Obstetrics and Gynecology Team

Team Leader SaraAlhaddab

From slides | Doctor's Notes | Team's Notes | From the book | Important

Special tanks to Abrar Allafi for her major contribution in summarizing some lectures.

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Contents

Obstetrics

Gynecology

Anatomy + Placenta +Fetal circulation.

Aims

- To fully understand the anatomy of the female pelvis in terms of bones and tissues, and fetal skull, this would help in explaining the mechanism of Labor
- To predict and thus prevent postpartumhemorrhage related to the placenta
- To understand the major events in fetal circulation; during pregnancy and after birth

Objectives

Student at the end of session should be able to:

- Explain the relationship between pelvic organs
- Comprehend the normal organs
- Understand the relationship between the female pelvis (Bones& Soft Tissue) and fetal skull, in order to understand the mechanism of labor
- Understand the major variant in the fetal circulation than that of the adult
- Know the significance of ductus venousus, ductus anteriousus and the first breath.
- Explain the changes that occur after birth.
- Familiarize yourself with the placental structure.
- Know the significance of placental and umbilical cord inspection after birth
- Differentiate between the different types of placental abnormalities and their significance

Anatomy of the Female Pelvic Organs

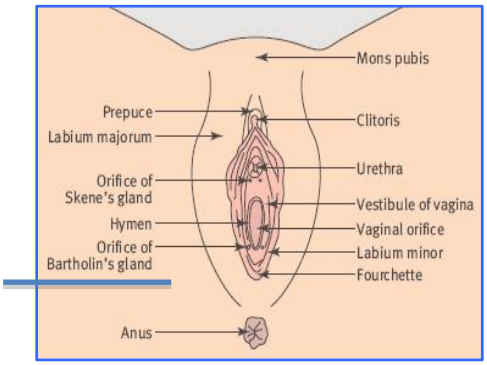
1- Vulva

- **External organs of the female include:**
  1-Mons veneris(Mons pubis) 2-Labia majora 3-Labia minora
  4-The clitoris 5-**The vestibule**
- **The vestibule has six openings:**
  1-Urethral meatus2-Two skene's ducts
  3-Vaginal orifice4-Two Bartholin ducts.
- **Bartholin glands:**
  Lies on each side of the vagina, in the posterior lower third (1/3) of the interiotus.
  Secrete mucus – **alkaline**

Vaginal duct is very important b/c when the duct becomes obstructed, the gland will become large and the woman will present with discomfort, but later on, the large gland will become inflamed then the woman will come to you with pain!

You must know the anatomy of the glands to differentiate them form any abnormal swellings.
And in case of (episiotomy= a man made cut) you must know the anatomy of the vulva to know where to cut.

Blood supply	-Pudendal artery from the femoral **aa** -Venous drainage in the corresponding vein
Lymphatic drainage	-Inguinal gland -External iliac glands
Nerves	-Branches of the pudendal nerve, perineal nerve (T12 L1-2, S2-4)

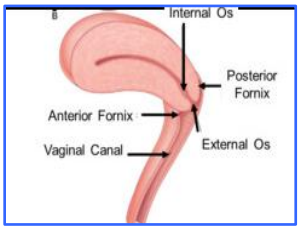


- **In labor:**
  Catheterization, Episiotomy, Anesthetic infiltration

If a woman comes to you in gyn/obstetric, you may end up with doing urethral catheterization to empty the bladder, so in labor, you have to know where is the urethral meatus to insert the cath. but not every woman in labor will end up with catheterization, but in gynecology, you must empty the bladder before starting any procedure.

2- Vagina

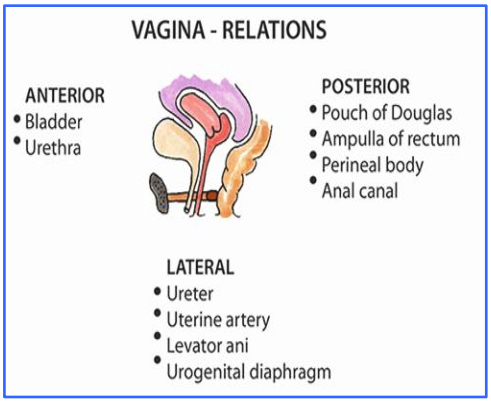
- A Canal/tube extend from the vulva to the uterus, runs upwards and backwards, and the walls lie in close contact, easily separated.
- The posterior vaginal wall is longer than the anterior 11.5 cm (4.5 in) vs 7.5 cm
- Cervix enters the vagina at a right angle.
- **Fornices:**they are four →1Anterior, 1posterior,2lateral
- **Relatins:**



Speculum examination

The vagina is closed, so you cannot inspect the vagina except by using an instrument, which is called (speculum). But you have to be careful when you insert the speculum and do not hurt the woman b/c if you hurt the woman, she will hate you and never come back to you!

Anterior	-Base of the bladder on upper ½ of vagina.
Posterior	-Pouch of Douglas in the lower ½ -Rectum centrally -Perineal body inferiorly
Lateral	**-Ureter** **-Utrine artery** **-Levator ani** **-Urogenital diaphragm**



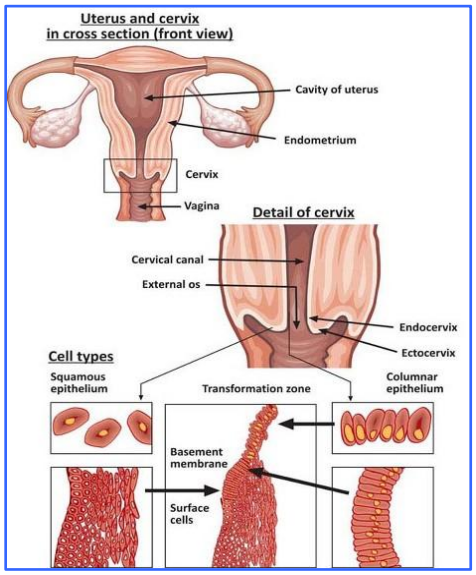
Blood supply	-Vaginal aa, uterine aa, middle haemorridal, inferior vesical, pudendal branch of the internal iliac aa. -Venous drainage to corresponding veins
Lymphatic drainage	-Inguinal, internal iliac, sacral glands
Nerves	-Symphatetic and parasymphatetic

3- Cervix

- Forms the lower 1/3 of the uterus, it enter the vagina at a right angle
- Barrel shape, 2.5cm (1 in) long
- The cervix is made up of fibrous and elastic tissue.
- **It consists of two parts:**
  - A-Supra-vaginal
  - B-Intra-vaginal
- **Transformation zone (**squamous-columnar junction):
  It is the junction between these 2 parts

You can also inspect the cervix by using the speculum. And at the end of your Ex, you have to write:

(The vulva in normal, the vagina is normal, and the cervix is normal).



You can protect the women form having cervical cancer by doing a screening test "**Pap smear",** cells form the squamous-columnar junction (squamous cells from the intra-vaginal part and columnar calls form supra-vaginal part)are gently scraped. Then the sample of cells is sent to a lab for examination.

- **The cervix has two cervical os:(2 openings)**
  - A-Internal os:
    (You cannot visualize itand you cannot enter your finger in it unless the woman is in labor or in a process of miscarriage/abortion). The internal opening is just like a tip of your pin in case of Nulliparous woman (woman who've never given birth), but in case of woman who had babies previously , it is like a slit. It helps in medico-legal cases, for example, when a woman had a baby and she denies it, you can tell!
  - B-External os:
    (You can visualize it)
- **Cervical canal** is between the internal os and the external os

Blood supply	- Uterine aa
Lymphatic drainage	-Internal iliac, sacral glands
Supports	- Cardinal ligaments - Pubocervical ligaments - Uterosacral ligaments

In early pregnancy and in non-pregnant state, the cervix is very hard and it's just like a carrot, but in late pregnancy, it will become softer.

The bluish color of the cervix helps in medico-legal cases when you want to know if the woman is pregnant but you're not having facilities to help you!

In pregnancy	- Rich blood supply – **bluish coloration** - Soft - Cervical glands – mucus plug "operculum**" This tells you that the cervix will dilate and it's very protective b/c it prevents infection**
Late in pregnancy	- Softer and starts to dilate.
In labor	- The longitudinal fibers of the uterus contract and retract pulling upward thus reducing the length of the cervix. -Full dilatation marks the end of the first stage of labor

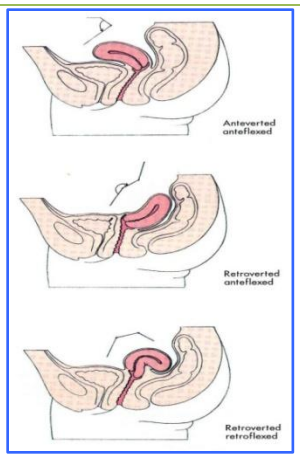
4- Uterus

- The uterus lies in the true pelvis.
- Anteverted (A/V)and anteflexed (A/F)in position.
- The body of the uterus lies above the bladder.
- **Size:** 7.5 cm length, 5 cm wide, 2.5 cm thick, 50 -75 gm weight.
- **Gross structure:**
  1- The cervix lower 1/3
  2- The isthmus,The cavity
  3- The corpus
  4- The cornua(at the level of fallopian tube)
  5- The fundus
- **Layers:**
  1-**Endometrium**
  (is the one that changes in menstrual cycles and sheds during menstruation)
  2-**Myometrium**(3 layers)
  3- **Perimetrium** (peritoneum)
  Adherent, where?**At the posterior part of the uterus.**
  It's**Loose**at the anterior part of the uterus, this lets the bladder to extend and expand. And the loose part helps us at the time of delivery by abdominal rote (C-section), so you can open the loose peritoneum and push the bladder and not let the bladder to be injured then open the uterus at the lower segment.

It is a mobile structure, if it is fixed; this is considered "pathology"
Q- what can make it fixed? Adhesions form a pelvic surgery, pelvis inflammatory disease (PID), endometriosis or cancers.
- Most of the female population are having Anteverted uterus, but 20% are having retroverted.

-You have to say five things when you examine the uterus:
1- position?4- mobile (normal) or fixed?
2- tender or no tenderness (normal) ? 5- size, Is it normal or not?
3- consistency, Is it firm or soft?

-In late pregnancy, the uterus is divided into 2 parts the upper and lower segments and you need to know them when you deliver the baby by abdominal rote.



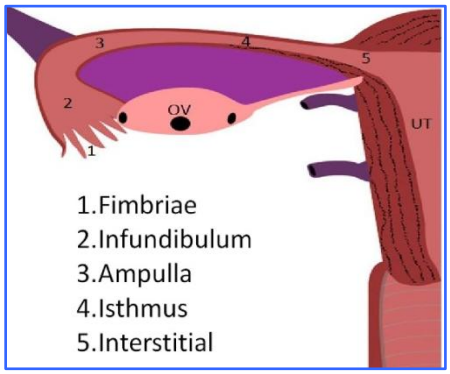
Blood supply	**- Arteries:** Fundus → Ovarian artery (aa) Body → Uterine aa, directlty from internal iliac (aa) **- The relationship between the ureter and uterine aa** Uterine aa runs behind the peritonieum, cross transeverse cervical ligament (Cardinal ligament) then the aa pass anterior to and above the uerter 1.5cm from lateral vaginal wall fornix **- Venous:** Right ovarian vein and inferior vena cava
Lymph	-Left ovarian vein -Renal vein -Internal and external iliac gland -Inguinal /Sacral gland

5- Fallopian tubes

- Extend from the cornua of the uterus, travels towards the sidewalls of the pelvis. Then turns downwards and backwards.
- The tube lies in the upper margin of the broad ligaments.
- **Communicate**: superiorly with the **uterine cavity**, Inferiorly with the **perineal cavity**
- **Length:**10 cm (4cm) **Thickness:**3 mm

It's important to know the anatomy of the fallopian tube b/c of Ectopic pregnancy and PID (pelvic inflammatory diseases)

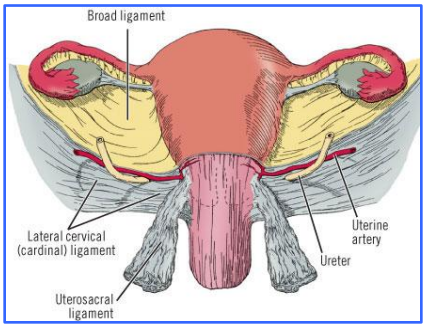
- **It consists of 4 PARTS:**(the parts are imp b/c the presentations are different in case of ectopic pregnancies)
  1. Interstitial
  2. **Ampulla**(it is the widest part,the ectopic pregnancy in this part will take up to 8 weeks!)
  3. Infundibulum
  4. Fimbriae
  5. Isthmus



Blood supply	-Ovarian aa - Uterine aa -Venous drainage is by corresponding veins

6- Ovaries(normally they are not palpable)

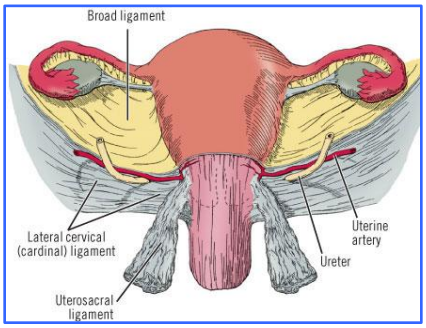
- Lie in the posterior wall of the broad ligament at the fiberal end of the fallopian tubes at the level of the pelvic brim.
- **Size:** almond like = 3 x 2 x 1 cm
- Dull white color, Corrugated surface ***(b/c of ovulation)***
- Structure varies with woman's age.



Blood supply	- Ovarian aa - Ovarian vein
Lymphatic	-Lumbar lymph nodes(pelvic and para-aortic nodes)
Nerves	- Ovarian plexus

- **Supports**: They lie in a fossa
  Attached to broad ligament – meso ovarian
  The meso salpinx is the broad ligament that extends between the fallopian tube and the ovary.
- **The Fallopion tubes, ovaries and broad ligments are called**Adnexa

7-Ligaments



1. **Round ligaments**

Maintains uterus in A/V + A/F

From the cornua of the uterus pass downwards and insert in the tissue of the labia majora.

2. **Broad ligaments**

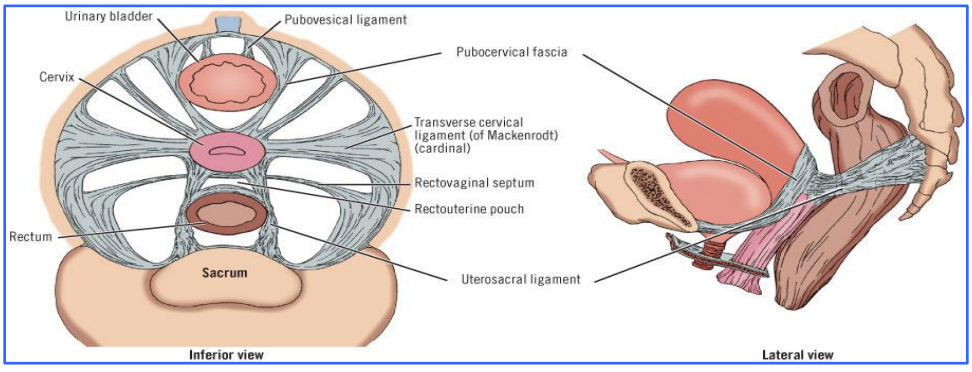
Not true ligament

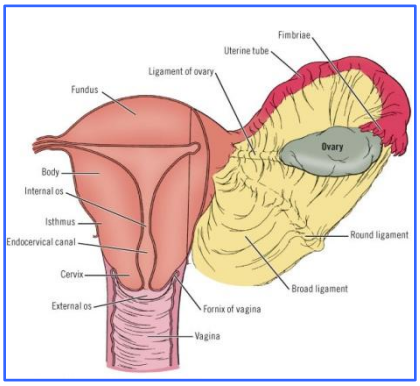
Folds of peritoneum extend laterally from the uterus to the pelvic sidewalls.

3. **Cardinal(transverse) ligaments**

4. **Pubocervicalligaments**

5. **Uterosacralligaments**



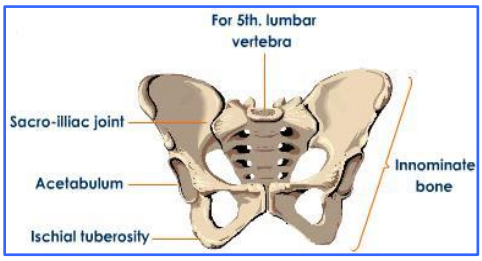


Normal Female Pelvis

- The pelvis articulates with the fifth lumbar vertebra above and with the head of each femur in the right and left acetabulum.
- **The most common female pelvic shapes is Gynecoid.**
- The weight of the trunk is transmitted through the pelvis into the legs.
- Gives protection to the pelvic organs.
- The pelvis is the largest bone in the body.
- **Gross structure**

It Consists of:

- 5 fused sacral vertebrae
- Coccyx
- Left & right innominate bones
- 4 pairs of holes
- Nerves, blood vessels, lymph

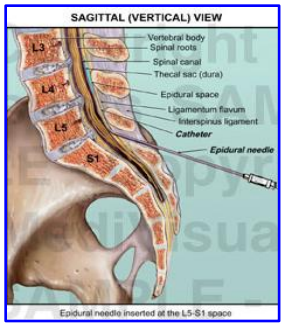


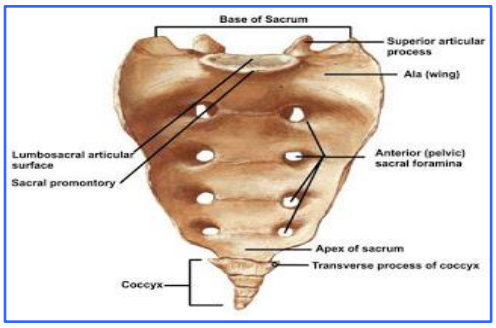
1- Sacrum

- 5 fused sacral vertebrae
- A triangular shape
- The hollow of the sacrum – smooth and concave
- The alaes of the sacrum - give the appearance of wings
- **The sacral promontory** is the center point of the upper border of the first sacral vertebrae.
- The sacral canal opens at the level of 5th sacral vertebra, a passage for spinal cord.
- At the level of the 2nd and 3rd sacral vertebrae, the nerves spread out to form the **cauda equina**.
- Anesthesia in labor!

Based on the shape of the sacrum, you can assess (if the woman can deliver vaginally or not).

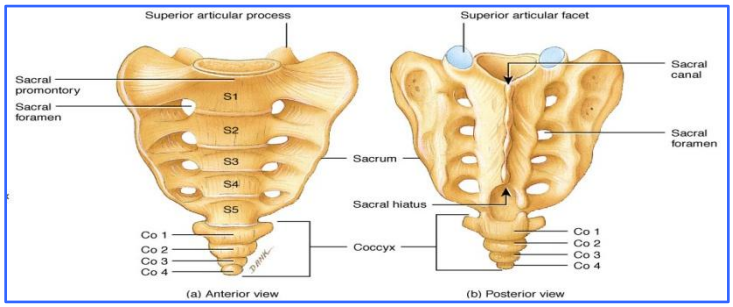
If a pregnant woman comes to me to assess her pelvis before labor, I would like to know from her sacrum if this woman can deliver vaginally or not, but this is only assessment!!! b/c you can't say 100% this woman cannot deliver vaginally. The best trial for this woman is that she should go into normal delivery (this is the real test) you should give her a chance!





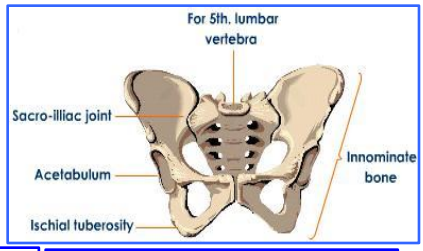
2- Coccyx

- 4 Fused coccygeal vertebrae.
- Triangular shape.
- Articulate with the sacrum.
- Muscles are attached to its tip.



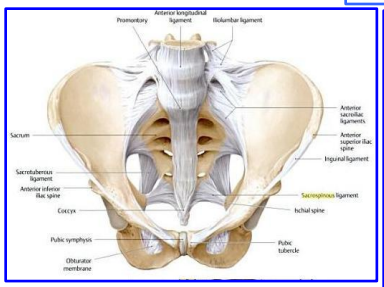
3-Right &Left in-nominate bones

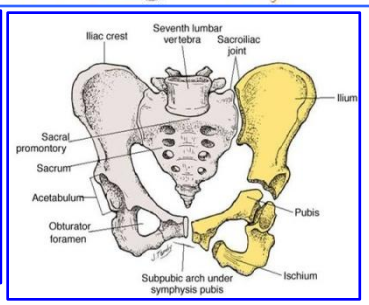
- **Each made of 3 separate parts meet in the acetabulum.**
- **The 3 parts are:**
  - **1-Ilium**→upper part is iliac crest (anterior and posterior, superior iliac crest)
  - **2-Ischium**→ ischial tuberosity, 2 cm above is the ischial spines.
  - **3-Pubis**→ both meet the pubic body fused by cartilage "symphysis pubis"



- **Pelvic joints:**

1-The two sacroiliac joints
2-The symphysis pubis
3-The sacro-coccygeal joints

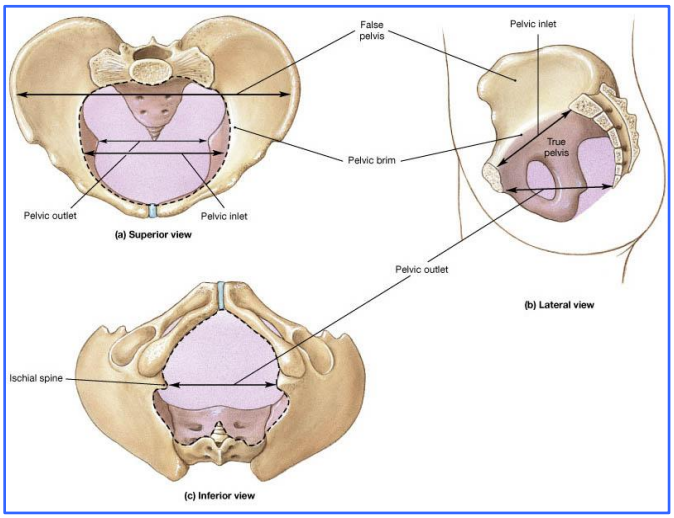




- **The pelvic ligaments:**
  1. Sacroiliac ligament →strongest in the body.
  2. Sacrotuberous-from sacrum–> ischial tuberosities
  3. Sacrospinous-from sacrum–> ischial spine
  4. Inguinal ligament.

Divisions of the pelvis

- The brim divides the pelvis into the parts:
  - The false: lies above the pelvic brim not important in obstetrics
  - The true: lies below the pelvic brim.
- It has a brim (inlet), a cavity, and outlet
  - Forms the curved canal through which the fetus passes during labor.



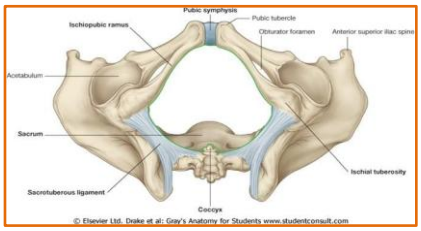
- The brim or inlet:
  - Round in shape, and has eight points as demonstrated
  - Bounded anteriorly by the pubis, laterally by illiopectineal lines, and posteriorly by ale and sacral promentary
  - Widest diameter is**Transverse**
  - True Conjugate (Antero-posterior diameter) from sacral promentary to upper inner border of Symphysis pubis
  - Diagonal Conjugate

- The Pelvic cavity:
  - Extend from the brim above to the pelvic outlet below
  - The posterior wall 11 cm formed by hollow of the sacrum and the anterior wall is formed by the symphysis pubis and obturator foramen 3.8 cm
  - The lateral walls sacrosciatic ligament and ischial spines
  - Interspines Diameter

- The pelvic outlet:

It has:

1. Anatomical outlet
   It is formed by fixed pointes, useful landmarks for taking pelvic measurement:
   - Bounded anteriorly by pubic Arch
   - Laterally by sacro-sciatic lig&Ischail Tuberosity
   - Posteriorly by tip of Coccyx
2. Obstetrical outlet
   The landmarks are:
   - The lower border of the symphysis pubis
   - The ischial spines
   - The sacro-spinous ligament
   - The lower border of the sacrum.

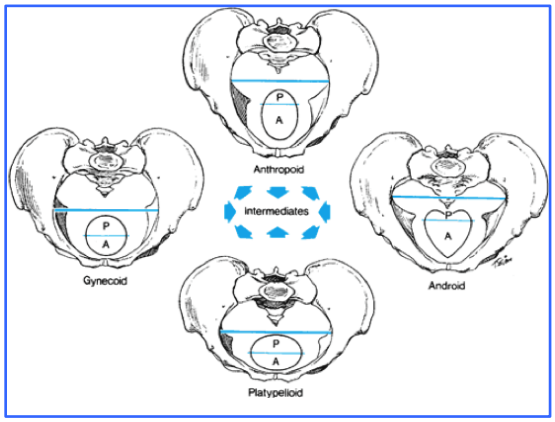


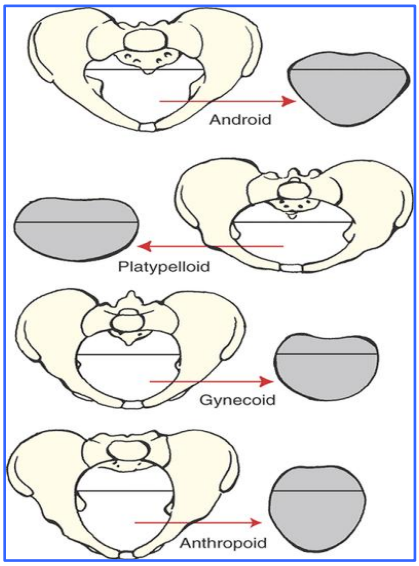
Average measurements of pelvis

- Brim (inlet):
  - Antero-posterior = 11.5 cm
  - Transverse = 13.0 cm
- Cavity:
  - Antero-posterior = 12.0 cm
  - Transverse (I/S) = 10.5 cm
- Outlet:
  - Antero-posterior = 12.5 cm
  - Transverse = 11.0 cm

Types of Female Pelvis

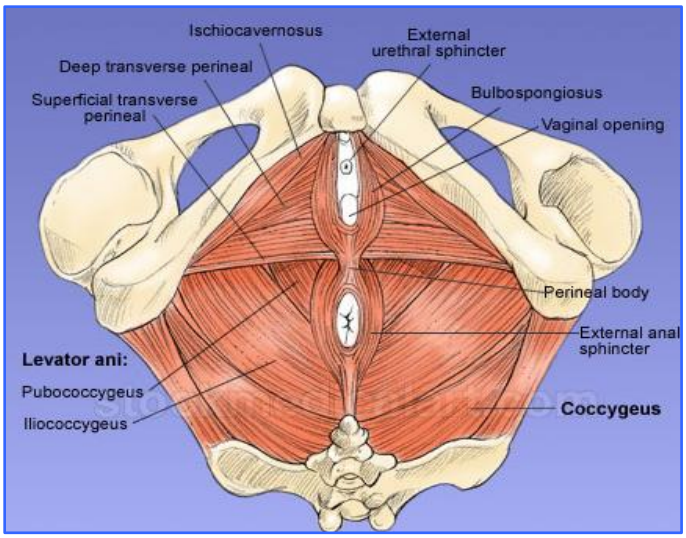
- There are four types of female pelvis shapes:
  1. Gynecoid Pelvis 50% →
  2. Anthropoid 25%
  3. Android Pelvis 20%
  4. Platypelloid (flat)5%





4- Pelvicfloor

- The outlet of the pelvis is filled with a soft tissue that supports the pelvic and abdominal organs.
- It forms as a gutler-shaped structure, and it is higher anteriorly than posteriorly.
- Three canals with external orifices run through the tissue:
  1. The urethra
  2. The vagina
  3. The rectum



- There are six layers of tissue:
  1. An outer covering of skin
  2. Subcutaneous fat
  3. Superficial muscles enclosed in fascia
  4. Deep muscles enclosed in fascia
  5. Pelvic fascia, thickened to form pelvic ligaments
  6. Peritoneum

- Superficial muscles:
  1. Transverse perinei
  2. Bulbo-cavernosus
  3. Ischio-cavernosus

- Deep muscles:
  - Three pairs of muscles all have their insertion around the coccyx
  - Their anatomical name is levator ani muscles, 5 mm thick
  1. Ilio coccygeus
  2. ischio coccygeus
  3. pubo- coccygeus

5-Perinea body

- Lies between the vaginal and rectal canals
- It is triangular, the base is the skin and the apex pointing upward each side is 3.8 cm in length
- Three layers of tissue:
  1. Outer covering of skin
  2. Superficial pelvic floor:
     - Bubo-cavernous
     - Transverse perinei
  3. Deep pelvic floor muscle.

- Episiotomy, types? indications? "Read about them"

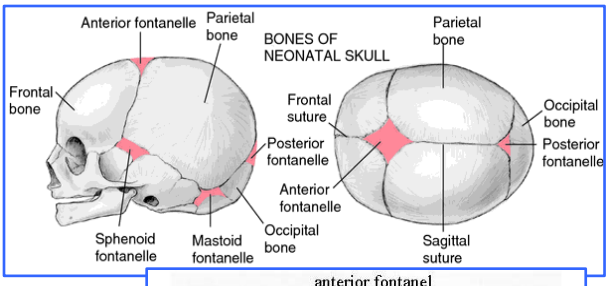
Fetal Skull

- Vault – formed from membrane and not cartilage
- There are 5 points – ossification centers
- Calcification begins as early as 5 weeks after conception
- Premature baby is born with a risk ofintracranial damage
- Fetal skull is divided into regions:
  1-The vault (cranium)
  It extends from the orbital ridges to the nape of the neckand it consists of: occipital bone, parietal bones, temporal bones, and frontal bones.
  2-The face.
  3-The base

Anencephaly is a neural tube defect characterized by **absence** of the cranial **vault** and cerebral hemispheres. And it can be diagnosed by**ultrasound in week 12**

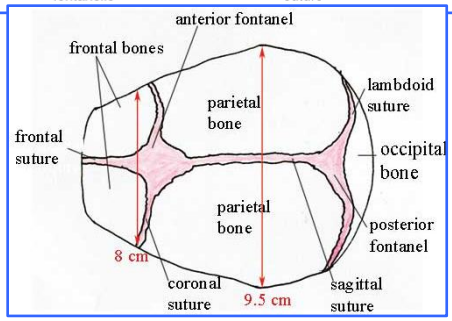
➢ Bones: Bones are separated by? "Sutures" they are imp in labor b/c they overlap during delivery, but we don't want too much overlapping b/c this is dangerous for the baby. At birth, the cranial bones touch each other, this process known as "molding" which is (grade one). And if they overlap and you can separate them, this is (grade two).But if they overlap and you cannot separate them, this is (grade three) and it's dangerous, grade 3 is a sign of "cephalopelvic disproportion" the head cannot go through the maternal pelvis, so it can damage the brain! .

- Two frontal bones
- Two parental bones
- One occipital bone
- Two temporal bones



➢ Suture: an area of membrane which has not ossified:

- Lambdoid suture
- Sagittal suture
- Coronal suture
- Frontal suture



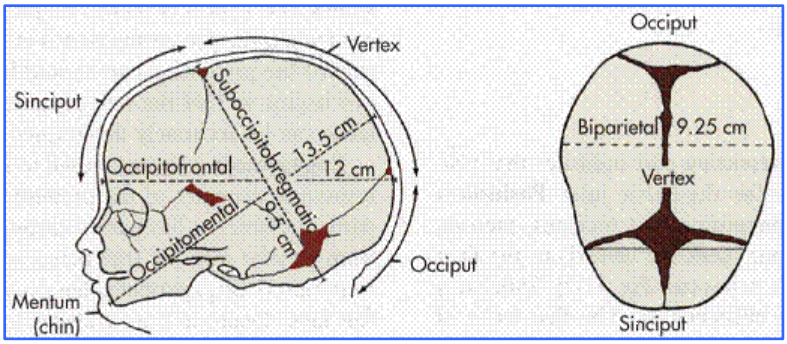
➢ Fontanels-areas where two or more sutures meet.-
They are very important landmarks:

- Anterior fontanel, diamond in shape where sagittal and frontal sutures meet
- Posterior fontanel, where lambdoidal and sagittal sutures meet.

