ Symptoms: weakness, fatigue, cold intolerance, constipation, hair loss, weight gain, **amenorrhea.**
  - ○ Signs: dry skin, periorbital edema, thick tongue, decrease reflexes, **hypertension**, and **bradycardia.**
  - ○ Lab values: high TSH low T4**.**
- Treatment:
  - ○ L-Thyroxin (Levothyroxine).
  - ○ Serial TSH and T4 monitoring to identify appropriate dosage.



❖ ***Hyperthyroidism***

- Epidemiology:
  - ○ Prevalence 0.8%
  - ○ Hyperthyroidisms from variety of causes each of which are managed differently.
  - ○ If uncontrolled>>preterm labor, preeclampsia, and low birth weight**.**
  - ○ If controlled has no impact on outcome**.**

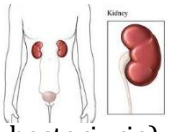


- Pathophysiology: most cases are graves disease and Plummer's disease.
  - **1.**Graves: autoimmune disease in which TSI (thyroid stimulating immunoglobulin) acts like TSH>>thyroid hyperfunction. Usually TSI activity↓in pregnancy.
  - **2.**Toxic nodular goiter (Plummer's): thyroid nodule produces thyroid hormone independent from hypothalamic-pituitary axis**.**
- Clinical findings and diagnosis:
  - ○ Symptoms: weakness, nervousness, diarrhea weight loss, heat intolerance, emotional instability, and **amenorrhea.**
  - ○ Signs: goiter, warm skin, eye stare, lid lag, exophthalmos, bring reflex, tremor, and **tachycardia.**
  - ○ Lab findings: low TSH and high T4 and presence of TSI in graves.
- Treatment:
  - ○ Anti-thyroid drugs (used for graves): PTU (propylthiouracil). Methimazole rarely used in pregnancy (causes aplasia cutis of scalp) both cross placenta and causes fetal hypothyroidism**.**
  - ○ Radioactive iodine-131 ablation: not used in pregnancy b/c it ablates fetal thyroid**.**
  - ○ Thyroidectomy: used if medical therapy failed.

❖ ***Postpartum thyroiditis***

- Epidemiology:
  - ○ Prevalence 5-8 %**.**
- pathophysiology:
  - ○ Destructive lymphocytic thyroiditis>>hyperthyroidism 1-4 month postpartum followed by hypothyroidism 4-8 month postpartum. Majority positive for microsomal autoantibodies**.**
- Clinical findings and diagnosis:
  - ○ Non-specific includes depression, carelessness, and impaired memory concentration.
- Treatment:
  - ○ Hyperthyroid case is managed conservatively and beta blockers may be used hypothyroid phase is managed by L-Thyroxin.

UTI in pregnancy



Classification:

1- **Lower tract diseases:** only bladder and includes ABS (asymptomatic bacteriuria) and acute cystitis.
2- **Upper tract diseases:** involves kidney and includes pyelonephritis

Pathophysiology:

It originates from organisms in vagina or rectum and it ascends from urethra to the bladder and then to the kidney

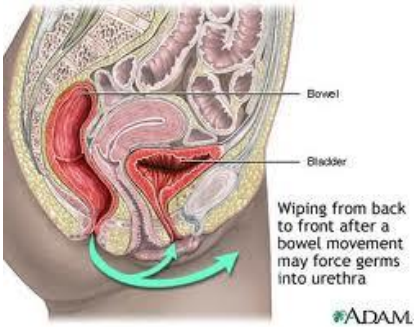
Most common causative organisms GRAM-NEGATIVE (80% E.COLI and 20% >>klebsiella, pseudomonas, enterococcus, and proteus)

Risk factors:

1-mechanical obstruction: uretropelvic junction, ureteral/urethral stenosis, and calculi.

2-functional obs.: PREGNANCY and vasicouretral reflux

3- **Systemic disease:** DM, gout, sickle cell trait/disease, and cystic renal disease.



****ABS****

Incidence: 8% and ↑ in women with sickle cell trait

30% of untreated may develop pyelonephritis

Diagnosis:

By culture (should contain more than 100000colony-forming units/ml of a single organism other than lactobacillus (normal vaginal contaminant)

Management:

Outpatient treatment: 7-10 days of oral antibiotics (nitrofurantoin) reculture after antibiotics should be performed.

****Acute cystitis****

Incidence: 1%

If treatment inappropriate it may progress to pyelonephritis

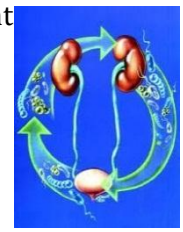
Diagnosis:

Symptoms: urgency, frequency, dysuria, and suprapubic discomfort.

Physical exam. : Unremarkable

Lab findings: +VE urine culture same ABS. hematuria may be present

Management: same ASB urinary tract analgesics may be indicated in first 24-48 hours of treatment



****Acute pyelonephritis****

Incidence: 1-2%

Most common serious medical complication of pregnancy. Preterm delivery is major concern.

Maternal comp.: anemia, pulmonary dysfunction, sepsis, and renal failure

Diagnosis:

Symptoms are abrupt and include dysuria, shaking, chills, headache, flank pain, tremor, anorexia, nausea, and vomiting

Physical exam: fever, costovertebral angle tenderness (more common on the right), dehydration, and UC's are common

Lab: pyuria, bacteriuria, WBC casts

Management: 1-Admission to hospital
2-IV hydration
3-Antipyretic agent
4-single agent IV antibiotic (ampicillin and cephalosporin) and oral AB are continued 7 days after discharge multiple AB are used if patient is septic
5-Periodic culture for any recurrence

Types of UTI recurrence

1. Relapse: reappear of the same organism 2-3 weeks of initial diagnosis
2. Reinfection: infection with new organism usually 12 weeks after completing initial therapy.
3. Superinfection: appearance of new organism while still on therapy

Prevention:

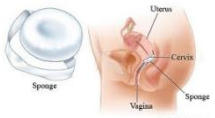
All pregnant women should undergo screening for ASB on 1st prenatal visit. Further testing is based on developing new symptoms.

Contraception Methods

=Abstinence: -Ideal for adolescents at high risk for pregnancy and STD's including HIV.

=Folk methods:-Coitus interruptus, but semen can escape into vagina before ejaculation.
-Postcoital douche, but sperms may enter cervical mucosa within 1,5 min.
-Lactation, ↑Prolactin=> ↓Estrogen & Ovulation. Effective in the first 6 months with 2% failure rate.

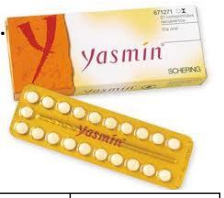
=Barrier and spermicidal methods:-Male condom, most widely used mechanical contraceptive.



-Female condom
-Diaphragm, Nanoxynol 9 spermicide causes epithelial irretation.
-Sponge, " " " " " .
-spermicide, " " " " " .
-Cervical cap, +/- spermicide.

All ↓ risk of STD, PID except cervical cap if no spermicide placed inside it.
All have no protection against HIV except for Male/Female condoms if used correctly.
All have 20% failure rate except Male condom with 15%.

Note: - Effect of estrogen & progesterone in contraception: -
-Suppression of LH, FSH leads to Anovulation (Estrogen & Progesterone).
-Thickening of cervical mucosa to inhibit migration & sperm penetration (progesterone).
-Thinning of endometrial lining to inhibit implantation (progesterone).



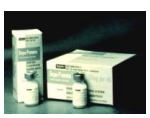
=Combined Oral Contraceptives Pills (COCP):
-Contains estrogen (M.C. is Ethinyl estradiol) & progesterone.
-Types: - Monophasic or multiphasic according to the amounts of hormone in each active pill.
-Most OCP's have 21 active pills and 7 placebo pills.
-Dependent upon gut bacterial flora for absorption .so they are affected by courses of antibiotics.
- Anticonvulsants (phenytoin, phenobarbital, primidone, and carpamazepine) and griseofulvin ↓ its effect.
-Advantages: -Continuous protection if taken properly
-Reversible
-Non contraceptive health benefits.**(see table)**
-Disadvantages:-Must be taken daily
-Intermenstrual bleeding
-Headaches (in the 1st few months)
-Weight gain (in high doses)
-No protection against STD's.
-Contraindications: - **(see table)**
-Failure rate: - 0.1%

Organ	Uses	Mechanism
Ovary	-Functional ovarian cyst -Epithelial ovarian carcinoma	Mainly As a result of suppression of ovulation, cervical Thickening, and endometrial Thinning
Uterus	-1ry & 2ry Dysmenorrhea -DUB -Endometrial carcinoma	
Oviduct	-PID -EP	
Breast	-Benign breast disease	
Blood	-Microcytic anemia (from↓bleeding)	
Skin	-Acne -Hirsutism	
Others	-Endometriosis -PMS -Hormone replacement Rx	

Absolute C/I	Relative C/I
Pregnancy	**Systemic diseases**
Undiagnosed vaginal bleeding	-HTN
Cardiovascular	-DM
-Complicated valvular heart disease	-Hyperlipidemia
-Coronary heart disease (or Hx)	-Sickle cell disease
-Hypertension sys. >160 dias. >100	**Neurologic**
-Cerebrovascular accident (or Hx)	-Migraine without aura
-DVT (or Hx)	-Depression
-Pulmonary embolism (or Hx)	-Seizure disorder,
Neurologic	-Anticonvulsant use
-Migraine with aura	**Others**
Malignancy	-Smoking, age >35 & <15 cig./day
-Breast ca. (Current)	-Elective surgery (needs 1 to 3 month discontinuation)
-Endometrial ca. (Current)	-Gall bladder disease
-Melanoma (Current)	
Liver	
-Any hepatic tumor (or Hx)	
-Abnormal LFT (current)	
-Viral hepatitis (Active)	
Others	
-Breast feeding <6 w. Post partum	
-Major surgery with prolonged immobilization	
-Smoking, age > 35 & >15 cig./day	

=Progestogen-Only Methods:
-can be used by every woman except if has breast ca. in the past 5 years (absolute C/I)

a-Progesterone-Only Pills (mini pills);
-They have more than 21 pills (unlike COCP)
-Must be taken daily in a strict time within 3 hours window.
-Not dependent upon gut bacterial flora for absorption .so they are not affected by courses of antibiotics.
-8% failure rate.

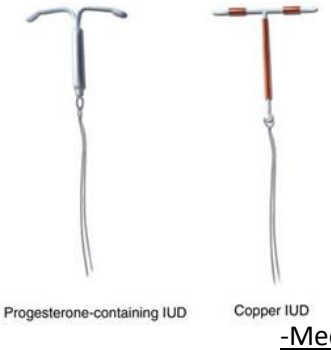


b-IM agents (Depo Provera):
-Also called depot medroxyprogesterone acetate (DMPA).
-Given every 3 months.
-3% failure rate.

#-Advantages & Disadvantages for Both: **(See table next page)**

Advantages	Disadvantages
-Estrogen-free [Both] no risk of DVT or hear disease. -Used in patients has C/I to Estrogen [Both] -Safe in breast-feeding [Both] -↑ milk quality in nursing mothers [Both] -Immediate reversible ovulation (menses) [POP] -Long acting [DMPA] -Pt. does not have to take daily [DMPA] -Given once per 3 months [DMPA] - Non contraceptive benefits:-Menorrhagia [DMPA] -Dysmenorrhea -Endometriosis -Endometrial ca. -Acute sickle cell crises	-Irregular bleeding [Both] in the beginning. -Weight gain [Both] -Breast tenderness [Both] -Mood swings [Both] -Anticonvulsants (phenytoin, phenobarbital, primidone, and carpamazepine) and griseofulvin ↓ its effect. [POP] -Late reversible ovulation (menses) [DMPA] (around 10 months). -↓ HDL cholesterol level [DMPA]

=Intrauterine Devices:-Types; -Copper IUD (Paragard): -↓ sperm motility and acrosomal enzyme action



-Lasts 10-12 years
-May increase bleeding and dysmenorrhea.
-0.8% failure rate.

-Hormonal IUD (Mirena): -Contains P.
-↑ thickness of cervical mucus => ↓ sperm migration
-Lasts up to 7 years.
-Improves menorrhagia.
-Causes amenorrhea.
-0.1% failure rate.

-Mechanisms: - Altering tubal motility prevents sperm-egg fertilization (P+ Copper).
-Endometrial inflammation prevents implantation (Copper).
- Thinning of endometrial lining to inhibit implantation (P).
#Note: no cervical thickening from Copper IUD.

-Complications of IUD :-Increased risk of PID within 1st 20 days
-Uterine perforation
-Fainting with insertion
-Expulsion
-Unexpected pregnancy following poor placement.

-Contraindications:

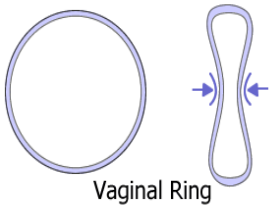
Absolute C/I	Relative C/I
-Pregnancy -Undiagnosed vaginal bleeding -Acute cervicitis -Acute endometritis -Acute Salpingitis -Hx of Salpingitis -Suspected gynecologic malignancy	-Nulliparity -Desired future pregnancy -Hx of EP -Hx of STD -multiple sexual partners -Abnormal uterine cavity -Moderate-severe Dysmenorrhea -Chronic menometrorrhagia -Iron deficiency anemia

= Transdermal patch (Ortho Evra):

- Contains E & P (ethinyl estradiol & norelgestromin)
-Takes 3 days to achieve a steady state of hormone in blood.
-Patch is replaced once per week for 3 consecutive weeks.
-Advantages:-Only has to be replaced once per week.
-May be taken continuously.
-Disadvantages: -May slip off.
-Less effective in women who are > 90 Kg.
-No protection against STD's.

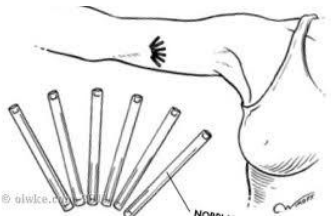
=Vaginal Contraceptive Ring (NuvaRing):

- Contains E & P.
- 8% failure rate.
-Place anywhere in vagina and remove after 3 weeks.
-Advantage:-Only has to be replaced once per month.

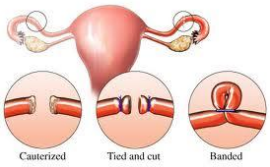

Vaginal Ring

=Subcutaneous implants (Norplant):-Contains P (L-norgestrel).

-Advantages and disadvantages: **(See table)**



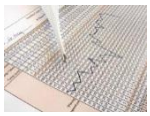
Advantages	Disadvantages
-Highly effective -Less than 1% failure rate -Replaced only every 5 years -Fertility returns rapidly	-Need professional -Removal can be difficult -Irregular bleeding -fluid retention -Weight gain



=Female Sterilization: -prevents fertilization by interrupting the patency of fallopian tubes.
-0.8-3.7% failure rate.
-May be performed through a mini-laparotomy incision, laparoscopically, or transcervically.

=Male Sterilization:-By ligating or cauterizing the vas deferens (Vasectomy).
-Prevents the passage of sperm into seminal fluid
-Failure rate: < 0.15%.
-Must use contraception until completely azospermic for two consecutive sperm counts.

=Natural family planning:-Avoidance of intercourse during the periovulatory fertile period of menstrual cycle.
-Prediction of ovulation; -keeping a calendar
-Basal body temperature (BBT)
-Cervical mucus changing
-Preovulatory symptoms (e.g, mittelschmerz [ovulation pain])



-Disadvantages: -Low effectiveness rate
-Possibility of variable cycles
-Hyperthermia arising from nonovulatory causes.

*****Emergency Contraception:**-Used after unprotected or inadequately protected sexual intercourse. Common in women after an assault or rape.

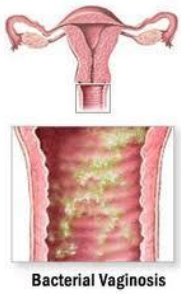
-Types: a-High dose progestin only (Plan B)
b-Yuzpe method- 13 different combined oral contraceptives (Preven)
c-Copper IUD (Paragard)
-Mechanism: Prevents fertilization and implantation it does not abort a pregnancy that is already implanted.
-They are given within the first 3-5 days of intercourse
-Most women will have a cycle 21 days after completing emergency contraception if not check a pregnancy test.

_____
M.C. = Most common
PMS=Premenopausal syndrome
LFT=Liver function test
C/I=Contraindication
E=Estrogen
P=Progesterone

Genital Tract Infections

Genital tract introduction: -The normal vaginal flora is predominately lactobacilli (aerobic organisms)
-The normal PH is <4.5 due to the H+ peroxide producing lactobacilli.

❖ **Bacterial vaginosis [BV];**-Caused by over-growth of anaerobic bacteria due to alteration of the normal flora & ↑ pH of the vagina (intercourse, douches).



-Recurrences are common
-Diagnosis; **(see table)**
-Treatment; -Flagyl (metronidazole)
-Clindamycin.
-Treatment of the partner is not recommended.

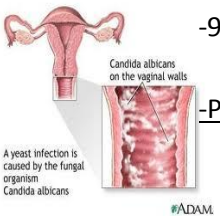
Diagnosis of BV
-PH >4.5.
-Fishy odor (especially after intercourse [↑ pH]).
- +ve whiff test (adding KOH to the vaginal secretions = fishy odor).
-Gray secretions.
-Presence of clue cells.

❖ **Trichomonas Vaginalis;** -It is an anaerobic parasite. 60% of patients also have BV.
-Patients should be tested for other STDs (HIV, Syphilis).

-Diagnosis; **(see table)**
-Treatment; -Flagyl (metronidazole)
-The partner should be treated.

Diagnosis of Trichomoniasis
-Profuse, purulent malodorous discharge.
-It may be accompanied by vulvar pruritis.
-Secretions may exudate from the vagina
-If severe → patchy vaginal edema and strawberry cervix
-PH >5
-Microscopy: motile trichomands and ↑ leukocytes
-Clue cells may if BV is present
-Whiff test may be +ve

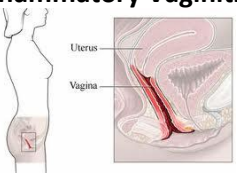
❖ **Candidiasis;** -75% of women will have at least once during their life.
-90% of yeast infections are secondary to Candida Albican.



-Predisposing factors: -Diabetes (↓immunity).
-Antibiotics (disrupting the normal flora by ↓ lactobacilli).
-Pregnancy (↓ cell-mediated immunity).
-Diagnosis: - **(See table)**
-Treatment: -Fluconazole
-Topical Azole
-1% hydrocortisone as adjuvant.

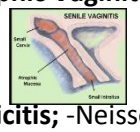
Diagnosis of Candidiasis
-Vulvar pruritis and burning
-The discharge vary from watery to thick cottage cheese discharge
-Vaginal soreness and dyspareunia
-Splash dysuria
-O/E: erythema and edema of the labia and vulva
-The vagina may be erythematous with adherent whitish discharge
-Cervix is normal
-PH< 4.5 budding yeast or mycelia on microscopy
-The culture will confirm the diagnosis

❖ **Inflammatory Vaginitis;** -Diffuse exudative discharge.



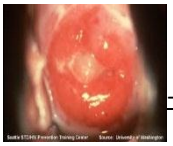
-Epithelial cells exfoliation.
-The cause is uncertain (strept!!)
-Rx is clindamycin cream
-30% of patients have relapsed.

❖ **Atrophic Vaginitis;** -Occur in post-menopausal women.



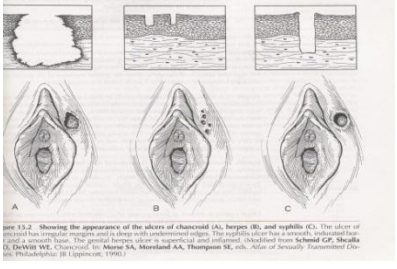
-May be accompanied by purulent discharge, dyspareunia and post-coital bleeding.
-It is treated with topical Estrogen cream.

❖ **Cervicitis;** -Neisseria Gonorrhea and Chlamydia Trachomatis infect only the glandular epithelium and are responsible for mucopurulent endocervisitis (MPC).