➢ Areas of the skull:

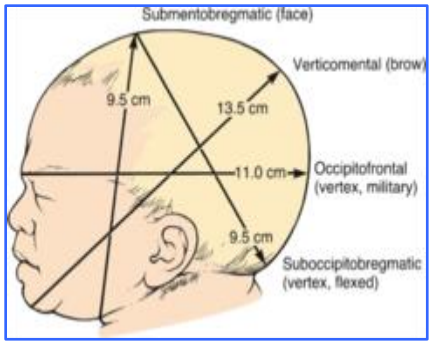
1. Glabella: the bridge of the nose
2. Sinciput : the forehead
3. Bregma: the anterior fontanels
4. Vertex
5. Lambda: the posterior fontanel
6. Occiput
7. Suboccipital area
8. Mentum: the chin

Malposition and malpresentation may happen



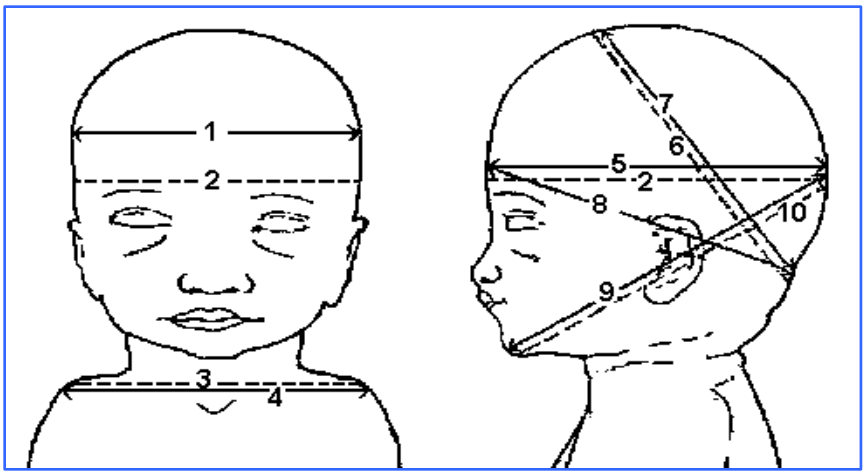
➢ Circumferences of the fetal skull:

- The engaging Diameter in a well flexed head:**suboccipito-bregmatic+Biparietal**
  →In **Vertex presentation**
- The engaging Diameter in a Deflexed head (partly extended): **Occipito-frontal+Biparietal**
  →In **Occipito posterior Position**



➢ Diameters of fetal skull

- Bipareital
- Bitemporal
- Suboccipital-bregmatic
- Occipito frontal
- Mentovertical
- Submento-bregmatic



Bi-parietal diameter
Occipito-frontal circumference
Bi-acromial circumference
Bi-acromial diameter
Occipito-frontal diameter
Suboccipito-bregmatic circumference
Suboccipito-bregmatic diameter
Suboccipito-frontal diameter
Occipito-mental diameter
Occipito-mental circumference

Effect of Labor and Delivery

- **Molding** (we've discussed it previously)
- **Caput succedaneum**

> **When there are good contractions and the baby fails to flex and rotate his/her head and there is increasing in molding, those will increase the caput succedaneum (it's edema 'collection of fluid' in the fetal head b/c there is pressure of the cervix on the fetal head)**

- **Cephalhematoma** **(blood between the skull and the periosteum of a newborn baby)**
  Cephalhematoma is one of the most common cranial injuries that an infant may suffer especially during a forceps-assisted delivery (instrumental delivery)

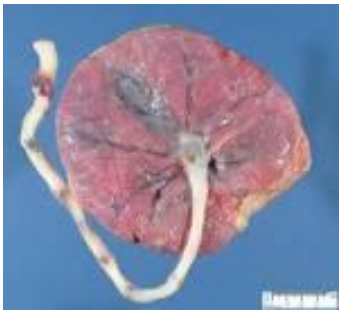
Placenta

It's very imp to know the structure of the placenta to prevent postpartum hemorrhage.

- Structure of the mature placenta:
  - ✓ It has 2 surfaces:

    A- Maternal surface
    - It lies next to the uterus on inspection, chorionic villi are arranged in lobes/cotyledons – **20 in number** – 200 lobules.
      After the delivery of the placenta you have to inspect it very well and check the 20 cotyledons carefully to prevent any complications from any missed parts inside the uterus b/c she may get infection or 2ry postpartum hemorrhage.
    - The grooves separating the lobes are sulci.
    - Dark – red color, and has rough surface.

    B- Fetal surface
    - It faces the baby.
    - Bluish gray color, and has smooth, shiny surface.
    - The umbilical cord is inserted in the fetal surface, usually in the center.
    - Blood vessels are radiating from the cord.
    - The amniotic membrane covers the fetal surface.

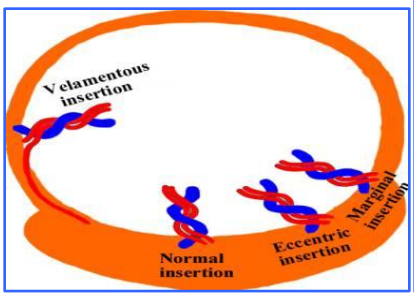


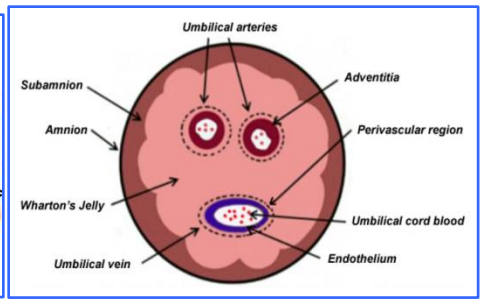
  - ✓ Flat, Roughly circular
  - ✓ 22 cm in Diameter
  - ✓ 2cm thick in the center
  - ✓ Weight: 1/6 of the baby's weight
- Abnormalities of placental development:
  - ✓ Placenta succenturiata
  - ✓ Placenta bipartita
  - ✓ Placenta circumvallata
  - ✓ Placenta velamentosa
  - ✓ Placenta succenturiata/ Placenta velamentosa
  - ✓ Vasa previa(it's very dangerous, may cause fetal death because the bleeding will be from the fetus NOT the mother!)
    Before labor, when you start artificial rupture of the membrane and you get bleeding, this can be vasa previa or premature separation of placenta)

Umbilical cord

After delivery, you have to inspect the umbilical cord and write that it contains 2 arteries and one vein.

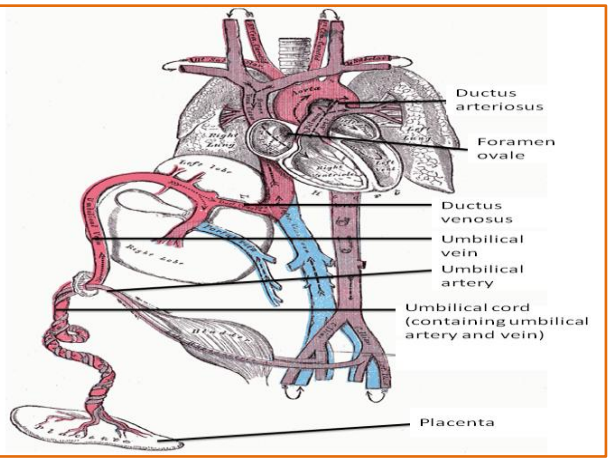
- At full term: 40-50 cm long
  1.5 cm in diameter
- Twisted in appearance
- Two umbilical arteries and one umbilical vein
- Wharton's jelly
  It is a gelatinous substance within the umbilical cord.It contains some fibroblasts and macrophages. As a mucous tissue it protects and insulates umbilical blood vessels. Wharton's jelly, when exposed to temperature changes, collapses structures within the umbilical cord and thus provides a physiological clamping of the cord (an average of) 5 minutes after birth.
- Abnormal insertion of the cord:
  - Battledore insertion **(Marginal Cord Insertion)**
  - Velamentous insertion





Fetal Circulation

Very helpful videos
Fetal circulation before birthhttp://www.youtube.com/watch?v=-IRkisEtzsk
Fetal circulation after birthhttp://www.youtube.com/watch?v=jFn0dyU5wUw
Other videos:
http://www.youtube.com/watch?v=uwswhoKfkmM
http://www.youtube.com/watch?v=A2oa65hB50c



Q-What are the two major events in fetal circulation?

1-Presence of umbilical-placental circulation

2- Absence of significant pulmonary circulation.

The fetus doesn't need pulmonary circulation b/c s/he gets O2 form the mother.

Q-What are the three shunts?

The fetal circulatory system uses three shunts:

1- Ductus venosus(in the liver)

2- Foramen ovale

3- Ductus arteriosus

The oxygenated blood moves from the mother to the fetus through the placenta→ umbilical vein →ductusvenosus (in the liver-)→foramen ovale (heart)→ductusarteriosus (what makes this duct open in utero? Is the Prostaglandin(PG))→brain!

After delivery of the baby, you have to clam the cord in both directions NOT CUT → there will be pressure changes in the shunts which will close them.

Q-How does the fetal circulatory system work?

- ✓ The respiratory function of the placenta requires that oxygenated blood to be returned via the umbilical vein and into the fetal circulation.
- ✓ High venous return from the placenta (oxygenated blood - O2 saturation 70-80% -) through the umbilical vein.
- ✓ This maintains the right-left shunt through the foramen ovale
- ✓ Delivers most oxygenated blood to fetal heart and brain (major organs)and upper extremities.
- ✓ Placenta →umbilical vein→ductus venosus,
- ✓ Most of the blood flows into the inferior vena cava (IVC), this mixes with returning non oxygenated blood from the lower limbs, kidney, and liver. However, only partial mixing of the two streams.
- ✓ Most of the oxygenated blood is directed to the crista dividens at the upper end of the inferior vena cava into the right atrium through the foramen ovaleand thus into the left atrium and hence to the left ventricles and ascending aorta to be directed to the brain, heart and upper extremities.
- ✓ The remainder of the blood from the superior vena cava mixes with that of IVC and passes directly to the right ventricle.
- ✓ 10% of it goes through the pulmonary artery to the lung.
- ✓ Most of this enters the systemic circulation via the ductus arteriosus and into the descending aorta beyond the vessels supplying the head
- ✓ It supplies the viscera and lower limbs
- ✓ It then passes into the umbilical arteries (branches of left and right internal iliac arteries)
- ✓ High pulmonary vascular resistance maintains the right-left shunt through the ductus arteriosus.

At birth:

- ✓ The closure of the shunts.
- ✓ Completes the transition of fetal circulation to newborn circulation.
- ✓ Umbilical vessels contract.
- ✓ Cessation of umbilical blood flow causes a fall in pressure in the right atrium.
- ✓ The foramen ovale is a valvular opening, the valve functioning from the right to left, the left atrial pressure rises and thus closure of the foramen ovula.
- ✓ Breathing:

Ventilation of the lung helps to create a negative thoracic pressure, this opens the pulmonary circulation and thus diverts blood from ductus arteriosus which then gradually closes.

Q-What maintains patency of ductus arteriosus in utero?

- ✓ It is the presence of prostaglandin **(PG)**

Fetal Circulation

Prior to birth the fetus is not capable of respiratory function and thus relies on the maternal circulation to carry out gas, nutrient and waste exchange. The fetal and maternal blood never mix, instead they interface at the placenta. Consequently the liver and the lungs are non-functional, and a series of shunts exist in the fetal circulation so that these organs are almost completely by-passed.

Shunt 1: The DuctusVenosus

Oxygenated blood travels from the placenta via the umbilical vein and most of it bypasses the liver by way of the ductusvenosus. The ductusvenosus links the umbilical vein to the inferior vena cava and the flow of blood is controlled by a sphincter, enabling the proportion travelling to the heart via the liver to be altered.

Shunt 2:The Foramen Ovale

The foramen ovale is an opening between the two atria enabling blood to be channeled directly into the systemic circulation thereby bypassing the lungs. The septum secundum directs the majority of the blood entering the right atrium through the foramen ovale into the left atrium. Here it mixes with a small volume of blood returning from the non-functional lungs via the pulmonary veins.

Shunt 3:The DuctusArteriosus

The ductusarteriosus connects the pulmonary artery to the aorta and allows equivalent ventricular function in the fetus. The blood from the right ventricle is pumped to the pulmonary trunk where, due to the high resistance in the collapsed fetal lungs, a larger volume passes through the ductusarteriosus to the caudal aorta. Most of the blood in the aorta is then returned to the placenta for oxygenation through the umbilical arteries. The ductusarteriosus empties blood into the aorta after the artery to the head has branched off thus ensuring that the brain receives well-oxygenated blood.

Circulatory Changes at Birth

Important circulatory changes occur at birth due to the replacement of the placenta by the lungs as the organ of respiratory exchange. When an newly born baby takes its first breath, the lungs and pulmonary vessels expand thereby significantly lowering the resistance to blood flow. This subsequently lowers the pressure in the pulmonary artery and the right side of the heart. On the other hand the removal of the placenta causes an increase in the resistance of the systemic circulation and hence an increase in the pressure of the left side of the heart.

The birth of the baby also triggers the closure of the fetal shunts:

Closure of the DuctusVenosus

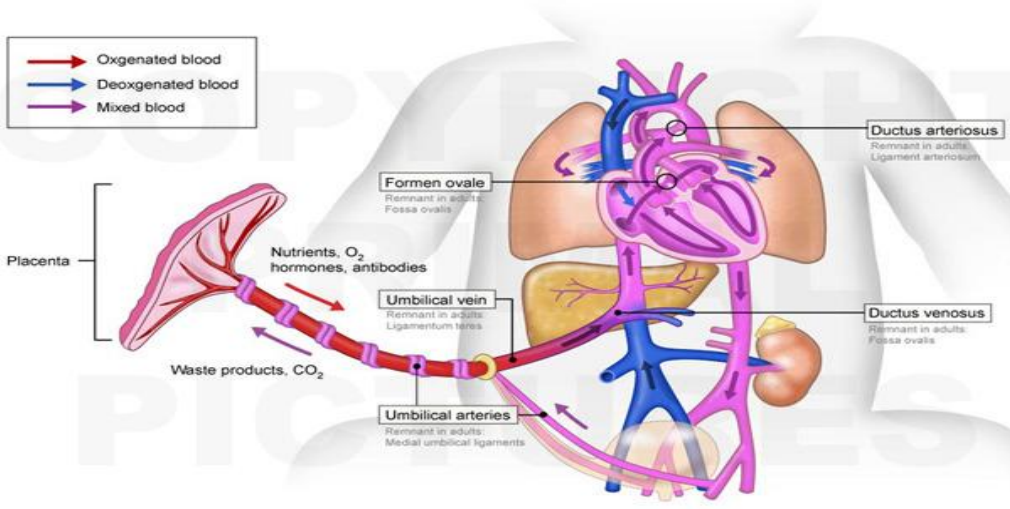
The ductusvenosus is weakly responsive to prostaglandin E2 (PGE_2) and prostacyclin (PGI_2) which behave as vasodilators. This influence is lost with the improved pulmonary clearance resulting from the absence of an umbilical blood supply. This loss of blood supply also causes the sphincter in the ductusvenosus to constrict thereby diverting blood to the liver. Closure of the ductusvenosus becomes permanent after two to three weeks. The remnants of the ductusvenosus form the ligamentumvenosum.

Closure of the Foramen Ovale

In the fetus the foramen ovale is kept open by the higher pressure of blood in the right atrium compared to the left atrium. At birth the blood pressure in the right atrium decreases due to termination of blood flow from the placenta, whilst pressure in the left atrium increases due to increased pulmonary flow. As a result, the flap of the septum primum presses against the septum secundum closing the foramen ovale. In most individuals, the foramen ovale closes a few months after birth. A scar remains between the two atria once the foramen ovale has closed and this is termed the fossa ovalis.

Closure of the DuctusArteriosus

The ductusarteriosus is a muscular artery and immediately after birth, contraction of the musculature closes the shunt. Factors which may contribute to the physiological closure of the ductusarteriosus include the increased oxygen content of the blood passing through it and the production of bradykinin, which causes smooth muscle



Embryology of Female Genital Tract, Congenital Malformation & Intersex.

Part 1: Embryology of ♀genital tract

SEXUAL DIFFERENTIATION

- The first step in sexual differentiation is the determination of 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
- ♀ exposed to androgens in- utero will be masculinized.

EXTERNAL GENITALIA

1-UNDEFERENTIATED STAGE (4-8 WK)

The neutral genitalia includes:

genital tubercle (phalus)
labioscrotal swellings
urogenital folds
urogenital sinus

Fetal assessment

2-♂ & ♀ EXTERNAL GENITAL DEVELOPMENT (9-12 WK)

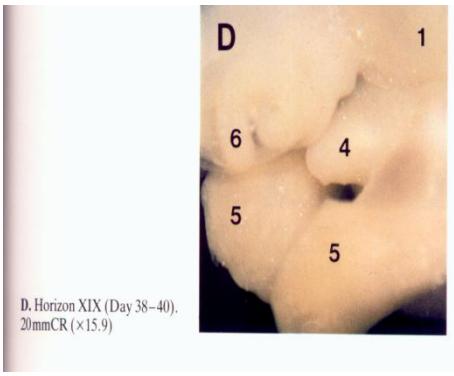
- By 12 wk gestation ♂&♀ genitalia can be differentiated
- In the absence of androgens ⇨♀ external genitalia develop
- The development of ♂ genitalia requires the action of androgens, specifically DHT

5 alpha reductase

Testosterone ⇨⇨⇨⇨⇨ DHT

EXTERNAL GENITALIA (INDIFFERENT STAGE)

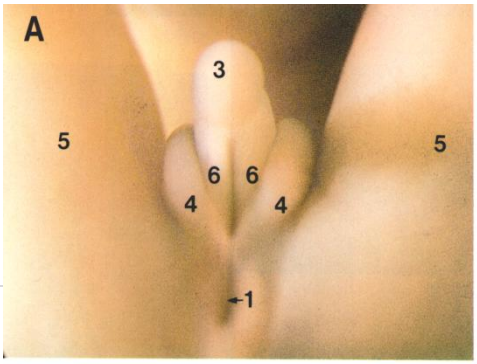
1-abdomen
4-genital tubercle
5-leg bud
6-midgut herniation to the umbilical cord



FEMALE EXTERNAL GENITALIA

Week 9

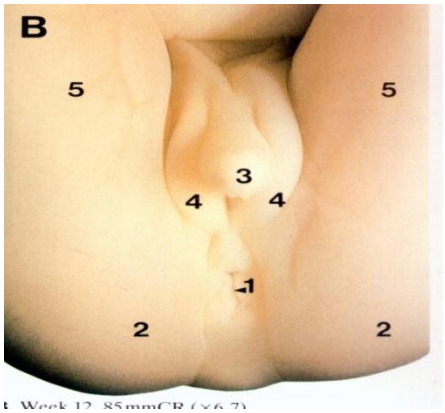
1-anus
2-buttocks
3-clitoris
4-labioscrotal swelling (labia majora)
5-leg
6-urogenital fold (labia minora)



Week 12

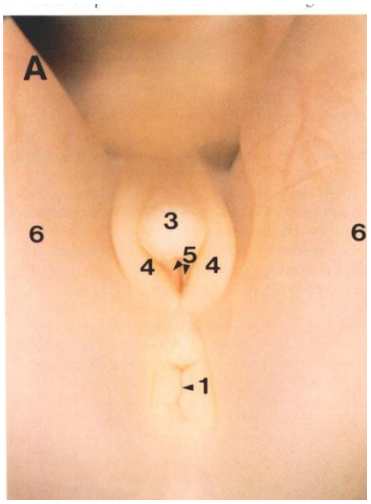
1-anus
2-buttocks
3-clitoris
4-labioscrotal swelling(labia majora)
5-leg
6-urogenital fold(labia minora)

The external genitalia of female is distinguishable at about 12 weeks



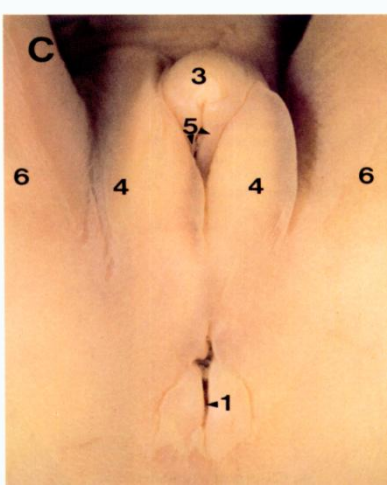
Week 13

1-anus
2-buttocks
3-clitoris
4-labia majora
5-labia minora
6-leg



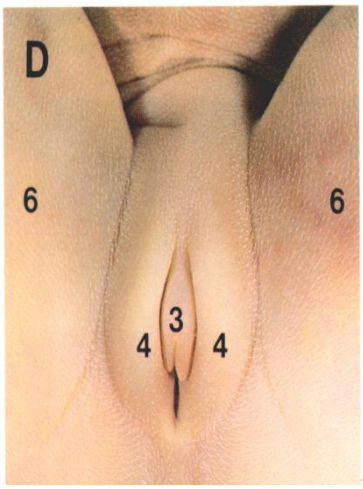
Week 17

1-anus
2-buttocks
3-clitoris
4-labia majora
5-labia minora
6-leg



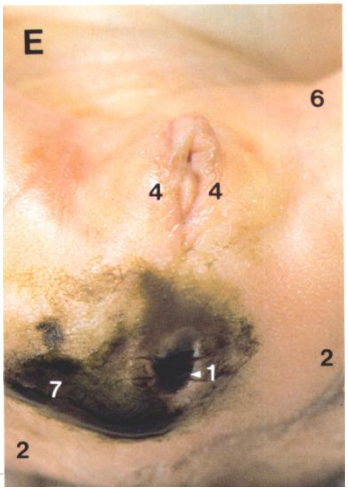
Week 20

1-anus
2-buttocks
3-clitoris
4-labia majora
5-labia minora
6-leg



Week 35

1-anus
2-buttocks
3-clitoris
4-labia majora
5-labia minora
6-leg
7-meconium



INTERNAL GENITAL ORGANS

1-GONADS

- Undifferentiated gonads begin to develop on the 5th wk
- Germ cells originate in the yolk sac & migrate to the genital ridge
- In the absence of Y chromosome the undiff gonad develop into an ovary
- 45XO embryo the ovaries develop but undergo atresia ⇨streak ovaries
- The gonads develop from the mesothelium on the genital ridge ⇨ 1ry sex cords grow into the mesenchyme ⇨ outer cortex & inner medulla
- The ovary develop from the cortex & the medulla regress
- The testes develop from the medulla & the cortex regress
- The development of the testes requires the presence of SRY gene (Sex determining region Y) found on Y chromosome
- The ovary contains 2 million 1ry oocytes at birth

Turner Syndrome

2-UTERUS & FALLOPIAN TUBES

- Invagination of the coelomic epithelium on the cranio-lateral end of the mesonephric ridge ⇨ Paramesonephric ducts
- Fusion of the two PMN ducts (mullerian ducts) ⇨ uterus, cx & F tubes (at 8-11 wk)
- 12-16 wks ⇨ proliferation of the mesoderm around the fused lower part ⇨ muscular wall
- In the male fetus the testes secrete the mullerian inhibiting factor ⇨ regression of the mullerian ducts

-The upper vagina, cervix & fallopian tubes are formed from the paramesonephric "PMN" (mullerian) ducts.

-the absence of Y chromosomal influence leads to the development of PMN system &total regression of the mesonephric system.

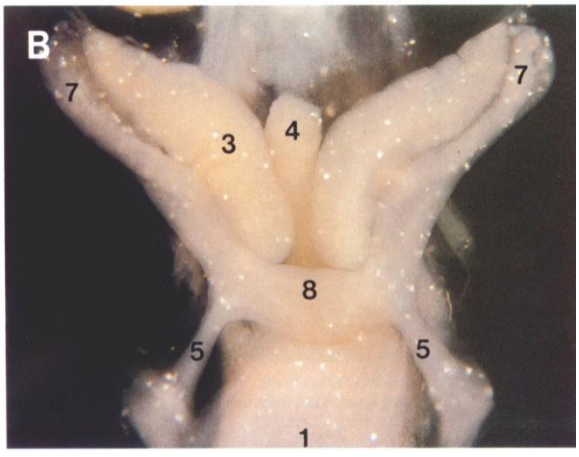
3-VAGINA

- The caudal ends of the mullerian ducts form the mullerian tubercle at the dorsal wall of the urogenital sinus
- Mullarian tubercle is obliterated ⇨ vaginal plate ⇨ 16-18 wk the central core breaks down ⇨ vaginal lumen
- The upper 2/3 of the vagina ⇨ formed by mullerian tubercle
- The lower 1/3 ⇨ urogenital sinus

FEMALE INTERNAL GENITAL ORGANS

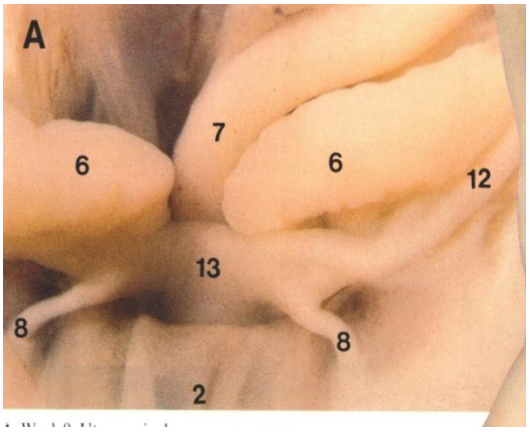
Week 8

1-bladder 2-kidney
3-ovary 4-rectum
5-round ligament of the uterus
6-adrenal gland 7-Fallopian tube
8-utero vaginal primordium



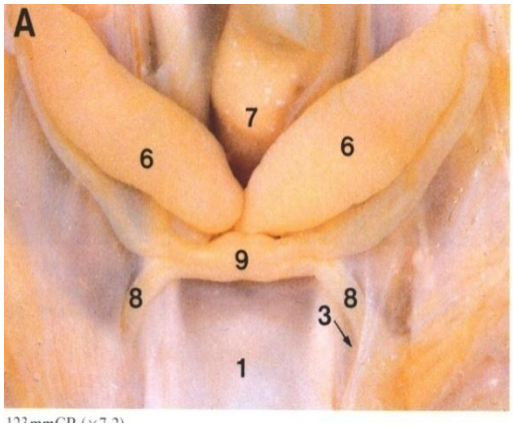
Week 9

2-bladder
6-ovary
7-rectum
8-round ligaments
12-uterine tube
13-uterovaginal primordium



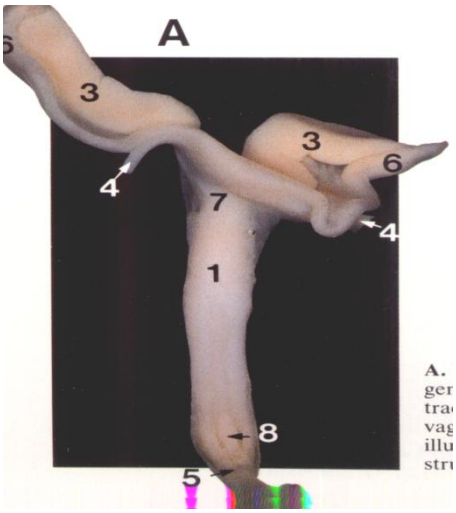
Week 15

1-bladder
2-clitoris
3-vaginal process
4-labia majora
5-leg
6-ovary
7-rectum
8-uterine round ligament
9-uterovaginal primordium

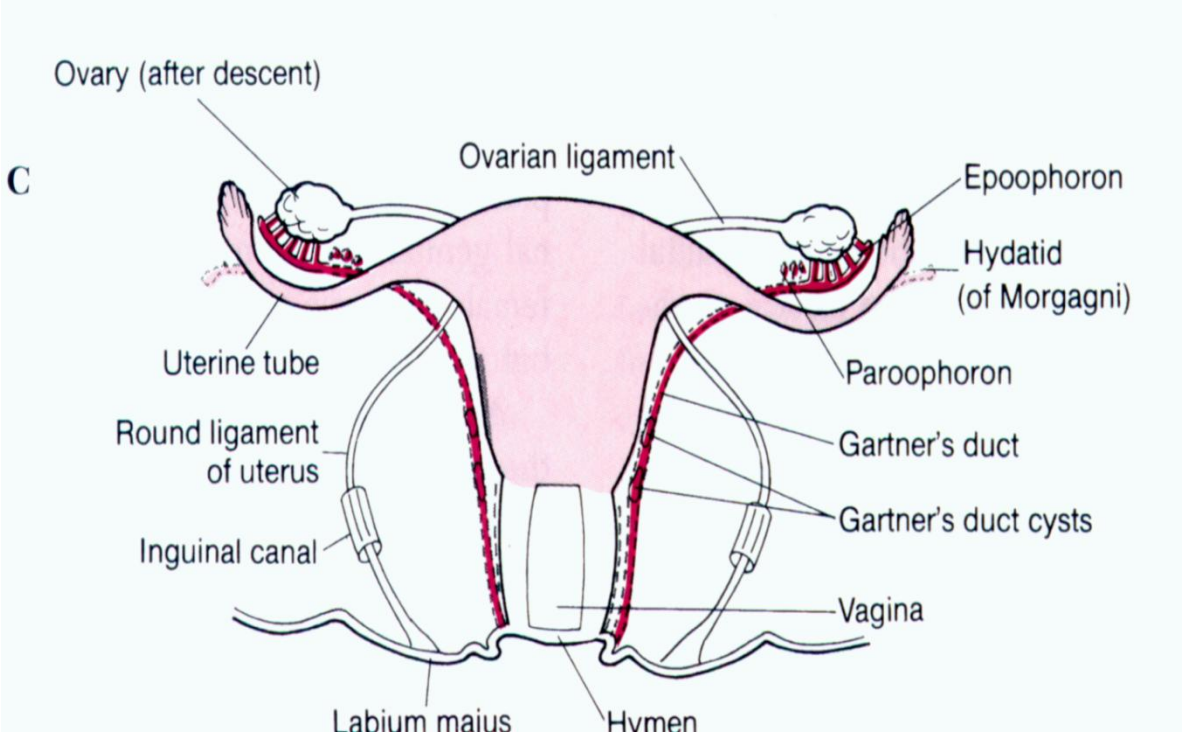


Week 13 (dissected genital tract)

1-body of uterus
2-clitoris
3-ovary
4-round ligament
5-solid epithelium (vagina meets urogenital sinus)
6-fallopian tube
7-uterus
8-vagina



FEMALE INTERNAL GENITAL ORGANS (Newborn)



Male & Female Derivatives of Embryonic Urogenital Structures

TABLE 1-2
Male and Female Derivatives of Embryonic Urogenital Structures

Embryonic Structure	Derivatives	
	Male	Female
Labioscrotal swellings	Scrotum	Labia majora
Urogenital folds	Ventral portion of penis	Labia minora
Phallus	Penis	Clitoris
	Glans, corpora cavernosa penis, and corpus spongiosum	Glans, corpora cavernosa, bulb of the vestibule
Urogenital sinus	Urinary bladder Prostate gland Prostatic utricle Bulbourethral glands Seminal colliculus	Urinary bladder Urethral and paraurethral glands Vagina Greater vestibular glands Hymen
Paramesonephric duct	Appendix of testes	Hydatid of Morgagni Uterus and cervix Fallopian tubes
Mesonephric duct	Appendix of epididymis Ductus of epididymis Ductus deferens Ejaculatory duct and seminal vesicle	Appendix vesiculosis Duct of epoophoron Gartner's duct —
Metanephric duct	Ureter, renal pelvis, calyces, and collecting system	Ureter, renal pelvis, calyces, and collecting system
Mesonephric tubules	Ductuli efferentes Paradidymis	Epoophoron Paroophoron
Undifferentiated gonad	Testis	Ovary
Cortex	Seminiferous tubules	Ovarian follicles
Medulla	— Rete testis	Medulla Rete ovarii
Gubernaculum	Gubernaculum testis	Round ligament of uterus

Part 2: Congenital Malformation of the ♀ Genital Tract

OBJECTIVES

- To be able to differentiate the various types of congenital malformation of female internal genital organs
- To know the abnormalities due to lateral and vertical fusion of the mullarian ducts as well as failure of mullarian duct development
- To know the clinical presentation and management of congenital anomalies of the female genital tract

1-MULLERIAN AGENISIS

To read more about Vaginoplasty click the link below:

http://en.wikipedia.org/wiki/Vaginoplasty

Mayer –Rokitansky-Kuster-Huser syndrome

Etiology ?

- Failure of mullerian duct development ⇨ absence of the upper vagina, cx & uterus (uterine reminants may be found)
- 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

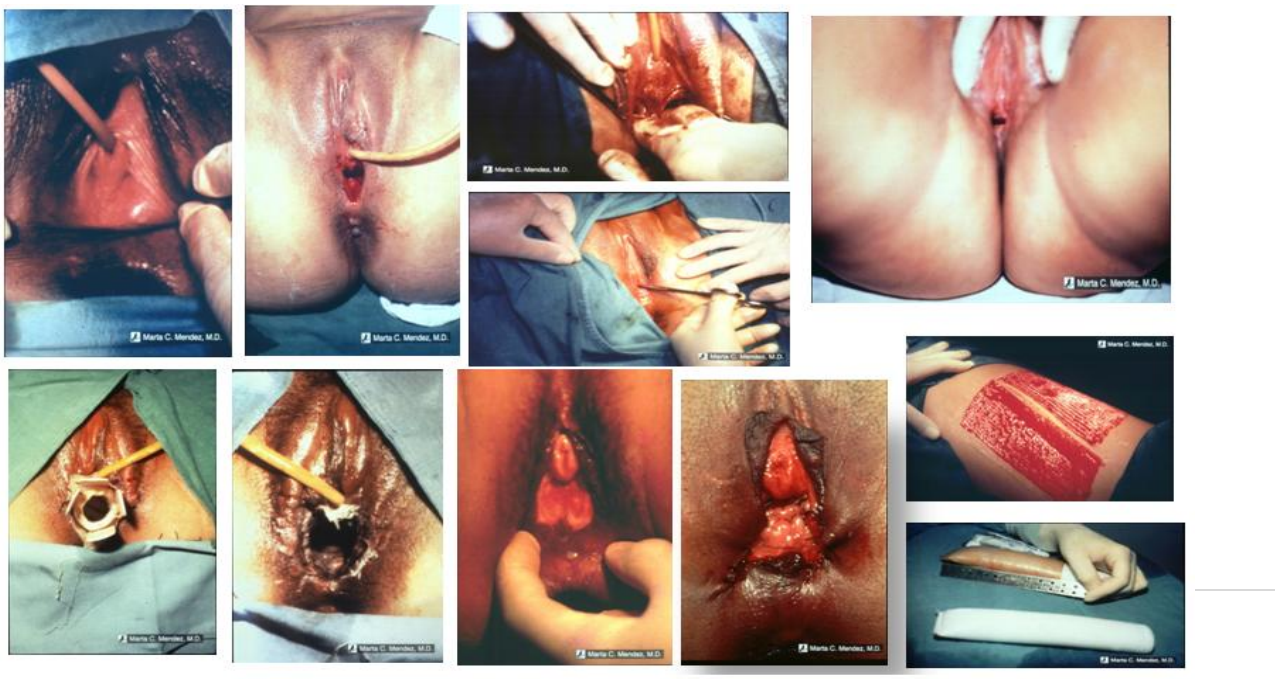
Surgical⇨- vaginoplasty

- Excision of utrine reminant (if it has Functioning endometrium)

-vaginal dilators

-Pt with 1ry amenorrhea, breast development & a 46XX karyotype have levels of testosterone appropriate of females.

-The clinical diagnosis may be caused by mullerrian defects that cause obstruction of the vaginal canal (e.g., imperforated hymen of a transverse vaginal septum) of by the absence of a normal cervix or uterus & normal fallopian tubes.



2-DISORDERS OF LATERAL FUSION OF THE MULL DUCTS

Incidence ? 0.1-2%

4% of infertile pt

6-10% recurrent abortion pt

Most pt can conceive without difficulty

↑ Incidence of:

◊ recurrent abortions

◊ premature birth

◊ fetal loss

◊ fetal malpresentation

◊ C S

◊ cx incompetence

CLINICAL PRESENTATION

♣Shortly after menarche ⇨if there is obstruction touterine blood flow

♣Difficulty in intercourse⇨ longitudinal vaginal septum

♣Dysmenorrhea or menorrhagia

♣Abnormality detected on D&C

♣U/S, laparoscopy or laparotomy

♣Palpable mass

♣Complications of pregnancy

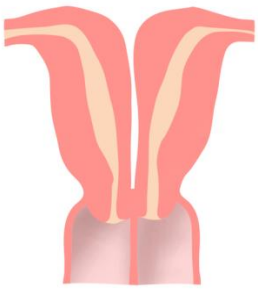
♣HSG ⇨ during infertility or RFL investigations

NON OBSTRUCTIVE MALFORMATIONS OF THE MULLERIANE DUCTS

2-DISORDERS OF LATERAL FUSION OF THE MULL DUCTS

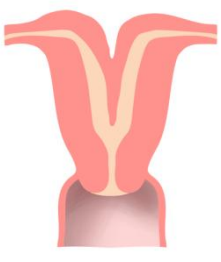
A-Uterus didelphus:

- Complete duplication of uterus, cx & vagina (due to failure of fusion of the two Mull ducts)
- ? ↑ pregnancy wastage
- Dx ⇨ HSG or at laparoscopy / laparotomy
- Rx ⇨ If affecting pregnancy outcome ⇨ surgical correction (metroplasty)



B-Bicornuate uterus

- Incomplete fusion of the two Mull ducts
- ↑ pregnancy wastage
- Dx ⇨ HSG or at laparoscopy / laparotomy
- Rx ⇨ If affecting pregnancy outcome ⇨ surgical correction (metroplasty)



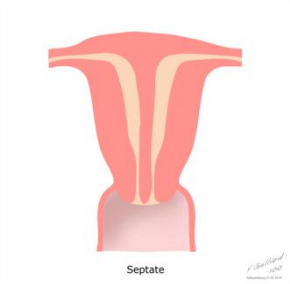
- C-Septate uterus

External contour of the uterus is normal but there is intrauterine septum of varying length & thickness

Worst pregnancy outcome

Dx ⇨ both HSG & laparoscopy

Rx ⇨ Hystroscopic excision of the septum



D-Unicornuate uterus

Due to development of only one Mull duct

Almost all pt have associated single kidney

Pregnancy outcome ⇨ similar to pt with didelphic uterus

Dx ⇨ HSG or surgery

Rx ⇨ NO corrective surgery

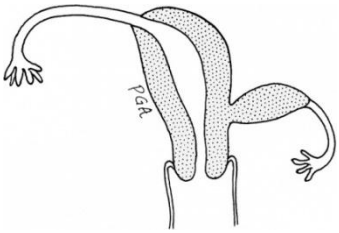
⇨ if pt has associated cx incompetence ⇨ cx cerclage

E-Unicurnuate with rudimentary horn

Noncommunicating horn 90%

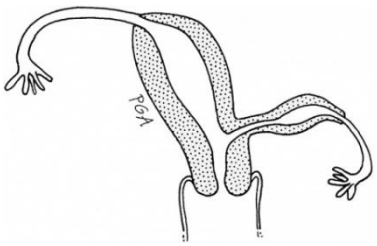
Present with cyclic pelvic pain, mass, ectopic pregnancy in the rud horn or endometriosis

Rx ⇨ surgical excision



Communicating horn

Present with ectopic pregnancy in the rud horn or ↑ pregnancy wastage



3-DISORDERS OF VERTICALE FUSION OF THE MULLERIAN DUCTS

A- VAGINAL SEPTUM

- Faults in the junction between the Mull. Tubercle & the urogenital sinus ⇨ could be very thick or thin
- 85% in upper two third of the vagina
- Pt present 1ry amenorrhea, hematocolpus, mass or cyclic abdominal pain
- ↑ incidence of endometriosis
- Rx ⇨ surgical excision

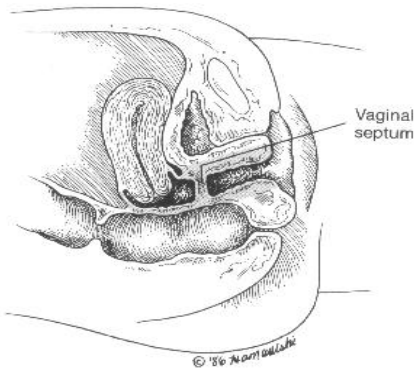
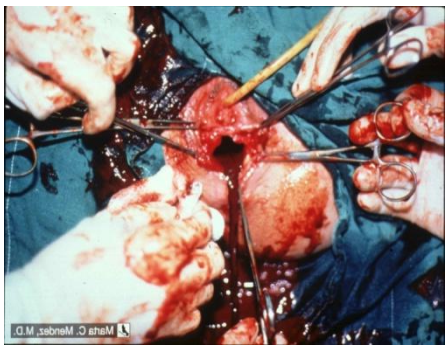
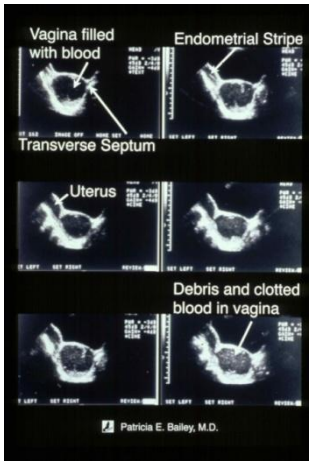


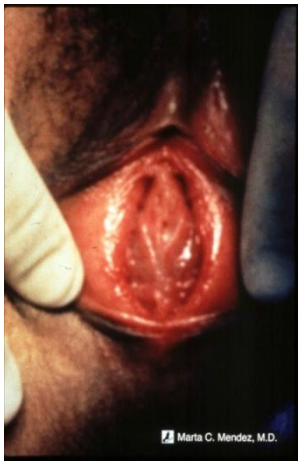
FIGURE 10-7
Diagram of transverse vaginal septum.

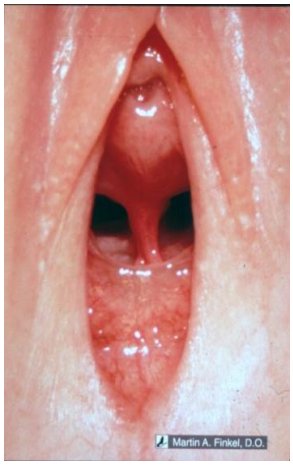
B-Cx AGENISIS / DYSGENISIS

- V rare
- Difficult, unsuccessful surgical correction
- Rx ⇨ hysterectomy





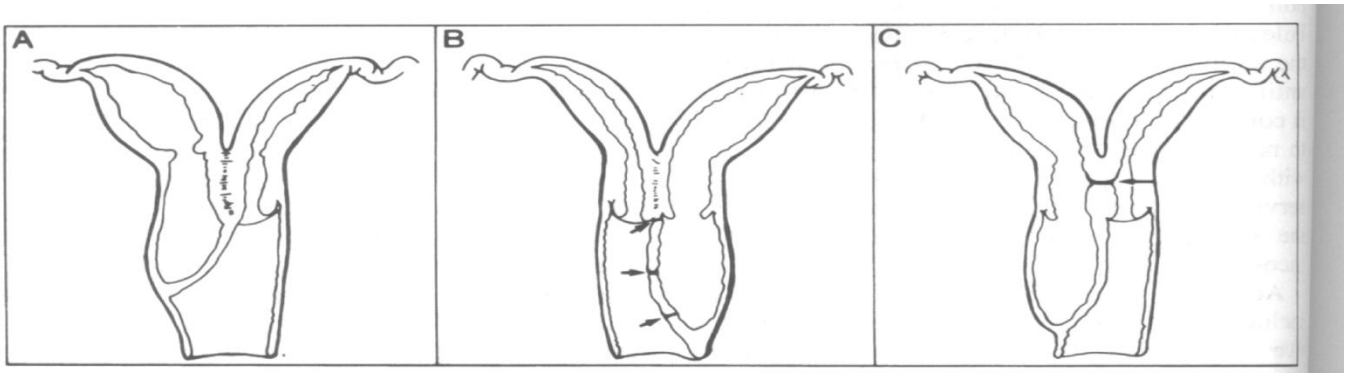




4-UNUSUAL CONFIGRATION OF VERTICAL/LATERAL FUSION DEFECTS

- Combined lateral & verticle defects
- Do not fit in other categories
- EXAMPLE, double uterus with obstructed hemivagina

Double uterus with vaginal obstruction



A-Complete vaginal obstruction B-Incomp vag obst C-Comp obst with comm double uterus

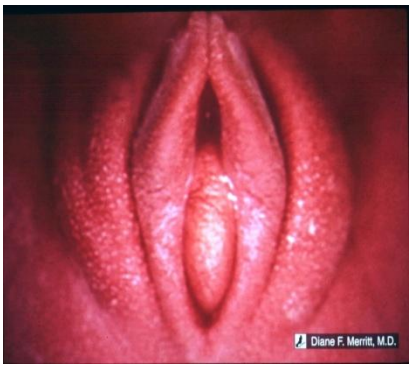
5-DEFECTS OF THE EXTERNAL GENITALIA

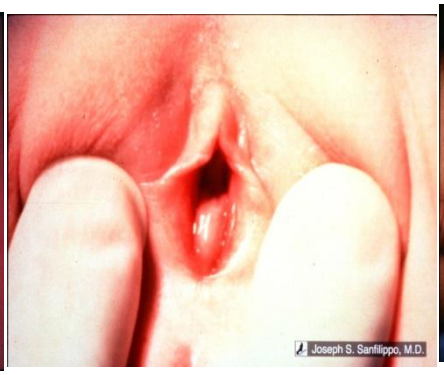
- Ambigious genitalia ⇨ congenital adrenal hyperplasia
  hermaphrodites
- Defects of the clitoris ⇨ uncommon ⇨ bifid clitoris
  hypertrophied ⇨ androgen effect
- IMPERFORATE HYMEN

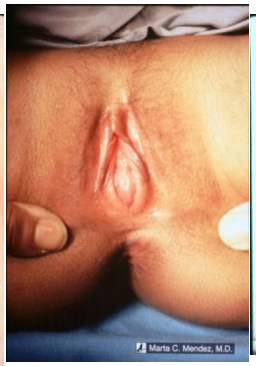
Hymen is formed at the junction of the urogenital sinus & sinovaginal bulbs

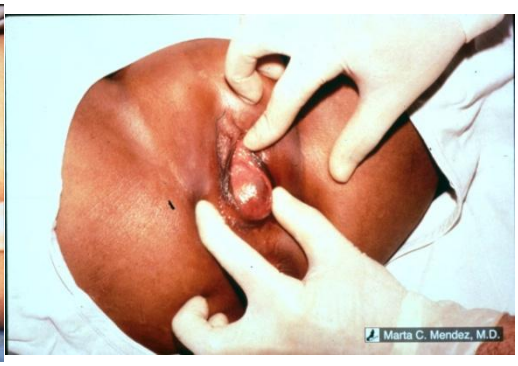
Pt presents with 1ry amenorrhea with cyclic abdominal pain or hematocolpus /hematometra

Rx ⇨cruciate incision









Part3: INTERSEXUALITY

OBJECTIVES

- To understands the defects that result in abnormal development of the external genitalia in male and female fetuses
- To be able to differentiate the types of male and female hermafrodites
- Causes presentations and management of various types of intersex

ABNORMAL SEXUAL DEVELOPMENT

1-Sex chromosome abnormality

Mosaicism associated with gonadal dysgenisis ⇨45X/46XY

2-Testis incapable of producing testosterone.

3-End organs incapable of utilizing testosterone eg. 5α reductase deficiency, failure of testosterone binding to receptors (androgen insensitivity)

MIF = Mullerian Inhibiting Factor.

4-Defficient production of MIF ⇨♀ internal genital organs in otherwise normal ♂

5-Musculanization of the ♀ external genitalia due to #↑ androgen eg. Congenital adrenal hyperplasia (CAH).

6-Rarely 46XX male due to the presence of a gene the SRY gene (Sex determining Region Y)

7-True hermaphroditism ⇨ the presence of testicular & gonadal tissue in the same individual

1-MUSCULINIZED ♀ (♀ PSEUDOHERMAPHRODITES)

-46XX

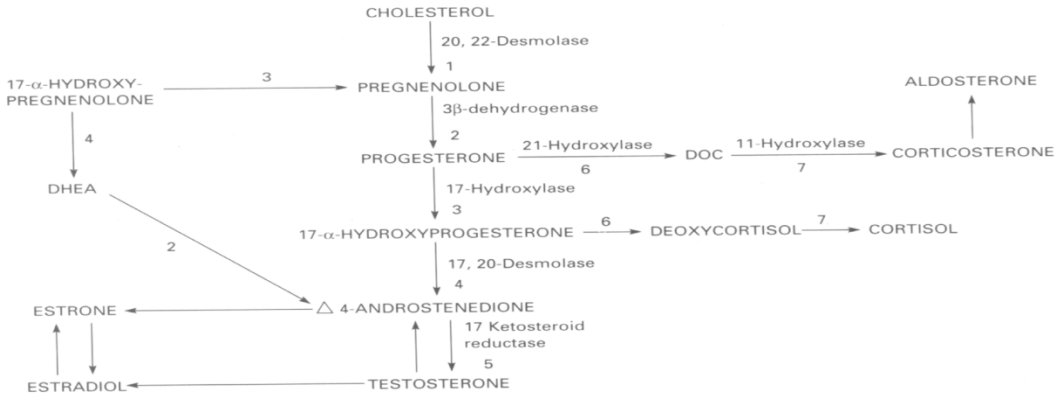
-Exposed to androgens in utero ⇨ varying degrees of musculinization of the external genitalia

A-CONGENITAL ADRENAL HYPER PLASIA (CAH)

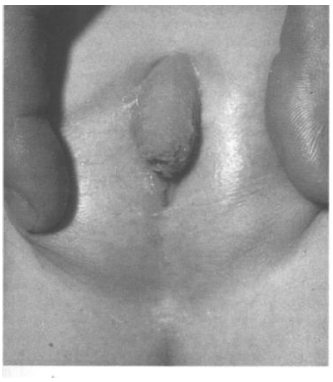
- The most common cause of ♀ intersex
- Deficiencies of the various enzymes required for cortisol & aldosterone biosynthesis (21-hydroxylase, 11β-hyroxilase , 3βhydroxisteroid dehydrogenase)
- 21-hydroxylase deficiency is the commonest defect 90%
- Affected ♀ may present at birth with ambiguous genitalia

-enlargement of the clitoris

-excessive fusion of the genital folds obscuring the vagina & urethra



A-CONGENITAL ADRENAL HYPER PLASIA (CAH)



-thickening & rugosity of the labia majora resembling the scrotum

- A dangerous salt losing syndrome due to deficiency of aldosterone may occure in some pt
- Delayed menarche & menstrual irregularities
- INVESTIGATIONS

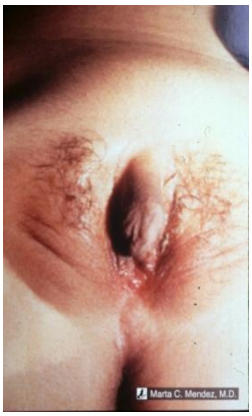
Karyotyping

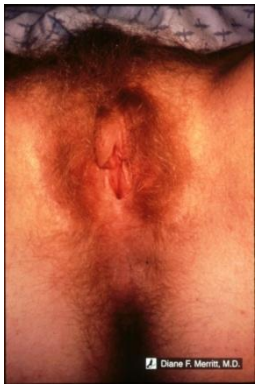
17-α-hydroxiprogestrone ↑

17-ketosteroids (androgens) in urine

Electrolytes

U/S





- Rx

1- Cortisol or its synthetic drevatives

⇨ suppress the adrenals ⇨↓ androgen production

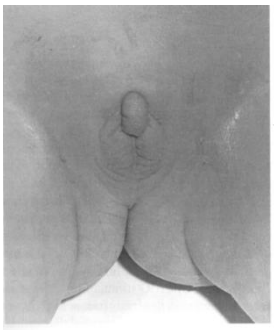
2-Corrective surgery

clitroplasty (neonatal period)

division of the fused labial folds

(delayed till puberty)

B- EXPOSURE OF THE MOTHER TO ANDROGENS



-Rare

-Androgen secreting tumours , eg. luteoma, arrhenoblastoma

-Drugs

2-UNDERMUSCULINIZED ♂(♂ PSEUDOHERMAPHRODITES)

A-ANATOMICAL TESTICULAR FAILURE

-Pure gonadal dysgenisis

*normal chromosomes 46XY

*variable features – mild-severe

(normal ♀ , ♀ with mild musculinization)

*uterus present

-Mosaicism 45X/46XY

*Variable features

(normal ♀, ambiguous genitalia, nearly normal ♂)

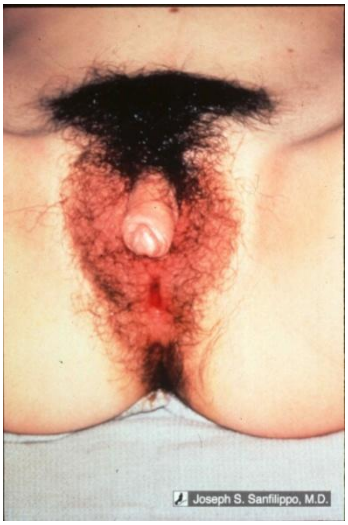
B-ENZYMETIC TESTICULAR FAILURE

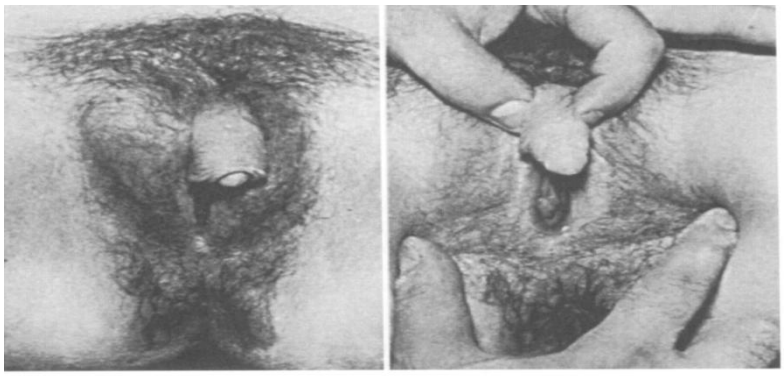
- Enzymetic defects in the biosynthesis of testosterone
- These defects are usually incomplete ⇨Varying degrees of musculinization of the external genetalia
-Uterus & tubes ⇨ absent (MIF produced by the testes)

C-END-ORGAN INSINSITIVITY

1-5α REDUCTASE DEFICIENCY

-Autosomal recessive
- Formation of the ♂ external genitalia requires 5α REDUCTAS testosterone ⇨⇨⇨⇨dihydrotestosterone
- Formation of the internal wollfiane structures respond directly to testosterone
  -External genitalia ♀ with mild musculinization
  -Absent uterus
  -At puberty ⇨↑ testosterone secretion ⇨ virilization





D-ANDROGENINSINSITIVITY

2-ANDROGEN INSINSITIVITY (TESTICULAR FEMINIZATION)

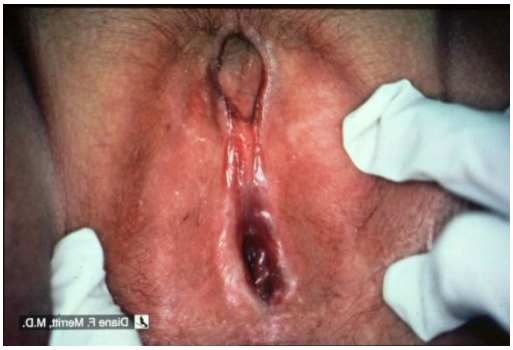
Etiology

- Lack of androgen receptors ⇨ complete (classical TF)
-Receptors are present but low in NO. or inactive

⇨ incomplete androgen insensitivity

Clinical features of Complete Androgen Insinsitivity

Normal ♀ external genitalia with blind vagina
Absent uterus
Breast development
Present with 1ry amenorrhea
Testes found in abdomen or inguinal canal
Normal ♂ Testosterone level

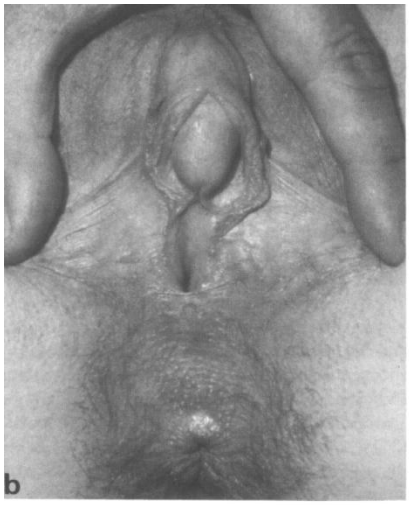


Rx Gonadectomy after puberty due to ↑ incidence of malignant change (5%)
Oestrogen replacement

INCOMPLETE ANDROGEN INSINSITIVITY

Ambiguous genitalia with varying degrees Breast development
Musculinization at puberty

3-TRUE HERMAPHRODITES



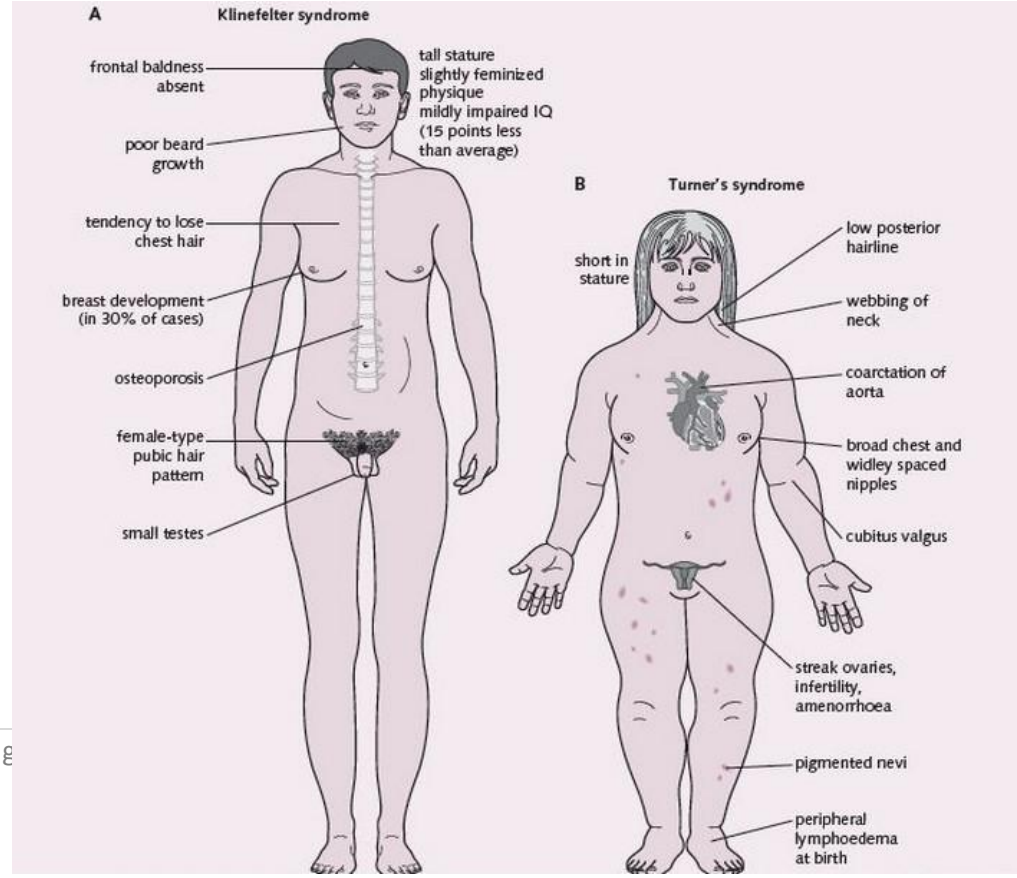
HAVE BOTH OVARIAN & TESTICULAR TISSUE
Ovotestes on one side & ovary or testes on the other
Ovary on one side & testes on the other
Bilateral ovotestes
Varying degrees of sexual ambiguity

KARYOTYPING

46XX ⇨ most common
46XX/XY
46XY
46XY/47XXY

Klinefelter Syndrome

47XXY
Normal male external genitalia
Tall stature
Gynecomastia
Azospermia (infertility)



♀ Pseudohermaphrodites	♂ Pseudohermaphrodites	True Hermaphrodites
Musculinized ♀ (46XX) ⬆ androgens in utero	Undermusculinized ♂	Have Both Ovarian & Testicular Tissue
A-Congenital Adrenal Hyper Plasia (CAH) -thickening & rugosity of the labia majora resembling the scrotum -A dangerous salt losing syndrome due to deficiency of aldosterone may occure in some pt -Delayed menarche & menstrual	A-Anatomical Testicular Failure Pure gonadal dysgenisis -normal chromosomes 46XY (normal ♀ , ♀ with mild musculinization) -uterus present -Mosaicism 45X/46XY -Variable features (normal ♀, ambiguous genitalia, nearly normal ♂)	Ovotestes on one side & ovary or testes on the other Ovary on one side & testes on the other Bilateral ovotestes
B- Exposure Of The Mother To Androgens: -Rare -Androgen secreting tumours , eg. luteoma, arrhenoblastoma -Drugs	B-Enzymatic Testicular Failure -Defects in the biosynthesis of testosterone -incomplete defect ⇨ Varying degrees of musculinization of the external genetalia -Uterus & tubes ⇨ absent (MIF produced by the testes	Varying degrees of sexual ambiguity
	C-Endorgan Insinsitivity (1-5α Reductase Deficiency) -Formation of the ♂ external genitalia requires 5α REDUCTAS testosterone ⇨dihydrotestosterone (DHT) - Formation of the internal wollfiane structures respond directly to testosterone -External genitalia ♀ with mild musculinization -Absent uterus -At puberty ⇨↑ testosterone secretion ⇨ virilization	KARYOTYPING 46XX ⇨ most common 46XX/XY 46XY 46XY/47XXY

D-Androgen Insinsitivity

- Lack of androgen receptors ⇨ complete (classical TF)
-Receptors are present but low in NO. or inactive.

Complete Androgen Insinsitivity

-Normal ♀ external genitalia with blind vagina
-Absent uterus
-Breast development Present with 1ry amenorrhea
-Testes found in abdomen or inguinal canal
-Testosterone level

Incomplete Androgen Insinsitivity

-Ambiguous genitalia with varying degrees Breast development Musculinization at puberty

Physiological Changes In Pregnancy.

- ***Volume Homeostasis***

  - **The factors contributing to fluid retention are:**
    1. Sodium retention > fluid retention > edematous pregnant lady (physiological not pathological because not associated with high BP nor proteinurea)
    2. Resetting of osmostat
    3. Decrease in thirst threshold
    4. Decrease in plasma oncotic pressure
  - **Consequences of fluid retention are**
    1. ☛ Haemoglobin concentration falls (that's why in pregnancy we see "physiological anemia" due to the increase in plasma volume being more than that of hemoglobin and RBCs)
    2. Haematocrit falls
    3. Serum albumin concentration falls that's why the resetting osmolarity will change thus fluid retention
    4. Stroke volume increases which also contributes to the fluid retention
    5. Renal blood flow increases that's why pregnant women have to urinate a lot!