-Trichomonas, HSV and Candida may cause ectocervix inflammation hence it is connected with the vagina.
-Diagnosis; -Culture on Thayer- martin media (for Gonorrhea).
-ELISA, direct IFA (for Chlamydia).
-Treatment; -Ceftriaxone, Ofloxacin, Cefixime, or Ciprofluxacin for Neisseria Gonorrhea endocervisitis.
-Doxycycline, Azithromycin, Ofloxacin, Erythromycin for Chlamydia Trachomatis // .

❖ **Genital ulcer disease;** -Mostly caused by HSV (Herpes simplex virus) or Syphilis, then chancroid, LGV (Lymphogranuloma venereum), and granuloma inguinale (donovanosis).
-Other causes: abrasions, drug eruptions, cancer and behcet's disease.
-Diagnosis; -R/O syphilis by serology, dark field examination or direct IF for Treponema pallidum.
-Culture for HSV.

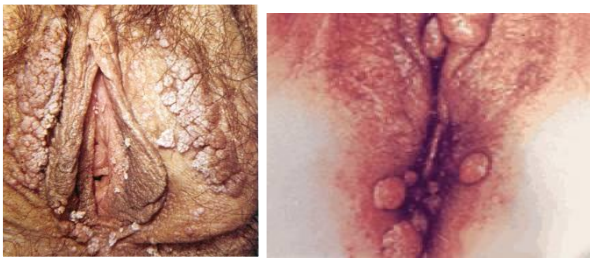


-Clinically; -Syphilis: non-painful, min. tender ulcer, not accompanied by LAP (Lymph adenopathy)
-HSV: grouped vesicles mixed with ulcers with a history of similar lesions.

-Chancroid: 1-3 extremely painful ulcers with tender inguinal LAP.
-LGV: inguinal bubo without ulcers.

-Treatment; -Chancroid: -Azithromycin
-Ceftazidime
-Erythromycin
-Herpes: -Acyclovir if it was the 1st episode (will not eradicate the infection).
-Recurrences are common
-Suppressive treatment (will not eliminate viral shedding and transmission) for patients with > 6 recurrences/year.

❖ **Genital Warts;** -Condyloma accuminata secondary to HPV infection (usually 6 & 11), these are non-oncogenic types.
-Usually at areas affected by coitus (posterior fourchette).
-Recurrences after treatment are secondary to reactivation of subclinical infection.
-Treatment; -Cryotherapy.
-Podophyllin.
-Podofilox.
-Triochloroacetic acid.
-Electrodesiccation or cautery.
-Laser.
-Interferon.



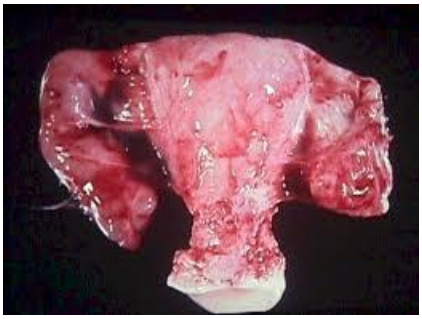
❖ **HIV;** -20-25% of patients are women.
-36% is secondary to heterosexual transmission.
-Median age between HIV infection and AIDS is 10 years.
-Diagnosis; -HIV1 antibody test.
-ELISA, if +ve → confirm by western blot.
-95% of the antibody is detected within 6 months of the infection.
-Patients are referred to as an infectious disease specialist for treatment.
-CD4 is the best indicator of disease progression.

Pelvic Inflammatory Disease (PID)

Definition: an infection of the uterus (endometritis, cervicitis), Fallopian tubes (Salpingitis), ovaries (Oophoritis) **and/or** peritoneum (peritonitis) that result in a spectrum of disorders affecting nonpregnant and occasionally pregnant women.

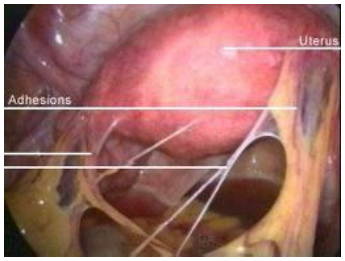
Epidemiology: 10% of reproductive-aged women have a Hx of PID.

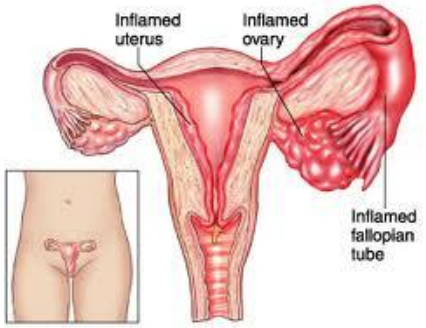
Etiology: (1)- Organisms **ascend** from the lower genital tract--to--cervix--to--endometrium--to--endosalpinx (fallopian tube) without deep invasion.
-This type called classic PID (Salpingitis-Oophoritis).
-Organisms: -Mainly N.Gonorrhea, Chlamydia.
-Findings: - Purulent salpingitis into peritoneal cavity.
(2)- Organisms invade endometrial **lymphatics** and penetrate deep into the myometrium and parametrium
-This type happens in Post-abortion and post-partum infections.
-Organisms: - Staphylococcus, streptococcus, and E-coli.
- Findings: - Cellulitis of the endo-, myo-, and parametrium (endomyoparametritis)
(3) - Organisms are carried from the lungs to the pelvic organs **through blood**.
- This type happens in pelvic TB
-Organisms: -M.tuberuclosis
- Findings: - Salpingitis, Oophoritis, and endometritis.



Risk factors: -Hx of PID
-Young age.
-Multiple sexual partners.
-Increased intercourse frequency.
-IUD use.
-Mucopurulent cervicitis.

Complications: # Adhesions, fibrotic changes and scarring in pelvic organs
-Infertility, due to tubal factor (× 14 times)
-Ectopic pregnancy (× 6-8 times)
-Endometritis (× 6-10 times)
-Chronic pelvic pain.
-TOA.
-Fitz-Hugh-Curtis syndrome.
-May require a hysterectomy
-Preterm labor, if Hx of Salpingitis.

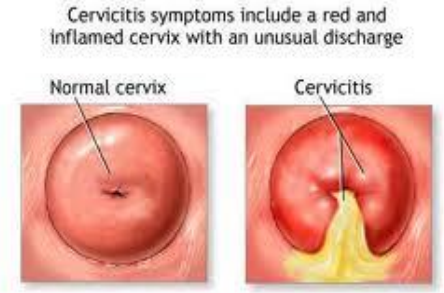




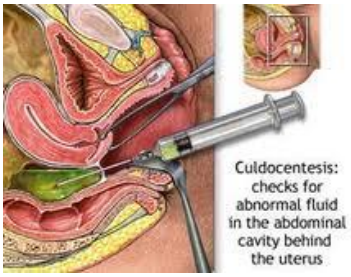
Diagnosis: -

=Symptoms: # No specific combination of symptoms is consistently associated with PID.
Some are asymptomatic
-Lower abdominal pain.
-Tenderness when walking or during coitus
-Abnormal vaginal discharge
-Fever / Chills
-Irregular vaginal bleeding (less common)
-Dysuria (less common)
-Nausea / Vomiting (less common)

=Signs: - Lower abdominal tenderness -/+ rebound tenderness.
-Uterine & adnexal palpation tenderness.
-Uterine & adnexal motion tenderness.
-Finding mucopurulent cervicitis.
-Fever (least common).



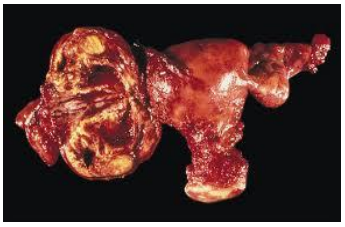
=*Investigations:* - Cervical culture, PCR, or antigenic tests (chlamydial & gonococcal infections)
-CBC (leukocytosis)
-ESR (increased)
-C-reactive protein (increased)
-Pelvic US (enlarged tubes and cul-de-sac fluid)
-Culdocentesis (cul-de-sac fluid-pus-)



Treatment: -Hospitalization is needed if -Surgical emergencies not ruled out (appendicitis).
-Pregnancy.
-Failed oral Rx
-Severe illness (high fever, nuasea, vomiting)
-Tubo-ovarian abscess shown or suspected
-Empiric antibiotics should be given:-Inpatient: IV (Cefoxitin, Doxycycline)or(Clindamycin,Gentamycin)
-Outpatient: Oral (" , ")or(Ceftriaxone,Metronidazole)

Prevention: - Routine screening and treatment if infection happened.

***Tubo-ovarian abscess (TOA):** *-Definition:* inflammatory mass due to inappropriately or untreated acute PID involving fallopian tubes, ovaries, bowel, and possibly other pelvic structures result from reactivation or repeated infections, whereas others may occur as a result of postpartum or postoperative infections.



-Characteristics: - It doesn't contain significant amounts of pus (so mainly no drainage needed)
- Easily treated with antibiotics.
-If ruptured causes spreading peritonitis.
-Epidemiology: -10% of hospitalized PID patients.
-Etiology: -Most commonly bacteroids and E.coli.
-Symptoms: -Severe pelvic and lower abdominal pain
-Nausea / Vomiting.
-Severe back pain
-Painful defecation
-Severe rectal pain
-Ex.: -High fever and tachycardia
-In abdominal guarding & rigidity
-In pelvic extreme tenderness
-Rectal Ex. **is the best method for palpation of the mass**
-Dx.: -US, CT, or MRI.
-Laparoscopy or laparatomy.
-Management: -Admit the patient.
-Medical Rx; -even large abscesses may resolve
- (Cefoxitin+Doxycycline or Clindamycin+Gentamycin)
-Cul-de-sac drainage; -if fever persists despite antibiotics
-Emergency laparatomy; -if Pt. deterioration or abscess rupture.
- **[TAH BSO]** **T**otal **a**bdominal **h**ysterectomy **B**ilateral **s**alpingio-**o**ophorectomy is advised.

***Fitz-Hugh-Curtis syndrome:** *-Definition:* a spread of pelvic infection in PID into the upper abdomen and cause inflammation of the capsule of the liver (Perihrpatitis) results in "violin-string adhesions" **see figure**.
-Symptoms: -Right upper quadrant pain
-(Mimic cholesystitis, pyelonephritis, or viral pneumonia.

Infertility

- **Fertility**: the ability to conceive after 12 months of regular intercourse .(50% after 3-month, 90% after 12-month)
- **Infertility:** no conception after **12 months** with regular unprotected intercourse and after **6 months** for women over age 35 years.
- **Primary infertility**: couple who has never achieved a pregnancy.
- **Secondary infertility**: infertility that occurs after previous pregnancy regardless of outcome.
- **Sterility:** Absolute and irreversible inability to conceive (hysterectomy and others)
- The **incidence** of infertility : **10-15%** in all couples
- Female fertility decline after the age of **35** and decline more rapidly after age of **40.**
- Origin of problem: **female**(35%), **male**(35%), **both** partners(20%), and **unexplained**(10%)"normal infertile couples"
- **Risk factors (imp)**:

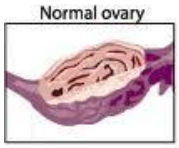
Age	Alcohol	Marijuana and cocaine use
Smoking	History of Pelvic inflammatory disease	Radiation treatment and chemotherapy
Poor diet	Overweight	excessive caffeine intake
Stress	Underweight	Too much exercise

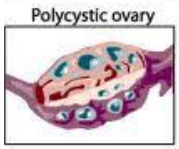
- **Requirements for Normal Reproduction :**
  1. Production of healthy egg and sperm.
  2. Normal pathway and transportation from the cervix to ampullary portion of the fallopian tube
  3. The sperms ability to penetrate and fertilize the egg
  4. Normal Implantation of the embryo into the uterus

- Female causes (imp):

A. Ovarian factor infertility (anovulation**):** 15-20% incidence.

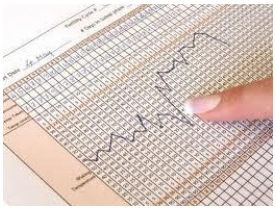
- **Causes :**
  1. Hypothalamus Dysfunction**:** Tumors, Stress, Trauma or radiation, Extreme exercise, anorexia nervosa, and drug abuse.
  2. Pituitary dysfunction**:** Hypothyroidism, hyperthyroidism, Sheehan's syndrome.
  3. **<u>Polycystic ovary syndrome</u>** (the most common)
  4. **<u>Premature ovarian failure</u>** (high FSH and LH and low estrogen) (MCQ)
  5. Resistant ovary syndrome (high FSH and LH and low estrogen)
  6. Menopause.
  7. Gonadal dysgenesis (Turner syndrome).
  8. Ovarian cancer



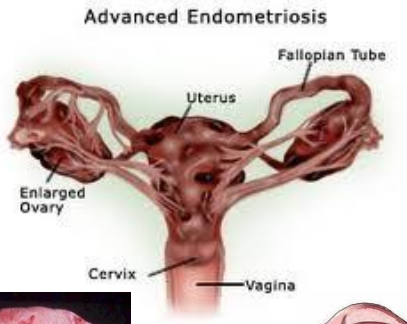


- **NB :** we can differentiate between premature ovary syndrome and resistant ovary syndrome by ovarian biopsy

- **Ovulation Detection :**
  1. Obtaining the history of regular menstrual cycle intervals without any medication that regulate menstrual cycle
  2. **<u>Progesterone levels at 21 day of menstrual</u> cycle** < 3 ng/ml = anovulation.
  3. The basal body temperature (BBT) record
  4. Cervical mucus characteristics
  5. Luteal phase endometrial biopsy to see the change of endometrium in luteal phase.
  6. Ovulation predictor kits test (detect LH surge in the urine)
  7. Mid cycle pain (one fifth of women) the condition, called mittelschmerz, may last a few minutes to a few hours.
  8. Frequent blood sampling to detect LH surge (The gold standard for **<u>research</u>**)
  9. Serial ultrasonography to see the collapse of follicular cyst after ovulation
  10. Ferning pattern in one day pre-ovulation

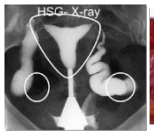


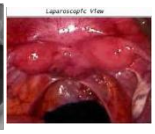
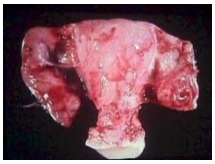
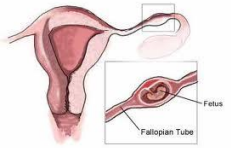
B. Tubal factors infertility: 30% incidence.



The fallopian tubes are blocked, damaged or there are adhesions:

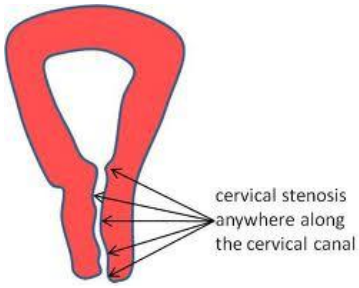
1. Endometriosis.
2. PID (**<u>most common is chlamydia</u>**)
3. Abdominal or gynecological surgery, such as bowel surgery, cesarean section or a ruptured appendix.
4. Tubal Surgery: Tubal ligation or salpingectomy.
5. Congenital absence of the fallopian tubes.
6. Ectopic pregnancy



- **Diagnosis**:
  A. Hystersalpingogram (HSG)
  B. Laparoscopy (by injection the dye throw the cervix and then seeing the dye getting out from the tube)

C. Cervical factor infertility: 5% incidence.



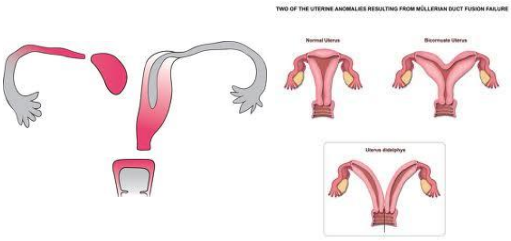
1. Unfavorable cervical mucus:
   Lack of estrogen or low-estrogen: results in thick mucus through which the sperms cannot pass.
2. Cervical stenosis
3. Antisperm antibodies→ post-coital test.
   - A postcoital test is performed 2 to 12 hours after intercourse to assess the number and motility of spermatozoa that have entered the cervical canal

- **Causes of cervical stenosis :**
  1. Surgical procedures performed on the cervix such as **<u>cone biopsy</u>**, cauterization
  2. Trauma to the cervix
  3. Repeated vaginal infections
  4. Atrophy of the cervix after menopause due to hypoestrogenism
  5. Cervical cancer
  6. Radiation

D. Uterine factor infertility: - 30% incidence
- Abnormalities of the uterine cavity are seldom (rare) the cause of infertility.

- Any problem with uterus may lead to implantation failure, early miscarriages, premature delivery, and abruption placentae

A- congenital defects :

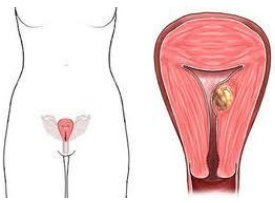


1. Müllerian agenesis (absent uterus+vagina)
2. Unicornuate uterus
3. Uterus didelphys
4. Bicornuate uterus
5. Septated uterus (the most common uterine malformation and a cause for miscarriages)
6. fetal exposure to DEX

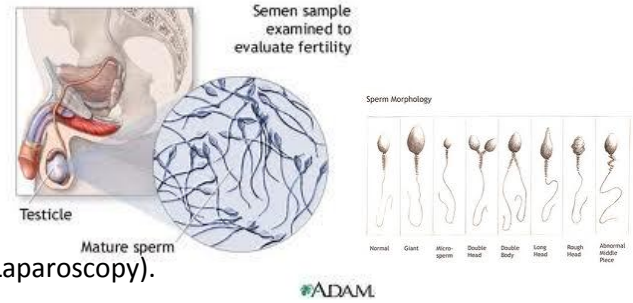
- **NB :** Laparoscopy to evaluate the exterior contour of the uterus and distinguish between a bicornuate and septate uterus (we can't differentiate between them by hysterosalpingogram)

B- Acquired defects :

1. Endometritis **:** associated with infection , a traumatic delivery, abortions, dilatation and curettage, intrauterine device, retention of placental fragments adhesion
2. **<u>Large</u>** submucosal fibroids
3. Uterine Polyps
4. Asherman syndrome (Adhesion due to excessive curettage)
5. Adenomyosis

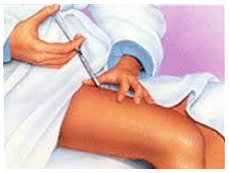


6. Luteal phase defect**:** an uncommon condition that involves inadequate development of the microscopic and cellular changes in the endometrial lining of the uterus after ovulation and exposure to progesterone→take biopsy during luteal phase to see endometrial changes→to know is it +/- normal**.**

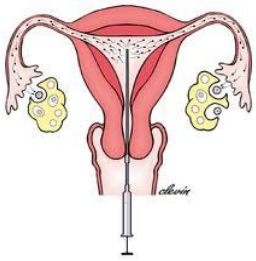


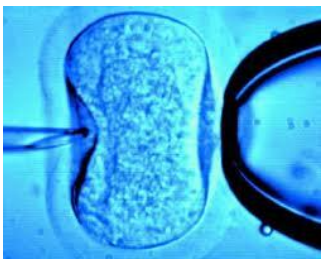
- **Types of Investigation for infertility :**
  1. **Semen analysis.**
  2. **Postcoital test:** for male and female.
  3. Assessment of **ovulation** (serum progesterone at 21 day).
  4. For **tubal factor** (Hysterosalpingography, Laparoscopy).
  5. Uterine factor (hysterosalpingography, hysteroscopy, and Laparoscopy).
  6. Blood concentration of **TSH and prolactin**
  7. Blood concentration of **FSH, LH and estradiol** (in the 3rd day of the cycle).
  8. Evaluation for Cushing's syndrome (either 24 hour urine for "urine free cortisol" or overnight 1 mg Dexamethasone suppression test) if clinical appearance is suggestive.
  9. Evaluation for acromegaly.
  10. In some situations, radiologic testing of the brain will be recommended

- **Treatment that used for infertility :**
  1. Ovulation induction (90% success rate):
     a. Clomiphene citrate (Clomid): weak estrogen that blocks the estrogen receptor and stops the estrogen negative feedback. Ovulation rate: 70% pregnancy rate: 50%.
     b. Human menopausal gonadotropin (HMG): when clomid fails, successful ovulation 90%, pregnancy rate: 50%.
     c. FSH: the same as HMG.
     d. Gonadotropin-releasing hormone (GnRH) analog: lower rates of hyperstimulation.
     e. Bromocriptine: when the anovulation caused by hyperprolactinaemia.