  - **Decreases in:**
    1. Red cell count
    2. Haemoglobin concentration
    3. Haematocrit
    4. Plasma folate concentration

    All decrease as the plasma volume increases
  - **Increases in:**
    1. white cell count in pregnancy it's ok to have slight increase of WBCs count and that doesn't mean infection.
    2. erythrocyte sedimentation rate
    3. fibrinogen

    That's why pregnant women are susceptible to have DVTs.

- ***Cardiovascular System***

  - **Normal changes in heart sounds during pregnancy:**

    1. Increased loudness of both s1 and s2 *
    2. Increased splitting of mitral and tricuspid components of s1
    3. No constant changes in s2
    4. Loud s3 by 20 weeks' gestation
    5. <5% with s4
    6. >95% develop systolic murmur which disappears after delivery *
    7. 20% have a transient diastolic murmur *
    8. 10% develop continuous murmurs due to increased mammary blood flow * that's why you should continue on observing the lady after delivery.

Here you **don't** have to memorize any number you only have to know that there is increased loudness of cardiac sounds and that >95% of pregnant women develop systolic murmur which's physiological but should be observed after the delivery to make sure it was physiological.

- **Cardiovascular Changes**

1. Heart rate increases (10-20%) that's why they have tachycardia
2. Stroke volume increases (10%)
3. Cardiac output increases (30-50%) as a consequence of the increase of both HR & SV
4. Mean arterial pressure decreases (10%) to allow the blood to flow easily into the placenta
5. Peripheral resistance decreases (35%) if it didn't decrease, the BP would increase and the blood flow to the fetus will be resisted reducing the blood going to the placenta.

- ***REPRODUCTIVE ORGANS***
  - The Uterus
  - The Cervix
- ***BREASTS AND LACTATION***

Will be discussed later.

- ***THE URINARY TRACT AND RENAL FUNCTION***

- **Renal changes**

1. Blood flow increases (60-75%)
2. Glomerular filtration increases (50%)
3. Clearance of most substances is enhanced
4. Plasma creatinine, urea and urate are reduced
5. Glycosuria is **normal** (physiological) but if recurrant and in each visit the lady has glycouria, you should investigate her for diabetes.

- **ENDOCRINE GLANDS**
  - **Pituitary gland**

1. FSH and LH ↓ normal decrease due to the –ve feedback by the high percentages of estrogen and progesterone.
2. ACTH, Thyrotrophin, melanocyte hormone and prolactin ↑ that's why melanocyte stimulating hormone increase and the pregnant women have hyperpigmentation of the neck and linea nigra.
3. Prolactin level ↑ until the 30th week of pregnancy then more slowly to term. To prepair the breasts for lactation , will increase steadily until the 30th week then will plateau.

There is a physiological enlargement of the pituitary gland, that's why when the lady during delivery has PPH there will be relative ischemia to the pituitary gland(Sheehan's Syndrome)

  - **Adrenal gland**

1. Total corticosteroids ↑ progressively to term. Due to the increase in ACTH
2. This will ↑ the tendency of pregnant women to develop abdominal strine, glycosuria and hypertension

- **Thyroid gland**
  1. Enlarges during pregnancy, occasionally to twice its normal size. This is mainly due to colloid deposition caused by a lower plasma level of iodine, consequent on the increased ability of the kidneys to excrete during pregnancy.
  2. Oestrogen stimulates or increased secretion of thyroxin in binding globulin.
  3. Both T3 and T4 levels rise. This rise will not indicate hyperthyroidism

That's why you can't investigate a women's thyroid during pregnany, there will be overlap between the symptoms of hyperthyroidism and that of pregnancy .

GENETAL TRACT CHANGES

- **UTERUS**
  1. Uterine muscles grow to 15 times than pre-pregnancy length.
  2. Uterine weight increases from 50 g before pregnancy to 950 g at term.
  3. In the early weeks of pregnancy the growth is by hyperplasia and more partially by hypertrophy of the muscle fibres.
  4. By 20 weeks growth ceases and the uterus expands by distension.
  5. The uterine blood vessels also undergo hypertrophy and become increasingly coiled in the first half of pregnancy but no further growth after that.
  6. The lower uterine segment is that part of the lower uterus and upper cervix lying between the line of attachment of the pertoneum of the utero vesical pouch superiorly and the histological internal os interiorly. Which is the part of the uterus which we'll cut though in case of caesarean section.
- **THE CERVIX**
  1. Becomes softer and swollen in pregnancy, with the result that columnar epithelium lining the cervical canal becomes exposed to the vaginal secretions due to collagen changes
  2. Prostaglandins act on the collagen fibres, especially in the last week of pregnancy. At the same time collagenae is released from leucocytes, which also helps in breaking down collagen. The cervix becomes softer and more easily dilatable the so called ripening of the cervix.
- **VAGINA**
  1. The vaginal mucosa becomes thickness, the vaginal muscle hypertrophies.
  2. There is alteration in the composition of the connective tissue, with the result that the vagina dilates more easily to accommodate the fetus during pentuntion.
  3. Oestrogen → desquamanation of the superficial vaginal mucosal cells with ↑ in vaginal discharge when pathogenesis entre the vagina (**CANDIDA**, trichomas) they will flourish rapidly.

     *****mcq*** What's the most common vaginal infection in pregnancy? CANDIDA**

The surface of the cervix in case of no pregnancy is covered by stratified sequamous externally, and columnar epithelium internally. In pregnancy however, there will be overgrowth of the columnar epithelium covering up the external sequamous epithelium, that's why pregnant ladies complain of redness in the cervix thinking it might be ulcers or so.

■ ALIMENTARY SYSTEM CHANGES:

TRICKY POINT !!

The regurgitation is due to the relaxation of the sphincter NOT due to increase of acidity! In fact the acidity is reduced!!

1. The mouth and the gum become spongy because of intracellular flood retention (prophylactic effect).
2. The lower oesophageal sphincter is **relaxed** which may permit regurgitation of gastric contents and cause heart burn.
3. Gastric secretion is **reduced** and food remains **longer** in the stomach.
4. The intestinal musculature is relaxed with **lower** motility → greater absorption and constipation.

****mcq*****The time of emptying of the stomach in pregnancy is …? INCREASED**

- All the changes in the alimentary system are to increase the digestion time.
- Very important to consider especially in anesthesia for appendectomy, cesarean section or for any reason , because if you anesthetized a lady with her stomach full she might vomit and get aspiration pneumonia.

■ RENAL SYSTEM

1. The smooth muscle of the renal pelvis and ureters **relaxes**, causing their **dilatation**. This increase the capacity of the renal pelvis and ureters from 12 ml to 75 ml allowing chance for urinary stasis and ↑ the chances of urinary **infection**. That's why MSU is a routine invetegation for every pregnant women, even with no symptoms because she might have asymptomatic bacterurea.
2. Urinary tract infection is more common in pregnancy. The muscles of the internal urethral sphincter relax and this together with the pressure of the uterus →degree of **incontinence**
3. The renal blood flow **increases** to the 16th week of pregnancy and the levels off.
4. GFT **increases** by 60% in early pregnancy and remains at the new level until the last 4 weeks of pregnancy when it falls.
5. Tubular reabsortion is unaltered.
6. **Clearance** of many solutes ↑ like urea and creatinine
7. Up to 300 mg of protein many be excreted in **24** hours. If >300 + high BP +edematous >>> might be pregnancy induced hypertension with proteinurea
8. ↑ GFT + progestogen effect → loss of Na.

■ IMMUNE STSTEM CHANGES

1. HCG →↓ **immune** response to pregnancy otherwise the mother's body will reject the baby, and also one of the main causes of recurrent miscarriages is no reduction in the immunity of the mother.
2. IgG, IgA and IgM ↓ from **10th** week of pregnancy reaching their lowest level at **30** weeks and remain so till the end of pregnancy →↑ risk of **infection** in pregnant women.

■ WEIGHT GAIN IN PREGNANCY

- Healthy women will gain around **12** kg of which **9 kg is gained in the last 20 weeks.**
- The elements of weight gain:

- Fetus	3300	gm
- Placenta	600	gm
- Uterus	900-950	gm
- Breasts	400	gm
- Blood	1200	ml
- Fat Deposited	2500	
- Fluid	2600	

The numbers are just for you to know, no need to memorize anything ☺

SUMMARY #1:What the doctor said at the end:

1. Increase in SV,HR, COP, plasma volume, renal blood flow, GFR, gastric emptying time, fibrinogen, UTI infection (especially candida).
2. Decrease in hemoglobin concentration, acidity, immunity, urea, creatinine.

SUMMARY #2 : **Kaplan:**

PHYSIOLOGIC CHANGES IN PREGNANCY

A. Skin

Striae graviclarum-"Stretch marks" that develop in genetically predisposed women on the abdomen and buttocks.
Spider angiomata and palmer erythema-From increased skin vascularity.
Chadwick sign-Bluish or purplish discoloration of the vagina and cervix as a result of increased vascularity.
Linea nigra-Increased pigmentation of the lower abdominal midline from the pubis to the umbilicus.
Chloasma-Blotchy pigmentation of the nose and face.

B. Cardiovascular

Arterial blood pressure-Systolic and diastolic values both decline early in the first trimester, reaching a nadir by 24-28 weeks, then they gradually rise toward term but never return quite to prepregnancy baseline. Diastolic falls more than systolic, as much as 15 mm Hg. Arterial blood pressure is never normally elevated in pregnancy.
Venous blood pressure-Central venous pressure (CVP) is unchanged with pregnancy, but femoral venous pressure (FVP) increases two- to threefold by 30 weeks' gestation.
Plasma volume-Plasma volume increases up to 50% with a significant increase by the first trimester. Maximum increase is by 30 weeks. This increase is even greater with multiple fetuses.
Systemic vascular resistance (SVR)-SVR equals blood pressure (BP) divided by cardiac output (CO). Because BP decreases and CO increases, SVR declines by 30%, reaching its nadir by 20 weeks. This enhances uteroplacental perfusion.
Cardiac output (CO)-CO increases up to 50% with the major increase by 20 weeks. CO is the product of heart rate (HR) and stroke volume (SV), and both increase in pregnancy. HR increases by 20 beats/min by the third trimester. SV increases by 30% by the end of the first trimester. CO is dependent on maternal position. CO is the lowest in the supine position because of inferior vena cava compression resulting in decreased cardiac return. CO is the highest in the left lateral position. CO increases progressively through the three stages of labor.
Murmurs-A systolic ejection murmur along the left sternal border is normal in pregnancy owing to increased CO passing through the aortic and pulmonary valves. Diastolic murmurs are never normal in pregnancy and must be investigated.

Table 1-2. Cardiovascular Changes

Arterial blood pressure	Systolic	↓
	Diastolic	↓↓
Venous pressure	Central	Unchanged
	Femoral	↑
Peripheral vascular resistance		↓

C. Hematologic

Red blood cells (RBC)-RBC mass increases by 30% in pregnancy; thus, oxygen-carrying capacity increases. However, because plasma volume increases by 50%, the calculated hemoglobin and hematocrit values decrease by 15%. The nadir of the hemoglobin value is at 28-30 weeks' gestation. This is a physiologic dilutional effect, not a manifestation of anemia.
White blood cells (WBC)-WBC count increases progressively during pregnancy with a mean value of up to 16,000/mm3 in the third trimester.
Erythrocyte sedimentation rate (ESR)-ESR increases in pregnancy because of the increase in gamma globulins.
Platelet count-Platelet count normal reference range is unchanged in pregnancy.
Coagulation factors-Factors VII, VIII, IX, and X increase progressively in pregnancy, leading to a hypercoagulable state.

D. Gastrointestinal ~

Stomach-Gastric motility decreases and emptying time increases from the progesterone effect on smooth muscle. This increase in stomach residual volume, along with upward displacement of intraabdominal contents by the gravid uterus, predisposes to aspiration pneumonia with general anesthesia at delivery. ~
Large bowel-Colonic motility decreases and transit time increases from the progesterone effect on smooth muscle. This predisposes to increased colonic fluid absorption resulting in constipation.

E. Pulmonary

Tidal volume (Vt)-Vt is volume of air that moves in and out of the lungs at rest. Vt increases with pregnancy to 40%. It is the only lung volume that does not decrease with pregnancy.
Minute ventilation ('Ve)-Ve increases up to 40% with the major increase by 20 weeks. Ve is the product of respiratory rate (RR) and Vt. RR remains unchanged with Vt increasing steadily throughout the pregnancy into the third trimester.
Residual volume (RV)-RV is the volume of air trapped in the lungs after deepest expiration. RV decreases up to 20% by the third trimester. To a great extent this is because of the upward displacement of intraabdominal contents against the diaphragm by the gravid uterus.
Blood gases--The rise in V t produces a respiratory alkalosis with a decrease in Pc02 from 40 to 35 mm Hg and an increase in pH from 7.40 to 7.45. An increased renal loss of bicarbonate helps compensate, resulting in an alkalotic urine.

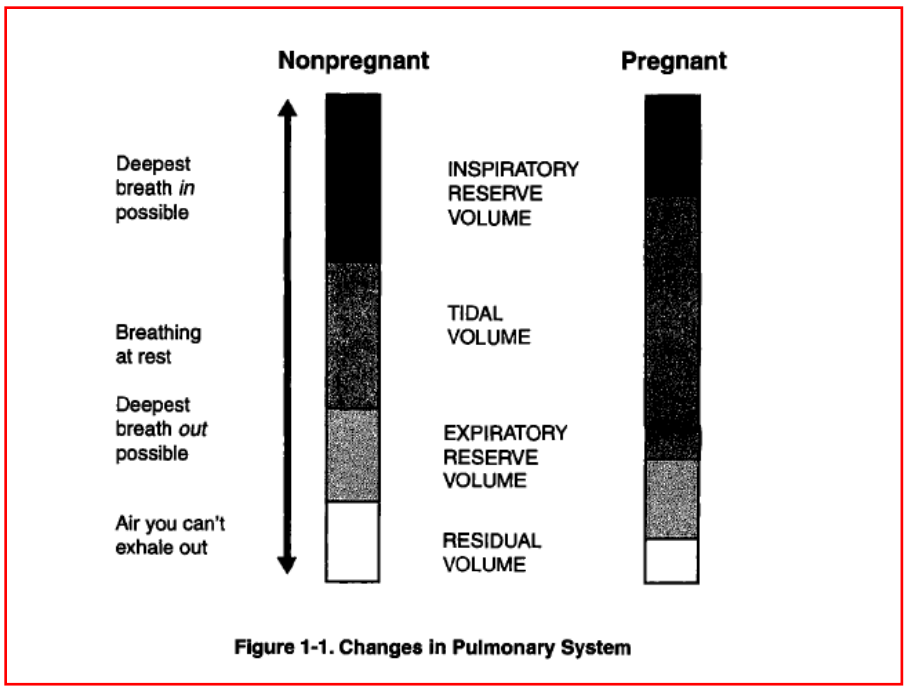


Figure 1-1. Changes in Pulmonary System

F. Renal

Kidneys-The kidneys increase in size because of the increase in renal blood flow. This hypertrophy doesn't reverse until 3 months postpartum.
Ureters-Ureteral diameter increases owing to the progesterone effect on smooth muscle. The right side dilates more than the left in 90% of patients.
Glomerular filtration rate (GFR)-GFR, renal plasma flow, and creatinine clearance all increase by 50% as early as the end of the first trimester. This results in a 25% decrease in serum blood urea nitrogen (BUN), creatinine, and uric acid.
Glucosuria-Urine glucose normally increases. Glucose is freely filtered and actively reabsorbed. However, the tubal reabsorption threshold falls from 195 to 155 mgldL.
Proteinuria-Urine protein remains unchanged.

G. Endocrine

Pituitary-Pituitary size increases by 100% by term from increasing vascularity. This makes it susceptible to ischemic injury (Sheehan syndrome) from postpartum hypotension.
Adrenals-Adrenal gland size is unchanged, but production of cortisol increases two- to threefold.
Thyroid-Thyroid size increases 15% from increased vascularity. Thyroid binding globulin (TBG) increases, resulting in increased total T3 and T4, although free T3 and free T4 remain unchanged.

H. Fetal Circulation

Three in utero shunts exist within the fetus. The **ductus venosus** carries blood from the umbilical vein to the inferior vena cava. The **foramen ovale** carries blood from the right to the left atrium, and the **ductus arteriosus** shunts blood from the pulmonary artery to the descending aorta.

Physiology of menstrual cycle.

OBJECTIVES

- Definition of the normal menstrual cycle
- Phases of the menstrual cycle
- Control of the menstrual cycle through thhypothalalmic pituitary ovarian axis
- Ovulation
- Hormones of the MC
- Two cell theory
- Endometrial changes during the MC

Normal Menstrual Cycle

- What is the mean duration of the MC?
  - Mean 28 days (only 15% of ♀)
  - Range 21-35
- What is the average duration of menses?
  - 3-8 days
- When does ovulation occur?
  - Usually day 14
  - 36 hrs after the onset of mid-cycle LH surge

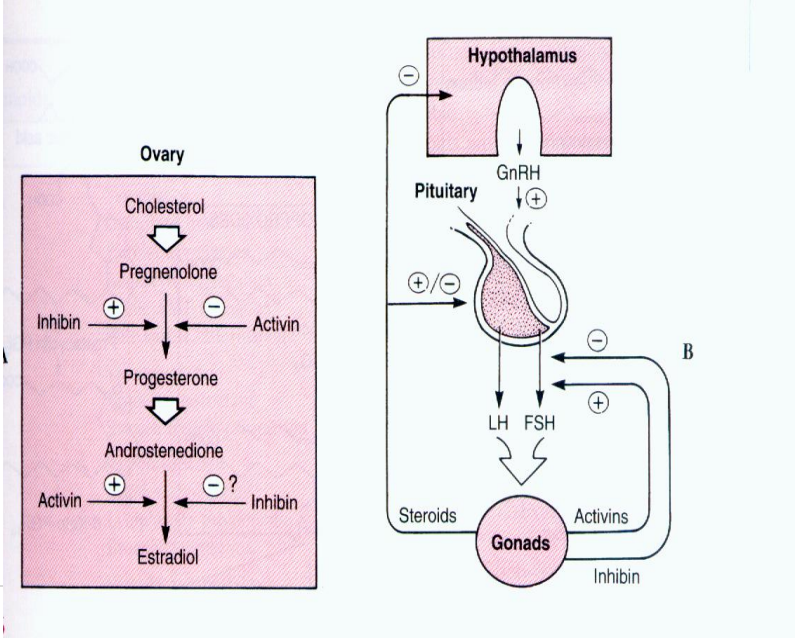
- What regulate the phases of the MC & ovulation?
  - Interaction between hypothalamus, pituitary & ovaries
- What is the mean age of menarche & menopause?
  - Menarche 12.7
  - Menopause 51.4

Gonadotropin releasing hormone is released from the hypothalamus in pulses, to the pituitary which will secrete FSH and LH.

FSH and LH will act on the ovaries (gonads).

In the ovaries under the effect of the FSH development of the follicles will occur then follicles will secrete estrogen.

LH will lead to progesterone secretion after ovulation.



Phases Of The Menstrual Cycle :

The first day of bleeding (mensuration) is day one of the cycle

- Ovulation divides the MC into two phases:
  1-FOLLICULAR PHASE
  - ✓ Begins with menses on day 1 of the menstrual cycle& ends with ovulation

▲RECRUITMENT(there is a certain number of follicles which will undergo for development and only one or sometimes two will reach ovulation)

FSH ⇨ maturation of a cohort of ovarian follicles "recruitment"

⇨only one reaches maturity

Maturation Of The Follicle (Folliculogenesis)

♥ FSH ⇨ primordial follicle

(oocyte arrested in the diplotene stage of the 1st meiotic division surrounded by a single layer of granulosa cells)

⇨⇨Primary follicle

(oocyte surrounded by a single layer of granulosa cells basement membrane &thica cells)

⇨⇨2ry follicle or preantral follicle

(oocyte surrounded by zonapellucida , several layers of granulosa cells &thica cells)

⇨⇨tertiary or antral follicle

2ry follicle accumulate fluid in a cavity "antrum" oocyte is in eccentric position surrounded by granulosa cells "cumulous oophorus"

SELECTION

- Selection of the dominant follicle occurs day 5-7
- It depends on
  - ✓ the intrinsic capacity of the follicle tosynthesize estrogen
  - ✓ highest/and ratio in the follicular fluid
- As the follicle mature ⇨↑ estrogen ⇨↓FSH

"-ve feed back on the pituitary"⇨⇨the follicle with the highest No. of FSH receptors will continue to thrive(The dominant follicle , the one having highest estrogen , even with FHS being reduced the dominant follicle will continue to grow)

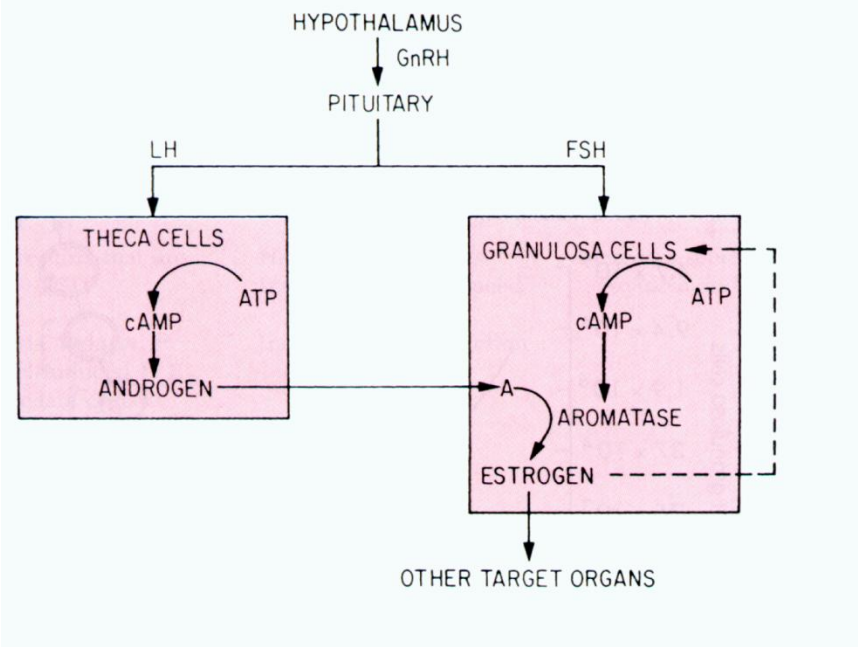
- The other follicles "that were recruited" willbecome atretic

- **FSH ACTIONS**
  - recruitment
  - mitogenic effect ⇨↑ No. of granulosa cells

    ↑ FSH receptor
  - stimulates aromatase activity ⇨ conversion of androgens ⇨estrogens "estrone& estradiol"
  - ↑ LH receptors
- **ESTROGEN**
  - Acts synergistically with FSH to :
    - induce LH receptors
    - induce FSH receptors in granulosa&thica cells
- **LH** ⇨thica cells ⇨ uptake of cholesterol & LDL ⇨androstenedione& testosterone

Two Cell Theory

FSH will act on granulosa cells, producing aromatase that will convert the androgen coming from the theca cells formed by LH leading to estrogen formation.



Folliculogenesis:

Factors that play a role in folliculogenisis:

-INHIBIN

- Local peptide in the follicular fluid(Not in the blood stream like the other hormones)
- -ve feed back on pituitary FSH secreation
- Locally enhances LH-induced androstenedione production (increase androgen production)

-ACTIVIN

- Found in follicular fluid
- Stimulates FSH induced estrogen production(increase estrogen production)
- ↑gonadotropin receptors
- ↓androgen
- No real stimulation of FSH secretion in vivo (bound to protein in serum)

Preovulatory Period

♥ NEGATIVE FEEDBACK ON THE PIUITARY

-↑estradiol&inhibin⇨-ve feed back on pituitary ⇨↓ FSH

-This mechanism operating since childhood

♥ POSITIVE FEEDBACK ON THE PITUITARY

- ↑↑estradiol (reaching a threshold concentration) ⇨⇨ +ve feed back on the pituitary (facilitated by low levels of progestrone) ⇨⇨ LH surge⇨ secretion of progestrone
- Operates after puberty
- +ve feed back on pituitary ⇨↑ FSH

When estrogen is increasing it will have a negative feedback suppressing FSH , but when it keeps increasing and reach a certain threshold it will have a positive feedback will result in LH surge.

♥LH SURGE

- Lasts for 48 hrs
- Ovulation occurs after 36 hrsof the onset of the LH surge.
- Accompanied by rapid fall in estradiol level
- Triggers the resumption of meiosis
- Affects follicular wall ⇨ follicular ruptureovulation
- Granulosa cells ⇨lutenization⇨progestrone synthesis

Ovulation

- The dominant follicle protrudes from the ovarian cortex
- Gentle release of the oocyte surrounded by the cumulus granulosa cells
- Mechanism of follicular rupture

1-↑ Follicular pressure

Changes in composition of the antral fluid ⇨↑ colloidosmotic pressure

2-Enzymatic rupture of the follicular wall

LH & FSH ⇨granulosa cells ⇨ production of plasminogen activator ⇨↑plasmin⇨↑fibrinolytic activity ⇨breake down of F. wall

LH ⇨↑prostglandin E ⇨↑ plasminogen activator ⇨↑ PG F2α⇨↑ lysosomes under follicular wall

Luteal Phase

LASTS 14 days (usually the luteal phase is constant, the variation is in the follicular phase)

FORMATION OF THE CORPUS LUTEUM

- After ovulation the point of rupture in the follicular wall seals
- Vascular capillaries cross the basement membrane & grow into the granulosa cells ⇨↑ availability of LDL-cholestrole(from the blood)

LH ⇨↑ LDL binding to receptors ⇨↑ 3α OH steroid dehydrogenase activity ⇨↑progesterone

- Marked ↑ in progestronesecretion (progesterone only AFTER ovulation while estrogen before and after)

- Progestrone actions:

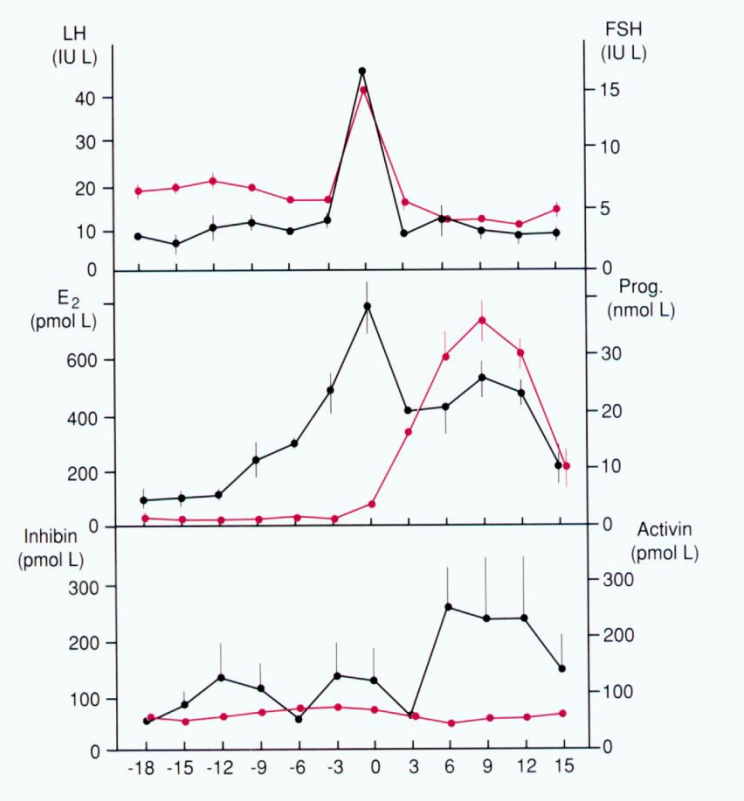
  -suppress follicular maturation on theipsilateral ovary

  -thermogenic activity ⇨↑ basal body temp

  -endometrial maturation

- Progestrone peak 8 days after ovulation (Day 22 MC)(That's why want to check for ovulation we check progesterone at day 22 , it should be the highest level)
- Corpus luteum is sustained by LH
- It looses its sensitivity to gonadotropins ⇨luteolysis⇨↓estrogen &progestrone level ⇨ desquamation of the endometrium "menses"
- ↓estrogen&progestrone⇨↑ FSH &LH(Beginning of the new cycle)
- The new cycle stars with the beginning of menses
- If prgnancy occurs ⇨hCGsecreation⇨ maintain the corpus luteum

Hormonal Profiles During The Menstrual Cycle



Endometrial Changes During The Menstrual Cycle

1-Basal layer of the enometrium

-Adjasent to the mometrium

-Unresponsive to hormonal stimulation

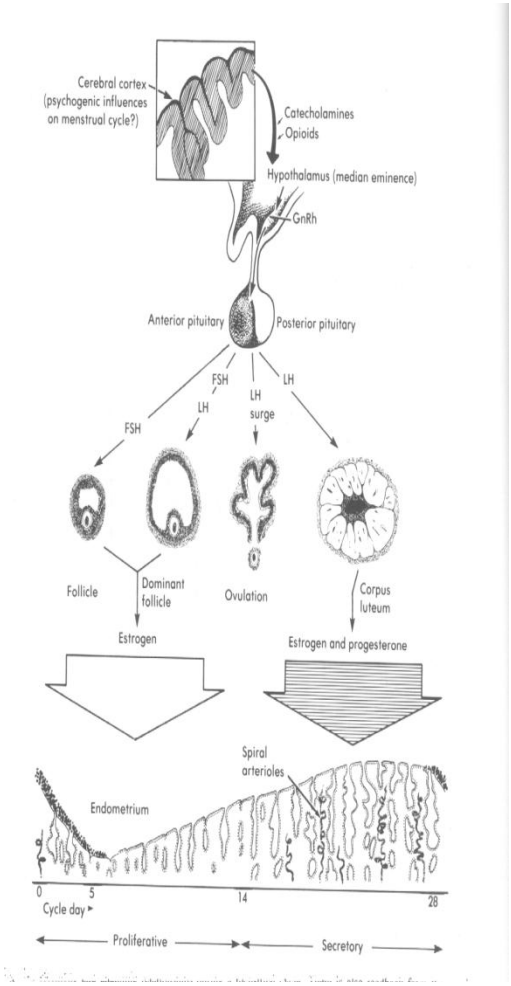
-Remains intact throughout the menstrual cycle

2-Functional layer of the endometrium

Composed of two layers:

-zonacompacta⇨ superficial

-Spongiosum layer



1-Follicular /proliferative phase

Estrogen ⇨ mitotic activity in the glands &stroma⇨

↑enometrial thickness from 2 to 8 mm

(frombasalis to opposed basalis layer)

2-Luteal /secretory phase

Progestrone⇨- Mitotic activity is severely restricted

-Endometrial glands produce then secrete glycogen rich vacules

-Stromal edema

-Stromal cells enlargement

-Spiral arterioles develop, lengthen & coil

Menstruation

- Periodic desquamation of the endometrium
- The external hallmark of the menstrual cycle
- Just before menses the endometrium is infiltrated with leucocytes
- Prostaglandins are maximal in the endometrium just before menses
- Prostaglandins ⇨ constriction of the spiral arterioles ⇨ischemia & desquamation

Followed by arteriolar relaxation, bleeding & tissue breakdown

Hypothalamic Role In The Menstrual Cycle

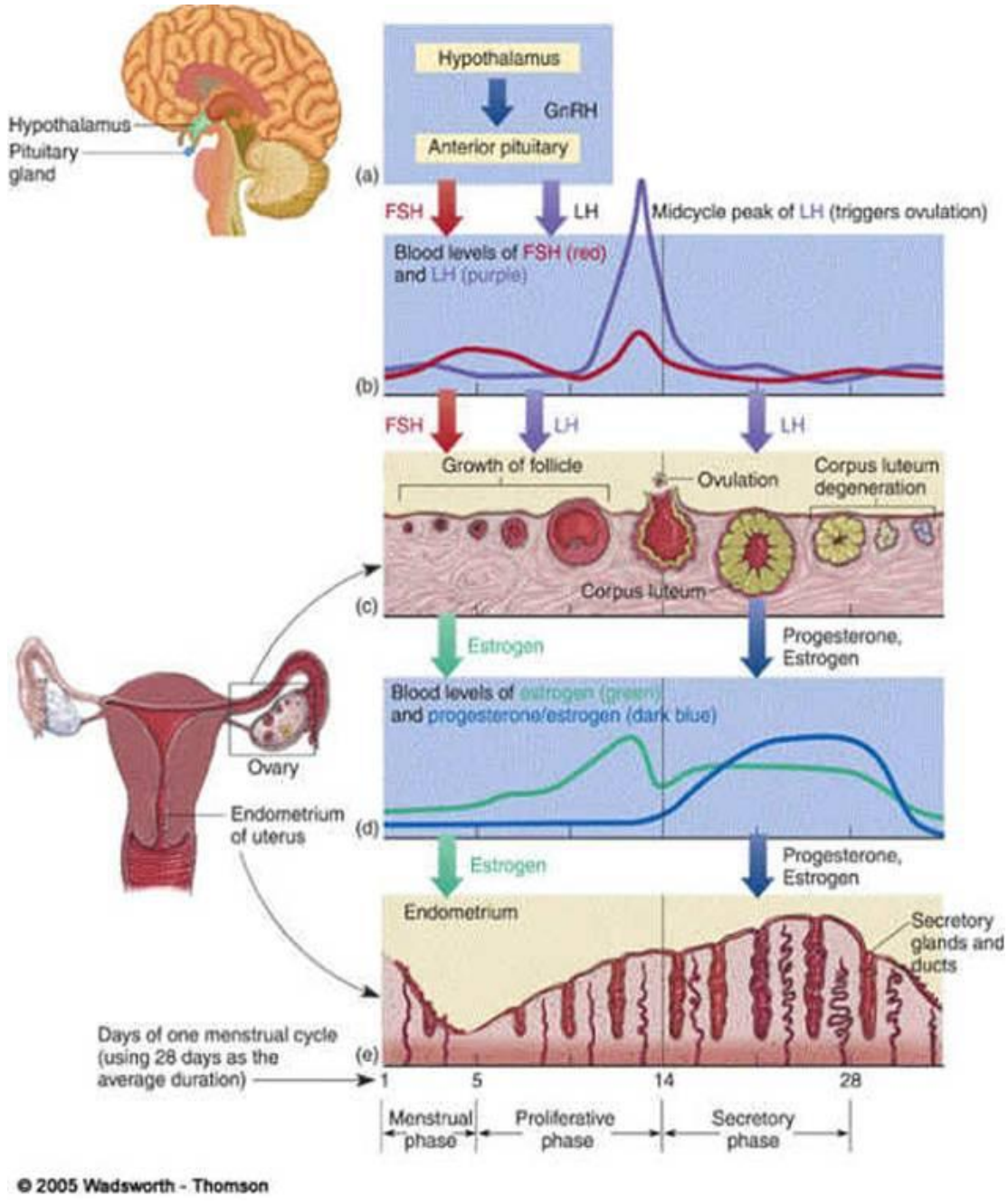
- The hypothalamus secretes GnRH in a pulsatile fashion(if steady it will suppress the ovaries)
- GnRH activity is first evident at puberty
- Follicular phase GnRH pulses occur hourly
- Luteal phase GnRH pulses occur every 90 minutes
- Loss of pulsatility⇨down regulation of pituitary receptors ⇨↓ secretion of gonadotropins
- Release of GnRH is modulated by –ve feedback by:

steroids

gonadotropins

- Release of GnRH is modulated by external neural signals

Summary:



Management of Labour.

Objectives:

- Managements of the stages of labour
- Pain relief in labour
- Fetal assessment (antenatal & intra-partum)

Physiology of labour: increasing frequency of contraction is associated with the formation of gab-junctions between uterine myometrial cells.

Uterine changes: the contractile upper uterine segment contain mostly smooth muscles fibers, becomes thicker as labour progress, exerting forces that expel the fetus down.

Management of labour

- **Definition of labour:**
  Progressive cervical effacement and dilatation resulting from regular uterine contractions that occur at least every 5 minutes and last 30-60 seconds

  It is MCQ'squestion so pay attention to it especially the criteria:

  1- Dilatation and effacement.
  2- Regular contraction
  3- Lasting for 30-60 seconds so if last 10 second this is not labour.

  Q- what is irregular contraction?
  If the patient has the contraction firstly every half hour and then every 10 minutes so she is not really in labour yet

- **Braxton Hicks:**
  contractions Not associated with cervical changes (Most of patient around 9 month of pregnancy = starting from 36 weeks of gestation experience Braxton Hicks because uterus getting prepared to go in labour however it is not a labour.)

- **Lightening:**
  Descent of the fetal head into the pelvis (at the beginning of 9 month the lightining take place especially in primigravida because abdominal muscle is strong not relaxed so puching the baby however in multigravida descent doesn't happen until they go into active phase of labour.)

Stages of Labour

First stage of labour

- Start from onset of true labour pain → full dilatation of cervix(equal to 10 cm any thing less than 10 cm is not consider to be full dilatation)
- In primigravida → 12 hour duration (it is very IMP to know duration if there is any change in the duration)
- In multigravida → 6 hours duration (in any stage you have to take an action)
  Chiefly concerned with preparation of the birth canal as to facilitate expulsion of the fetus in the second stage

- **It has 2 phases:**
  1. **A latent phase** up to 3 cm dilatation of cervix:
     - is variable: up to 8 hours in primi
     - 4 hours in multi
  2. **An active phase** from 3 cm to full dilatation of cervix.
     Rate of dilatation :

     - 1 cm/hour in primigravida
     - 1.5 cm/ hour in multigravida
       - (with epidural anesthesia things may take longer)

Purpose of latent phase is to prepare the cervix for rapid dilation through effacement

Purpose of active phase is rapid cervical dilation

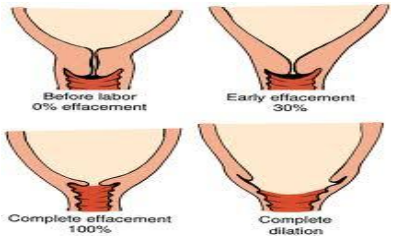
❖ **Dilatation of the cervix:**

- Dilatation usually measured by fingers but recorded in cm.
- Dilatation relates with dilatation of internal os.
- If internal os is opened you should feel the head of the baby.
- If the membrane is rupture you could feel the skull of the baby.
- Our recordingif the cervix is dilated depend on the internal os if it is opened or not so we can tell the cervix is dilated or not.

Dilatations occur as the passive lower uterine segment is thinned and pulled up by the contractile upper segment.

❖ **Effacement or taking up of cervix**

- (It is very IMP to know the difference between effacement and dilatation).
- Muscle fibers of cervix are pulled upward and merges with the fibers of the lower uterine segment.
- Cervix becomes thin during first stage.
- In primi: effacement precedes dilatation of the cervix.
- In multi: both occur simultaneously.
- Effacement is determined by the length of the cervical canal in the vagina
  (There is no canal anymore when you put your finger, as it is around a ring not a slender)
- Effacement is expressed in terms of percentage.



Effacement Cervical softening and thinning occur as increasingly levels of oxytocin and prostaglandin lead to breakage of disulfide linkages of collagen fibers resulting in increasing water content

First stage of labour

Maternal system	Fetal system
• General condition remains unaffected • Pulse rate increases by 10-15 bpm during contraction with the settle down to its previous rate in between contractions • Systolic BP increase by 10 mm Hg during contraction • Temperature remains unaffected • We have something called Patrogram documenting every thing in labour if any thing is not normal the nurse should inform the physician	• As so long as the membranes are intact, usually there is no adverse effect on the fetus BUT However, during contraction there may be slowing of FHR by 10-20 bpm which soon returns to its normal as the intensity of contraction diminishes CTG is like an ECG for the fetus Qs why there is decrease in FHR during contraction? Because during contraction there is a reduction in placental blood flow which lead to drop in FHR and after contraction it goes to normal

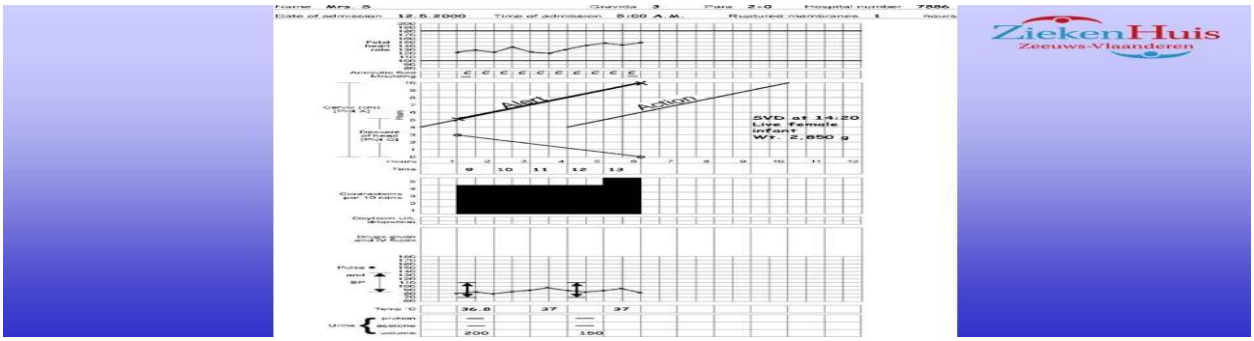
❖ Management of labour

- **Initial assessment:**
  - **History**: Onset, strength, frequency of contractions.
  - **Leakage of fluid** it is very IMP to ask when fluid start leaking? Because any body who has been leaking fluid for a long time (24-48hours) should induce or start labour especially for term pregnancy as well as cover them with antibiotics.
  - **Vaginal bleeding** patient should not bleed in labour but it is ok to have blood stain mucous however frank bleeding is not ok.
  - **Fetal movement.**
  - **Medications** Heparin had to be stopped in labour.
  - **Last oral intake** patient must fast even vaginal delivery because we don't know what's gonna happen we may need cesarean section.
  - **Review** of past obstetric history, prenatal lab tests, gestational age, parity, size of previous infants, any antenatal complications.

❖ Management of the first stage of labour

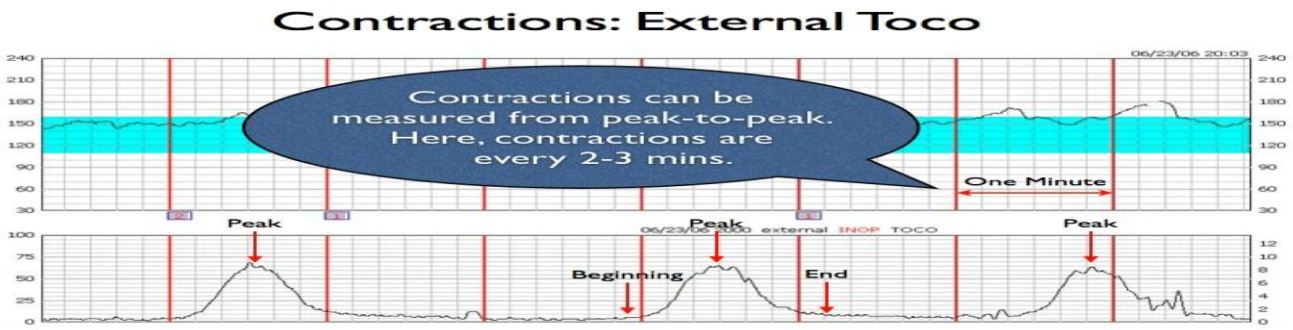
- Informed consent on management of labour& delivery.
- Maternal position: lateral recumbent position, avoid supine hypotension(don't make patient laying back because it may compress on pelvic vessels and cause hypotension).
- **Partogram**:
  - Iv fluids & avoid oral intake
  - Maternal vital signs every 1-2 hours
  - Input-output monitoring
  - Analgesia
  - Fetal heart rate monitoring (CTG)we have two ways monitoring FHR:
    1. External we put it on the belly and see recording
    2. Internally put the electrodes on the scalp of the baby the membrane must be ruptured
  - Uterine contractions monitoring
  - Vaginal examination for cervical dilatation &poistion in
  - active phase every 2 hours
  - Amniotic membranes status & amniotic fluid colour(color – amount – smell – quality of fluid)

Monitoring progress of labour (Partogram)



Mechanics of labour

The Power: force generated by uterine noitcartnoc

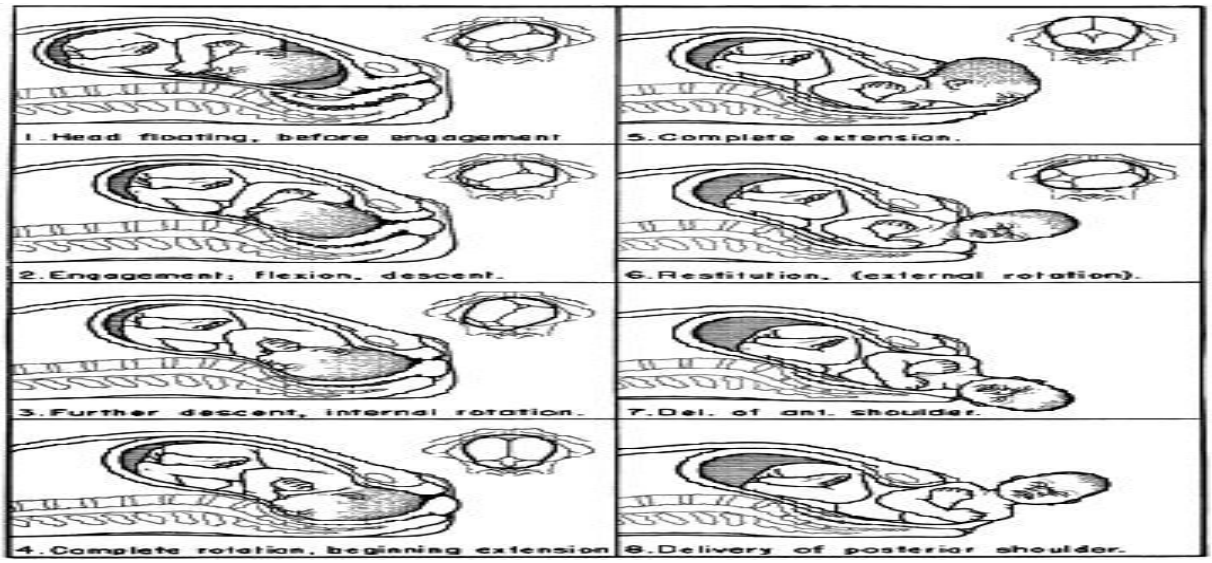


Second Stage of labour

- From full dilatation of cervix till delivery of the neonate.
- The mother has a desire to bear down with each contraction.
- Last from:
  - 30 minutes to 3 hours in primigravida.
  - 5-30 minutes in multigravida.

Purpose of second stage is descent of the fetus through the birth canal as maternal pushing efforts augment the uterine contractions

Mechanism of labour

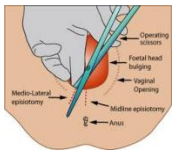


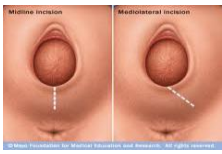
❖ Management of the second stage of labour

- **Molding** (alteration of the relationship of the fetal cranial bones to each other as a result of compression forces by the bony pelvis).
- **Caput** (localized edematous swelling of the scalp caused by pressure of the cervix on the presenting portion of the fetal head)--- gives false impression of fetal descent.
- **Crowning**(when the largest diameter of the fetal head is encircled by the vulvar ring)it is IMP because this is the time to do an Episiotomy
- Vaginal examination every 30 minutes
- Maternal position– any comfortable position for bearing down
- Bearing down---with each contraction
- Delivery of the fetal head---manual perineal support
- Fetal airway clearance
- Umbilical cord clamping
- Place the infant under warmer

Episiotomy

- Incision in the perineum after crowning to aid delivery and avoid laceration of perineum.
- **Types:**
  1. Right mediolateral.
  2. Left mediolateral.
  3. Central.





PERINEAL LACERATION

- **4 degrees :**
  - **First degree:** laceration involving the vaginal epithelium or perineal skin.
  - **Second degree:** laceration extending into the sub-epithelial tissues of the vagina or perineum with or without involving the perineal body.
  - **Third degree:** laceration involving anal sphincter.
  - **Fourth degree:** laceration involving rectal mucosa.

❖ Third stage of labour

- The interval between the delivery of the infant and complete delivery of the placenta & membranes.
- Duration is 5-30 minutes (if it is more than 30 minutes you are dealing with a condition called retained placenta).
- **Signs of placental separation**: IMP !!
  (If you pulled the placenta that is not separated it will lead to uterine inversion the uterus become like sock so please don't do that)
  1. Fresh blood show from vagina.
  2. The umbilical cord lengthens outside the vagina.
  3. The fundus of the uterus rises up.
  4. The uterus becomes firm & globular.
- Make sure that you removed all the placenta if there is a remnant of the placenta this is not good it will lead to infection and it is a life threatening condition .
- The placenta should be examined to ensure that it is complete.
- The blood loss should be estimated.

Purpose is delivery of the placenta

❖ Forth stage of labour

- The hour immediately after the delivery.
- Needs close observation of: blood pressure, pulse rate and uterine blood loss.
- Watch for post partum hemorrhage.

Pain relief in labour

- Goal: effective pain relief to the mother that is safe for her & the fetus with minimal side effects on the progress & outcome of labour
- **Non pharmacological method**:
  - ✓ Back massage
  - ✓ Acupuncture
  - ✓ Hypnosis
  - ✓ Breathing exercises
- **Pharmacological methods**:
  - ✓ Narcotic analgesics– cross the placenta – cause fetal respiratory depression (Nitrous oxide, pethidine)
  - ✓ Epidural analgesia: The most effective

    Contra indicated if-coagulo-pathy, infection at needle site, severe hypo-volemia

    Side effects: Hypotension, headache, impaired ability to push, prolonged second stage (15 Minutes)

  - ✓ Pudendal block: for S2-S4

    for the second stage of labour

    for instrumental delivery

SUMMARY

- **Definition of labour:**

Progressive cervical effacement and dilatation resulting from regular uterine contractions that occur at least every 5 minutes and last 30-60 seconds

- **Braxton Hicks:** contractions Not associated with cervical changes
- **Lightening:** Descent of the fetal head into the pelvis
- **Dilatation of the cervix**

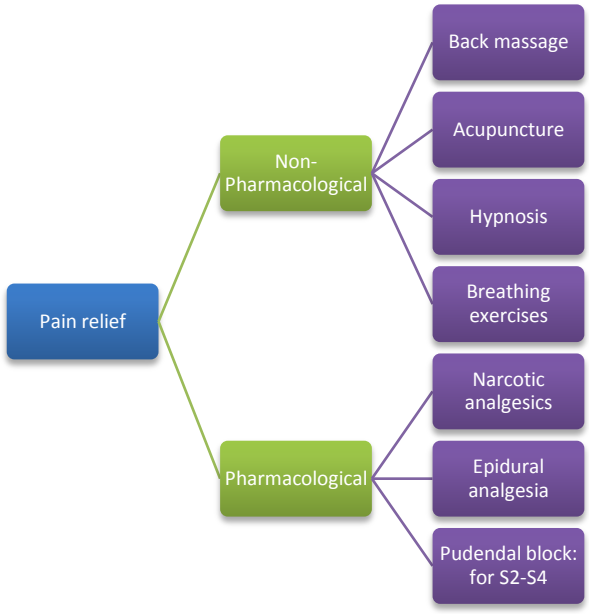
Dilatation usually measured by fingers but recorded in cm

Dilatation relates with dilatation of internal os

- **Effacement or taking up of cervix**

Muscle fibers of cervix are pulled upward and merges with the fibers of the lower uterine segment

Cervix becomes thin during first stage



From 428 booklet :stages of labour

	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.

Mechanisms of labor (Cardinal movements of labor):-

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.

Preterm Labour& Premature Rupture Of Membrane.

◆ **Extra notes from Essentials of obstetrics and gynecology 5th edition**

PRETERM LABOUR

❖ Definition:

- Labor that occurs after **24** weeks but before **37** completed weeks,
- Although it has an incidence of 10%, its contribution to neonatal morbidity and mortality is high ranges from 50 – 70%.
- Any delivery before 24* weeks is a miscarriage not a preterm labor
- *In some countries or centers preterm labor is any labor that occurs after 20 weeks and before 37 weeks, so it depends on the health care system status
- Preterm birth is defined as that occurring after **20 weeks and before 37** completed weeks of gestation. Labor that occurs between these gestational ages is defined as preterm labor. Internationally, the lower boundary defining preterm birth varies between **20 and 40** weeks.

I. Etiology and Risk factors

A] Idiopathic: is the commonest one.

- Low socioeconomic class.
- Previous preterm labor. With one PTL "preterm labor" the relative risk in the next pregnancy is 3.9, it increases to 6.5 with two.
- Repeated spontaneous abortions.

B] Obstetrics causes:

1- Multiple pregnancy "twins". As long as the incidence of multiple pregnancy is rising the incidence of PTL will rise as well

2- Premature preterm rupture of membrane.

3- Genital tract infection as bacteria vaginosis and **B streptococcus "MCQ"**.

4- Cervical incompetence.a medical condition in which a pregnant woman's cervix begins to dilate (widen) and efface (thin) before her pregnancy has reached term

5- Uterine anomalies.

C] Iatrogenic causes

- Induction of labor or CS. for obstetrics causes as PET "Pre-eclamptictoxaemia", placenta Previa and Abruptioand antepartum hemorrhage.

II. DIAGNOSIS

- Documented uterine contractions"4 contractions per 20 minutes".By history, physical examination & CTG.
- Documented cervical changes as cervical effacement "thinning" of 80%, or cervical dilatation of 2 cm or more. Note the progression of the changes

MANAGEMENT

- Put the patient on CTG to confirm uterine activity
- Assess cervical status, progress of labour and presenting part "cephalic ,breech..etc".
- Vaginal swab for bacteria vaginosis and B streptococcus and give antibiotic
- Hydrate the patient Maternal dehydration may trigger the secretion of ADH by the posterior pituitary. It is thought that oxytocin may also be released at the same time, bringing about uterine contraction before the optimum time. These uterine contractions, or uterine "irritability" (low intensity, high frequency contractions) of preterm labor are often treated with maternal hydration. Women at risk for preterm labor are encouraged to drink copious amounts of water throughout the day. And, if hospitalized for contractions, hydration with a bolus of IV fluid is often effective to "quiet" the uterus.

TOCOLYTIC THERAPY

❖ B-Adrenergic agonist (B-sympathomimetic agent):

- **Mechanism:**

  Convert ATP into cAMP in the cell causing decrease of the free calcium ion.
  Required close monitoring
- **Side effects:**
  - Mainly cardiovascular as increased heart rate and hypotension
  - Chest pain in 1-2% from myocardial ischemia.
  - Rarely pulmonary edema particularly with concurrent corticosteroid therapy.
  - Increased liver and muscle glycogenolysis causing hyperglycaemia. 2nd increase in insulin causehypokalaemia.
- **Most commonly used drug in this group is Ritodrin hydrochloride (Yutopar)**

❖ Magnesium sulphate:

- **Mechanism:**

  Compete with calcium for entry into the cell at the time of depolarization so there is decrease of intracellular calcium. Easily reaches the toxic level so, you have to monitor the patient closely

- **Side effects:** more serious than Ritodrin's
  - Warm and flushing
  - Respiratory arrest
  - Fetal hypotonia due to decrease calcium

❖ Prostaglandin synthetase inhibitors:

- **Side effects:**
  - Decrease fetal renal blood flow and cause oligohydraminose.
  - Premature closure of ductusarteriosus, which lead to pulmonary Hypertension.
  - Necrotizing enterocolitis.
  - Fetal intracranial hemorrhage.
- **Indomethacin is the most commonly used.**

❖ Calcium channel blockers:

- **Nifedipine:**

  Inhibits the inward current of calcium iron during the 2nd phase of the action potential of uterine muscle.

- **Side effects:**
  - Headache
  - Hypotension
  - Flushing
  - Tachycardia

❖ Oxytocin Antagonist: very effective

- **Side effects:**
  - Nausea, dizziness, headache, and flushing.
  - Expensive drug.
- **Most commonly used drug in this group is Atosipan(tractocil)**

❖ CONTRAINDICATIONS TO TOCOLYTIC THERAPY:

1- Severe PET "Pre-eclamptictoxaemia"
2- IUGR "Intrauterine Growth Restriction"
3- Severe APH "Antepartum Hemorrhage"
4- Fetal anomalies "major anomalies"
5- Chorioamnionitis"inflammation of the fetal membrane" you have to stabilize the mother then deliver her.
6- Maternal heart disease "some times it is contraindicated to give tocolytic but you can try to give another drugs"

CORTICOSTEROID THERAPY

- Reduces fetal mortality, incidence of RDS "respiratory distress syndrome", and intracranial hemorrhage.
- Stimulate fetal pnemocyte 2 cell to produce surfactant
- Statistically sig.effect up to 34 weeks.
- Betamithasone IM 12 mg given twice 24 h. Apart.
- Optimal benefit is from 24h – 7 days. "Wait or try to delay the labor for At least 24 hours after receiving the drug to get benefit"

LABOUR AND DELIVERY

- Should be in a well equipped center with good SCBU "NICU"
- Continuous fetal monitoring
- Forceps and episiotomy for cephalic presentation
- C.S. for breeches if weight is less than 1500 gms.

Premature Rupture of Membrane

•Definition:

- Rupture of the membrane before the onset of labor at any stage of gestation.
- It is defined as amniorrhexis (spontaneous rupture of membranes as opposed to amniotomy) before to the onset of labor at any stage of gestation.

•CAUSES:

- In majority of cases no clear cause can be found.
- Vaginal infection, bacteria vaginosis and group B streptococcus.
- Cervical incompetence.
- Abnormal membrane.

•DIAGNOSIS:

- History of fluid loss per vagina.
- Visualization of amniotic fluid in the vagina by sterile speculum.
- +Ve NITRAZIN test.Alkaline amniotic fluid turns yellow nitrazin reagent to blue colour. Blood, cervical mucus and alkaline urine give false +ve results.
- +ve fern test. Required slide and microscope so, it is Not used any more.
- USS "ultrasound": Marked decrease or absent liquor.
- USS "ultrasound": Confirm gestation age and exclude fetal anomalies.

•COMPLICATIONS:

1- **Premature labor**: Amniotic fluid contains prostaglandins.
2- **Chorioamnionitis**:The amniotic fluid has bacteriostatic properties and acts as a mechanical barrier against infection.
3- **Fetal sepsis**.
4- **Lung hypoplasia** if occurs before 24 weeks.

•MANAGEMENT:

- The management depends mainly on the gestation age:

A] 36 weeks or more → IOL "Induction of labour".
B]< 36 weeks →expectant management, unless there evidence of chorioamnionitis.

- **EXPECTANT MANAGEMENT**
  - Rest in hospital.
  - Early detection of Chorioamnionitis (immediate delivery) by checking WBCs and C reactive protein twice weekly.
  - High vaginal swab for culture.
  - Prophylactic antibiotics for 10 days.

- **Rule of tocolytics:** it is not advisable
  1- Allow time for corticosteroids to work.
  2- Contraindicated in the presence of infection.

- **Rule of corticosteroids:**
  1- Significant value for pregnancy less than 34weeks.

- **ChorioamnionitisSymptoms:**
  1- Maternal pyrexia >38 C.
  2- Tender irritable uterus.
  3- Foul smelling vginal discharge.
  4- Fetal tachycardia.

SURFACTANT

- Produced by pnemocyte type 2 cells.
- Consists mainly of phospholipids, neutral lipids, proteins and carbohydrates.
- Measured as a ratio (lecithin / sphyngomyelin) mature lung >2.
- Decreases alveolar surface tension, maintains alveoli open at a low internal alveolar diameter and decrease intra alveolar fluid.

Post Term + Induction of Labor.

POSTTERM PREGNANCY

A 21-year-old primigravida at 42 weeks' gestation by dates comes to the outpatient prenatal clinic. She has been seen for prenatal care since 12 weeks' gestation, confirmed by an early sonogram. She states that fetal movements have been decreasing. Fundal height measurement is 42 cm. Her cervix is long, closed, posterior, and firm. Nonstress test is reactive, but amniotic fluid index is 4 cm.

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Definition

- **Academic.** The most precise definition is a pregnancy that continues for ≥40 weeks or ≥280 days postconception. This includes 6% of all pregnancies.
- **Practical.** Because most of the time the date of conception is not known, a practical definition is a pregnancy that continues ≥42 weeks or ≥294 days after the first day of the last menstrual period.
- **Statistics.** Generally, 50% of patients deliver by 40 weeks, 75% by 41 weeks, and 90% by 42 weeks. These statistics assume ovulation occurred on day 14 of a 28-day menstrual cycle. These figures probably overstate the actual number because up to half of these patients had cycles longer than 28 days.

Etiology. The most common cause of true postdates cases are idiopathic (no known cause). It does occur more commonly in young primigravidas and rarely with placental sulfatase deficiency. Pregnancies with anencephalic fetuses are the longest pregnancies reported.

Significance. Perinatal mortality is increased two- to threefold. This is a direct result of changes on placental function over time.

- **Macrosomia syndrome.** In most patients, **placental function continues** providing nutritional substrates and gas exchange to the fetus, resulting in a healthy but large fetus. **Cesarean rate is increased** owing to prolonged or arrested labor. Shoulder dystocia is more common with risks of fetal hypoxemia and brachial plexus injury.
- **Dysmaturity syndrome.** In a minority of patients, **placental function declines** as infarction and aging lead to placental scarring and loss of subcutaneous tissue. This that is responsible for the increased perinatal morbidity and mortality. **Cesarean rate is increased** owing to nonreassuring fetal heart rate patterns. Oligohydramnios results in umbilical cord compression. Hypoxia results in acidosis and in utero meconium passage.

Management. Management is based on 2 factors.

- **Confidence in dates.** Identify how much confidence can be placed on the gestational age being truly >42 weeks.
- **Favorableness of the cervix.** Assess the likelihood of successful induction of labor by assessing cervical dilation, effacement, position, consistency, and station. The Bishop score is a numerical expression of how favorable the cervix is and the likelihood of successful labia induction.
  - Favorable cervix is dilated, effaced, soft, and anterior to mid position. Bishop score is ≥8.
  - Unfavorable cervix is closed, not effaced, long, firm, and posterior. Bishop score is ≤5.

Bishop Scoring Method

Parameter\Score	0	1	2	3
Position	Posterior	Intermediate	Anterior	-
Consistency	Firm	Intermediate	Soft	-
Effacement	0–30%	31–50%	51–80%	>80%
Dilation	0 cm	1–2 cm	3-4 cm	>5 cm
Fetal station	-3	-2	-1, 0	+1,+2

Patients can be classified into 3 groups.

- **Dates sure, favorable cervix.** Management is aggressive. There is no benefit to the fetus or mother in continuing the pregnancy. Induce labor with IV oxytocin and artificial rupture of membranes.
- **Dates sure, unfavorable cervix.** Management is controversial. Management could be aggressive, with cervical ripening initiated with vaginal or cervical prostaglandin E_2 followed by IV oxytocin. Or management could be conservative with twice weekly NSTs and AFIs awaiting spontaneous labor.
- **Dates unsure.** Management is conservative. Perform twice weekly NSTs and AFIs to ensure fetal well-being and await spontaneous labor. If fetal jeopardy is identified, delivery should be expedited.