  2. Intrauterine insemination (IUI): used for unexplained infertility, Male infertility (mild) , Failure to conceive after ovulation induction treatment, Immunological (anti sperm antibodies), Ejaculatory failure and retrograde ejaculation.
  3. In vitro fertilization (IVF)(ICSI) : indicated in **<u>blocked fallopian tubes</u>**, Failure to conceive after ovulation induction treatment, sperm only in the testes or low amount of sperm and sever endometriosis





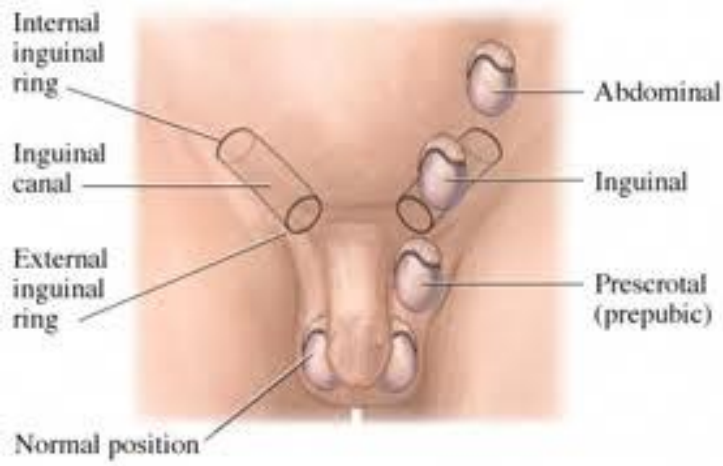
- **NB:** -No fertility treatment has officially been found to effectively increase fertility in women with premature ovarian failure or resistant ovary syndrome.
  -Hyprerstimulation from clomiphine and hMG (the later is more) result in multiple ovria follicles, and can produce large theca lutein cyst with ascites, treatment is conservative.

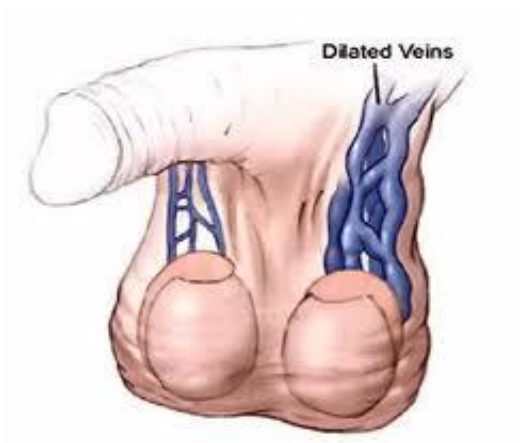
- **Male infertility (imp):**
- **Causes:**

Age	Malaria	**Testicular Trauma** (excess heat, radiation, torsion)
Radiation	**Mumps**	Cryptorchidism (undescended testes)
Orchitis and prostatitis.	Hydrocele	**Lifestyle** → smoking, alcohol, Drugs*, Obesity
Varicocele	Testicular cancer	Idiopathic oligospermia (30% of male infertility)
Drugs: Chemotherapy, anabolic steroids, Cimetidine, Spironolactone, and Phenytoin.		
Altered Sperm Transport : Absent vas deferens or obstruction + Erectile dysfunction + Retrograde ejaculation + Hypospadias + Impotence		

- **Investigations:**
  1. **Semen Analysis: (see table)**
  2. **Hormonal**
     a. Testosterone level.
     b. FSH level.
  3. **Chromosome Karyotype**

Volume	2-5 ml
Density	> 20 million/ml
Motility	> 50% (speed)
Morphology	> 30%
pH	7.2-7.8
Liquefaction	Within 30 min.





Hirsutism

- **Hirsutism**: is the excess growth of male pattern, pigmented, terminal hairs of the body midline (face, chine, chest, abdomen, back, inner thighs).
- **Hypertrichosis**: the excess growth of nonsexual hair (eyebrows, eyelashes, forearm, lower legs).
- **Defemininzation**: is the early stage of virilization (decreased breast size, loss of vaginal lubrication).
- **Virilization**: is an excess of male-pattern hair with: Increased muscle mass, Clitromegaly, temporal balding, and deepening voice.

- **Causes of Hirsutism:**

  A. Ovaries:
  1. Hormonally active tumor. (sertoli-leydig cell tumor [Hilar cell tumor]).
  2. PCO syndrome.
  3. Luteoma of pregnancy.

  B. Adrenals:
  1. Hormonally active tumor.
  2. Congenital adrenal **h**yperplasia [CA**H**] (21-**h**ydroxylase deficiency).
  3. Cushing syndrome.

  C. Exogenous medication:
  1. Anabolic steroids.
  2. Danazol (estrogen antagonist & has androgenic effect).
  3. Methyl-testosterone

  D. Hair follicle sensitivity: (excessive conversion of androgens to **DHT** in the hair follicle**).**

- **Laboratory testing:**
  1. **Serum total testosterone:** identifies androgen producing **ovarian** tumor.
  2. **Serum DHEAS:** androgen producing **adrenal** tumor**.**
  3. **Serum 17-OH progesterone: CAH.**
  4. **Overnight dexamethasone suppressing test: Cushing's syndrome.**
  5. **Serum LH:FSH ration: PCO syndrome.**
  6. **Serum 3α diol glucuronide:** product OF **DHT.**

- **Management of Hirsutism :**
  1. **Stop the offending medication.**
  2. **Surgical removal of the androgen secreting tumors.**
  3. **Medical medication :**
     - i. **Glucocorticoid replacement:** for **CAH.**
     - ii. **Oral contraceptive agents**: for **PCO.**
     - iii. **Spironolactone:** for **hair follicle sensitivity.**
  4. **Cosmetic measures:** (waxing, depilatory creams, shaving, bleaching).

- **Notes:**
  1. **Medications** that cause **hypertrichosis: (**Phenytoin, Diazoxide, Minoxidil, Streptomycin, and penicillamine**).**
  2. **Defemininzation** and **Virilization** symptoms: suggest a **tumor [pathology].**
  3. **Cushingoid findings:** include centripetal obesity, extremity wasting, abdominal striae, buffalo hump, and moon face.

Amenorrhea

- **Primary Amenorrhea :** absence of normal menstruation in a patient without previously established cycles
  - by age 16 in female with 2ry sexual characteristics
  - by age of 14 in female without 2ry sexual development
- **Secondary amenorrhea:** absence of normal menstruation in a patient with previously established cycles.
  - No menses for 3 months, if previous menses were regular.
  - No menses for 6 months, if previous menses were irregular.
- Hypothalamus, pituitary gland, ovarian follicle, corpus luteum, endometrium, and out flow tract are the anatomical components of a normal menstrual physiology.
- **Causes of Primary amenorrhea :**

Anatomic site	Pathology	LH, FSH level	Dx
Hypothalamus	-Hypothalamus Dysfunction -Kallmann syndrome	Low	-Hx -MRI -CT scan
Pituitary	-Pituitary dysfunction	Low	-Hx -GnRH stimulation test
Ovarian follicle	-XX & XY gonadal dysgenesis -Turner syndrome (XO) [THE MOST COMMON] -Vanishing testes -5-alpha-reductase deficiency - 17-α hydroxylase deficiency in CAH -Galactosaemia (galactosaemia is toxic to oocytes)	High	-Hx -Karyotyping -Gonadal biopsy -FSH level
Corpus luteum (**Anovulation**)	-Hyperprolactinemia (tumors, drugs)	Normal	-Hx -Progesterone challenge test -MRI -CT scan
Uterus or endometrium	-Androgen insensitivity (resistance) syndrome -Mullerian agenesis (absent uterus)	Normal	-Hx & Ex -Testosterone level -Karyotyping -Hysteroscopy
Outflow tract	-Imperforate hymen -Vaginal agenesis (absent vagina) -Transverse vaginal septum -Congenital cervical stenosis	Normal	-Hx & Pelvic Ex.
Constitutional delay (Unknown)	-There is no abnormality but she is a little later than others in reaching her menarche		-Family Hx of delayed menarche

Another classification of Causes of Primary amenorrhea: -

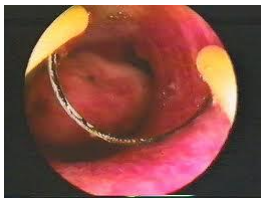
Patient type	DDx	Evaluation
-Breast present -Uterus present	-Outflow tract anomaly -Anovulation -Current hypothalamic-pituitary failure	-Pelvic Ex. -Serum prolactin -Progesterone challenge test
-Breast present -Uterus absent	-Congenitally absent uterus -Androgen insensitivity	-Karyotype -Serum testosterone
-Breast absent -Uterus present	-Gonadal dysgenisis -Hypothalamic-pituitary failure	-Karyotype -FSH level -GnRH stimulation test
-Breast absent -Uterus absent	-Agonadism (vanishing testes) -17,20-desmolase deficiency -17-hydroxylase deficiency	-Karyotype -Gonadal biopsy -HCG stimulation of testosterone

- **Causes of secondary amenorrhea :**

Anatomic site	Pathology	LH, FSH level	Dx
Hypothalamus	-Tumors -Anorexia nervosa -Severe weight loss, stress, Trauma, radiation, drug abuse , and exercise.	Low	-Hx -MRI -CT scan
Pituitary (anterior)	-Panhypopituitarism -Hypothyroidism -Hyperthyroidism -Pituitary tumors -Sheehan's syndrome -Empty sella syndrome -Haemochromatosis -Medications as progestagenic medications	Low	-Hx -GnRH stimulation test
Ovarian follicle	-Postradiation, infection, or chemotherapy. -Premature ovarian failure -Resistant ovary syndrome -Autoimmune disease -Menopause -Ovarian cancer	High	-Hx -Karyotyping -Gonadal biopsy
Corpus luteum (**Anovulation**)	-Polycystic ovary syndrome -Hyperprolactinemia (tumors, drugs, chest wall stimulation) -Weight loss, stress, and exercise	Normal	-Hx -Progesterone challenge test -MRI -CT scan -US
Uterus or endometrium	-Pregnancy [THE MOST COMMON] -Intrauterine adhesions (Asherman's syndrome) blocks the passage of blood	Normal	-Hx -β-hCG -Hysteroscopy
Outflow tract	-Cervical stenosis from conization or cryotherapy	Normal	-Pelvic Ex.

Treatment:

1. Hypothalamic :
    a. Increase the weight to normal in case of low weight.
    b. Clomiphene → failure→ then go with FSH and HCG
    c. Anorexia nervosa ⇨ Psychiatric Rx + HRT
2. Pituitary :
    a. HRT
    b. In case of hyperprolactinaemia or pituitary adenoma → bromocriptine → failure →surgery
    c. Thyroxin in case of hypothyroidism
    d. SHEHAN'S SYNDROME→ HRT
3. Asherman's syndrome : Hystroscopic resection of the adhesions followed by estrogen therapy
4. Premature Ovarian Failure or resistant Ovarian Failure : HRT → to prevent osteoporosis
5. Uterus absent : Vaginoplasty + Vaginal dilators
6. Testes present : Gonadectomy
7. Polycystic ovarian syndrome
    a. Reduce weight
    b. Metformin (Hyperinsulinemia)
    c. Spironolactone(Hirsutism)
    d. Combined oral contraceptive pill
    e. Clomiphene citrate
    f. Laser therapy (Hirsutism)
    g. Ovarian drilling is an option for patients with PCO not responding to medical treatment



_____
HRT=Hormonal replacement therapy

Urinary Incontinence

-Definition: involuntary loss of urine. Incidence ↑with age and parity.

-Normal physiology: normal continence occurs if the intralvesical pressure is lower than urethral sphincter, HOW?

-By a relaxed detrusor muscle (muscle within bladder wall) with contracted bladder neck and urethra.

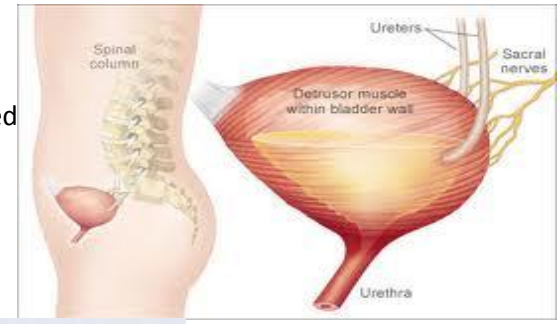
-Micturition (opposite): occur when intralvesical pressure exceeds

-Urethral sphincter pressure, by contracting detrusor and relaxing sphincter.

1-Urethral length: longer urethra→better continence, so incontinence in ♀ (5cm) more than ♂ (12cm).

2-Pubourethral ligaments: support the urethra, loss of support of these ligaments → incontinence.

-Innervations of lower urinary tracts:



1- Parasympathetic Fibers from S2-S4 → detrusor muscle contraction to promote **micturition**, acute cystitis, radiation injury cause uncontrolled stimulation leading to incontinence.

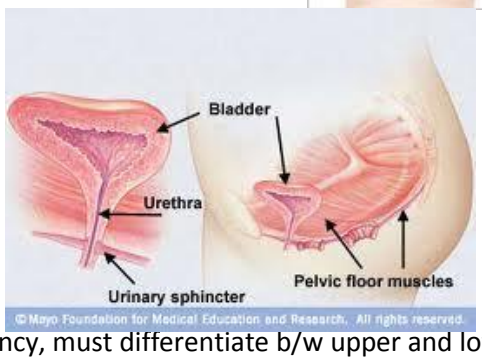
2-Sympathetic fibers from T10-T12 and L1-L2 to promote **continence**:

a- α-adrenergic: contract bladder neck and urethra

b- β-adrenergic: relax detrusor muscle.

-Normal cystometric measurement: (see table)

Residual volume	**<50 ml**
Sensation of fullness	**150-200 ml**
Urge to void	**400-500 ml**



-Urinary incontinence in pregnancy:

-Infection: cause urinary incontinence in pregnancy, must differentiate b/w upper and lower UTI.

-<u>Asymptomatic bacteriuria criteria:</u> 1- Mid stream, clean-catch specimen.
2- Colony count > 100,000/ ml on culture.
3- Isolation of single organism (not lactobacillus which is vaginal contaminant).

-Urinary tract anomalies: urethral diverticulum, duplicated ureter), predispose to infection. Bladder inflammation irritates the uterus →uterine contraction→preterm labor.

-Urinary incontinence in nonpregnant women:

❶**-Irritative incontinence**: should be ruled out as first step.

<u>-Causes:</u> a- Infection (cystitis)
b- Neoplastic (bladder tumors).
c- Foreign body in bladder.

<u>-Symptoms:</u> urgency, frequency, dysuria.

<u>-Physical examination finding:</u> leakage with urgency, stable bladder with normal anatomy, normal neurologic Ex.

<u>-Diagnostic workup:</u> 1- High WBC and bacteria on **urine analysis** →infection, +ve urine culture confirm diagnosis.
2- RBC on urine analysis → tumor or foreign body, urethrocystoscopy confirms diagnosis.
3- Normal cystometrogram examination, (which is distending the bladder with known volume and observe the pressure change, when the volume reach about 450-500ml ,sudden rise in intravesical pressure appear due to detrusor contraction, normal patient can inhibit this reflex, but abnormal can't).

<u>-Management:</u> treat the cause (antibiotics, resection of tumor, remove foreign body).

❷**-Stress Urinary Incontinence**: occur when intraabdominal pressure ↑ and transmitted more to bladder and less to urethra.

-Mechanisms: 1-Weakness of pelvic diaphragm→ loss of bladder support and descent of proximal urethra.

2-Change of urethroviscal angle so any stress activity→great ↑of intravisecal pressure more than urethral pressure. **(See the figure)**.

a- **Spurts** loss of urine

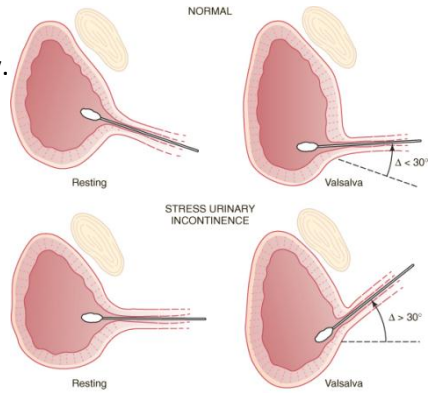
b- Detrusor muscle contractions **do not** occur→ bladder does not empty completely

-Clinical findings:

1-Hx of loss urine loss because of coughing, sneezing, laughing, or physical activity.

2-No loss of urine when patient is supine or sleep (unique to stress type)

3-Cystocele or urethrocele, anterior vaginal wall prolapsed (maybe).



-Diagnosis: include all of the following tests:

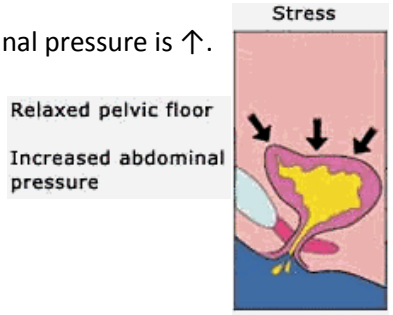
1-Urine culture to roll out infection.

2-Cystometrogram is normal (no involuntary detrusor contraction, (residual volume, bladder capacity, sensation) all are normal).

3-Urinary leakage seen with voluntary stress.

4- Positive cotton-tipped test (Q-tip or Bonney), which shows poor anatomic support, the angle of Q-tip will change more than 30 degree when Intraabdominal pressure is ↑.

5-Normal neurologic examination.



-Management: =Medically:

a- Kegel exercise: strength the pelvic floor muscles.

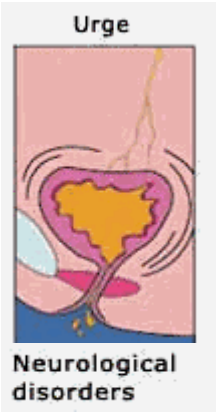
b- ERT therapy: dilates the periurithral venous plexus → elevate resting urethral pressure.

=Surgically:

a-Elevation urethravesical angle (urethropexy), is the definitive treatment, restore normal anatomy, so the intra-abdominal pressure will distribute through bladder, urethra, bladder neck **equally**.

b-Collagen injection: elevate resting urethral pressure.

❸**-Urge incontinence:** 2nd most common, results from detrusor instability, (hypertonic bladder, uninhibited bladder) are other names.



-Mechanisms: 1- Uninhibited involuntary detrusor muscle contraction (only type of incontinence in which detrusor muscle contraction take place).

2- Coughing, sneezing, exercise; induce detrusor contraction-→complete emptying.

-Symptoms: urgency, frequency, and unpredictable loss of volume (urine).

-Physical examination: normal.

-Diagnosis: confirmed by all of the following in cystometrogram:

1- **Involuntary bladder contraction associated with leakage**.

2- Normal residual volume and sensation.

3- ↓urge-to-void volume.

-Management: **only medical** (no surgical):

1- Anticholinergic: like oxybutynin, panthline, to suppress parasympathetic stimulation→↓tone of detrusor muscle.

2- β-agonist: flavoxate →to relax detrusor muscle.

3-NSAID: (ibuprofen) inhibit bladder contraction.

4- Bladder training: to help patient control the bladder.

❹**- Overflow incontinence:** leakage occurs when intravesical pressure exceeds urethral pressure, and stop when pressures are equal, bladder **never empties detrusor contractions do not occur.**

-Mechanisms: **1- Areflexia or hypotonia of detrusor muscle**:

a- Denervated bladder (DM, LMN lesion).

b- Medications: Anticholinergic, α-agonist, epidural anesthesia).

2-Urethral obstruction: urethral knick, pelvic mass.

-Cystometric finding: ↑residual volume, ↑bladder capacity (>1000ml), ↓sensation, poor detrusor contractility.