Table 8-4. Placental Function in Post-term Pregnancy

Maintained	Deteriorates
Macrosomia (80%)	Dysmaturity (20%)
Difficult labor and delivery	Placental insufficiency
↑ C section (forceps, vacuum extractor, shoulder dystocia, birth trauma)	↑ C section (acidosis, meconium aspiration, oxygen deprivation)

Management of Meconium. Previous recommendations to prevent meconium aspiration syndrome (MAS) included:

- **In labor**, amnioinfusion (with saline infused through an intrauterine catheter) to dilute meconium and provide a fluid cushion to prevent umbilical cord compression.
- **After the head is delivered**, suction the fetal nose and pharynx to remove any upper airway meconium.
- **After the body is delivered**, visualize the vocal cords with a laryngoscope to remove meconium below the vocal cords.

Newer recommendations (American Heart Association, American Academy of Pediatrics):

- **Amnioinfusion** may be helpful to prevent umbilical cord compression; okay to perform it.
- **Suctioning of fetal nose and pharynx** makes no difference in preventing MAS; do not routinely perform.
- **Laryngoscopic visualization** of vocal cords is only indicated if the neonate is depressed; perform selectively.

Induction of labor

DEFINITION➡Induction of labor is defined as an intervention designed to artificially initiate uterine contractions leading to progressive dilatation and effacement of the cervix and birth of the baby. This includees both women with intact membranes and women with spontaneous rupture of the membranes but who are not in labor.But delivery will come faster with the SROM.

INDICATIONS:

- Post-term pregnancy ➡ most common (after 40w is considered post term, but we wait until 41 w then we deliver her in the 42).
- PROM (premature rupture of membranes)
- IUGR (intra-uterine growth retardation) (so they don't die)
- Non-reassuring fetal suvillence. (e.g. baby is not moving …….)
- Maternal medical conditions ➡ DM (2nd most common, we don't wait until 42w she shouldn't even reach 40w), renal disease, HPT, gestational HPT, significant pulmonary disease, antiphospholipid syndrome
- Chrioamnionitis
- Abruption
- Fetal death

RISKS of IOL:

- ⬆ rate of operative vaginal deliveries (because vagina might not be prepared)
- ⬆ rate of CS (also because vagina might not be prepared)
- Excessive uterine activity
- Abnormal fetal heart rate patterns
- Uterine rupture
- Maternal water intoxication
- Delivery of preterm infant due to incorrect estimation of GA (that's why we should compare gestational age with US).
- Cord prolapse with artificial rupture of membrane (ARM) (umbilical cord is damaged when exposed to air and vagina, this is an emergency they will take her immediately to CS).

gnigami eht reilrae eht fo noitamitse retteb eht AG☺

CONTRAINDICATIONS:★

(mostlycontraindications of IOL are Contraindications to labor or vaginal delivery)

- Previous myomectomy entering the cavity (because contractions might be very strong on the scar causing uterine rupture).
- Previous uterine rupture
- Fetal transverse lie
- Placenta previa

- Vasa previa
- Invasive Cx Ca
- Active genital herpes
- Previous classical or inverted T uterine incision
- 2 or more CS (they have to do CS they are contraindicated to vaginal).

PREREQUISITES:

- assess the following
- Indication / any contraindications
- GA
- Cxfavourability (Bishop score) (if score >6 then its ok, if less than it might not be successful)
- Pelvis, fetal size & presentation
- Membranes status
- Fetal heart rate monitoring prior to IOL
- Elective inductions

Cervix (Cx) ripening prior to IOL

Indication➡ if the Bishop score is ≤ 6

- The state of the Cx is an important predictor of successful IOL

Methods :

1. **Intracervical** PGE2 gel ➡0.5 mg/6hrs----3 doses
2. **Intravaginal**PGE2 gel ➡1-2 mg/6hrs----3doses
   - PGE2 gel ⬇ the rate of not being delivered in 24 hrs
   - ⬇ the use of oxytocin for augmentation of labor
   - PGE2 gel ⬆ the rate of uterine hyperstimulation
3. **Misoprostol**➡ Should not be used for term fetuses
4. **Mechanical**methods:

➢ Foley Catheter:

- It is introduced into the cervical canal past the internal os, the bulb is inflated with 30-60 cc of water
- It is left for up to 24 hrs or until it falls out

- Contraindications ➡Low laying placenta, antepartum Hg, ROM, or cervicitis
- No difference in operative delivery rate, or maternal or neonatal morbidity compared to PG gel.

➢ Hydroscopic dilators (Eg.Laminaria tents): Higher rate of infections.

Induction Of Labor

1-Oxytocin with Amniotomy:

- IV, mix it with saline, Half life 5-12 min. (titrate it SLOWLY increasing the dose until we reach 3 contractions/10minutethen maintain the dose).
- A steady state uterine response occurs in 30 min or >
- Fetal heart rate & uterine contractions must be monitored.
- If there is hyperstimulation or nonreassuring fetal heart rate pattern ➡ D/C infusion (ie: decelerate it or even stop it).
- Women who receive oxytocin were more likely to be delivered in 12-24 hrs than those who had amniotomyalone & less likely to have operative delivery.

So: do amniotomy if not that effective give oxytocin.

2-PGE2(most commonly used):

- For women with favorable Cx➡ PGE2 ⬇ the rate of operative delivery & failed IOL when compared to Oxytocin
- PGE2 ➡⬆ GIT side-effects like nausea, pyrexia & uterine hyperactivity (specially the local because she might have the labor & the drug hasn't melted completely, so be careful with women with previous CS or high parity).

3-Sweeping of the membranes:(mcq) ★

- Vaginally the examining finger is placed through the os of the Cx& swept around to separate the membranes from the lower uterine segment
  ➡ ⬆ local PGF2 α production & release from decidua & membranes ➡ onset of labor
- ⬆ the rate of delivery in 2-7 days
- ⬇ the rate of post-term
- ⬇ the use of formal induction methods
- If there is urgent indication for IOL sweeping is not the method of choice

specific circumstances or indications:

Prelabor SROM at term:

- 6-19%
- IOL with oxytocin ➡⬇ risk of maternal infections (chorioamnionitis&endometritis) & neonatal infections
- PG also ➡⬇maternal infections & neonatal NICU admissions

IOL after CS:

- PG should not be used as it can result in rupture uterus
- Oxytocin or foley catheter may be used

Summary:

- **Indications of IOL are**: Post-term pregnancy, PROM, IUGR, Maternal medical conditions like DM HPT, Fetal death, Abruption &Chrioamnionitis.

- **Contraindications of IOL** are the contraindications of vaginal delivery, like Previous myomectomy entering the cavity, Previous uterine rupture, Fetal transverse lie, Placenta previa, Vasa previa, Invasive Cx Ca, Active genital herpes &Previous classical or inverted T uterine incision.

- **Cx ripening**prior to IOL is indicated if the Bishop score is ≤ 6, using Intracervical or intravaginal**PGE2**, misoprostol, or mechanical methods (**foley catheter**& hydroscopic dilators).

- **IOL**: we can use oxytocin with amniotomy, **PGE2** (most commonly) or sweeping of membranes.

- **PG should not be used for IOL after CS as it can result in rupture uterus**, we can use oxytocin or foley catheter instead.

MCQ:

A 34 year old lady G7P6+0 at 42 week. Her last caesarean section was done due to small for gestational age fetus. Which of these is a contraindication for induction of labour?

a) Hypertension
b) Grand multiparty
c) Intra uterine growth restriction
d) Transverse lie of the fetus

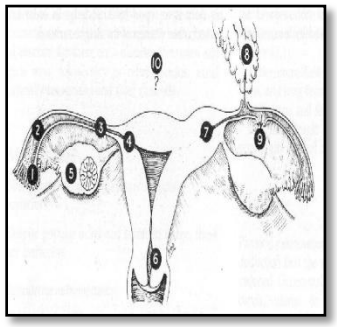
Bleeding in Early Pregnancy.

Is it important?? Yes because it can cause maternal death

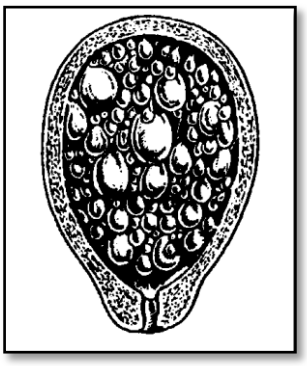
Aims:

1- To know that bleeding in early pregnancy is common and the differential diagnoses are extensive.
2- To critically assess the women with early pregnancy bleeding as this can kill the women.

❖ Theunderlying reasons of bleeding in early pregnancy:



Ectopic pregnancy "most common"



Local causes: in the cervix (polyps, infections or cancer),

Trauma (RTA)

❖ Pregnancy Loss:

- Definition: Termination of the conceptus from the time of conception till the time of fetal viability (24 weeks). Why not 20 weeks?
- Biochemical pregnancy
- Clinical pregnancy

Viability:

- Fetal weight >500 grams
- Incidence: 15-20% of clinically recognized,
- Can be much higher if consider chemical pregnancies, before clinical recognition

- Miscarriage is spontaneous while abortion is induced either by the doctor or the mother.
- Miscarriage or abortion is loss of pregnancy before 20 weeks which is the period of fetal viability (period of viability: can I resuscitate the fetus or not? Can he survive?)
- Because our country is following the WHO so we will say loss of pregnancy before 24 weeks (instead of 20 Ws) is miscarriage/abortion.
- Bleeding after 24 weeks is considered "antepartum hemorrhage"
- Biochemical pregnancy: by testing B HCG either in urine (urine pregnancy test) or blood with no sign of pregnancy in the US
- Clinical pregnancy: signs of pregnancy in US (first sign is the gestational sac).

❖ Pathology:

- Hemorrhage into the decidua basalis.
- Necrotic changes and inflammation in thetissue, adjacent to the bleeding.
- Detachment of the conceptus.
- The above will stimulate uterine contractions resulting in expulsion.

❖ Causes of abortion:

Immunological:

- Alloimmune response: failure of a normal immune response in the mother to accept the fetus for duration of a normal pregnancy.
- Autoimmune disease: antiphospholipid antibodies especially lupus anticoagulant (LA) and the anticardiolipin antibodies (ACL).

Uterine abnormality:

- Congenital: septate uterus → recurrent abortion.
- Fibroids (submucus)
- Polyp > 2 cm diameter.
- Cervical incompetence: → second trimester abortions.
  "The internal os is not constricted.

This condition is diagnosed mainly by history. Patient will present with painless abortion."

Endocrine:

- Diabetes Mellitus
- Hypothyroidism.
- Luteal Phase Defect (LPD): a situation in which the endometrium is poorly or improperly hormonally prepared for implantation. (Questionable).

Infections:

TORCH infections,

Environmental:

Toxins: alcohol, smoking, drug abuse, ionizing radiation.

Chromosome Abnormality:

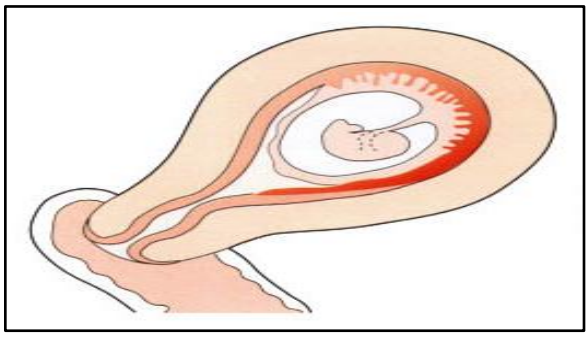
50% of spontaneous losses are associated with fetal chromosome abnormalities:

- Autosomal trisomy
- Monosomy
- Triploids

❖ Classification and subtypes:

1- Spontaneous "something goes wrong in her body or uterus"

a. Threatened abortion:



- 25% of pregnancies
- This refers to bleeding from placental bed, minimal bleeding.
- The pregnancy is sound. In practice any case of bleeding before the 24th weeks may be classified as threatened abortion in the absence of any other explanation.

The patient will come to your clinic with:

- A period of amenorrhea.
- Mild bleeding (spotting). "Might be heavy bleeding"
- Mild pain.
- Internal cervical os is closed.
- Gestational age/ pregnancy test/ Ultrasound
- **In bimanual exam:**Vulvae, Vagina and Cervix are healthy,
- Uterus corresponds toperiod of gestation,
- **USS:**viable intra uterine fetus.

- Closed internal os
- The fetus is intact. She might loose him.
- Diagnosed by history and examination.
- She DOES NOT pass any tissue.
- Confirmed by US

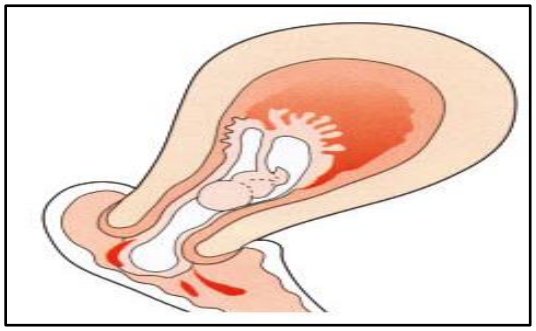
- 97% of the threatened abortions with viable fetus, reassurance and care will end up with normal delivery.

Management

- Expectant; reassurance.
- Anti D if Rhesus negative to protect the next pregnancy
- Hormones; Progesterone and Rest ??? "Prof. Lulu said no need to give her hormones"

b. Inevitable abortion:

Clinical features:



- A period of amenorrhea.
- Heavy bleeding accompanied with clots (may lead to shock).
- Severe lower abdominal pain no passage of tissue.
- Internal cervical os is open and product of conception may be felt in the cervical canal.
- **Bimanual Exam:**Vulvae, Vagina and Cervix are healthy
- Uterus corresponds to period of gestation.

- She does not pass any tissues but she will loose her fetus

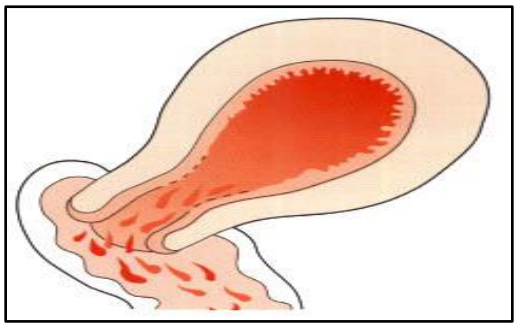
- If there is heavy bleeding we have to enhance the procedure by oxytocin

Management:

- Intravenous fluids
- Cross Match blood.
- Oxytocin; Syntocinon Intravenous infusion.
- Evacuation of the uterus
- Anti D if Rhesus negative

c. Incomplete abortion:

Clinical feature:



- Partial expulsion of products of Conception
- Bleeding and colicky pain continue.
- P.V.: cervix os is open, retained products of conception(RPOC) may be felt in the canal.
- Uterus is smaller.
- **USS:** retained products of conception.

Management:

- If bleeding: go for Evacuation and Curettage(E&C). Complication of the procedure?
- If no bleeding: give oxytocic medication: Ergometrine/ cytotic and repeat the USS in 7-10 days.
- Give Anti D
- Check Rubella immunity
- Advices.

d. Complete abortion:

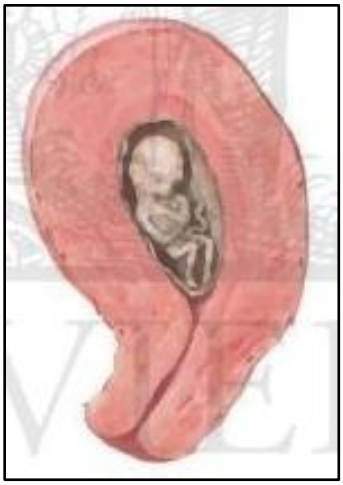
Clinical features:

- A period of amenorrhea.
- Gestational age
- Heavy bleeding accompanied with+/-clots
- Severe lower abdominal pain with passage of tissue expulsion of all products of conception.
- Cessation of bleeding and abdominal pain.
- P.V.: cervix; internal os is closed
- Uterus is bulky smaller than gestational age.
- **USS:** empty uterus.

Management:

- Anti D, Rubella, Advises.

e. Missed abortion:



Clinical features:

- Gradual disappearance of pregnancy Signs and Symptoms.
- Brownish vaginal discharge.
- Pregnancy test: may be + ve for 3-4 weeks after the death of the fetus.
- **USS:**Absent fetal heart pulsations. Empty Gestational sac

Complications:

- Infection (Septic abortion).
- Disseminated intravascular coagulation (DIC).

- Spontaneous expulsion means termination of pregnancy
- If abortion occurs before 4 weeks we have to worry about DIC, otherwise no.
- Less than 12 weeks: we can do surgery but there will be complications.
- More than 12 weeks: induce labor by medications
- Why 12 weeks? Because the fetus has bones at this time

Management:

- Wait 4 weeks for spontaneous expulsion
- Terminate the pregnancy if:
  Spontaneous expulsion does not occur after 4 weeks, or if there is:Infection or bleeding.

- **Manage according to size of uterus:**
  - **Uterus < 12 weeks:** dilatation and suction evacuation (D&C).
  - **Uterus > 12 weeks:** Oxytocic medications, cytotic drugs.

f. Septic abortion:

- Uterine infection at any stage of abortion

Causes:

- Delay in evacuation of uterus
- Delay seeking advice
- Incomplete surgical evacuation followed by infection from vaginal organisms:
  - Anaerobic bacteroids
  - Clostridium welchin
  - Bacterial fragilis
  - Coliform bacillus

Infected abortion that becomes septic causing septicemia then death.

g. Recurrent abortion:

When a woman has had 3 consecutive miscarriages.

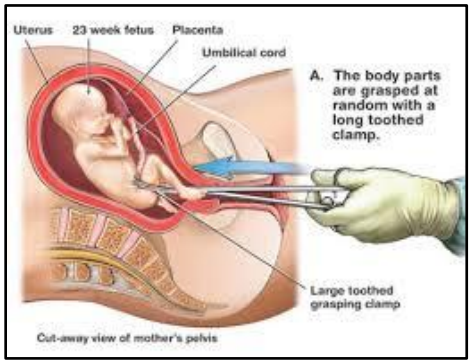
Etiology:

- **Genetic factors**
  Karyotyping of both partners will reveal chromosomal anomalies
- **Anatomical factors**
  Uterine anomalies.
  Cervical incompetence.
  Hysteroscopy & HSG – Septum / Fibroid
- **Endocrine problem**
- **Immunological factors**
  Recurrent miscarriage is common in couples with similar HLA types.
  Common in women with antiphopholipid antibodies syndrome.
  Anticardiolipid ant. & Lupus anticoagulant.

2. Induced abortion:

a. Therapeutic abortion:

Termination of pregnancy before time of fetal viability for the purpose of safe guarding the health of the mother.
For example in case of heart disease, invasive cancer of cervix.



An agreement for termination is given by 2 heart consultants and obstetricians.

b. Elective (voluntary)"criminal"abortion:

The interruption of pregnancy before viability at request of the women but not for reason of ill-health of either mother or fetus**.**
This is not done in this country

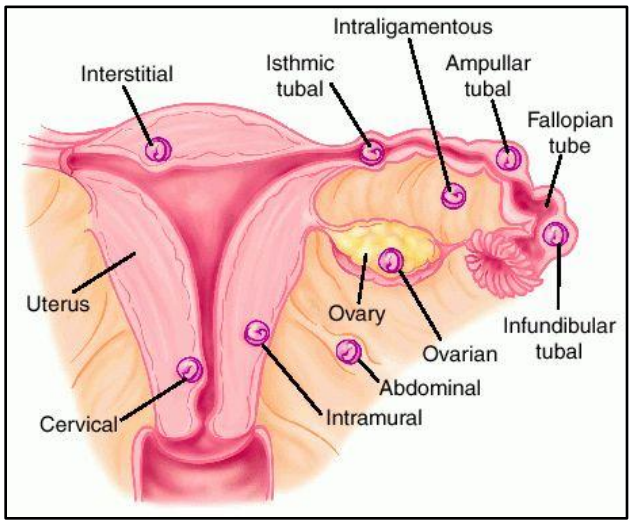
❖ Abortion technique:

- **Medical:**
  - Oxytocin
  - Prostaglandins; misoprostol
  - Anti progesterone RU 486: (Mifepristone)

- **Surgical:**
  D & C, E&C, Suction Evacuation.

❖ Ectopic pregnancy:

Fertilized embryo implanted outside the uterine cavity.



- ❖ **Sites of ectopic pregnancy:**
  1. **Fallopian tube (98%)**
     - Ampullary (most frequent)
     - Isthmic
     - Infundibular and fimbrial
     - Interstitial (least frequent)
  2. **Other sites**
     - Abdominal
     - Ovary
     - Cervical

**Step up to OB/GYNE

- Leading cause of maternal deaths in the first trimester
- Constituting 1-2% of all conceptions
- Subsequent infertility
- Incidence increasing
- Mortality decreasing with better detection and early awareness

❖ Risk factors:

Women are at higher risk for tubal pregnancy:

- Prior history of PID (pelvic inflammatory disease)

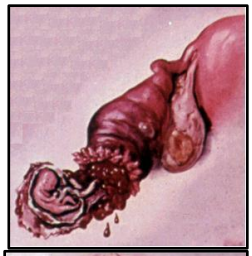
Tubal pregnancy:

- May occur before she misses her period
- A woman who had a history of previous ectopic pregnancy should inform her doctor immediately when misses her period
- She'll present with rupture
- No x ray pregnancy
- No intrauterine pregnancy on US
- Asymptomatic
- So check and repeat B HCG: if it is going down then it's dying pregnancy
- Repeat 48 platelet: if doubled then it's normal pregnancy. If not then it's abnormal pregnancy.

- Tubal Surgery
- Previous Ectopic Pregnancy
- IUD (intrauterine device)
- Tubal abnormalities
- Assisted conception, IVF
- Tubal sterilization depends on type
- Pelvic surgery

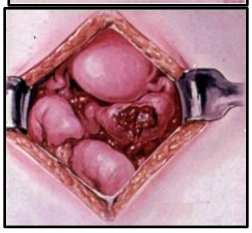
- These are factors that lead to tubal damage or dysfunction and thus prevent or delay passage of the fertilized ovum into the uterine cavity.

❖ Outcome:

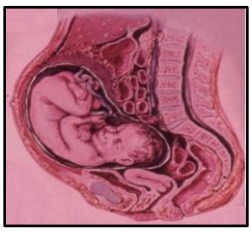


1. Spontaneous resolution
2. Tubal abortion
3. Rupture of tubal pregnancy
4. Secondary abdominal pregnancy(may reach 9months)

❖ Symptoms:



Ectopic pregnancy often confused with those of a miscarriage or pelvic inflammatory disease.



- The most common symptoms "classic triad":

  Amenorrhea
  Abdominal/pelvic pain
  Irregular vaginal bleeding

- A ruptured ectopic pregnancy is a true medical emergency.
- Common symptoms of a ruptured ectopic pregnancy include the following:

  Dizziness, pale complexion, sweaty, fast heart beat.
  Abdominal/pelvic pain.
  Shoulders tip pain.
  Pain at defecation

❖ Examination:

- Examine the woman from top to toe
- Vital signs
- Abdominal examination
- Pelvic examination; should be extremely careful

❖ Diagnosis:

- An ectopic pregnancy should be considered in any woman with abdominal pain or vaginal bleeding who has a positive pregnancy test. High index of suspicion
- An ultrasound showing:
  - Gestational sac with fetal heart in the fallopian tube is clear evidence of ectopic pregnancy.
  - Pseudo sac and a gestational sac in the tube
  - Empty uterus and positive pregnancy test
- An abnormal rise in blood β-hCG levels may also indicate an ectopic pregnancy.

❖ Management:

Once diagnosed, patient needs to be fully aware of the risks involved.

1. **Expectant**
   There are conditions that needs to be fulfilled
2. **Surgical**(if there is sever abdominal pain or bleeding and no medical therapy. "Laparoscopy or laparotomy")
   - Laparoscopy is performed for:
     Symptomatic patient
     Fluid/blood in the Pouch of Douglas
     Negative laparoscopy: follow-up with β HCG for the reasons:
     - Intrauterine pregnancy
     - Ectopic pregnancy that has been missed
   - Laparotomy
   - Salpingostomy/ salpingotomy
   - Salpingectomy
3. **Medical**(chemotherapy because they are chorionic villi (rapidly dividing cells)).
   Methotrexate (1 mg/kg): is an anti metabolite that interferes with the synthesis of DNA by inhibiting the action of Dihdrofolatereductase.

❖ Indications:

- Haemodynamically stable, no active bleeding, no haemoperitneum, minimal bleeding and no pain
- No contraindications to methotrexate
- Able to return for follow up for several weeks
- Unrupturedadenexal mass < 4 cm in size by scan
- No cardiac activity by scan
- βhCGdoes not exceed 5000 IU/L
- Willing for treatment
- Facility for USS monitoring
- Facility of βhCG monitoring

❖ Contraindications:

- Breastfeeding
- Immunodeficiency / active infection
- Active pulmonary disease
- Peptic ulcer or colitis
- Blood disorder
- Hepatic, Renal or Haematological dysfunction

❖ Side effects:

- Nausea & Vomiting
- Stomatitis
- Diarrhea, abdominal pain
- Photosensitivity skin reaction
- Impaired liver function, reversible
- Severe neutropenia
- Reversible alopecia
- Haematosalpinx and haematoceles

❖ Treatment Effects:

- ↑ Abdominal pain (2/3 of patient)"it will be dislodged so uterus will contract expelling the products".
- ↑βhCGduring first 3 days of treatment
- Vaginal bleeding
- Increase in the size

❖ Signs of Treatment failure and tubal rupture:

- Significantly worsening abdominal pain, regardless of change in serum βhCG, check CBC)
- Haemodynamic instability
- Level of βhCGdoes not decline by at least 15% between Day 4 & 7 post treatment
- ↑or plateauing βhCGlevel after first week of treatment

❖ Follow up:

- Repeat βhCGon Day 5 post injection if <15 % decrease – consider repeat dose
- If βhCG>15 ↓ recheck weekly until <25 ul/l
- Surgery is considered in women presenting with severe pain in the first few days after methotrexate and careful clinical assessment is required. If there is significant doubt surgery is the safest option

❖ Tubal procedures:

- **Salpingotomy (or -ostomy):** Making an incision on the tube and removing the pregnancy.
- **Salpingectomy:** Cutting the tube out.
- **Fimbrial expression:** "Milking" the pregnancy out the fimbrial end of the tube. Care of bleeding

- In the future: The chance of recurrent ectopic pregnancy is about 10%.

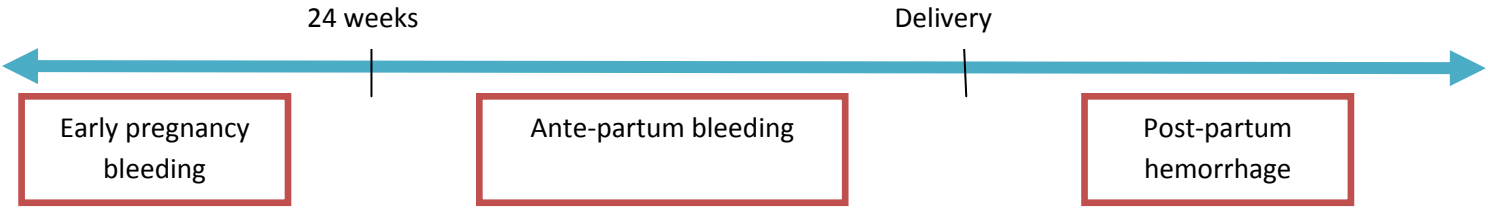
SUMMARY 1

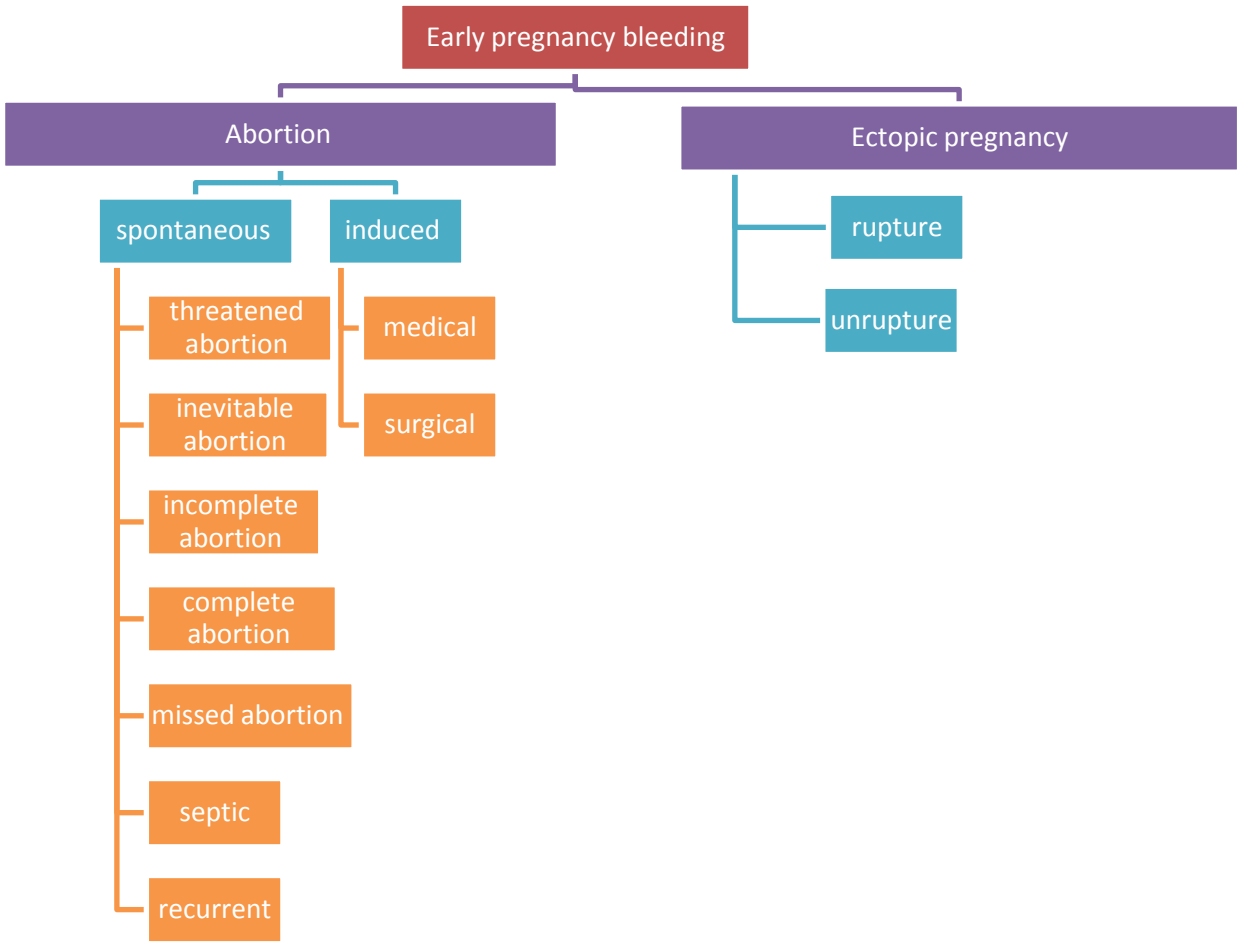
- Ectopic pregnancy is the most common cause of bleeding in early pregnancy.
- Pregnancy loss: before 24 weeks
- Biochemical pregnancy: testing B hCG in urine/blood. Clinical pregnancy: US signs
- Causes of abortion: (fetal): chromosomal abnormality. (maternal): uterine abnormalities such as, septate uterus (recurrent abortion), polyps, fibroids and cervical incompetence.
- We have two types of abortion:

1. **Induced:**elective (voluntary, criminal) and therapeutic.
   - Abortion techniques:
     Medically by: oxytocin, prostaglandins (misoprostol) or anti progesterone RU 486: (Mifepristone).
     Surgically: D & C, E&C and Suction Evacuation.
2. **Spontaneous:**
   a. **Threatened:** Closed internal os, the fetus is intact (she might loose him), she DOES NOT pass any tissue, mild pain and confirmed by US.
   b. **Inevitable:** Heavy bleeding with clots, severe pain, no passage of tissue, internal cervical os is open and she will lose her fetus.
   c. **Incomplete:** Partial expulsion of products of conception, bleeding, colicky pain and cervical os is open.
   d. **Complete:**Heavy bleeding, severe pain with passage of tissue expulsion of all products of conception, internal os is closed and uterus is empty on US.
   e. **Missed:** Gradual disappearance of pregnancy Signs and Symptoms, brownish vaginal discharge, absent fetal heart pulsations and empty gestational sac on US.
   f. **Septic.**
   g. **Recurrent:** 3 consecutive miscarriages.