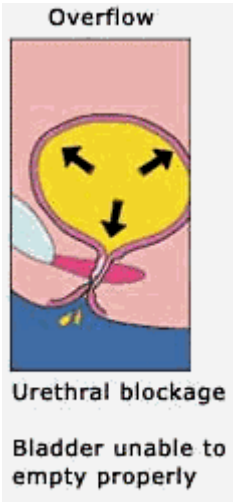
-Management: only medical:

1- Intermittent self-catheterization

2- Cholinergic agents: (bethanecol) to stimulate detrusor contraction.

3- α-blockers: phenxybenzamine, to ↓outlet resistance.

4-Stop causative medication.



❺- **Bypass incontinence:** when urethral sphincter function is bypassed.

-Mechanisms:

1-Fistula: usually, uretrovaginal, vesicovaginal, urethrovaginal.95% of fistulas result from pelvic surgeries or radiation.

2-Urethral diverticulum: sac-like outpunch from urethral which creates reservoir of urine and empty unpredictably.

-Symptoms:

1- Fistula: urinary drainage after pelvic surgery or radiation.

2- Diverticulum: post-void urinary incontinence, urgency, frequency.

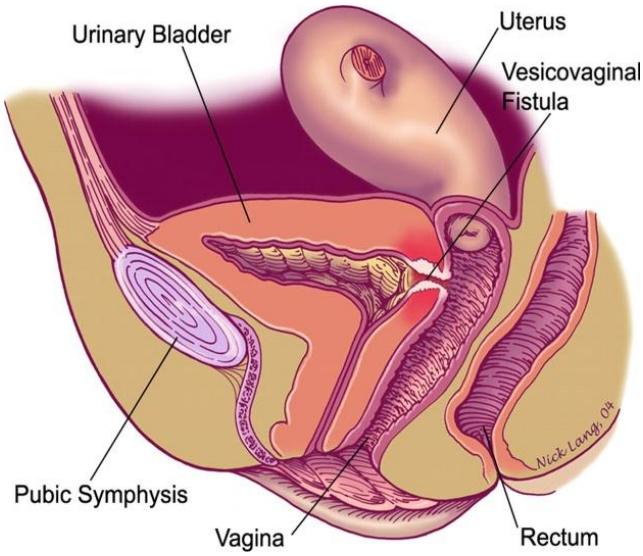
-Diagnosis:

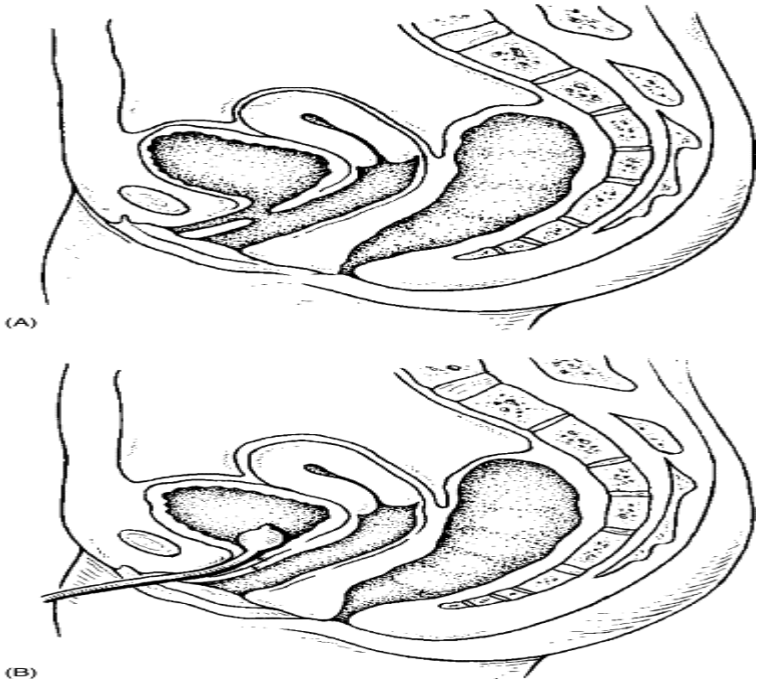
1-IVP with contrast, look for contrast leakage.

2-IV indigo carmine dye injection, dye leakage.

3-Urethroscopy: for diverticulum.

-Management: surgery is only definitive treatment.





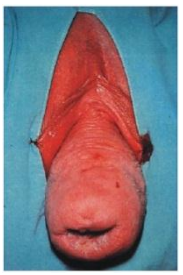
Urethro-vaginal fistula

Genital prolapse

Definition: protrusion of the pelvic organs into or out of the vaginal canal. They result from **weakness** in the pelvic muscular (levator ani) and fascial structural support.

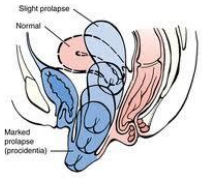
Predisposing factors:

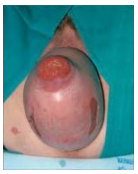
1. Stretching of the pelvic support by **pregnancy** and **labor** especially with use of **forceps** or **Ventouse extractor**.
2. Chronically increase of **intra-abdominal pressure** (chronic cough, constipation, heavy lifting, ascites).
3. Pelvic connective tissue **weakness**.



Types of pelvic organ prolapse:

1. **Uterine prolapse:**
   Symptoms: heaviness or fullness in pelvis, feeling of something falling out, difficulty walking.
   Staging:
   0 = No prolapse
   1 = Distal portion is -1 cm above hymen.
   2 = Distal portion is ≤ 1 cm above or below hymen.
   3 = Distal portion is ≤ 2 cm below hymen.
   4 = Complete eversion, which is called procidentia and results in purulent discharge, decubitus ulceration, and bleeding.

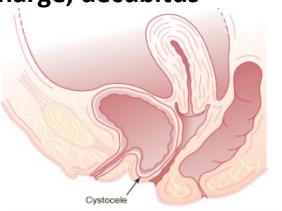




2. **Cystocele:** (anterior vaginal wall herniation**)**
   Symptoms: urinary **frequency** and urgency, urinary **incontinence**, urinary **retention**.
   Nature of the prolapse: the **bladder** is within the vagina.

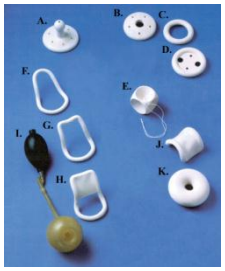
3. **Rectocele: (**lower posterior vaginal wall herniation**)**
   Symptoms: difficulty emptying her rectum, constipation, and the need to splint.
   Nature of prolapse: the rectum is within the vagina.

4. **Entrocele:** (upper posterior vaginal wall herniation)
   Symptoms: 1. Backache or pulling sensation when standing relieved by lying down.
   2. Uncomfortable pressure with falling out sensation in the vagina.
   Nature of prolapse: the pouch of Douglas is herniated and contains loops of bowel.

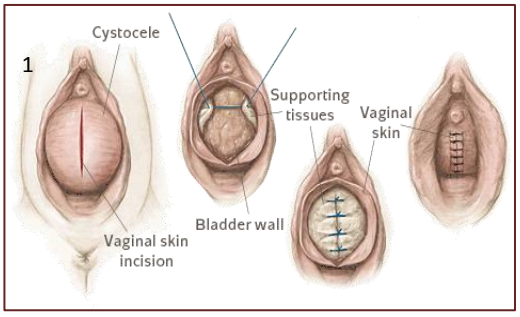


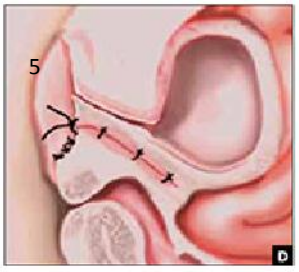
Management of genital prolapse:

1. **Nonsurgical approach:**
   1. **Kegel exercises:** the patient tightens the pelvic muscles as of to stop the flow of urine.
   2. **Passaries:** when the patient is not fit for surgery, pregnant or is postpartum.
      Side effect: vaginal infection or discharge.
   3. **Estrogen replacement therapy (ERT):** in menopausal women**.**



2. **Surgery:**
   1. **Anterior colporrhaphy:** plication of the pubocervical fascia (cystocele and urethral disp.)
   2. **Posterior colporrhaphy:** plication of endopelvic fascia (rectocele).
   3. **Entrocele repair:** hernia repair and approximating uterosacral ligament and levator ani muscles.
   4. **LeFort's partial colpocleisis:** suturing anterior and posterior vaginal walls together to support the uterus.
   5. **Complete colpocleisis:** total obliteration of the vagina.





Menopause

Perimenopause: is 3 to 5 years period with increasingly frequent **irregular** anovulatory **bleeding**. Followed by episodes of **amenorrhea** lengthening with **intermittent menopausal symptoms.**

Menopause: is the point at which menstrual cycles **permanently** cease. A woman is classified as menopausal after **12 months of amenorrhea**, and the mean age for menopause is **51 years**.

1. **Natural menopause:** > 40 years, due to depletion of ovarian follicles.
2. **Premature menopause:** 30 - 40 years, mostly **unknown cause**, may occurs after: Oophorectomy, or **ovarian** infection, radiation or chemotherapy.
3. **Ovarian failure:** < 30 years, **autoimmune** condition.

Risk factors: Smoking and **high altitude residence.**

Early effects of menopause [occur in Climacteric]**:** due to decreased estrogen levels.

i. **Symptoms :**
   1. **Cessation of menses:** the **most common** symptom and happens gradually.
   2. **Hot flashes:** in **75%** of women, sensation of heat begins is the face, neck, chest, followed by profuse sweating the upper body lasts for **4 minutes** and for **1-5 years** duration, due a **cutaneous vasodilation**.
   3. **Reversal of PMS:** decreased breast tenderness, abdominal bloating, edema, headache, and cyclic emotions.
   4. **Urinary symptoms:** of **urgency**, **frequency**, and **nocturia** due to **urothelium atrophy.**
   5. **Psychological change: depression** and **irritability** are common, happens due decreased levels of central neurotransmitters and endogenous opioids.

ii. **Physical findings:**
   a. **Reproductive tract:**
      1. **Vagina:** flattening and thinning of the epithelium, increase in pH, and decrease in lubrication.
      2. **Cervix:** decrease in size and cervical mucus, squamoclumnar junction retreats into endocervical canal.
      3. **Uterus:** Decrease in size, shrinkage of myomas, and atrophy of adenomyosis.
      4. **Ovaries:** decrease in size and become nonpalpable.
   b. **Urinary tract:** urothelium atrophy leads to **loss of urethral tone** and cause urethral caruncle. The bladder may become hypertonic (**detrusor instability**).
   c. **Pelvic floor:** pelvic relaxation which could lead to uterine and vaginal **prolapse.**
   d. **Breasts:** decrease in size and fewer benign cysts than non-menopausal.
   e. **Skin:** thickness and collagen content decrease resulting in **loss of elasticity** and **wrinkling**.

iii. **Hormonal changes:** related to loss of activity of ovarian follicles (estrogen and progesterone) with **continued** secretion of **testosterone** from ovarian **stroma** [sertoli-lydig cell tumor]**.**

Late effects of menopause:

i. **Osteoporosis:** is disorder of decreased bone density, leading to decreased skeletal strength and fractures.
   a. **Vertebral crush fractures**: the **most common**, untreated women lose 2.5 inches in height.
   b. **Hip and wrist fractures:** the **next most common** sites.

Types of osteoporosis:

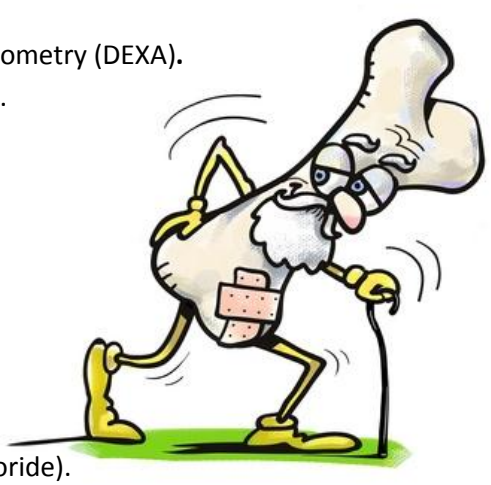
1. **Type 1:** affects **trabecular** bone mostly **vertebral bodies**, **postmenopausal** women mostly affected.
2. **Type 2:** affects **trabecular** and **cortical** bone, mostly **hip bones**, both **men** and **women** are affected.

- Risk factors:

gender	genetic	Life style	steroids
Female (3:1)	Positive family history	smoking	Exogenous steroids
Fair skinned, slender, women	Highest In whites Lowest in black	↑ Caffeine intake ↑ Alcohol use ↑ Protein in diet	Cushing's syndrome

- Diagnosis:

1. **Current bone density:** assessed by dual-energy x-ray absorptiometry (DEXA)**.**
2. **Rate of bone loss:** assessed by 24-hour urinary hydroxproline.



Prevention:

- **Lifestyle:**
    1. Dietary calcium and supplements.
    2. Vitamin D supplements.
    3. Weight-bearing exercise.
    4. Cigarettes and alcohol cessation.
- **Medications:**
    1. **Estrogen replacement therapy (ERT).**
    2. **Other medication:** (alendronate, ralxifene, calcitonin, fluoride).

ii. **Cardiovascular disease:** the number one cause of death in women:
Epidemiology: before menopause the risk of a heart attack is only one-third that of a man's. After menopause, it increases until it is the same for both genders as the age of 70 years.

Management of menopause: by **E**RT.

- **Benefits:**

a. **Vagina:** estrogen thickens the epithelium which decreases dyspareunia, and produces an acidic pH.
b. **Urinary tract:** decreases atrophy of urothelium.
c. **Osteoporosis:** fractures reduced by 50%.
d. **Cardiovascular disease:** decrease the risk of heart attack by 50%.
e. **Alzheimer's disease:** reduced by 50%.
f. **Colon carcinoma:** reduced up to 50%.

- **Risks:**

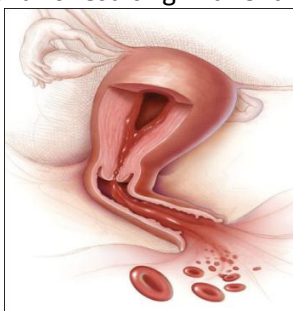
a. **Endometrial carcinoma:** up to **10 folds**, adding progestin to ERT eliminates the increased risk.
b. **Breast cancer.**
c. **Thrombosis:** by 100%.
d. **Stroke:** by 41%.
e. **Gallbladder disease.**

Follow up in HRT –No risk in the 1st 5 years of menopause.
-After that annual mammography is needed to rule out breast ca.

Abnormal menstrual bleeding

❖ **Definitions:**

- **Menorrhagia:** excessive and/or prolonged menses (>80 mL, >7 days) occurring at normal intervals.
- **Hypomenorrhea:** decreased amount and/or duration menses (<10 mL, <2 days) occurring at normal intervals.
- **Polymenorrhea:** abnormally frequent menses at intervals <24 days.
- **Oligomenorrhea:** abnormally prolonged intervals > 35 days.
- **Metrorrhagia:** irregular episodes of uterine bleeding.
- **Menometrorrhagia:** Heavy and irregular uterine bleeding.
- **Dysfunctional uterine bleeding (DUB**): abnormal uterine bleeding between menarche and menopause that is **not** attributed to medications, blood dyscrasias, systemic diseases, trauma, uterine neoplasms, or pregnancy. **Caused** by aberrations in the hypothalamic-pituitary-ovarian hormonal axis resulting in **anovulation.**



- **Characteristics of normal menstrual cycles:**
  1. Cycle length (21-35 days).
  2. Cycle duration (2-5 days).
  3. Volume of menstrual blood loss (10-80 ml).
  4. Cycles are regular and predictable.

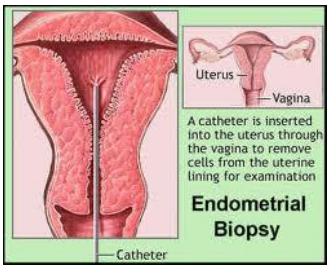
- **Ovulatory cycles:** are **self-limited** due:
  1. **Endometrium is structurally stable:** due the proliferation effect of estrogen is **followed** by progesterone.
  2. **With progesterone withdrawal:** the **vasoconstriction** leads to tissue collapse from **all parts** of the endometrium, in **orderly and progressive** way and **gives enough time** for the **clotting factors** to seal of the exposed bleeding sites.
- **Anovulatory cycles:** are **not self-limited** due:
  1. **Endometrium is structurally unstable:** due the effect of unopposed estrogen proliferation which could lead to a random breakdown.
  2. **Without progesterone withdrawal:** there is **no vasoconstriction** and bleeding occurs from **spontaneous**, **random breakdown** of the hyperproliferative endometrium.

- **Characteristics** of ovulatory cycle:
  1. Retained cycle predictability and regularity.
  2. Serum progesterone level > 500 ng/dl.
  3. Endometrial biopsy indication secretory changes.

- **Anovulatory bleeding** or DUB which effect usually **the extremes** of the reproductive years:

a. **Adolescent women:** may experience DUB in the first few years after menarche.
   1. Endometrial biopsy: is not necessary, because malignancy is rare.
   2. Management :
      -Cyclic progestin therapy is used in chronic bleeding.
      -Parenteral estrogen for stopping the acute hemorrhaging by rapid endometrial proliferation.

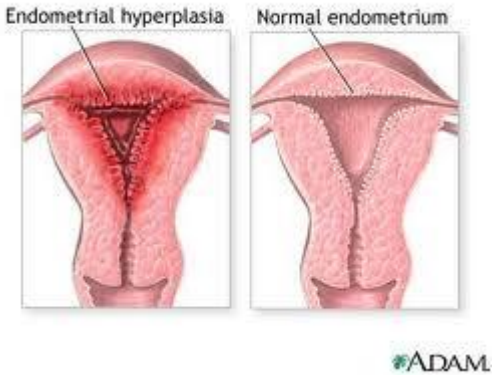
b. **Reproductive age women:** may experience DUB as a result of chronic anovulation syndrome:
   1. Endometrial biopsy : performed on in high risk patient(> 35 years, obese, chronically hypertensive, or diabetic)
   2. Management:
      a. Cyclic progestins.
      b. Endometrial ablation or hysterectomy: when hormonal management fails.



c. **Premenopausal women:** experience increasingly frequent anovulatory cycles(DUB):
   1. Endometrial biopsy: should be performed to rule out hyperplasia.
   2. Management is based on histological findings.

d. **Postmenopausal women:** can never experience DUB. Malignancy must be ruled out by endometrial biopsy.

- **If cycles appear to be anovulatory: a** progestin trial should be initiated:
  Following changes should be anticipated if the cycles are anovulatory:
  1. Bleeding should stop within 48 hours of starting the progestin trial.
  2. Bleeding should remain stopped until progestin course completed.
  3. Normal withdrawal bleeding occurs after progestin is completed.



- **If anovulation is confirmed:**
  1. **Fasting serum prolactin:** to rule out pituitary prolactinoma.
  2. **TSH level:** to rule out hypothyroidism.
  3. **Periodic progestin cycling:** to prevent endometrial hyperplasia.

- **If cycles appear to be ovulatory:** structural abnormality of the uterus should by investigated:
  a. **Hysterosonography:** endometrial polyps and uterine submucosal leiomyomas.
  b. **Hysteroscopy** with **D & C**: endometrial polyps and uterine submucosal leiomyomas.

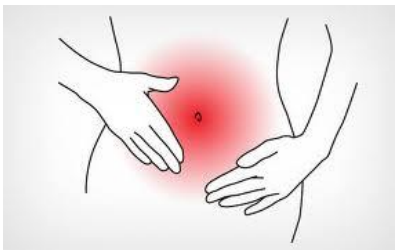
Dysmenorrhea

- **Definition:** is the presence of **painful menstrual periods**, prevalence: 50% of menstruation women.