- Sites of ectopic pregnancy: fallopian tube common (ampullary, isthmic, infundibular and interstitial), ovary, abdomen and cervical.
- Risk factor: prior history of PID.
- Symptoms: classic triad (amenorrhea, abdominal/pelvic pain and irregular vaginal bleeding)
- Managed by:
  a. Expectant therapy
  b. Medical (methotrexate)
  c. Surgical (laparoscopy and laparotomy)

Summary 2


24 weeks
Delivery
Early pregnancy bleeding
Ante-partum bleeding
Post-partum hemorrhage


Early pregnancy bleeding
Abortion
Ectopic pregnancy
spontaneous
induced
rupture
unrupture
threatened abortion
inevitable abortion
incomplete abortion
complete abortion
missed abortion
septic
recurrent
medical
surgical

1-spontaneous abortions : All are diagnosed by history and confirmed by US

Type	In history ?	Treatment
threatened abortion	Close cervix , no tissue passed	Reassurance +anti D
inevitable abortion	Open cervix , no tissue passed	Oxytocin(syntocinon),evacuation +Anti D
incomplete abortion	Open cervix , tissue passed	Bleeding: EC- -No bleeding : oxytocin(Ergometrin)
complete abortion	Close cervix , tissue passed , clots	Reassurance +anti D
missed abortion	Brownish discharge	1- wait 4 W for spontaneous expulsion. 2-if nothing happened or there's infection or bleeding terminate the pregnancy: A- IF fetus <12 weeks : D&C B- if fetus >12 weeks : oxytocic medication

2-induced abortion:

Medical	1-Oxytocin 2-Prostaglandin:Misoprostiol 3-Anti progesterone :Mifepristone
Surgical	D&C ,E&C,Suction Evacuation

3-Ectopic pregnancy

Symptoms: 1-early pregnancy bleeding- vaginal bleeding
2-secondary amenorrhea
3-pelvic-abdominal pain.

Common site : fallopian tube

Rx:1-expectant
2-surgical : laproscopy –laprotomy
3-medical : methotrexate if un-ruptured

Ante Partum Haemorrhage.

Ante Partum Haemorrhage (APH):

- Affects 3-5 % of pregnancies.
- Bleeding from or into the genital tract.
- Occurring from 20 weeks of pregnancy and prior to the birth of the baby.

Bleeding in early pregnancy (first 20 weeks of gestation) causes:

- Miscarriage

- Ectopic pregnancy

- Molar pregnancy

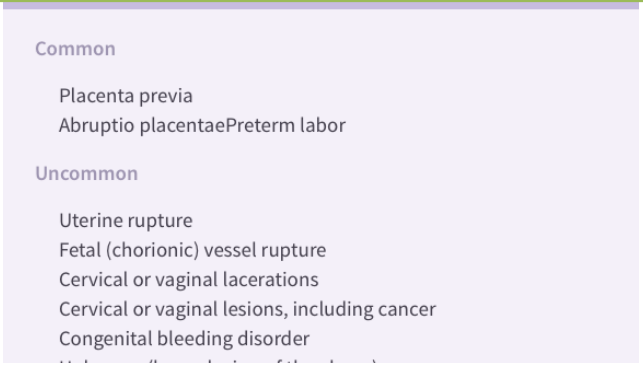
- Local causes: tumor, trauma etc.

Causes:

- Placenta previa.
- Placenta abruption.
- Local causes (cervical or vaginal lesions, lacerations).Trauma, tumor and infections.
- Unexplained (SGA, IUGR).SGA: small for gestational age.
- Vasa previa.
- Uterine rupture.

-APH is the leading cause of prenatal and maternal morbidity and prenatal mortality (mainly prematurity).

- Obstetrics hemorrhage remains one of the major causes of maternal death in the developing countries.



Source: Essentials of Obstetrics and Gynecology.

Management:

In the hospital maternity unit with facilities for resuscitation such as:

- Anesthetic support.
- Blood transfusion resources.
- Performing emergency operative delivery.
- Multidisciplinary team including (midwifery, obstetric staff, neonatal and anesthetic).

Investigations:

- Tests if suspecting vasa previa are often not applicable
- Tocolysis: shouldn't be used in:
  - Unstable patient.
  - Fetal compromise.
  - Major APH.

  It's a decision of a senior obstetrician.

  Senior (consultant) anesthetic care needed in high-risk hemorrhage.
- Risk of PPH: patient should receive active management of 3rd stage of labor using syntometrine (in absence of high BP).Syntometrine→active uterine contraction after deliveryto prevent PPH.
- AntiD Ig should be given to all non sensitized RH –ve if the have APH, at least 500 IU AntiD Ig followed by a test of FMH if it is more than 40 ml of RBC additional AntiD required. AntiD Ig should be given at minimum of 6 weeks intervals.
- Vaginal speculum examination should be done to rule out local causes.(e.g: polyps)

Vasa previa:

- 1:2000.
- Rare but very serious cause of vaginal bleeding.
- Bleeding is fetal in origin associated with velamentous cord insertion where fetal blood vessels in the membranes cross the cervix.
- Rupture of membranes can lead to tearing of fetal B.V with exsanguination of the fetus.

Diagnosis by color flow Doppler ultrasound.

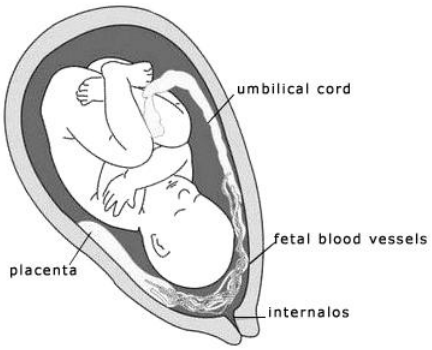
* The term velamentous insertion is used to describe the condition in which the umbilical cord inserts on the chorioamniotic membranes rather than on the placental mass.

*B.V: blood vessels.

* Exsanguination is the process of blood loss, to a degree sufficient to cause death.

Risk factors: **all are important.**

- Velamentous insertion.
- Bi-lobed or succenturiate lobed placenta.
- Multiple pregnancies.
- Low-lying placenta.
- IVF pregnancy.



Vasa Previa

Placenta Abruption (abruptio placentae):

- **Definition:** bleeding at the decidual-placental interface that causes partial or total placental detachment (by forming a decidual hematoma) prior to delivery of the fetus over 20 weeks of gestation.(It is called a miscarriage if it is before 20 weeks.)
- **Types:** Concealed and revealed hemorrhage.Blood may either dissect upward toward the fundus, resulting in a concealed hemorrhage, or extend downward toward the cervix, resulting in an external or revealed hemorrhage.(Source: Essentials of Obstetrics and Gynecology)

Incidence:

- 0.4%-1% of pregnancies.
- 40-70% occurs before 37 weeks.
- It is a significant cause of maternal morbidity and perinatal morbidity and mortality (PN mortality:12% and 77% occurs in utero)(PNM: perinatal mortality).
- PNM Rate: the number of stillbirths and deaths in the first week of life per 1000 live birth.

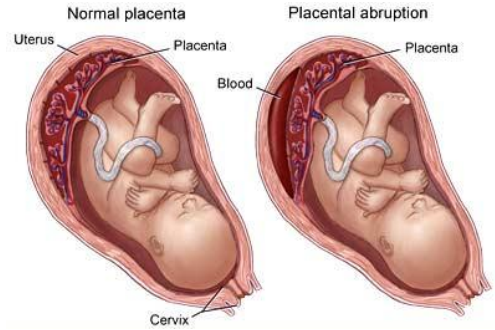
Risk factors: **all are important.**

- Abdominal trauma and accidents.
- Cocaine or other drug abuse.
- Poly hydramnios.
- Hypertensive disease during pregnancy.
- Premature rupture of membranes.
- Chorioamnionitis, IUGR.
- Previous abruptio.
- With increasing age, parity and smoking.
- Uterine anomalies, leiomyoma, uterine synchiae.
- First trimester bleeding.

The diagnosis of Placenta Abruption is made**clinically.**
Suspect this diagnosis if a patient presents with **painful vaginal bleeding** in association with **uterine tenderness, hyperactivity, and increased tone**. Rigid.
- US can only detect 2% of Placenta Abruption but we still use US to **exclude co-existing Placenta Previa.**
- The use of **tocolytics or uterine relaxants is not advised**. Uterine tone must be maintained to control bleeding following delivery, or at least to control the bleeding sufficiently to allow a safe hysterectomy to be performed, if necessary.
Source: Essentials of Obstetrics and Gynecology.

Clinical presentation:

- Vaginal bleeding (mild,moderate or severe).Most common finding (80%).
- Abdominal pain or back pain (if posterior placenta).
- DIC occurs in 10-20% of severe abruption and death of fetus(severe if placenta separate >50%).
  - BP,FH abnormalities or death.
  - Tender or rigid or firm abdomen (woody feel).
  - Hypertonic uterine contractions.
  - DIC.Placenta Abruption is the most common cause of DIC in pregnancy.
  - Hypovolemic shock, renal failure, ARDS multi-organ failure.
  - Blood transfusion, rarely death.



Suspecting abruptio, severe pain, dead fetus in utero with no bleeding→think of DIC

Fetal & neonatal outcome:

- Increased mortality and morbidity due to asphyxia, IUGR and preterm delivery.

Recurrence:

Several-fold higher risk of abruption in subsequent pregnancy: 5-15%.Risk of third rises: 20-25%.

Chronic abruption:

Light, chronic, intermittent bleeding, oligohydroamnious, IUGR, pre-eclampsia and preterm rupture of membrane.

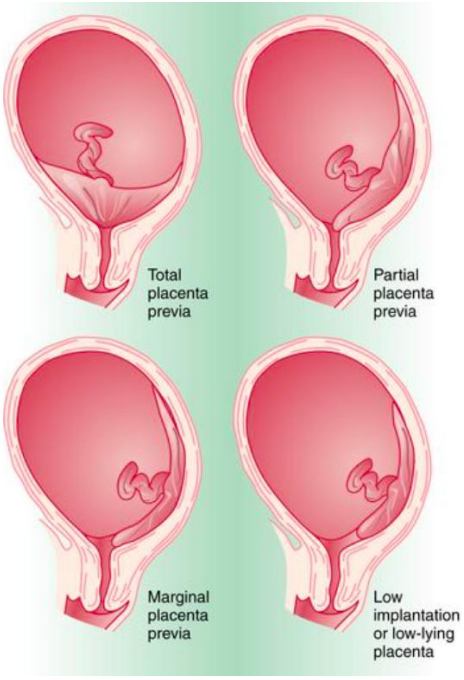
Coagulation studies usually normal.

Placenta previa:

Definition: the presence of placental tissue that extends over or lies proximate to the internal cervical os(I.O). (Beyond 20 weeks of gestation).

Degrees:

i. Total or complete placenta previa: the placenta completely covers the I.O.
ii. Partial previa: the placenta partially covers the I.O.
iii. Marginalprevia: the edge of the placenta extends to the margin of the I.O.
iv. Low-lying placenta: placental margin is within 2cm of I.O.



Presentation:

Painless, recurrent vaginal bleeding in 70-80%.

Uterine contractions in 10-20%.

Prevalence:3.5-4.6/1000 births.

Recurrence: 4-8%.

- Predisposing factors to Placenta Previa:
    1. Multiparity.
    2. Increasing maternal age.
    3. **Prior Placenta Previa**.
    4. Multiple gestations.
- **Transvaginal US** can accurately diagnose placenta previa in virtually all cases.
- Placenta Previa predisposes to preterm delivery, which poses the greatest risks to the fetus.
- Placenta Accerta: implies an abnormal attachment of the placenta through the uterine **myometrium** as a result of defective decidual formation (absent Nitabuch's layer).
- Superficial (accreta), or the placental villi may invade partially through the myometrium (increta) or extend to the uterine serosa (percreta).
- Those with a prior c-section have a 25% risk of having Placenta Accerta.

Source: essentials of Obstetrics and Gynecology.

Risk factors:

- Previous c/s, placenta previa.
- Multiple gestation, multiparity, advanced maternal age.
- Infertility treatment, previous abortion.
- Previous intrauterine surgical procedures.Site for abnormalzygote implantation.
- Maternal smoking, cocaine use.
- Non-white race, male fetus.

Associated Conditions:

Placenta accreta: complicated 1-5% patients with placenta previa.Doctor saidyou need to memorize these numbers.

- If previous c/s: 11-25%.
- Two c/s: 35-47%.
- Three c/s: 40%.
- Four c/s: 50-67%.

Preterm labor, rupture of membrane, mal presentation, IUGR, vasa previa, congenital anomalies, amniotic fluid embolism.

Diagnosis:

Soft abdomen, normal fetal heart, mal presentation.Unlike abruptio.
Avoid vaginal, rectal examination or sexual intercourse.May cause severe bleeding.
Investigation:

- Abdominal u/s: false +ve 25% due to over distended bladder or uterine contractions, or can be missed if fetal head is low in pelvis.
- Transvaginal u/s: (if diagnosis by abdominal u/s not certain), or trans perineal u/s.
- MRI: High cost.

Any pregnant woman who comes with vaginal bleeding has to have an US to rule out Placenta Previa.

Management:

Treatment depends on gestational age, amount of vaginal bleeding, maternal status and fetal condition.

Consent for hysterectomy should be ready early.

Expectant management:

- If fetus is preterm less than 37 weeks:
    - Hospitalization.
    - Investigations (CBC, RFT, LFT, coagulation factors, blood grouping and RH).
        - Steroids (between 24-34 weeksgestation).Give dexamethasone for lung maturity.
        - AntiDIg if the mother is RH negative.
    - Cross match blood and blood products.At least 4 units.
    - CTG.
- Elective c/s: if fetus more than 37 weeks.
- Emergency c/s: if severe bleeding or fetal distress.

Morbidity and Mortality:

- Hemorrhage.
- Hypovolemic shock (renal failure, Sheehan's Syndrome, death).
- Blood transfusion risk.
- Hysterectomy, uterine/iliac artery ligation or embolization of pelvic vessels.
- Increase mmR.Maternal Mortality Rate.
- Increase neonatal morbidity.

Summary:

Ante Partum Haemorrhage

- Occurring from 20 weeks of pregnancy and prior to the birth of the baby.
- APH is the leading cause of prenatal and maternal morbidity and prenatal mortality (mainly prematurity).

Causes:

- Placenta previa.
- Placenta abruption.
- Local causes (cervical or vaginal lesions, lacerations). Trauma, tumor and infections.
- Unexplained (SGA, IUGR). SGA: small for gestational age.
- Vasa previa.
- Uterine rupture.
- APH has a risk of PPH: patient should receive active management of 3rd stage of labor using syntometrine (in absence of high BP). Syntometrine → active uterine contraction after delivery to prevent PPH.
- AntiD Ig should be given to all non sensitized RH –ve if the have APH, at least 500 IU AntiD Ig followed by a test of FMH if it is more than 40 ml of RBC additional AntiD required. AntiD Ig should be given at minimum of 6 weeks intervals.
- Vaginal speculum examination should be done to rule out local causes. (e.g: polyps)

Vasa previa

Rare. Bleeding is fetal in origin associated with velamentous cord insertion where fetal blood vessels in the membranes cross the cervix.

Diagnosis: by color flow Doppler ultrasound.

Risk factors:

- Velamentous insertion.
- Bi-lobed or succenturiate lobed placenta.
- Multiple pregnancies.
- Low-lying placenta.
- IVF pregnancy.

Placenta Abruption (abruptio placentae)	Placenta Previa
Definition: bleeding at the decidual-placental interface that causes partial or total placental detachment (by forming a decidual hematoma) prior to delivery of the fetus over 20 weeks of gestation. **Types:** Concealed and revealed hemorrhage.	**Definition:** the presence of placental tissue that extends over or lies proximate to the internal cervical os (I.O). (Beyond 20 weeks of gestation). **Degrees:** I - Total or complete placenta previa: the placenta completely covers the I.O. II - Partial previa: the placenta partially covers the I.O. III- Marginal previa: the edge of the placenta extends to the margin of the I.O. IV - Low-lying placenta: placental margin is within 2cm of I.O.

	Placenta accreta: complicated 1-5% patients with placenta previa. If previous c/s: 11-25%. Two c/s: 35-47%. Three c/s: 40%. Four c/s: 50-67%. Can cause: preterm labor, rupture of membrane, mal presentation, IUGR, vasa previa, congenital anomalies, amniotic fluid embolism.
Risk factors: - Abdominal trauma and accidents. - Cocaine or other drug abuse. - Poly hydramnios. - Hypertensive disease during pregnancy. - Premature rupture of membranes. - Chorioamnionitis, IUGR. - Previous abruptio. - With increasing age, parity and smoking. - Uterine anomalies, leiomyoma, uterine synchiae. - First trimester bleeding.	**Risk factors:** - Previous c/s, placenta previa. - Multiple gestation, multiparity, advanced maternal age. - Infertility treatment, previous abortion. - Previous intrauterine surgical procedures. Site for abnormal zygote implantation. - Maternal smoking, cocaine use. - Non-white race, male fetus.
Presentation: Suspect this diagnosis if a patient presents with painful vaginal bleeding in association with uterine tenderness, hyperactivity, and increased tone. Rigid. **Diagnosis:** is made clinically. - US can only detect 2% of Placenta Abruption but we still use US to exclude co-existing Placenta Previa.	**Presentation:** Painless, recurrent vaginal bleeding **Diagnosis:** Soft abdomen, normal fetal heart, mal presentation. Unlike abruptio. Avoid vaginal, rectal examination or sexual intercourse. May cause severe bleeding. **Investigation:** Transvaginal US is the gold standard. **Management:** ◆If fetus is preterm less than 37 weeks: -Hospitalization. - Investigations (CBC, RFT, LFT, coagulation factors, blood grouping and RH). -Steroids (between 24-34 weeks gestation). Give dexamethasone for lung maturity. -AntiD Ig if the mother is RH negative. - Cross match blood and blood products. At least 4 units. -CTG. ◆Elective c/s: if fetus more than 37 weeks. ◆Emergency c/s: if severe bleeding or fetal distress.

Summary 2:

Causes	Information
1-vasa previa	**Risks**: artificial rupture of membrane, bi-lobed or succenturate lobed placenta **Symptoms**: **painless** vaginal bleeding **Fetus** is on danger because of fetal bleeding and bradycardia >>death **Diagnosed by** :Doppler ultra sound **Rx**: immediate C section
2-placenta Abruptio	**Risks** :truma , HTN , coccine……etc **Symptoms** :**painful** vaginal bleeding , normal placenta implantation ,DIC **Fetus** is on danger **On examination:** abdomen rigid **Diagnosed by:** clinical presentation mainly
3-Placenta previa	**Risks**: previous intrauterine surgical procedure , multiple gestation **Symptoms : painless** vaginal bleeding , lower segment placenta implantation **Fetus** :No serious risk , usually fetus on transverse presentation **On examination:** abdomen is soft . **Diagnosed by :**vaginal ultra sound **Complications** :accreta , increta, perceta .

Postpartum Haemorrhage.

What does PPH? Bleeding after childbirth, Antepartum hemorrhage? bleeding before delivery

-Definition: bleeding from genital tract which lead to presence of symptoms

Any blood loss than has potential to produce or produces hemodynamic instability.

- symptoms of hemodynamic instability (dizzinis, weak, palpitatetion, pale) if there is symptoms then measure the vital signs blood pressure and pulse rate.
- So we have to observe the pt after 2 hs of delivery
- The most common cause of maternal death worldwide is PPH
- Second cause is PE and the third is HTN

-Incidence

About 5% of all deliveries

- 2type of PPH: primary and secondary:
- primary: from 3rd stage of delivery to 24 h (after child birth) before that not PPH
- secondary:24 hs to 6 wks
- measuring the blood loss done by symptoms and seeing blood

Definition:

- from the placental site, vagina and perineum specially in episiotomy
- >500ml after completion of the third stage, 5% women loose >1000ml at vag delivery
- >1000ml after C/S
- >1400ml for elective Cesarean-hyst
- >3000-3500ml for emergent Cesarean-hyst
- woman with normal pregnancy-induced hypervolemia increases blood-volume by 30-60% = 1-2L
- therfore, tolerates similar amount of blood loss at delivery
- hemorrhage after 24hrs = late PPH
- In normal non regnant ladies they can't tolerate 300 ml why in pregnant tolerate up to 500 ml of blood loss ?because already there is increase in blood volume physiologically .

Hemostasis at placental site:

- At term, 600ml/min of blood flows through intervillous space
- Most important factor for control of bleeding from placenta site = contraction and retraction of myometrium to compress the vessels severed with placental separation.
- Incomplete separation will prevent appropriate contraction
- The coagulation cascade will start immediately after delivery.
- So any one of these doesn't work, will lead to PPH
- Symptoms of placental delivery:
  Gush of blood
  Elongated umbilical cord
  Change the uterus shape (contraction)

Etiology of Postpartum Haemorrhage(very important)

Tone	Uterine atony 95% (the most common)
Tissue (Retained part of placenta, Placenta previa, placenta accreta)	Retained tissue/clots
Trauma (Laceration of the vagina or cervix, uterine rupture)	laceration, rupture, inversion
Thrombin (Coagulation cascade should start immediately after delivery)	Coagulopathy

Predisposing factors- Intrapartum (bleeding during the delivery)

what will cause the previous causes?

- Operative delivery (asc-section, ventose, forceps)
- Prolonged or rapid labour(depend on the time of every stage of labour, also it differ from parmi and multi) will end with uterine atony
- Induction or agumentation(increase medication for induction)
- Internal podalic version
- Choriomnionitis(infection)
- Shoulder dystocia
- Coagulopathy

Definitions of internal podalic version:maneuver to deliver the fetus by inserting a hand into the uterine cavity, grasping one or both feet, and drawing them through the cervix; rarely indicated today except for the delivery of a second twin.

Predisposing Factors- Antepartum

- Previous PPH or manual removal (imp in history)
- Abruption/previa
- Fetal demise = (fetal death)
- Gestational hypertension
- Over distended uterus
- Bleeding disorder

Postpartum causes

- Lacerations or episiotomy
- Retained placental/ placental abnormalities(imp to check and inspect the placenta after delivery for any missing part)
- Uterine rupture / inversion
- Coagulopathy

Prevention

- Be prepared
- Active management of third stage
  - Prophylactic oxytocin+ Ergometrinein delivery , after placental delivery assess the uterus, fundal high and confirm if it well contact, if not give extra oxytocin
  - 10 U IM
  - 5 U IV bolus
  - 10-20 U/L N/S IV @ 100-150 ml/hr
  - Early cord clamping and cutting
  - Gentle cord traction with surapubiccountertraction
  - Put new pads and check after few mins and check the uterus contraction and vital signs.

Remember!

- Blood loss is often underestimated
- Ongoing trickling can lead to significant blood loss
- Blood loss is generally well tolerated to a point
- If there is still bleeding after the previous precutions with decrease in BP and paleness and contracted uterus we check the 4 Ts:
- Check episiotomy, suturing, any hematoma and any laceration in the vagina
- If everything is ok :So may be internal bleeding from the uterus then start the resuscitation
- If it is Internal bleeding, what should I do? We do laparotomy and look for the bleeding (surgical intervention), we back the uterus and use the balloon or blood vessel legation.

Management

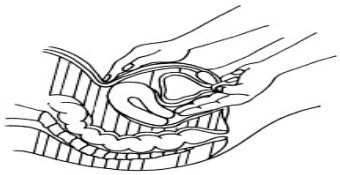
- talk to and assess patient
- Get HELP!
- Large bore IV access
- Crystalloid-lots!
- CBC/cross-match and type
- Foley catheter

Diagnosis

- Assess in the fundus (Palpate the abdomen to see if the uterus contacted or not)
- Inspect the lower genital tract (Check externally : vagina, episiotomy, then internally: uterus, cervix)
- Explore the uterus
  - Retained placental fragments
  - Uterine rupture
  - Uterine inversion
- Assess coagulation

Management:

1- Assess the fundus

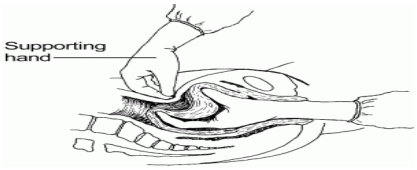


- If she still bleed we resuscitate
- Simultaneous with ABC's
- Atony is the leading case of PPH>>so treat it withBimanual Massage
- Bimanual massage
- Rules out uterine inversion
- May feel lower tract injury
- Evacuate clot from vagina and/ or cervix ,feel the bleeding from the vagina
- May consider manual exploration at this time

2- Manual Exploration

- Manual exploration will:
  - Rule out the uterine inversion
  - Palpate cervical injury
  - Remove retained placenta or clot from uterus
  - Rule out uterine rupture or dehiscence

Replacement of Inverted Uterus ⟶



3- Oxytocin

- 5 units IV bolus
- 20 units per L N/S IV wide open
- 10 units intramyometrial given transabdominally

4-Additional Uterotonics

- Ergometrine (caution in hypertension)
  - .25 mg IM 0r .125 mg IV
  - Maximum dose 1.25 mg
- Hemabate (asthma is a relative contraindication)
  - 15 methyl-prostaglandin F2 alfa
  - O.25mg IM or intramyometrial
  - Maximum dose 2 mg (Q 15 min- total 8 doses)
- Cytotec (misoprostol) PG E1
  - 800-1000 mcg pr

TABLE 3

DRUG THERAPY FOR PPH

Drug	Dose	Side Effects	Contraindications
Oxytocin	10 units IM/IMM 5 units IV bolus 10 to 20 units/litre	Usually none painful contractions nausea, vomiting, (water intoxication)	hypersensitivity to drug
Methylergonovine maleate	0.25mg IM/0.125mg IV repeat every 5 mins as needed maximum 5 doses	peripheral vasospasm hypertension nausea, vomiting	hypertension hypersensitivity to drug
Carboprost (15-methyl PGF_2 alpha)	0.25 IM/IMM repeat every 15 mins as needed maximum 8 doses	flushing, diarrhea, nausea, vomiting bronchospasm, flushing, restlessness, oxygen desaturation	active cardiac, pulmonary, renal, or hepatic disease hypersensitivity to drug
Vasopressin	20 units diluted in 100 ml normal saline = (0.2 units/ml) inject 1 ml at bleeding site avoid intravascular injection	acute hypertension, bronchospasm nausea, vomiting, abdominal cramps angina, headache, vertigo death with intravascular injection	coronary artery disease hypersensitivity to drug

5- Bleeding with Firm Uterus

- Explore the lower genital tract
- Requirements
  - Appropriate analgesia
  - Good exposure and lighting
- Appropriate surgical repair
  - May temporize with packing

6- ABC's

<u>ENSURE</u> THAT YOU ARE ALWAYS AHEAD WITH YOUR RESUSCITATION!!!!

- Consider need for Foley catheter, CVP, arterial line, etc.
- Consider need for more expert help

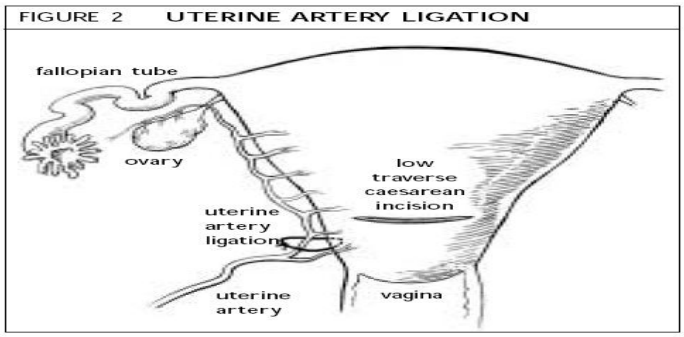
Evolution

- Panic
- Panic
- Hysterectomy
- Pitocin
- Prostaglandins
- Happiness

MANAGEMENT OF PPH

Management- Continued Uterine Bleeding

- Consider coagulopathy
- Correct coagulopathy
  - FFP, cryoprecipitate, platelets
- If coagulation is normal:
  - Consider embolization
  - Prepare for O.R



Surgical Approaches

- Uterine vessel ligation
- Internal iliac vessel ligation
- Hysterectomy

Conclusions

- Be prepared
- Practice prevention
- Assess the loss
- Assess the maternal status
- Resuscitate vigorously and appropriately
- Diagnose the cause
- Treat the cause

dr didn't read it

THROMBIN

- Check labs if suspicious**.**

CONSUMPTIVE COAGULOPATHY (DIC)

- A complication of an identifiable, underlying pathological process against which treatment must be directed to the cause

Pregnancy Hypercoagulability

- ↑coagulation factors I (fibrinogen), VII, IX, X
- ↑plasminogen; ↓ plasmin activity
- ↑fibrinopeptide A, b-thromboglobulin, platelet factor 4, fibrinogen

-Pathological Activation of Coagulation mechanisms

- Extrinsic pathway activation by thromboplastin from tissue destruction
- Intrinsic pathway activation by collagen and other tissue components
- Direct activation of factor X by proteases
- Induction of procoagulant activity in lymphocytes, neutrophils or platelets by stimulation with bacterial toxins

-Significance of Consumptive Coagulopathy

- Bleeding
- Circulatory obstruction→organ hypoperfusion and ischemic tissue damage
- Renal failure, ARDS
- Microangiopathic hemolysis

-Causes

- Abruptio placentae (most common cause in obstetrics)
- SeverHemorrhage (Postpartum hge)
- Fetal Death and Delayed Delivery >2wks
- Amniotic Fluid Embolus

Septicemia

- Treatment
- Identify and treat source of coagulopathy
- Correct coagulopathy
    - FFP, cryoprecipitate, platelets

Fetal Death and Delayed Delivery

- Spontaneous labour usually in 2 weeks post fetal death
- Maternal coagulation problems < 1 month post fetal death

- If retained longer, 25% develop coagulopathy
- Consumptive coagulopathy mediated by thromboplastin from dead fetus
- tx: correct coagulation defects and delivery

Amniotic Fluid Embolus

- Complex condition characterized by abrupt onset of hypotension, hypoxia and consumptive coagulopathy
- 1 in 8000 to 1 in 30 000 pregnancies
- "anaphylactoid syndrome of pregnancy
- Pathophysiology: brief pulmonary and systemic hypertension→transient, profound oxygen desaturation (neurological injury in survivors) → secondary phase: lung injury and coagulopathy
- Diagnosis is clinical
- Management: supportive
- Prognosis:60% maternal mortality; profound neurological impairment is the rule in survivors
- fetal: outcome poor; related to arrest-to-delivery time interval; 70% neonatal survival; with half of survivors having neurological impairment

Septicemia

- Due to septic abortion, antepartum pyelonephritis, puerperal infection
- Endotoxin activates extrinsic clotting mechanism through TNF (tumor necrosis factor)
- Treat cause

Abortion

Coagulation defects from:

- Sepsis (*Clostridium perfringens* highest at Parkland) during instrumental termination of pregnancy
- Thromboplastin released from placenta, fetus, decidua or all three (prolonged retention of dead fetus)

Kaplan notes:

1- Uterine atony :

- Most common cause of PPH
- **Risk factors :** rapid or protracted labor, chorioamnionitis, medication (MgSO4, B-adrenergic agonists), and overdistended
- **Clinical findings:** soft uterus palpable above the umbilicus
- **Management:** uterine massage and uterotonic agents (e.g: oxytocin, methylergonovine, carboprots)

2- Lacerations :

- **Risk factors :** uncontrolled vaginal delivery and operative vaginal delivery.
- **Clinical findings:** identifiable laceration (cervix, vagina, perineum) in the presence of contracted uterus.
- **Management :** surgical repair

3- Retained placenta:

- **Risk factors:** accessory placental lobe and abnormal trophoblastic uterine invasion (cervix, vagina, perineum)
- **Clinical findings:** missing placenta cotyledons in the presence of contacted uterus
- **Management :** manual removal or uterine curettage under US guidance.