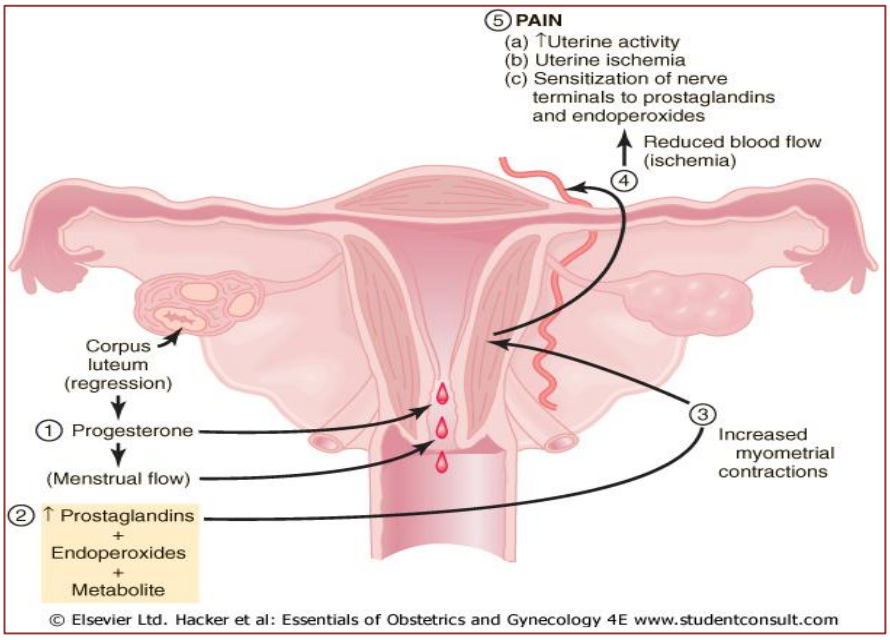
- **Classification:**

	Primary	Secondary
Onset	Within 2 years of menarche **Prior** or **at** menses, Lasting for **48-72** hours	20-30 years of age. May extend **pre-** or **post**menstually.
Description	**Cramping** in lower abdomen, radiating to lower back, thighs	**Dull**, **aching** often
Associated symptoms	Nausea and vomiting Fatigue Diarrhea headache	Dyspareunia Infertility Abnormal bleeding
Pelvic examination	**normal**	Variable, depending on cause.
Etiology *	1. Excessive endometrial prostaglandins (**PGF_{2a}, PGE2**). 2. Excessive myometrial **contractions.** 3. **Decreased** uterine blood **flow.** 4. Uterine **ischemia.**	**Endometriosis.** **PID.** **Adenomyosis.** **Leiomyoma.** **Pelvic congestion syndrome.** **Ovarian cysts.** **Cervical stenosis**.
Management	**1. NSAIDs** (suppress prostaglandin). **2. OCP.** **Avoid** surgical procedures.	Depend on the cause.

- **Cervical stenosis:** if severe, can impede menstrual flow, and cause retrograde menstrual flow which could lead to endometriosis.



- **Causes:**
  1. **Congenital.**
  2. **Injury:** infection, scarring, or operative trauma.
- **Management: cervical dilation** under anesthesia**.**
- **Pelvic congestion syndrome:**
  Results from **vascular engorgement** (congestion) of uterus and vessels of the broad ligament.
  The pain is chronic (burning or throbbing).
  Appears to be Stress**-related** with **psychosomatic features**.
- **Management: stress reduction** and counseling.



Premenstrual syndrome

- **Definitions:**
- **PMS:** adverse **physical, psychological, and behavioral symptoms** during the **luteal phase of** the menstrual cycle. (90% of women experience some degree of PMS: highest at 25-35 years).
- **PMDD (premenstrual dysphoric disease):** PMS symptoms are so **severe** that they **interfere** with usual daily functioning or personal relationships (5% of women).

- **Criteria:**
  1. Symptoms present only in the **second half** of the menstrual cycle (luteal phase).
  2. The first week of the cycle (follicular phase) is **symptom free**.
  3. Symptoms are **recurrent** (>3 consecutive cycles).
  4. Symptoms **sever enough** to interfere with normal functioning.

- **Theories of pathogenesis:**
  1. **Estrogen-progesterone imbalance.**
  2. **Renin- angiotensin -aldosterone activity**: causing fluid retention.
  3. Endogenous **endorphin deficiency.**
  4. **Prostaglandin excess.**
  5. **Vitamin deficiency.**
  6. **Serotonins deficiency.**

- **Diagnosis:** by **history** and confirmed by **menstrual diary.**

- **Symptoms and management:** see the table:

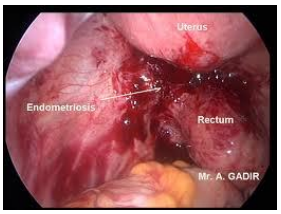
Symptoms clusters (fame)	Treatment options
1. Emotional: • Nervous tension. • Forgetfulness. • Mood swings. • Depression. • Irritability. • Anxiety. • Crying.	**A. Medications** 1. **SSRIs:** fluoxetine (prozac) only **effective** drug. 2. **Spironolactone:** for fluid retention. 3. **NSIDs:** for musculoskeletal complaints. 4. **Bromocriptine**: for breast tenderness. 5. Pyridoxine (v. B6). 6. Progesterone: **not** very effective.
2. Fluid retention: • Breast tenderness. • Extremity edema. • Weight gain. • Bloating.	**B. Lifestyle:** 1. Stress reduction. 2. Relaxation techniques. 3. Regular exercise. 4. Support groups.
3. Autonomic: • Heart pounding. • Confusion. • Dizziness. • Insomnia. • Fatigue.	**C. Nutritional:** 1. Balanced diet. 2. Decrease caffeine/sugar/ salt
4. Musculoskeletal: • Muscle aches. • Joints ache. • Headaches. • Cramps.	**SSRIs:** selective serotonin reuptake inhibitors. **Spironolactone:** potassium-sparing diuretics.

Endometriosis

a. **Definition:** is a benign condition in which the endometrial **glands and stroma** are present **outside** the uterine cavity. (**15%** of women).

b. The most common gynecological responsible for hospitalization in the reproductive years.

c. Pathogenesis theories:



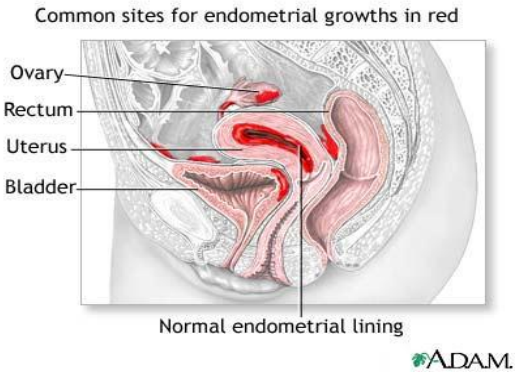
1. **Retrograde menstruation:** through the oviduct.
2. **Lymphatic and vascular drainage:** transporting endometrial tissue to other sites.
3. **Metaplastic transformation**: peritoneal mesothelium into endometrial tissue.

d. **Most frequent sites**: ovaries, cul-de-sac, uterosacral ligaments, broad ligaments, and fallopian tubes.

e. **Pathological** appearance: **vary** from **small**, red petechial implants in the peritoneum to **thickened**, scarred "powder burn", endmetriomas of the ovary (**chocolate cysts**).

f. **Symptoms:** could be asymptomatic and could be severe **despise** the extent of the disease.

1. **Painful intercourse (dyspareunia):** Especially with deep penetration.
2. **Painful defecation (dyschesia):** caused by rectum involvement.
3. **Infertility.**



g. **Diagnosis**: confirmed by **laparoscopy.**

h. Management:

i. **Medical**:

1. **Pseudopregnancy:** (putting the body in menopause state)
   -Continuous progestins
   -OCP.
2. **Pseudomenopause:** (putting the body in pregnancy state)
   -Danazol (Androgenic side effect is deepening in voice [MCQ])
   -GNRH agonist.

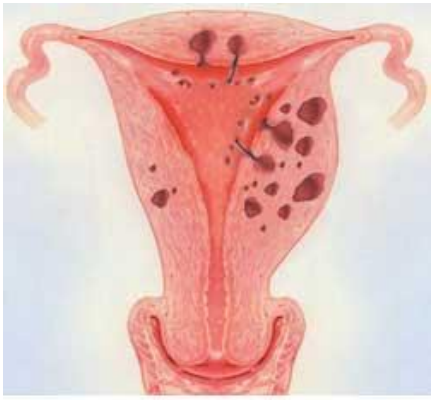
ii. Surgical:

1. **Conservative surgery:** (excision, cauterization, or ablation) to lyse adhesions to enhance **fertility**.
2. **Radical surgery: TAH+BSO** (total abdominal hysterectomy, bilateral salpingo-oophorectomy).

Adenomyosis

Definition: It is a benign ectopic endometrial **glands** and **stroma** found within the myometrium without a direct connection with the endometrial cavity.

Epidemiology: -It is the 2nd most common benign cause of enlarged nonpregnant uterus.
-50% of pts. with Adenomyosis have coexistent leiomyomas.
-15% of pts. with Adenomyosis have coexistent endometriosis



Adenomyosis. Note thickened wall of uterus which can be mistaken for fibroids.

Types: They can be diffused or localized forming an adenomyoma.

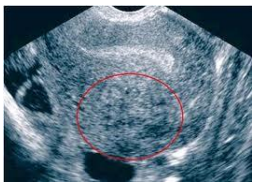
Risk factors:-Past Hx.
-Family Hx
- Multiparity
- Prior uterine surgery (C/S, Myomectomy)

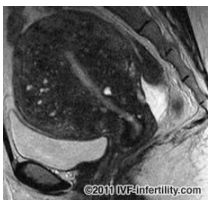
Symptoms:-Abnormal menstrual bleeding (Prolonged, Heavy, with large blood clots).
-Secondary dysmenorrhea.
-Infertility due to obstruction from extension of adenomyosis into isthmus (Salpingitis isthmica nodosa).

Ex.:-Usually unremarkable
-Enlarged uterus
-Tender uterus

Complications: -Globular enlargement of the uterine fundus due to the myometrial hyperplasia and hypertrophy around it.
-Anemia from excessive bleeding

Dx.: Using US or MRI.





Rx.: -NSAIDS
-Natural Progesterone Cream
-Hysterectomy is the only definitive Rx.

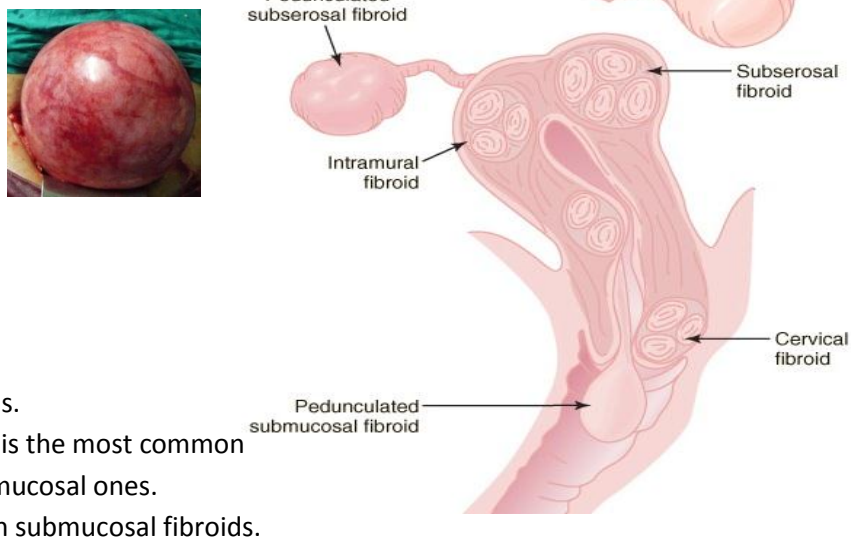
Uterine Fibroids

Other names: Uterine leiomyoma, myoma, fibromyoma, leiofibromyoma, fibroleiomyoma, and fibroma.

Definition: Fibroids are benign tumors derived from the smooth muscles of the myometrium.

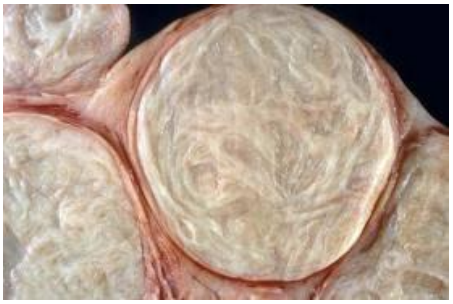
Epidemiology: -They are the most common neoplasm of uterus.
-Found in 25% of woman older than 35 years old.
-Their malignant potential is minimal (0.1%)

Types: -**See figure**.
-They always arise within the myometrium (intramural) but some migrate.
-They can grow to impressive sizes. Also they can invade the vena cava.



Risk factors:-Past Hx.
-Family Hx.
- ↑ Age during reproductive years.
- Ethnicity (high in African)
-Nulliparity
-High BMI.
-OCP (low risk)
-Mydroxy-progesterone (low risk)

Symptoms:- Most are asymptomatic even large ones.
-Prolonged & heavy menstrual bleeding is the most common if symptomatic with intramural & submucosal ones.
-Intermenstrual bleeding may occur with submucosal fibroids.
-Pelvic pressure (Urinary frequency, retention, hydronephrosis).
-Pelvic Pain (due to red degeneration especially in pregnancy).
-Lower back pain.
-Dysmenorrhea.
-Dyspareunia.
-Infertility can occur especially submucosal ones.
-Might obstruct labor and lead to malpresentation if it was cervical.

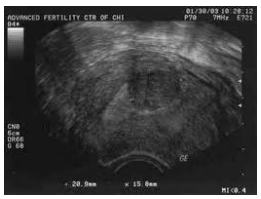


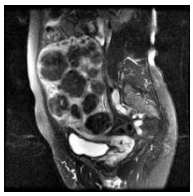
Pathogenesis: -They are strongly dependent on Estrogen & Progesterone for their growth.

Characteristics: -They are spherical, well-circumscribed, white, firm lesions with a whorled appearance on a cut section.
-They can undergo degenerative change such as: 1-Hyaline (most common).
2-Cystic degeneration.
3-Fatty degeneration.
4-Red degeneration (painful in pregnancy).
5-Calcification degeneration.
6-sarcoma degeneration (malignant).
-Fibroids can enlarge dramatically during pregnancy.

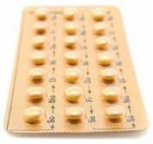
Ex.:-Usually unremarkable
-Irregularly enlarged uterus with smoothly rounded masses if the tumor is subserosal or intramural. It moves with the cervix in palpation.
-Tender uterus if red degeneration happened.

Dx: US, MRI.





Rx.: -Rx not necessary unless excessively large, causes infertility.



-Medical Rx:-For heavy, prolonged menstruation or Dysmenorrhea: -COCP

-Hormonal IUD.

-Progesterone-only pills.

-To reduce uterine Leiomyomas' size: -GnRH agonists

-Selective antiprogesterone receptor antagonists (mifepristone)

-Surgical Rx:-Myomectomy, either hysteroscopic or laparoscopic (if she desires to preserve fertility).

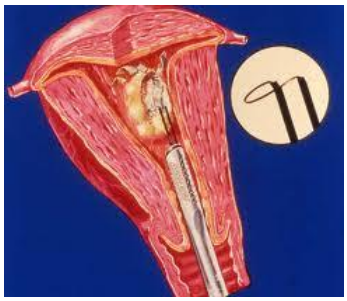
-Uterine A. embolization (if she desires to preserve fertility).

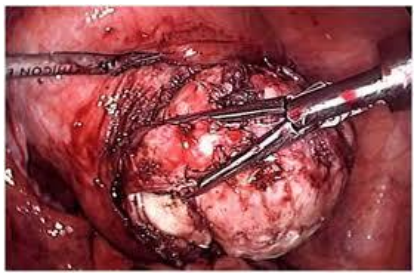
-Endometrial ablation (if completed family).

-Hysterectomy is the definitive Rx.

-Cryomyolysis.

-Radiofrequency ablation.





Endometrial cancer

Epidemiology: is the most common gynecologic malignancy in the US. It is the 4th most common malignancy after breast, colorectal, and lung cancer in women.

Risk factors: (PELOT BNDH)

i. **Chronic unopposed estrogen** stimulation **(PELOT):**
   1. **Obesity**.
   2. **Late menopause.**
   3. **Polycystic ovarian syndrome**.
   4. **ERT usage** without a progestin (**2- to 14-fold**).
   5. **Tamoxifen use for breast cancer:** Even though it is an **estrogen antagonist** in breast tissue it acts as **partial estrogen agonist** on the endometrium (**2- to 3-fold**).

ii. **Nulliparity.**

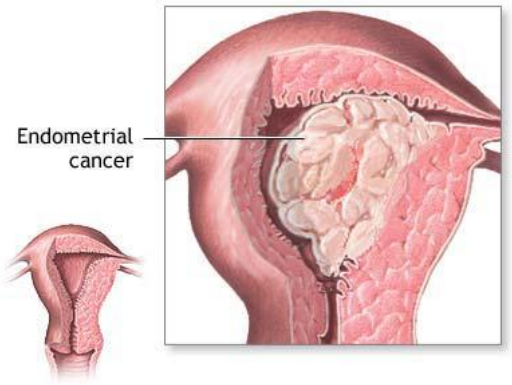
iii. **Diabetes mellitus.**

iv. **Hypertension.**

v. **Breast, colon, or ovarian cancer**.

Screening for asymptomatic women:
Population screening is **not feasible**, because there is no simple method for detection available. Screening only could be for high risk women.

Symptoms: abnormal vaginal bleeding (90% of patients).



Etiology of postmenopausal bleeding:

1. **Atrophic endometritis, vaginitis** (30%).
2. **Exogenous estrogens** (30%).
3. **Endometrial cancers** (15%).
4. Endometrial or cervical polyps (10%).
5. Endometrial hyperplasia (5%).

Diagnosis:

- Any woman who presents with postmenopausal bleeding should have a transvaginal ultrasound. If the endometrial thickness is **greater than 5 mm** (Tamoxifen use could lead to false positive reports) endometrial sampling is necessary**.**
- If the endometrial biopsy is **negative** for cancer or reveals **endometrial hyperplasia**, a fractional dilatation and curettage (**the gold standard**) should be performed under general anesthesia. Specimens should be submitted to histological evaluation.

FIGO staging: -Stage I**:**

Stage Ia: Tumor limited to endometrium.
Stage Ib: Invasion through less than one half of the myometrium.
Stage Ic: Invasion equal to or more than half of the myometrium.

-Stage II**:**

Stage IIa: Endocervical glandular involvement only.
Stage IIb: Cervical stroma invasion.

- Stage III**:**

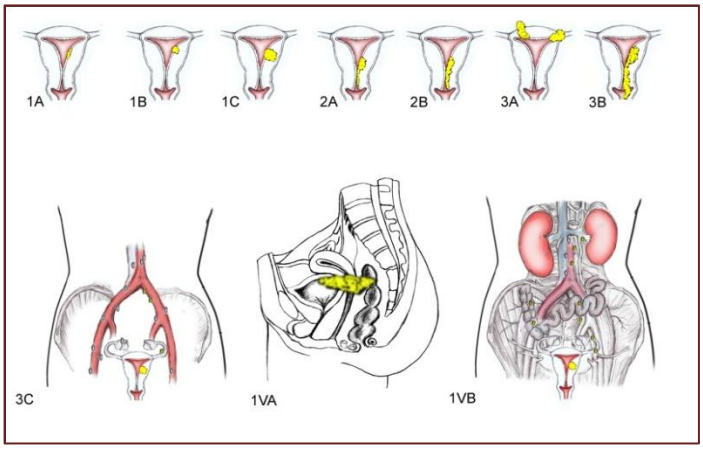
Stage IIIa: invades serosa or adnexa, or both, or positive peritoneal cytologic findings.
Stage IIIb: Vaginal metastases.
Stage IIIc: Metastases to pelvic or para-aortic lymph nodes, or both.

-Stage IV**:**

Stage Iva: Tumor invasion of bladder or bowel mucosa, or both.
Stage IVb: Distant metastases including intraabdominal or inguinal lymph nodes, or both

***Histologic grade does not change the stage*:**

Grade 1: Well differentiated.
Grade 2: Moderately differentiated.
Grade 3: Poorly differentiated.



Pathology:

1. **Adenocarcinomas (75%).**
2. **clear cell, squamous, or serous carcinomas,** all carry a **worse prognosis.**

Pattern of spread:

1. **Direct extension adjacent structures: most common** route of spread.
2. **Exfoliation of cells that are shed through the fallopian tubes**: implant on the ovaries, the visceral or parietal peritoneum, or the omentum.
3. **Lymphatic dissemination:** occurs to the pelvic lymph nodes and subsequently to the para-aortic lymph nodes, although simultaneous spread to both nodal groups may occur. In patients with deeply invasive, poorly differentiated stage I adenocarcinomas, however, pelvic lymph node metastases occur in up to 40% of cases.
4. **Hematogenous dissemination: less common**.

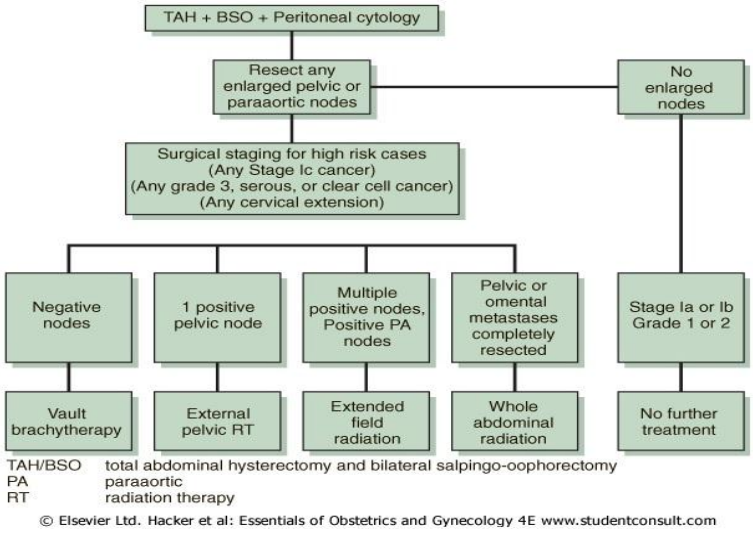
Treatment:

Stage I**:**

1. **Surgery**: TAH+BSO+ pelvic lymphadenectomy, in case of high risk patients (e.g. serous, clear cell or grade 3 histology; outer half myometrial invasion; or cervical extension).

2. **Radiation:**
   1. No need for **adjuvant therapy** for stage Ia or Ib, grade 1 or 2 patients.
   2. Patients with high risk carcinomas with negative pelvic nodes (i.e., any stage Ic cancer; any grade 3, clear cell or serous cancer; or any stage II cancer) may have vault **brachytherapy** (without external beam pelvic radiation).
   3. **External pelvic radiation:** for Patients with one positive pelvic node.
   4. **Extended field radiation:** for patient with multiple positive pelvic or proven para-aortic nodes.
   5. **Whole abdominal radiation** for patient with adnexal or omental metastases completely resected.
   6. **Combination of intracavitary + external beam radiation:** In patients medically unfit for surgery**.**

STAGE II: see the picture:



Advanced stages:

For advanced disease, treatment is individualized. The uterus, tubes, and ovaries should be removed, if possible, for palliation of bleeding and other pelvic symptoms.

Recurrent disease:

Seventy-five percent of recurrences develop within 2 years of treatment.
Metastases in other sites, such as the upper abdomen, lungs, or liver, are treated initially with high-dose progestins or antiestrogens.
If disease progresses while the patient is receiving progestins, chemotherapy may be offered.

Prognosis: see the table.

Stage	5-year Survival (%)
Ia	88.9
Ib	90.0
Ic	80.7
IIa	79.9
IIb	72.3
IIIa	63.4
IIIb	38.8
IIIc	51.1
IVa	19.9
IVb	17.2

Uterine sarcomas:

Classified as:

Pure: malignant tissue is of mesenchymal origin.

Mixed: malignant mesenchymal and malignant epithelial tissues**.**

Or classified as:

1. **Homologous:** the malignant tissue is **normally** present in the uterus (e.g., endometrial stroma, smooth muscle).
2. **Heterologous:** the malignant tissue is **not** normally present in the uterus (e.g., bone or cartilage).

Types:

1. **Leiomyosarcomas:**
   - Could be associated with a benign leiomyoma of the uterus, but the risk of malignant transformation in a benign fibroid is less than **1%.**
   - The histologic criteria for distinguishing leiomyosarcomas from leiomyomas are the **mitotic count (usually greater than 10 per 10 high-power fields), the presence or absence of coagulative necrosis, and the presence or absence of cellular atypia.**
   - Most cases are **not** diagnosed preoperatively but are discovered at the time of exploratory surgery for a probable fibroid.
   - If a known fibroid uterus appears to be rapidly enlarging, especially postmenopausally, malignancy should be suspected.

   Management: TAH+BSO.

2. **Endometrial stromal tumors:**
   a. **Endometrial stromal nodule**: **a** rare benign condition.
   b. **Endometrial stromal sarcoma:** is a low-grade lesion.
      Causes: abnormal vaginal bleeding and often with pelvic pain.
      Management: TAH+BSO.
   c. **High grade endometrial sarcoma:** causes abnormal uterine bleeding
      Diagnosis: endometrial biopsy or uterine curettage.
      Management: TAH+BSO.

3. **mixed müllerian tumors (**40% of uterine sarcomas):
   Up to 50% of patients with this lesion have evidence of metastatic disease at the time of diagnosis if surgically staged. Uterine sarcomas are poor because of the propensity for hematogenous dissemination. The overall 5-year survival rate is about **35%.**

Benign Ovarian Tumors (BOT)

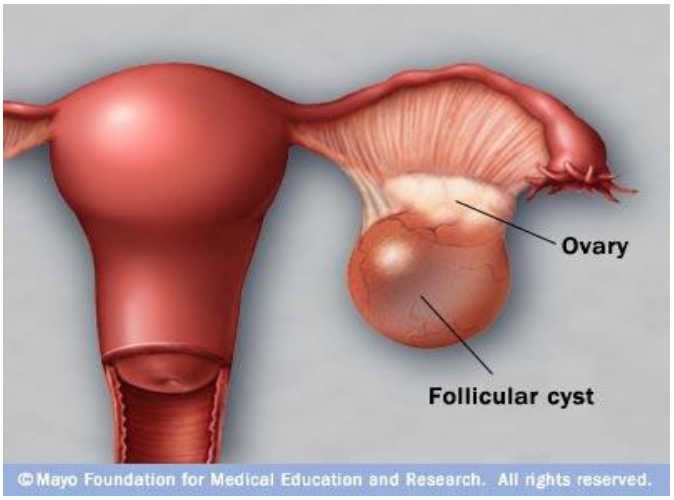
- Generally benign Tumors more common than malignant.

- Prevalence: 70% of **BOT** are functional, 20% neoplastic, 10% endometriomas.

***Functional cysts*:** - For a cyst to be classified as functional, the follicle diameter must ≥ 3 cm.
- Generally they cause pelvic pain, heaviness, dull sensation.

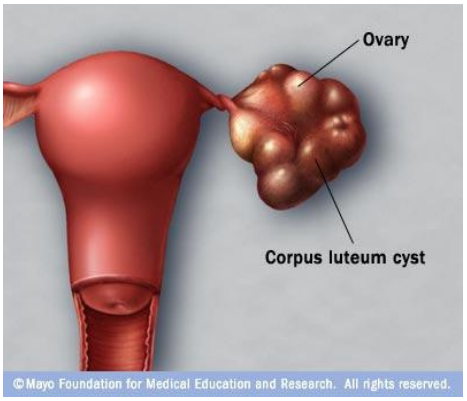
1-Follicular cyst: lined by one or more layer of granulosa cells.

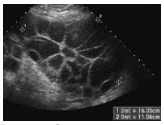
-Arise when physiologic release of the mature ovum fails (follicle fail to rupture).
-Follicular growth continues.
-Excessive stimulation by FSH**.**
-Lack of the normal preovulatory LH surge**.**
-Rarely grow larger than 10 cm.
-Most are asymptomatic**.**
-Larger cysts may cause pelvic discomfort or heaviness.
-Thin-walled, **unilocular**, and appear simple on ultrasound.
-Usually unilaterally.
-Management:
-Observation (70% to 80% resolve spontaneously)
-Oral contraceptives.
-If persist → surgical.



2-Corpus luteum cyst (lutein):

-Occur when the corpus luteum fails to involute and continues to enlarge after ovulation (after 14 days)
-Cysts may produce dull, unilateral pelvic pain
-Hemorrhagic corpus luteum cysts results from invasion of ovarian vessels into corpus luteum, they are more likely to cause symptoms and more likely to rupture.
-Ruptured hemorrhagic corpus luteum cysts can result in a Hemoperitoneum requiring surgery
-Most corpus luteum cysts are asymptomatic and resolve with observation and analgesia but If persist → surgical.
-Usually unilaterally.



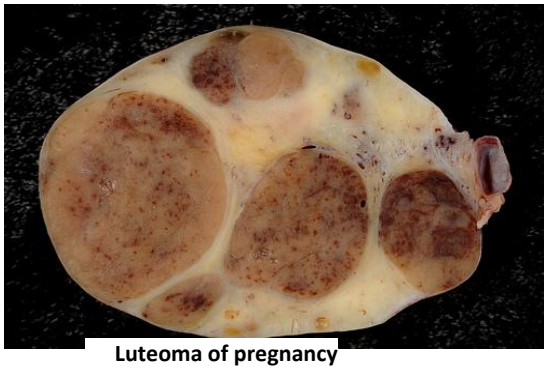


3- Theca-Lutein cysts:

-Occur due to high level of human chronic gonadotropin (HCG) or increased ovarian sensitivity to HCG, (Hydatidiform mole, multiple gestation, choriocarcinoma) and ovulation induction agents (clomiphene, gonadotropin).
-Usually **bilateral**, regress after gonadotropin or HCG levels fall.

4-Luteoma of pregnancy:

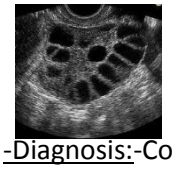
-Hyperplasic reaction of ovarian theca cells from prolongs response to HCG during pregnancy.
-Multifocal and usually bilateral, they may cause maternal virilization or ambiguous genetalia in female fetus.
-They regress after pregnancy.


Luteoma of pregnancy

5-Polycystic ovarian syndrome:

-Functional disorder associated with chronic anovulation, and hyperandronegsim.

-Clinical feature: -Generally are asymptomatic and unilocular, they continue to produce progesterone → delayed menses.



-If they undergo torsion or rupture it will cause acute lower abdominal pain, tenderness, hemoperitonium.

-Diagnosis:-Confirmed when the lesion regresses over the course.

-In general they are mobile and not associate with ascites.

Criteria	Scoring system
A- menopausal states Premenopausal postmenopausal	 1 3
B- US findings: Multiloculated Solid Bilateral Ascites	 1 feature = 1 ≥2 features = 3
c- serum CA-125 titer	Absolute value

-Risk of Malignancy Index (RMI).

-RMI= AxBxC. 200 is a cutoff value b/w benign and malignant.

-RMI has high sensitivity (87%) and specificity (97%).

-Management: conservative and OPC, but if RMI is high → refer to specialist, laparoscopic cystectomy for histologic evaluation.

-Aspiration of fluid is controversial, because possibility of spreading malignant cells to peritoneal cavity.

***Benign Neoplastic tumors*:**

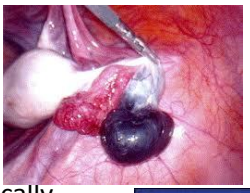
Epithelia (80%-85%, most common), stromal, and germ cell (10%-15%).

❶- **Epithelial**:

1-Serous cystadenoma: 20%-25% are malignant, depend on patient's age.

-Commonly unilocular, derived from ciliated tubal epithelium, and form **psmmoma bodies**.

-Excision is definitive, but if they are small (<6cm), conservative is reasonable.



2-Endometrioid neoplasm: -Resembles the endometrium.

3-Mucinous cystadenoma:

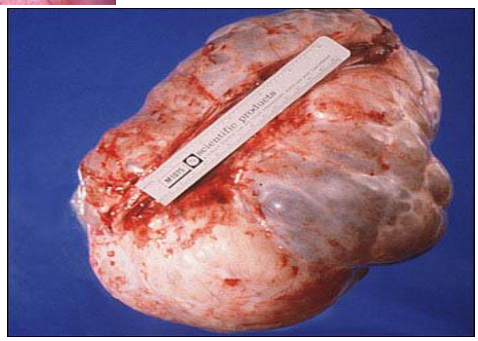
-Can attain very huge size (up to 150 pounds), histologically similar to endocervical columnar epithelium.

-Maybe is complicated by pseudomyxoma peritonei. And often associated with appendiceal mucocele.

-Generally Multiloculated, and 15% of mucinous are malignant.

-They may form a honeycomb structure with thin septa.

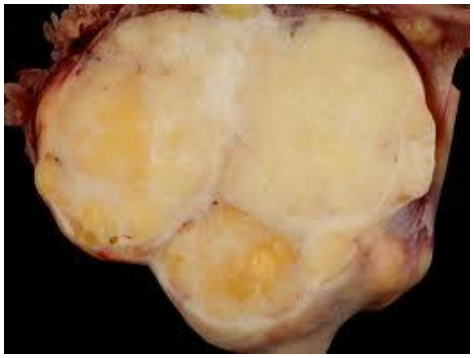
-They often need surgical excision because of their large size.



4-Transitional (Brenner) Tumors:

-Small Solid ovarian neoplasm, almost always benign, histologically encases epithelioid cells that resemble transitional cells of the bladder(urothelium).

-Because these are solid tumors, excision is indicated and curative.



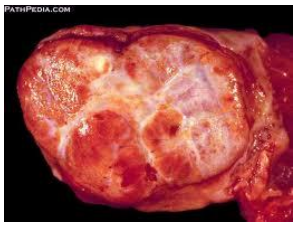
❷- **Sex cord-Stromal ovarian neoplasm**:

1-Granulosa-cell tumors, theca-cell tumors: solid-yellow appearance

-They are functioning tumors (not functional) → they produce estrogen.

-They promote feminizing sings: precocious menarche or telarche, and in reproductive years → menorrhagia, endometrial hyperplasia. And may cause endometrial cancer and breast tenderness.

-Management: 1- Unilateral salpingo-oophorectomy if future pregnancy is considered.
2- Bilateral salpingo-oophorectomy with hysterectomy.



2-Sertoli-leydig cell tumors (Hilar cell tumor MCQ): produce androgens.

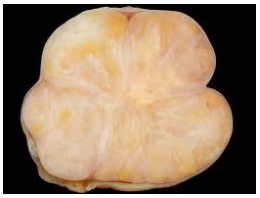
-Virializing effects like: hairsitisum, temporal baldness, deepening of voices, clitromegaly.

3-Fibroma:

-It is benign.

-Form encapsulated, solid, smooth-surfaced tumor, composed of **spindle- shaped cells.**

-**Usually asymptomatic, it is not hormonally active.**



-Meig's syndrome: -Ascites and hydrothorax associate with fibroma.
-Ascites caused by transudation of fluid from ovarian fibroid.
-Flow of ascites through transdiaphragmatics lymphatics to right plural cavity →hydrothorax.

❸- **Germ cell tumor:**

-Occur at any age, account 60% of ovarian neoplasm in infants.

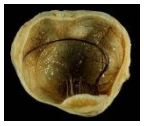
-Benign teratoma is the most common.

-Mature cystic teratoma:

-Not just the most common germ cell tumor, but also the most common ovarian neoplasm.

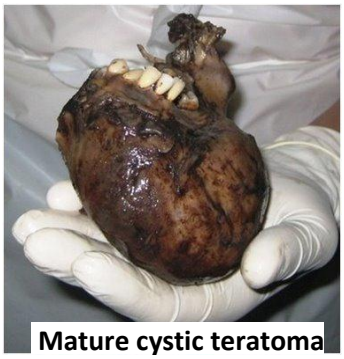
-Composed of mature adult-type tissues; known as **dermoid cysts**.

-Mature teratoma composed primarily of ectodermal tissue (hair, sweat and sebaceous gland, teeth) with some mesoderm and endoderm.



-Almost always benign.

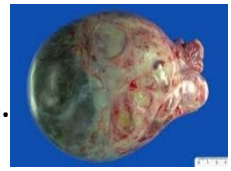
-Clinical manifestations: -Most are asymptomatic
-Symptoms depend upon the size of the mass
-Torsion is may occur
-Rupture can occur (Shock and hemorrhage)


Mature cystic teratoma

-Treatment: - Ovarian cystectomy
- Salpingo-oophorectomy

❹-**Mixed ovarian neoplasm:**

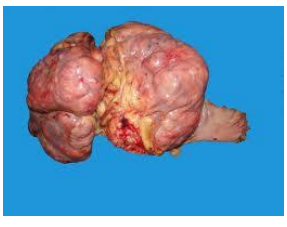
-Most common ovarian tumor that originates from more than one cell type is **a cystadenofibroma.**

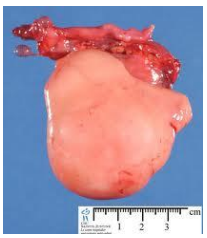


- Gonadoblastoma: here almost all patients have dysgenetic gonads, and Y chromosome has been detected in 90% of cases.

-It is benign but it may predispose to malignant dysgerminoma.

-Management: Bilateral salpingo-oophorectomy.





-Diagnosis Of benign Tumors generally: (see tables)

-Most benign tumors are asymptomatic, unless torsion or rupture occurs.

-US, serum CA-125 are helpful.

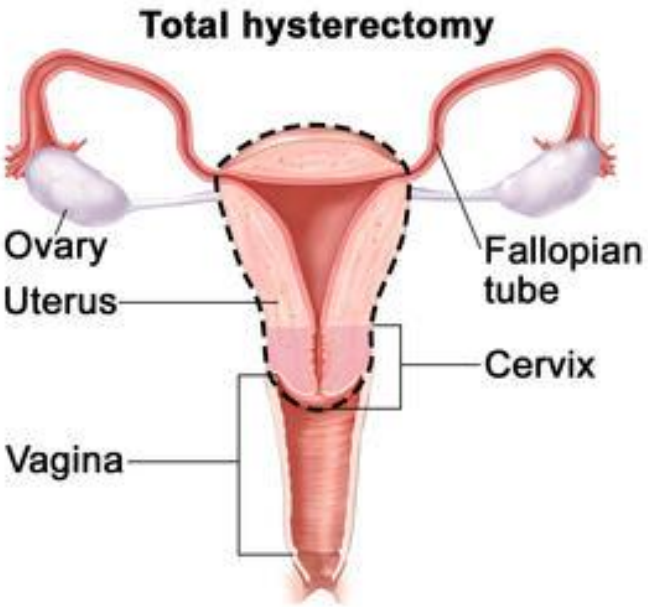
-Laparoscopy: it will not distinguish b/w functional cyst and ovarian neoplasm.

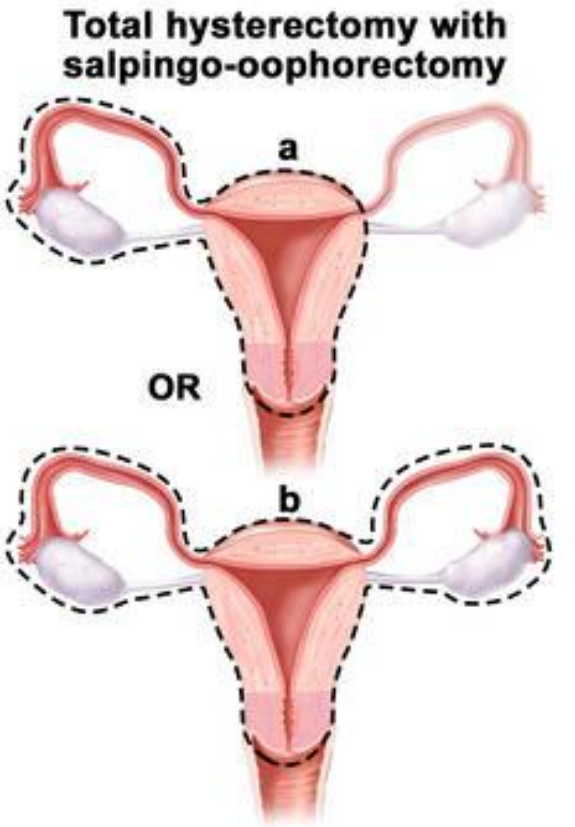
Age	Malignancy chance
Prepubertal	10%
Reproductive years	15%
Postmenopausal years	50%

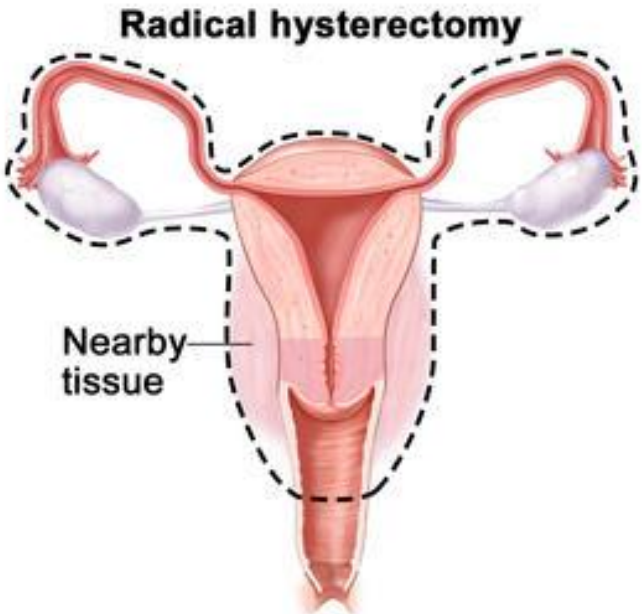
Benign tumor	Malignant tumor
-Cystic	-Solid
-Simple cyst	-Loculated cyst
-Mobile	-Fixed
-Unilateral	-Bilateral
-Small	-Large

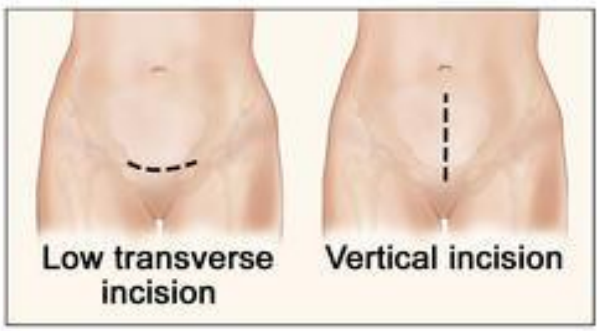
-Management of benign tumors:

-Epithelial ovarian neoplasm treated by unilateral salpingo-oophorectomy .and contralateral ovary should be inspected, and in old woman bilateral salpingo-oophorectomy with hysterectomy maybe appropriate.



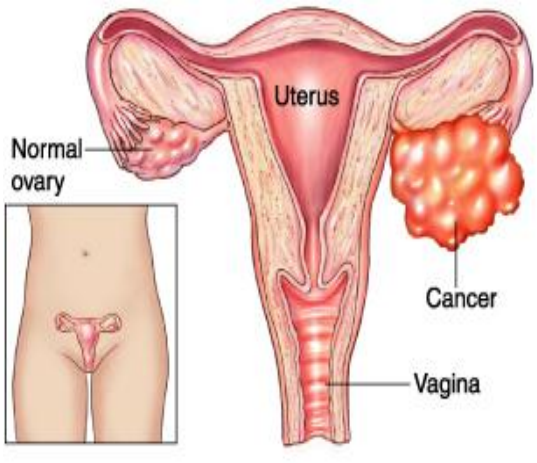




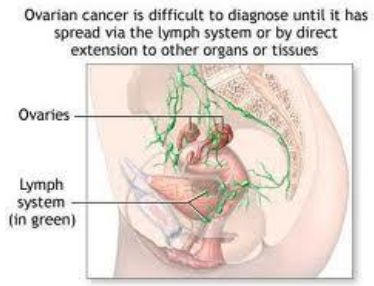


Ovarian Cancer

- It is the leading cause of death from gynecologic cancer because it is difficult to detect before it spread
- It could be arise from the surface (epithelium) of the ovary, from the egg cells (germ cell tumor) or supporting cells (stroma).
- Most ovarian cancer is **epithelial** in origin



- **Risk Factors of ovarian cancer : [LEEMON]**
  - **O**lder women
  - Race
  - Geographic locations
  - **E**arly menarche
  - **L**ate menopause
  - Family Hx of ovarian, breast, or bowel cancer
  - **N**ulliparity
  - Genetic **m**utation (*BRCA1* and *BRCA2*, but also in genes for hereditary nonpolyposis colorectal cancer [breast-ovarian cancer syndrome])
  - Postmenopausal **e**strogen replacement therapy
- **Factors that decrease the risk :**
  - Combined oral contraceptive pills
  - Tubal ligation
  - Hysterectomy
  - Salpingo-oophorectomy
  - Increase in the number of pregnancy
  - Breastfeeding
- **Screening**
  - only to women who has family history of ovarian cancer due to high false positive
  - serial transvaginal ultrasonography
  - serum CA-125 titers
- **CLINICAL FEATURES :**
  - Irregular menses if she is premenopausal
  - Symptoms of a mass compressing the bladder or rectum, such as urinary frequency or constipation
  - lower abdominal or pelvic "fullness" or of dyspareunia
  - Acute symptoms : pain secondary to torsion, rupture, or intracystic hemorrhage
  - In advanced-stage disease : abdominal pain or swelling (ascites or from tumor)
  - Decrease in weight
  - Loss of appetite
  - Signs : solid, irregular, fixed pelvic mass with or without ascites
- **Investigation and Diagnosis :**
  - Start with Hx + Ex → blood test (for CA-125 and sometimes AFP & B-HCG), and transvaginal ultrasound → Definitive diagnosis is confirmed by Biopsy (frozen section) [laparotomy or laparoscopy]
  - Routine preoperative : CBC , Liver function test , Urea and electrolyte
  - CT scan → liver metastases
  - Endometrial biopsy
  - Endoscopy in patient with blood stool
  - U/S for pelvis, kidney and liver
  - Abdominal radiograph: may be useful in a younger patient to locate calcifications (bone or teeth) associated with a benign cystic teratoma (dermoid cyst), which **is the most common neoplasm in patients younger than 25 years of age**.



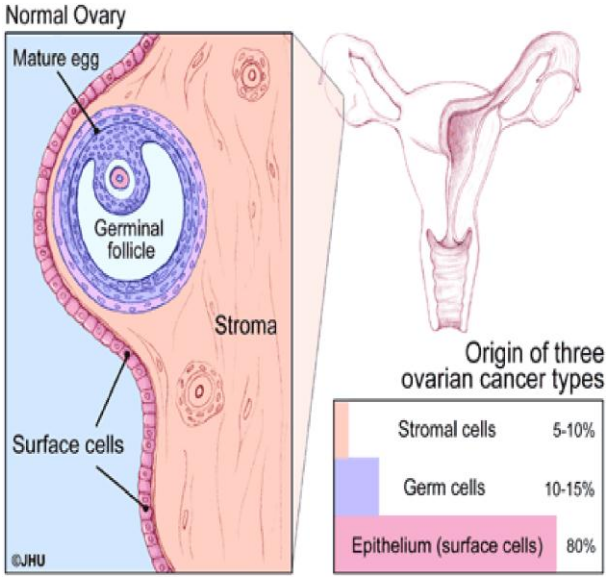
- **Metastatic ovarian spread:**
  - Direct : tubes→ uterus → bladder
  - Exfoliating along peritoneal surface [Typical pattern of spread].
  - Lymphatic spread → pelvic and para aortic lymph nodes
  - Hematogenous spread → liver- lung
- **Staging :**
- Ovarian cancer staging is by the FIGO staging system and uses information **obtained after surgery**, which can include a total abdominal hysterectomy, removal of (usually) both ovaries and fallopian tubes, (usually) the omentum, and pelvic (peritoneal) washings for cytopathology.

Medscape

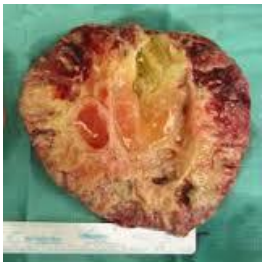
- IA: growth limited to one ovary
- IB: growth limited to both ovaries
- IC: IA or IB with capsule involvement, tumor rupture, positive washings/ascites
- IIA: extension or metastasis to uterus and/or fallopian tubes
- IIB: extension to other pelvic tissues
- IIC: IIA or IIB with capsule involvement, tumor rupture, positive washings/ascites
- IIIA: microscopic abdominal peritoneal implants
- IIIB: abdominal peritoneal implants ≤2 cm
- IIIC: abdominal peritoneal implants >2 cm, positive retroperitoneal or inguinal lymph nodes
- IV: distant metastasis (cytologic positive pleural effusiona and parenchymal liver metastasis)

Source: Expert Rev of Obstet Gynecol © 2009 Expert Reviews Ltd

- **Histological classification of ovarian Tumors :**
  - **Epithelial Tumors**
    - Serous tumor (40%)
    - Mucinous tumor (25%)
    - Endometrioid tumor (20%)
    - Clear tumor (5%)
    - Brenner (3%)
    - Mixed tumor
    - Undifferentiated and Unclassified tumor
  - **Sex-Cord Stromal tumors**
    - Granulosa –theca stromal cell tumors.
    - Sertoli–stromal cell tumors
    - Gynandroblastoma
    - Lipid cell tumor
    - Sex – cord tumor with annular tubules
    - Unclassified sex-cord tumors Steroid cell tumors
  - **Ovarian germ-cell tumors**
    - Dysgerminoma
    - Teratoma (immature, mature and monodermal)
    - Yolk sac tumor (endodermal sinus tumor)
    - Embryonal carcinoma
    - Polyembryoma
    - Choriocarcinoma
    - Mixed germ-cell tumor.



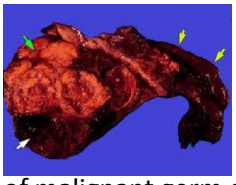
- **Epithelial tumor :**
  - Arise from surface epithelium of ovary account from 60-65 % of ovarian tumor and approximately 90% are malignant
  - common bilateral
  - Serous tumors resemble fallopian tube epithelium histologically
  - Mucinous tumors histologically resemble endocervical epithelium and are **<u>often large</u>**
  - Endometrioid tumors closely resemble carcinomas of the endometrium and arise in association with **<u>primary endometrial cancer</u>** in about 20% of patients.



  - Clear cell carcinomas:
    - Worse prognosis
    - In about 25% of cases, they occur in association with endometriosis.
  - Borderline ovarian tumor:
    - Account approximately 15% of epithelial ovarian cancer.
    - They are low malignant potential.
    - Affecting young women and may present in pregnancy
    - Microscopically they show malignant features but no stromal invasion.
    - They have good prognosis.

- **Sex cord stromal tumors:**
  - They are composed of granulosa, theca, and sertoli cells.
  - Estrogen and progesterone are typically associated with granulosa-theca cell tumors, whereas testosterone and other androgens may be secreted by many Sertoli-Leydig cell tumors
  - Most of them are benign and most clinically malignant are granulosa cell T.
  - **<u>Meigs' syndrome, is the triad of ascites, pleural effusion and benign ovarian tumor (fibroma)</u>**

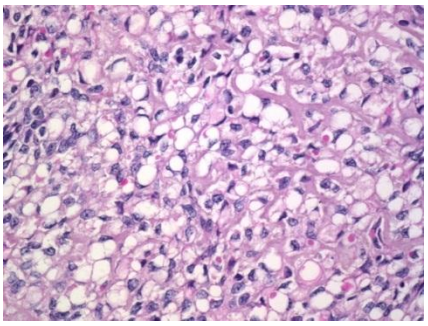
- **Germ Cell tumor:**
  - Account approximately 30% of ovarian tumor.
  - Germ cell tumor rare and they occur predominantly in young patients and frequently produce either human chorionic gonadotropin (hCG) by ovarian choriocarcinoma or α-fetoprotein (AFP) by Yolk sac tumor.



  - **Dysgerminoma :**
    - 75% present in stage I disease.
    - 10-15% Bilateral
    - Dysgerminoma is the commonest type of malignant germ cell ovarian cancer
    - 5-10% occur in female with abnormal gonads **(gonadal dysgenesis or the testicular feminization syndrome**)
    - Commonly produce lactate dehydrogenase
    - Pure dysgerminomas do not produce the tumor markers hCG and AFP.

  - **Teratomas (mature & immature) :**
    - Derived from 2 -3 embryonic layers.
    - Mature teratoma is typically benign (grade 0) and found more commonly in women, while an immature teratoma is typically malignant and is more often found in men
    - **Mature teratoma (Dermoid Cysts) : <u>Commonest ovarian tumor</u>** ,Benign , Leading to torsion , Contain teeth and hair in the cyst , Malignant transformation 2%
    - Pure immature teratomas do not produce hCG or AFP
    - Immature Teratoma 2nd commonest germ cell malignancy

- **Secondary ovarian malignancy:**
    - Account up to 10%.
    - Metastases form :
        - Colon
        - Stomach
        - Breast
        - Female genital tract
    - **The Krukenberg tumor** : specific type of metastatic tumor
        - Ovarian metastatic tumor from gastric or colon cancer.
        - Microscopic assessment shows **signet ring cells** (see figure)
        - CEA marker increase
        - Bilateral

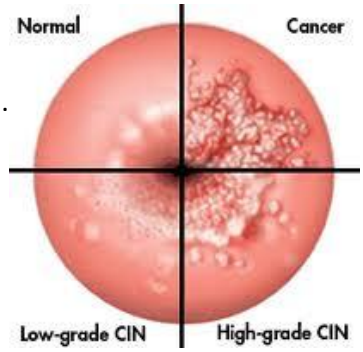


Management

1. **Initial management is staging surgery by laparotomy:**
    - Total abdominal hysterectomy, bilateral salpingo-oophorectomy, infracolic omentectomy , washing bowel
    - Young Patients who wish to preserve fertility may have a unilateral salpingo-oophorectomy →Delaying procedure until histopathology is available
    - If there is gross cancer outside the ovary → debulking (cytoreductive surgery :cutting out as much of the tumor as possible) and sampling of lymph nodes and other tissues

2. **Borderline tumor :**
    - only staging surgery
    - Young Patients who wish to preserve fertility may have a unilateral salpingo-oophorectomy
    - chemotherapy or radiation not indicated
3. **Early stage [I]**
    - confined to one or both ovary grade 1 or 2: only **staging surgery**
    - Patients with poorly differentiated (grade 3) tumors : **staging surgery + chemotherapy**
4. **Advanced-Stage Disease [II,III,IV]:**
    - **Primary cytoreductive surgery followed by chemotherapy** (carboplatin and paclitaxel) is current gold standard→ monitoring by serial CA 125 →within 12 months if there is rising or plateau → change to second line chemotherapy → progression has been greater than 12 months → paclitaxel or carboplatin chemotherapy + Secondary cytoreduction
    - **Interval debulking**
        - **Definition :** 3 cycles of chemotherapy then → surgery then → 3 more cycles of chemotherapy
        - **Indication :** medically unfit , large ascites , severe malnutrition
        - **Aims :** to improve patient condition as it to resolve the malignant effusions → after the resolve → surgery and if there is failure resolve → palliative care only (pain management like radiotherapy :Treatment by radiotherapy only for palliation)
5. **Follow up**
    - Three times monthly for one year then six monthly then yearly
    - History, examination, and CA125
    - Imaging if recurrence is suspected clinically or by CA125
6. **Additional note :**
    - Optimal cytoreduction if all macroscopic disease cannot be removed, an attempt should be made to reduce individual tumor nodules to 1 cm or less in diameter. which can be achieved in about 70% of patients (**optimal cytoreduction = tumor nodules ≤ 1 cm)**
    - Optimal cytoreduction have longer median survivals.

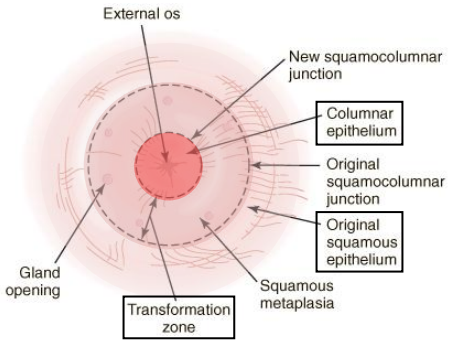
Cervical Dysplasia (CIN) & Invasive Cancer



Statistics: -Cervical cancer is the most common cause of death from cancer in women.
-Cervical cancer is the second most common malignancy in women worldwide.
-The disease is rare before 20 years and the mean age about 47 years

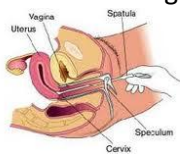
Etiology: -All cervical cancers are caused by persistent infection with a high-risk HPV.
-There are 15 high-risk HPV types.
-HPV Type 16 & 18 are responsible for 70% of cervical ca.
-HPV Type 6 & 11 are responsible for cervical condylomas and low-grade CIN.

Pathophysiology: - **See figure** for cervical topography in adolescence.
- "Squamous" metaplasia occurs normally during periods of endocrine change in adolescent cervix at the transformation zone (T-zone). It makes the cervix more susceptible to carcinogenic stimuli.
- HPV influence cellular alterations that result in an atypical T-zone. Result in CIN which is the pre-invasive phase of cervical ca.



Risk factors: # risks that are related to high-risk HPV exposure.
- Young age at first coitus (<20 yr.)
- Multiple sexual partners
- Sexual partner with multiple sexual partners
- Young age at first pregnancy
- High parity
- Lower socioeconomic status
- Smoking
- Immunodeficiency.

Prevention: -Screening: -Regular screening with Pap smears markedly decreased the incidence of the disease.



-It is recommended that all women undergo annual physical Ex. including Pap smear within 3 years of sexual intercourse, or by age 21 years.
-In Pap smear both the endocervical canal & the ectocervix should be sampled.

-Vaccinations: -Prevent effectively HPV 6, 11, 16, & 18 infections for females aged 9-45 years.

-Thus decreasing the incidence of precancerous lesions and cervical cancer.
-Most effective if given before the onset of sexual activity.
-Less effective after HPV exposure.
-After the vaccination Women should have regular cervical screening because it doesn't protect against the other high-risk HPV types.

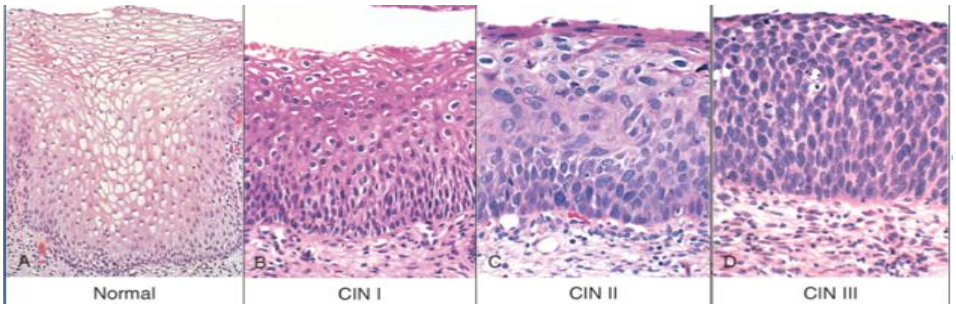
A-Cervical Intra-epithelial Neoplasia (CIN)

Definition: Abnormal epithelial proliferation and maturation <u>above</u> the basement membrane which is the pre-invasive phase of cervical ca. ranging from mild dysplasia (CIN I) to severe and carcinoma in situ (CIN III).

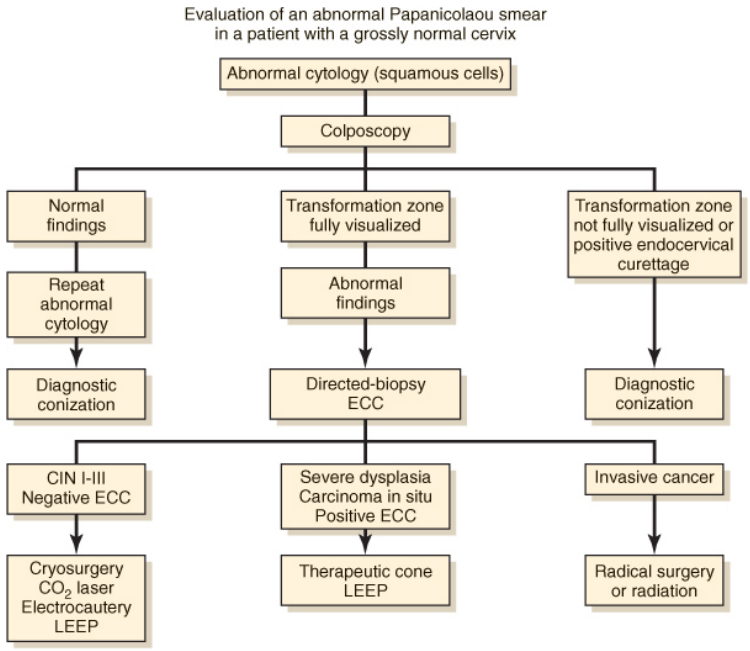
Statistics: -CIN I & CIN II often spontaneously regress.
-At least 35% of patients with CIN III develop invasive cancer within 10 years.

Grades:-CIN I = involvement of inner 1/3 of the epithelium.
-CIN II = involvement of inner 1/2 to 2/3 of the epithelium.
-CIN III = involvement of full thickness of the epithelium.

Symptoms: - None (asymptomatic).



Dx.: -Pap smear; -Is the first thing to do, then send it for cytology. **(Then see algorithm)**



-Colposcopy; - Colposcope is magnifying & has a strong light. Speculum is inserted to expose the cervix.

- In normal Colposcopy; -The original squamous epithelium (OSE) appears gray and homogeneous.

-The columnar epithelium appears red and grapelike.

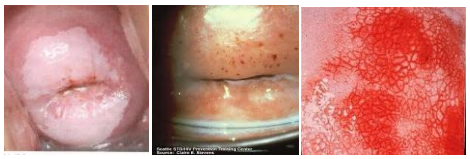
-The T-zone appears -Paler than OSE.

-Has gland openings.

-Nabothian follicles maybe seen.

-Normal blood vessels branch like a tree.

-In abnormal Colposcopy;-Hallmark of CIN is an area of sharply delineated **acetowhite epithelium** after the application of acetic acid.



-Vascular changes;-Punctation: single-looped capillaries seen as a "dot".

-Mosaicism: network of capillaries parallel to the surface.

-↑ Irregularity as the disease becomes more invasive.

-Punch biopsy; - Performed if any patient has a grossly abnormal cervix regardless of Pap smear results.

-Endocervical curettage; -Not performed in a pregnant.

-HPV testing; -Hybrid Capture assays (Digene, Silver spring, Md).

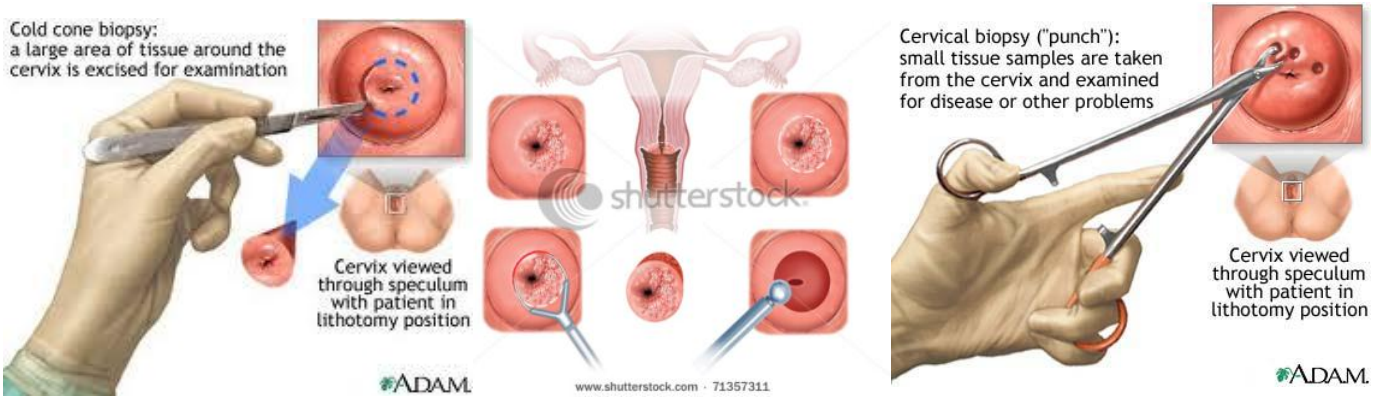
-Diagnostic cone biopsy; -Indications: -Pap smear (high grade) & Colposcopy unsatisfactory (invisible T-zone).

-Pap smear (high grade) & Punch biopsy not confirmed.

-Pap smear indicates adenocarcinoma in situ.

-Micro-invasion is present on the punch biopsy.

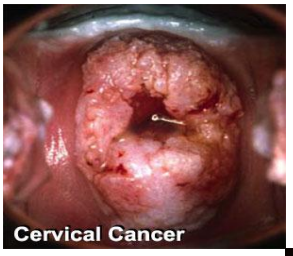
-Endocervical curettage shows a high-grade lesion.

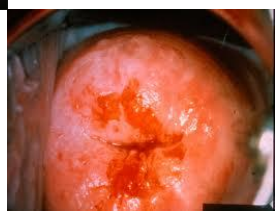


Rx.: -CIN I = Observation
-CIN II & III = If the entire T-zone is visible;
-Superficial ablation, such as large loop excision of the transformation zone (LLETZ).
-Cryosurgery.
-Co2 laser.
= If not;
- Diagnostic cone biopsy used here as a treatment.

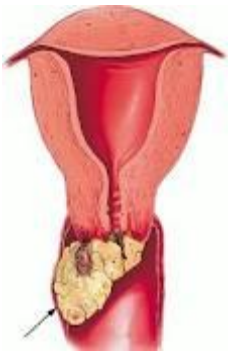
B-Invasive Cervical Cancer

Symptoms: -Postcoital, intermenstrual, or postmenopausal bleeding. They are late symptoms unlike endometrial ca.
-Persistent vaginal discharge.
-Pelvic pain
-leg swelling
-Urinary frequency
-Loss of urine or stool from vagina, because of fistula formation.





Ex.: -General Ex.; -Weight loss
-Loss of appetite
-Enlarged inguinal or supraclavicular lymph nodes
-Leg edema
-Hepatomegaly
-Pelvic Ex.; - Cervix is ulcerative or exophytic (growing outward).
-Cervix bleeds on palpation.
-Cervical serous, purulent or bloody discharge.
-Lesions involving the vagina and extending toward the introitus.
-Rectovaginal Ex.; -Essential to determine the extent of the disease.
-May found extension into uterosacral ligaments.



Pathology: -Most Cervical cancers are squamous in origin.
-Adenocarcinomas and adenosquamous carcinomas account for 20-25% of cases.
-Melanomas & sarcomas occur rarely.

Patterns of spread: -Direct invasion, to cervical stroma, corpus, vagina, and parametrium.
-Lymphatic spread, to pelvic and then para-aortic lymph nodes.
-Hematogenous spread, to lungs, liver, and bone.

Preoperative investigations: -To assess the patient's wellbeing, like CBC, Blood urea nitrogen & Creatinine levels.
-Ureteric obstruction occurs in about 30% of patients with stage III & in 50% with stage IV disease. Thus untreated patients may develop renal failure & uremia.
-Clinical staging:-FIGO staging is **based on physical examination** and noninvasive testing.
-Tests could be used in FIGO : -Biopsies
-Cystoscopy
-Sigmoidoscopy
-Chest & Skeletal radiographs.
-Intravenous pyelography.
-Liver function tests.
-Planning management: Abdominal & Pelvic radiographs.
-Hypercalcemia may donate bone metastasis.

FIGO Staging: =Stage I : confined to the cervix.

-Stage Ia: seen only microscopically.

_Stage Ia1: Stroma invasion is ≤ 3mm in depth & ≤ 7mm in width.

_Stage Ia2: Stroma invasion is ≤ 5mm in depth & ≤ 7mm in width.

-Stage Ib: seen grossly.

_Stage Ib1: Lesion is ≤ 4cm in size.

_Stage Ib2: Lesion is > 4cm in size.

=Stage II: Extended beyond cervix but didn't reach to pelvic wall or lower 1/3 of vagina.

-Stage IIa: No parametrial involvement.

-Stage IIb: Present parametrial involvement.

=Stage III: Extended to the pelvic wall or reached the lower 1/3 of vagina.

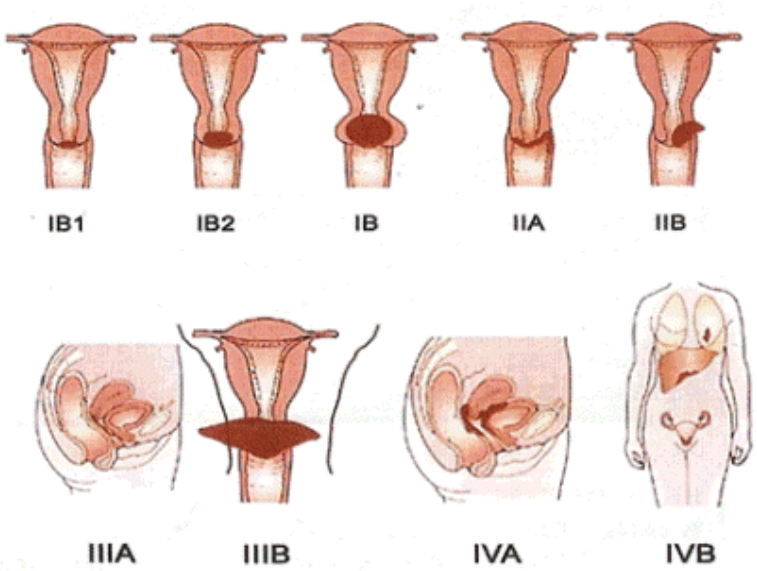
-Stage IIIa: Just reached the lower 1/3 of vagina.

-Stage IIIb: Extended to the pelvic wall +/- hydronephrosis or nonfunctioning kidney.

=Stage IV: Extended beyond true pelvis or has involved the bladder or rectum.

-Stage IVa: Spread to adjacent organs.

-Stage IVb: Spread to distant organs.



Rx: Stage Ia = Cervical conization

= Hysterectomy if family completed.

Stage Ib + IIa = Primary surgery (radical hysterectomy & bilateral pelvic lymph adenectomy) **or** Chemoradiation.

Stage IIb + III + IV = Chemoradiation (Brachytherapy).

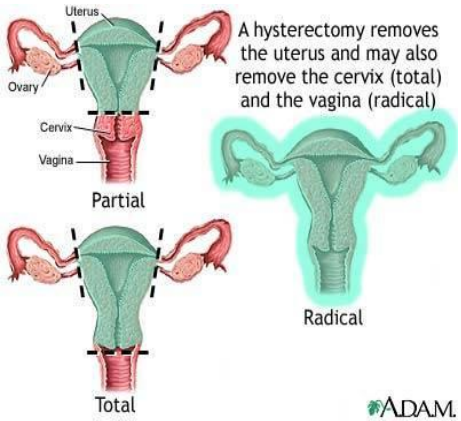
#NOTE; Stage IIa is the last stage that can be treated surgically.

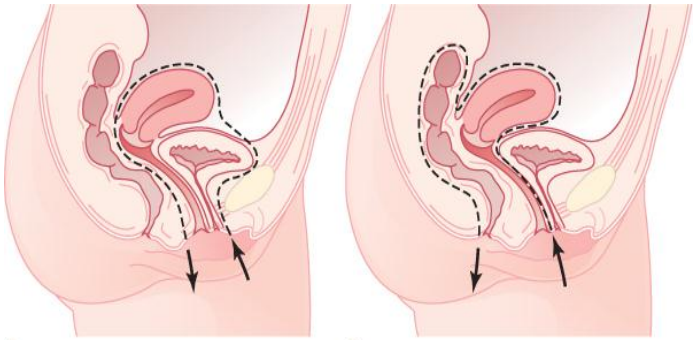
Stage IVa chemoradiation, if results in only partial tumor regression, do pelvic exenteration.

Stage IVb chemoradiation is just palliative to ↓ bleeding.

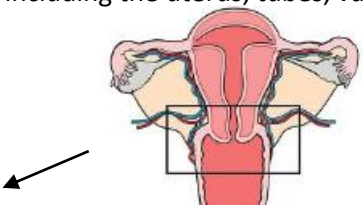
Radical hysterectomy: - the uterus is removed along with adjacent portions of the vagina, cardinal ligaments, uterosacral ligaments, and bladder pillars

- The most common complication is bladder dysfunction.
- The most serious complication is ureteric fistula or stricture, which occurs in 1% to 2% of cases.





Pelvic exenteration: -Total exenteration involves removal of the pelvic viscera, including the uterus, tubes, vagina, ovaries, bladder, and rectum.
-Following the surgery, pelvic reconstruction is necessary.



Radical trachelectomy: -Used for young women with early cancer (≤2cm in diameter). It allows fertility preservation.

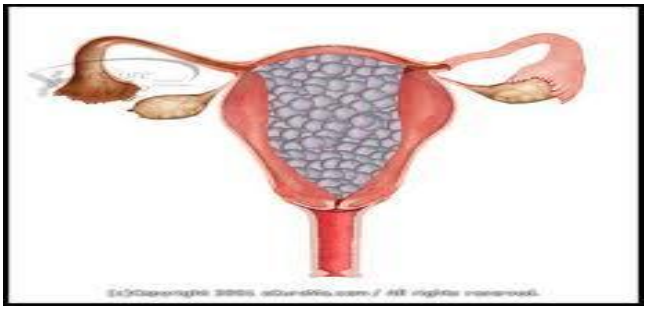
Chemoradiation Therapy: -Attempt to shrink the tumor and improve therapy with the addition of cisplatin.

Cervical carcinoma in pregnancy: - Diagnosis during pregnancy or 6 months postpartum.
-Symptoms: -Painless vaginal bleeding.
-Dx.: -Pap smear in antenatal care.
-Cone biopsy could be performed in the 2nd trimester.
-Management: - CIN I-III = conservatively until 6 weeks postpartum.
- Invasive cancer = if after 22-26 weeks, continue pregnancy.
= if less, start radiation therapy. Abortion usually occurs spontaneously during therapy.

Prognosis: <u>Five-year survival;</u> -Stage I = 79-99%
-Stage II= 65-69%
-Stage III= 40-43%
-Stage IV= 15-20%

Gestational Trophoblastic Neoplasia

- **Gestational trophoblastic disease (GTD)** is the term for uterine tumors that develop from the trophoblast.
- **Types of GTN**



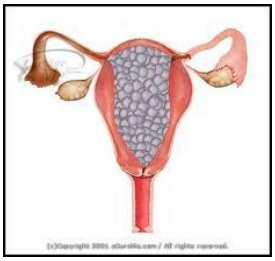
1. BENIGN :
    - Hydatidiform moles (Vesicular Mole)
2. MALIGNANT:
    - Invasive mole
        - usually confined to uterus
    - Choriocarcinoma
        - usually with extra uterine spread
        - It can develop from a hydatidiform mole, or it can occur after a full-term pregnancy or abortion
    - Placental site trophoblastic tumor
    - Epithelioid trophoblastic tumor

Hydatidiform moles (molar pregnancy)

- **Molar pregnancy :** is an abnormal form of pregnancy wherein a non-viable, fertilized egg implants in the uterus, and thereby converts normal pregnancy processes into pathological ones characterized histologically by:
    - Trophoblastic proliferation
    - Edema of the villous stroma (Hydropic)
- **Molar pregnancy is categorized into : (see table)**
    - Complete moles (common)
    - Partial mole

	Complete (classical)	Incomplete (partial)
Karyotype	Paternally derived euploidy 46 XX > 46 XY	Triploidy or quadraploid 69 XXY (80%) 69 XXX (20%)
Mechanism	1 or 2 sperms fertilize empty ovum	1 or 2 sperms fertilize X ovum
Fetus present	No	Yes
Vessels villii	Absent	Normal
Edema of villii	Yes	Some
Trophoblastic cells	Proliferative	Atrophic
β-hCG level	Very very high	Very high
Malignancy potential	20%	5%

- **Complete mole**
    - is caused by a single (90%) or two (10%) sperm combining with an egg which **has lost its DNA** (the sperm then reduplicates forming a "complete" 46 chromosome set)
    - The genotype is typically 46,XX (diploid) due to subsequent mitosis of the fertilizing sperm, but can also be 46,XY (diploid)
    - 46,YY has never been observed
    - Microscopically Enlarged, edematous villi and abnormal trophoblastic proliferation that diffusely involve the entire placenta
    - No fetal or embryonic tissue are produced
    - Uterine enlargement in excess of gestational age.
    - Theca-lutein cyst associated in 30%
    - 20 % → become malignant (GTT gestational trophoblastic tumor)



- **Partial mole :**
    - Occurs when an egg is fertilized by two sperm or by one sperm which reduplicates itself yielding the genotypes of 69, XXY (triploid) or 92, XXXY (quadraploid).
    - In this type the mole may be **combined with a fetus** But the fetus usually don't survive.
    - Microscopically: The enlarged, edematous villi and abnormal trophoblastic proliferation are slight and focal and did not involve the entire villi.

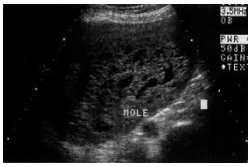
- Uterine enlargement in excess of gestational age is uncommon.
- Theca-lutein cysts are rare
- 5% → become malignant.

- **Presentation :**

1. Painless irregular vaginal bleeding in 16-20 week → 85%
2. Passing tissue that looks like grapes
3. The uterus may be larger than expected →40%
4. The ovaries may be enlarged →30%
5. Hyperemesis→because of the increased HCG→10%
6. Signs of hyperthyroidism→ nervousness, tachycardia, and sweating
7. Early onset pre-eclampsia may accompanies molar pregnancy→ 1%

- **Diagnosis :**

1. U/S : the mole resembles a bunch of grapes ("cluster of grapes" or "honeycombed uterus" or "snow-storm"
2. β-hCG levels > 2 multiples of the median may be of value in the diagnosis (It is more important in the follow up than in diagnosis)
3. Biopsy : the definitive diagnosis
4. CXR: for detecting any metastasis to the lungs



- **Management :**

1. Start with the A B C and treat the hyperemesis with hydration and stabilize the vital signs
2. Uterine **<u>suction</u>** and curettage as soon as possible after diagnosis, in order to avoid the risks of choriocarcinoma.(oxytocin can be given IV to contract the uterus and close the large venous sinuses)
3. Hysterectomy: is an opinion in women who have completed child bearing or old(and have a higher risk of malignancy)
4. Invasive mole or metastatic moles (cancer) may require chemotherapy and often respond well to methotrexate or actinomycin-D (what is the indication of chemotherapy? [osce])
5. Follow up : obtain weekly HCG titers until they're negative for 3 weeks then monthly until they're negative for 12 month
6. Contraception are recommended to avoid pregnancy for at least 6 to 12 months.

- **Prognosis :**
- The prognosis is very good and excellent (nearly 100% cure)
- 90% of women with malignant, non-spreading cancer are able to survive and retain their ability to conceive and bear children.
- Metastatic cancer, remission remains at 75 to 85%, although their childbearing ability is usually lost.
- In 10 to 15% of cases, hydatidiform moles may develop into invasive moles. This condition is named **<u>persistent trophoblastic disease</u>** (PTD). The moles may intrude so far into the uterine wall that hemorrhage or other complications develop.
- **Risk factors that associated with poor prognosis**: high levels of hCG (>40,000 mIU/mL) at the onset of therapy (not at diagnosis), late initiation of therapy (> 4 months), metastatic cancer (brain, liver), choriocarcinoma after full-term delivery.
- **Risk Factors of GTN : (see table)**

Non-metastatic risks	Metastatic risks
-Hx of similar problem. -Family Hx. -Maternal age : <20y. (2fold) and >40y. (10fold). -Blood group: type A women impregnated by type O men. (10 fold to have choriocarcinoma). -Vitamin A deficiency (low β-carotene). -Folate deficiency. -Race→ more common in south east Asian. -Contraception: COC double the incidence. -Hx of spontaneous abortion: double the incidence	-Large uterus. -High β-hCG titer. -Low gestational age. -Presence of theca lutein cyst.