4- Disseminated intravascular coagulation:

- **Risk factors:** abruption placenta, sever preeclampsia, aminiotic fluid emberiolism, and prolonged retention of dead fetus.
- **Clinical findings:** generalized oozing or bleeding from IV sites or lacerations in the presence of contracted uterus
- **Management:** removal of pregnancy tissue from the uterus, ICU support, and selective blood- product replacement

5- Uterine inversion:

- **Risk factors:**myometrial weakness and previous uterine inversion
- **Clinical findings :** beefy appearing bleeding mass in the vagina and failure to palpate the uterus abdominally
- **Management:** uterine replacement by elevating the vaginal fornices and lifting the uterus back into its normal anatomic position, followed by IV oxytocin

Summary 1: Remember 4 Ts

- Tone
- Tissue
- Trauma
- Thrombin

TONE: Rule out Uterine Atony :

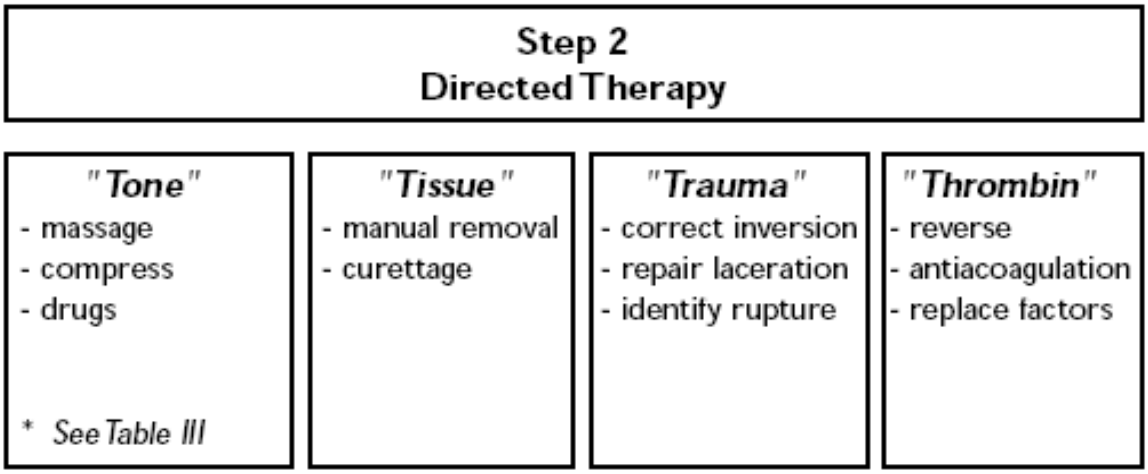
- Palpate fundus.
- Massage uterus.
- Oxytocin
- Methergine
- Hemabate

Tissue: R/O retained placenta:

- Inspect placenta for missing cotyledons.
- Explore uterus.
- Treat abnormal implantation.

TRAUMA: R/O cervical or vaginal lacerations.

- Obtain good exposure.
- Inspect cervix and vagina.
- Worry about slow bleeders.
- Treat hematomas



Summary 2

Postpartum hemorrhage:

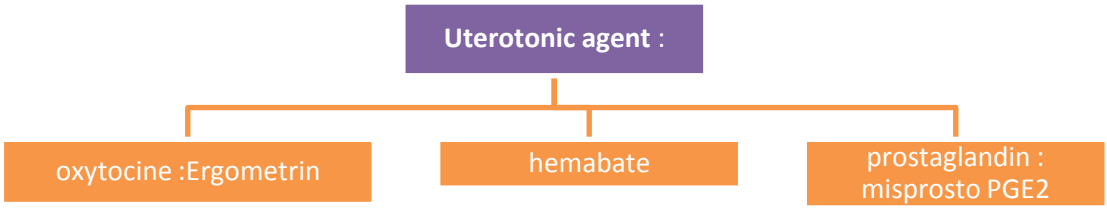
- Primary :3rd trimester >>> 24 hours after delivery
- Secondary: 24 hours after delivery >>>6 weeks

Normal blood loss:

- Vaginal delivery: 500 ml
- Cesarean section : 1000 ml

Causes : 4Ts

Tone : uterine atony	Most common cause of PPH Risk factors : tocolytics medications Rx: uterine agent , uterotonic agent
Tissue: retained placenta	Risk factors: accessory placenta lobe Rx: manual removal , uterine curettage under us guidance
Truma :1-uterine inversion 2- laceration	Rx: surgical repair .
Thrombin: Disseminated intravascular coagulation	Risk factors: abruption placenta , preeclampsia . Rx :selective blood product replacement



Antenatal Care(Prenatal Care).

Is the complex of interventions that a pregnant woman receives from organized health care services (its goal is to prevent complication to fetus or mother).

What should happen at antenatal appointments?

The schedule of appointments as the first (booking), and then at 16,
18–20, 25, 28, 31, 34, 36, 38, 40 and 41 weeks is organized and modified according to the need of the pregnant woman.

In booking (first visit for pregnant): physical examination include BMI, BP, sings of pregnancy(skin changes , enlarge of breast ...) ,fundal examination (at 12 weeks and above) ,abdominal examination
Investigation : CBC (looking for haemoglobin),glucose challenge test , serology (TORCH), urine analysis (protein and glucose) and US (in first at 16-18 weeks to know gestational age and look for congenital malformation and third visit in 28-34 weeks looking for placenta localization)

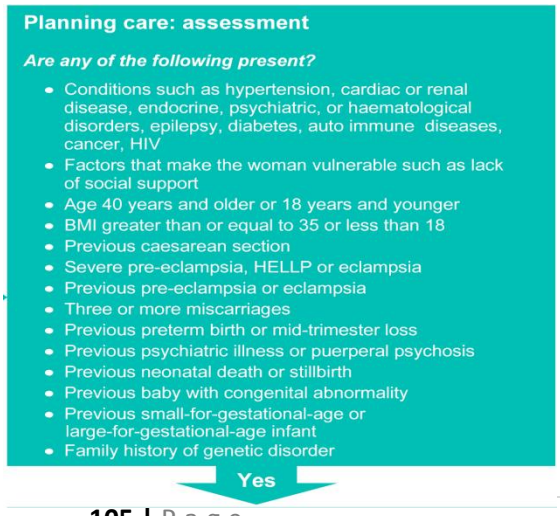
The PURPOSE of antenatal care is:

-To prevent or identify and treat conditions that may threaten the health of the fetus/newborn and/or the mother-
To help a woman approach pregnancy and birth as positive experiences-
- To help provide a good start for the newborn child

Nulliparous (1st pregnancy) porous : have been pregnant before

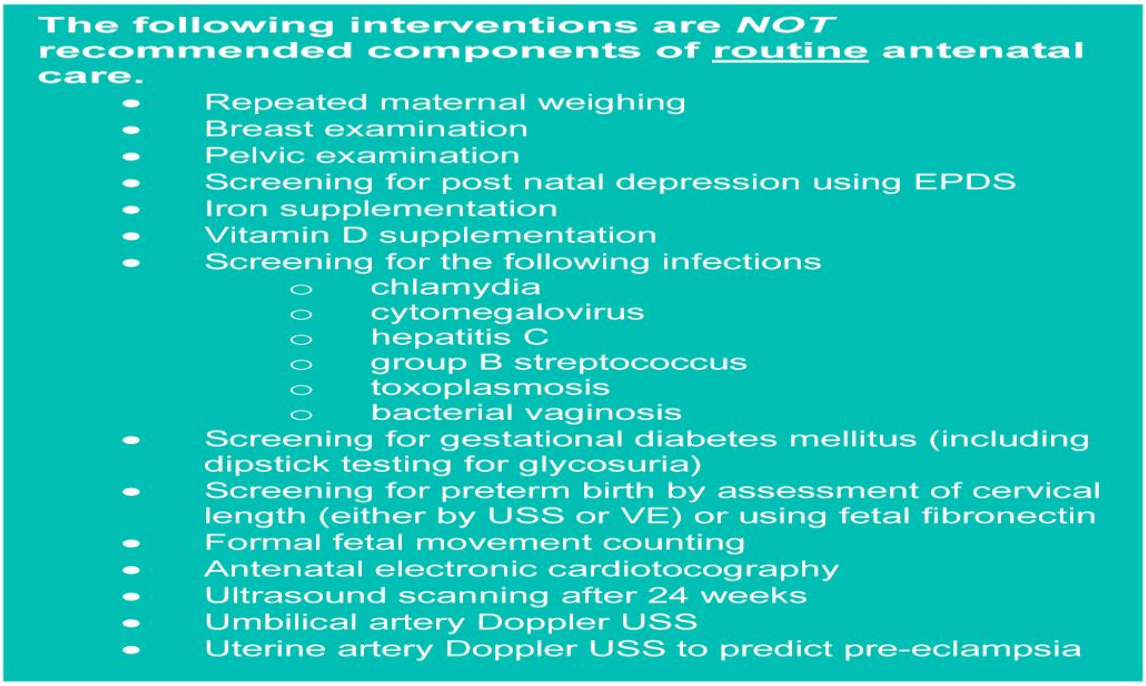
Pregnancy is divided into trimesters :
First trimester last until 12 weeks but also defined as up to 14 weeks
Second trimester last from 12 to 14 until 24 to 28 weeks
Third trimester from 24 to 28 until delivery
An infant delivered after 24 consider to be viable

Women who may need additional care



These women are likely to need additional care which is outside the scope of this guideline. The care outlined here is the **'baseline care'.**

week	Early in pregnancy (before 16 weeks)	2 visit	3rd visit (28 weeks)	32 weeks	36 weeks	38 weeks	41 weeks
Screening test	Blood test to screen for : -Blood group , rhesus status and red cell antibodies . -Hemoglobin for anemia -HBV, rubella, syphilis Urine test to screen for asymptomatic bacteriuria US to determine gestational week Down's syndrome screening 11-14 week nuchal translucency Serum screening 14-20 week	Review and discus the result Measure BP and test the urine and symphysis fundal height	-Measure symphysis fundal height+BP ,urinanalysis , -repeat screening for anemia and atypical red cells antibodies -Offer 1st does of anti-D if rhesus negative	-Measure symphysis fundal height+BP ,urinanalysis -Offer 2nd does of anti-D if rhesus negative	-Measure symphysis fundal height+BP ,urinanalysis -check presentation -offer ECV if breech	-Measure symphysis fundal height+BP ,urinanalysis	-Measure symphysis fundal height+BP ,urinanalysis -offer membrane sweep -offer induction after 41
Total appointment for nulliparous 10 parous women 7							



Important indications of health care

1-Perinatal deaths: refers to the number of stillbirths and deaths in the first week of life (early neonatal mortality).

2-Maternal deaths : The death of a woman while pregnant or within 42 days of termination of **pregnancy**, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes.

Antepartum Intrapartum Fetal Monitoring.

Fetal assessment

- **Aim Of Fetal Assessment:** Ensure fetal wellbeing (Identify patients at risk of fetal asphyxia)> to prevent prenatal mortality & morbidity.
  - Asphyxia: a lack of oxygen or excess of carbon dioxide in the body that is usually caused by interruption of breathing and that causes unconsciousness
- **Screening For High Risk Pregnancy**
  - History(history of previous abnormal baby, past medical history, any associated risk factor)
  - Age (about 35 and more associated with higher risk of mortality and morbidity rate, higher risk of chromosomal abnormalities and higher risk of malformation, chronic diseases)
  - Social burden
  - Smoking
  - Past medical conditions e.g D.M, HTN
  - Past Obstetric history (previous abnormal baby, IUGR , fetal death..)
- **Fetal And Neonatal Complications Of Antepartum Asphyxia**

- Stillbirth (Mortality)(it is a death of the fetus after 24 weeks of pregnancy / within labor or at the first day after delivery)
- Metabolic acidosis at birth
- Hypoxic renal damage
- Necrotizing enterocolitis
- Intracranial haemorrhage
- Seizures
- Cerebral palsy

- **Conditions Associated With Increased Perinatal Morbidity/Mortality**
  - Small for gestational age fetus (IUGR)
  - Decreased fetal movement
  - Postdates pregnancy (>294 days)
  - Pre-eclampsia/chronic hypertension
  - Pre-pregnancy diabetes
  - Insulin requiring gestational diabetes
  - Preterm premature rupture of membranes
  - Chronic (stable) abruption
- **When To Start Fetal Assessment Antenatally**

- Risk assessed individually
- For D.M. fetal assessment should start from 32 weeks onward if uncomplicated
- If complicated D.M. start at 24 weeks onward..**MCQ**
- For Post date pregnancy start at 40 weeks
- For any patient with decrease fetal movements start immediately
- Fetal assessment is done once or twice weekly

▪ Antenatal Fetal Assessment

1. Fetal movement counting
2. Non stress test
3. Contraction stress test
4. Ultrasound fetal assessment
5. Umbilical Doppler Velocimetry

➢ Fetal Movement Counting .

1. **Cardiff technique**: Done in the morning, patient should :calculate how long it takes to have 10 fetal movement

**10 movements should be appreciated in 12 hours if it less than 10 movements the patient at risk.

2. **Sadovsky technique**: For one hour after meal the woman should lie down and concentrate on fetal movement ,4 movement should be felt in one hour **..**If not, she should count for another hour

** If after 2 hours four movements are not felt, she should have fetal monitoring

➢ Non Stress Test .

This test assesse the frequency of fetal movements using an external fetal heart rate (FHR) monitoring device
to detect the present or absent of acceleration. "From Kaplan Lecture Notes"

*The base line fetal heart rate 120-160 beats/minute..**MCQ**

Done using the CardiotocometryRecord for 20 minuteswith the patient in left lateral position. Why?

- (Cardiac output is the lowest in the supine position because of the inferior vena cava compression resulting in increased cardiac return. CO is the highest in the left lateral position) "From Kaplan Lecture Notes"

1. **Reactive CTG**:
   - At least two accelerations from base line of 15 bpm for at least 15 sec within 20 minutes
     - Accelerations mean the fetal heart rate will increase more than the basement
2. **Non reactive**:
   - No acceleration after 20 minutes >proceed for another 20 minutes
   - If non reactive in 40 minutes >proceed for contraction stress test or biophysical profile
   - The positive predictive value of NST to predict fetal acidosis at birth is 44%

Table 12-1. Nonstress Test (NST)

Reactive NST	**Criteria:** ≥2 accelerations in 20 min: ↑ FHR ≥15 beats/min and lasting ≥15 seconds
	Assessment: reassuring of fetal well-being
	Follow-up: repeat weekly/biweekly
Nonreactive NST	**Criteria:** no FHR accelerations or did not meet criteria
	Assessment: sleeping, immature, or sedated fetus; acidotic, compromised fetus?
	Follow-up: VAS
	If still NR: do CST or BPP

Definition of abbreviations: BPP, biophysical profile; CST, contraction stress test; FHR, fetal heart rate; VAS, vibroacoustic stimulation.

➢ Contraction Stress Test .

- Fetal response to induced stress of uterine contraction and relative placental insufficiency
- The aim of that to see the response of fetal heart rate with those contractions
- Should not be used in patients at risk of preterm labor or placenta previa
- Should be proceeded by NST.. Why?To check if there are already contractions
- Contraction is initiated by nipple stimulation or by oxytocin I.V.
- The objective is 3 contractions in 10 minutes"Definition of normal labor"
- If late deceleration occur >positive CST

1.

Table 12-2. Contraction Stress Test (CST)

Negative CST	No **late decelerations** are seen in the presence of 3 uterine contractions in 10 min
	Assessment: reassuring of fetal well-being
	Follow-up: repeat CST weekly as needed
Positive CST	Repetitive **late decelerations** are seen in the presence of 3 uterine contractions in 10 min
	Assessment: worrisome, especially if nonreactive non-stress test
	Follow-up: prompt delivery

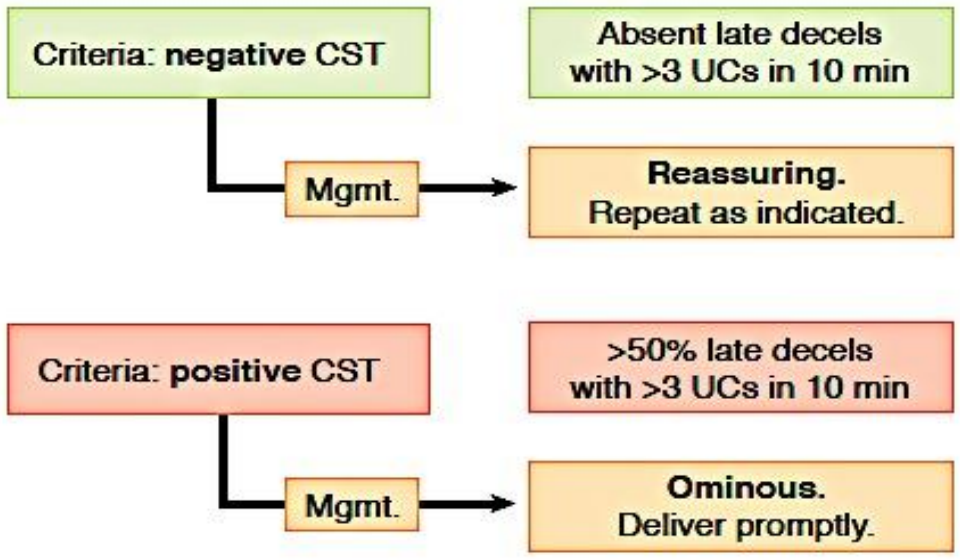


Figure I-12-2. Contraction Stress Test

- **Interpretation of CTG**(important and you have to memorize all the definitions)

  - Normal Baseline FHR 110–160 bpm
  - Moderate bradycardia100–109 bpm
  - Moderate tachycardia 161–180 bpm

    Occure with Hypoxia, Chorioamnionitis, Maternal fever , B-Mimetic drugs, Fetal anaemia,sepsis,htfailure,arrhythmias
  - Abnormal bradycardia< 100bpm
  - Abnormal tachycardia >180bpm

- **Deceleration IMP *****

1. **Early deceleration**: occurring at the same time as the contractions.
   Ex: Head Compression..**OSCE & MCQ**
   - That occur at delivery at the second stage when the head pass through the bone pelvis
2. **Late deceleration** :persisting after contraction has finished.
   Ex: U-Placenta Insufficiency
3. **Variable deceleration** :variation in shapes and timing.
   Ex: Cord Compression, Primary CNS Dysfunction.

- **Partogram**

It is a sheet of paper in the delivery room to Evaluate the progression of labor**QCM** ،including :

- Iv fluids & avoid oral intake-
- Maternal vital signs every 1-2 hours
- Input-output monitoring
- Analgesia
- Fetal heart rate monitoring (CTG)
- Uterine contractions monitoring
- Vaginal examination for cervical dilatation &poistion inactivephase every 2 hours
- Amniotic membranes status & amniotic fluid colour

Ultrasound Fetal Assessment

- From kaplan lecture notes

MODALITIES

- **Transvaginal sonogram:** used in first trimester, producing high-resolution images that are not influenced by maternal BMI. Dating accuracy of first trimester sonogram is +/- 5 days.
- **Transabdominal sonogram:** used any time during the pregnancy, but image quality may be limited by maternal obesity. No adverse fetal effects have been noted during decades of research studies. Dating accuracy of early second trimester sonogram is +/- 7-10 days.
- **Doppler** ultrasound studies: used to assess umbilical artery (UA) and middle cerebral artery (MCA) blood flow. This modality assesses fetal well-being in IUGR pregnancies as well as fetal anemia in alloimmunized pregnancies.

- **Assessment Of Growth**

1. Biometry:
    1. Biparietal diameter (BPD)
    2. Abdominal Circumference (AC)
    3. Femur Length (FL)
    4. Head Circumference (HC)
2. Amniotic fluid
3. Placental localization, why is it important?

    To detect placenta previa (land marks: bladder and cervix)

➢ Biophysical Profile (BPP)

- **" from Kaplan lecture notes" A complete BPP measures 5 components of fetal well-being:NST, amniotic fluid volume, fetal gross body movement, fetal extremity tone, and fetal breathing. The last 4 components are assessed using obstetric ultrasound. Score given for each component are 0 or 2, with maximum possible score 10 and minimum score 0.**
    1. **Score of 8 – 10highly reassuring of fetal well-being.Management is to repeat the test weekly or as indicated.**
    2. **Score 0f 4 – 6 worrisome. Management is delivery if the fetus is ≥ 36 weeks or repeat the BPP in 12-24 h if < 36 weeks. An alternative is to perform a CST.**
    3. **Score of 0 – 2 highly predictive of fetal hypoxia, management is prompt delivery regardless of gestational age.**

" From First Aid"

A biophysical profile (BPP) is the combination of the non-stress test andan ultrasound exam, for a total of five components:

1. NST: Appropriate variation of fetal heart rate.

2. Breathing: ≥ 1 episode of rhythmic breathing movements of 30 sec or more within 30 min.

3. Movement: ≥ 3 discrete body or limb movements within 30 min.

4. Muscle tone: ≥ 1 episode of extension with return to flexion oropening/closing of a hand.

5. Determination of amniotic fluid volume: Single vertical pocket of amniotic fluid measuring ≥ 2 cm is considered adequate* (or an amniotic fluid index > 5 cm).

Each of the category is given a score of 0 or 2 points:

0: Abnormal, absent, or insufficient.

2: Normal and present as previously defined.

Test Score Result	Interpretation	Management
10 of 10 8 of 10 (normal fluid) 8 of 8 (NST not done)	Risk of fetal asphyxia extremely rare	Intervention for obstetric and maternal factors
8 of 10 (abnormal fluid)	Probable chronic fetal compromise	Determine that there is functioning renal tissue and intact membranes. If so, delivery of the term fetus is indicated. In the preterm fetus less than 34 weeks, intensive surveillance may be preferred to maximize fetal maturity.
6 of 10 (normal fluid)	Equivocal test, possible fetal asphyxia	Repeat test within 24 hr
6 of 10 (abnormal fluid)	Probable fetal asphyxia	Delivery of the term fetus. In the preterm fetus less than 34 weeks, intensive surveillance may be preferred to maximize fetal maturity
4 of 10	High probability of fetal asphyxia	Deliver for fetal indications
2 of 10	Fetal asphyxia almost certain	Deliver for fetal indications
0 of 10	Fetal asphyxia certain	Deliver for fetal indications

□ Umbilical Doppler Velocimetry..

Use a free loop of umbilical cord to measure blood flow in it

- **Indication:**

- IUGR
- PET
- D.M.
- Any high risk pregnancy

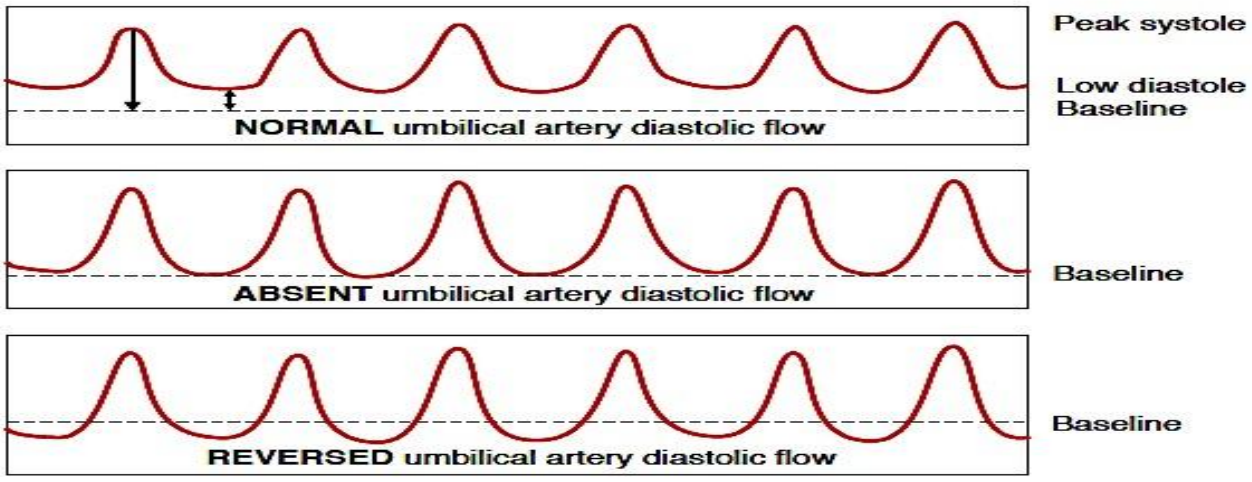


Figure I-12-3. Umbilical Artery Doppler Waveform Patterns

1. Doppler Velocimetry " From First Aid"

Doppler sonography is a noninvasive technique used to assess fetal hemodynamicvascular resistance by imaging specific fetal vessels:

- □ Umbilical artery (UA) and umbilical vein.
- □ Aorta.
- □ Heart.
- □ Middle cerebral artery (MCA).
- ○ **Commonly measured flow indices are:**
- Peak systolic frequency shift (S).
- Peak diastolic frequency shift (D).
- Mean peak frequency shift over the cardiac cycle (A).
- Systolic to diastolic ratio (S/D).
- Resistance index (S-D/S).
- Pulsatility index (S-D/A).
- ○ **Flow velocity waveforms differ in normal-sized fetuses as compared to those suffering from growth restriction:**

➢ Fetuses with normal growth: High-velocity diastolic flow.
➢ Fetuses with restricted growth: ↓ velocity diastolic flow, ↑ flow resistance (↑ S/D) in umbilical artery and ↓ resistance (↓ S/D) in MCA.
➢ Very severe intrauterine growth restriction: Flow may be absent or even reversed.

1. **Abnormal flow is usually the result of placental insufficiency and dysfunction, resulting in fetal hypoxia and acidosis. This may induce the phenomenon of brain sparing:**

- ↑ S/D in umbilical artery (↑ resistance).
- ↓ S/D in MCA (↓ resistance).
- Adaptive response to fetal hypoxemia.

Intra Uterine Growth Restriction (IUGR) + Intra Uterine Fetal Death (IUFD)

Intra Uterine Growth Restriction (IUGR) + Intra Uterine Fetal Death (IUFD)

- **Low Birth weight:** Is defined by the WHO simply as birth weight < 2.5kg, so does not correct for gestation.
- **Small for gestation age:** is used variably prenatally and postnatally to describe a fetus or neonate with growth parameters (e.g EFW, AC, birthweight) below a given centile for gestational age.

Implications of Growth Restriction

Growth restricted fetuses

- Have a higher risk of still birth and mortality
- Are most at risk of
  - hypothermia
  - hypoglycaemia
  - pulmonaryhaemorrhage
  - infection
  - encephalopathy
  - necrotisingenterocolitis
- Incidence of fetal heart rate abnormalities
- Higher incidence of operative delivery

Aetiology

There is a wide range of associations:

- Fetal
- Maternal
- Placental

These in turn may have a genetic or environmental basis

Fetal Causes ..

1. Chromosomal

Abnormal fetal karyotype can be responsible for up to 20% of growth restricted fetus

- Early pregnancy, triploidy58%
- Trisomy 46%
- Trisomy 21 and Turners – second trimester

The reason is probably because of lack of cell division or cell growth in either the fetus or placenta

2. Structural anomalies / Structural defects

- Central nervous system
- Cardiovascular system
- Gastro intestinal system
- Genito urinary system
- Muskilo skeletal

Are associated with an increased risk of IUGR

3. Infection:

- Malaria – major cause of IUGR – its treatment reduces the incidence of IUGR
- Rubella
- Cytomegalovirus
- Toxoplasmosis
- Syphilis

Can affect cell division and have all been implicated

4. Nutrient Supply

Inadequate maternal nutrition can restrict growth in the 3rd trimester

- examples are the Dutch Famine and the Leningrad sledge
- Leningrad sledge 700gmGlucose, amino acids and lactate are the major substrates for the fetus
- Oxygen : Babies born at higher altitude are smaller than those born at sea level

➢ Placental Causes

1. Lack of conversion of spiral arteries into utero placental arteries
2. The low-resistance circulation thus created allows high blood flow to the placenta.

In normal pregnancies, end diastolic flow is usually present (umbilical arteries by the early second trimester

And increases until term

3. Growth restricted fetuses often have absent or reversed end-diastolic flow in the umbilical artery – this suggests increased resistance in the feto-placental circulation

➢ Maternal Causes

1. Smoking

- Active and passive smoking is a major cause of IUGR
- Such babies weigh between 100-300gm less than other babies
- > 10 cigarettes/ day is significant
- Male fetus more affected than females
- Mechanism is probably via the higher levels of carboxyhaemoglobin in such fetuses.

2. Alcohol:

Moderate to heavy alcohol consumption can reduce fetal weight by up to 500 gm.

3. Drugs

Heroin and methadone use are associated with growth restriction <490 gm and 280 gm respectively.

4. Chronic Diseases

- Congenital heart disease – especially if cyanotic
- Chest disease e.g. cystic fibrosis, bronchitasis, kyphoscoliosm and asthma in severe cases where there is marked respiratory compromis.
- Chronic renal diseases – especially if there is hypertension, proteinuria
- Diabetes mellitus – if there is renal disease and vascular disease.

▪ Screening:

- **Clinical Examination**
  - Palpation
  - Symphyseal – fundal height – higher sensitivities than palpation
- **Ultrasound**
  - Has a better detection rate for IUGR than clinical examination
  - Only disadvantage is that the work load will be great if all pregnant women were to be subjected to it too often.

▪ Management

The terms symmetric and asymmetric growth restrictions are descriptive

- Growth restriction detected at any gestation without associated anomaly is most likely to represent true growth restriction as a result of utero placental dysfunction
- The earlier the gestation the more likely the fetus is to be aneuploidy or infected.

The gestational age should be checked using the last menstrual period and any early scans.

- Diagnosis of IUGR should be made on serial scans – every 2 weeks
- Thorough survey of the fetus for associated anomalies is undertaken
- Liquor volume should be quantified (amniotic fluid index)
- Doppler waveforms of the uterine and umbilical artery should be obtained.

➢ Early –Onset Growth Restriction (<32 Weeks)

The principle differential diagnosis are:

(a) Chromosomal abnormality or some other genetic problem
(b) Congenital infection
(c) Utero placental dysfunction

Findings that would make a chromosomal problem more likely include:

- Normal uterine artery doppler findings
- Normal liquor volume
- Presence of a structural abnormality

The commonest infection associated with IUGR is **cytomegalovirus** (CMV)

- Mother may have complained of flu-like illness
- Fetus has sonographic findings compatible with CMV (e.g.microcephaly and cerebral calcification).

Utero placental dysfunction is a diagnosis of exclusion, Factors supporting this are:

- A history of growth restriction in a previous pregnancy
- Reduced liquor volume
- Abnormal uterine umbilical artery waveforms

➢ Late-Onset Growth Restriction (>32 Weeks)

Most likely cause is utero placental insufficiency, often associated with the development of pre-eclampsia

▪ Fetal Monitoring

Monitoring the growth-restricted fetus involves serial fetal measurement

- Abdominal circumference
- Amniotic fluid index
- Cardiotocography
- Doppler ultrasound

- Fetuses with absent end-diastolic flow are hypoxaemic, these changes may appear up to 5 weeks before demise
- Reversed end-diastolic flow is suggestive of preterminalcompromise ; the fetus may die within 1-2 days if not delivered.

▪ Amniotic Fluid Index

- Reduction in amniotic fluid index (the sum of the four deepest vertical pools in each quandrant) is associated with an increase in perinatal mortality.
- Fetal urine production is significantly lower in the SGA fetus than in the AGA fetus.
- Decreased renal perfusion results in oligohydramnios

- **Biophysical Profile**
  - Breathing
  - Tone
  - Movement
  - Amniotic fluid volume
  - Cardiotocography
    - Requires about 40 mins observation of fetal breathing movements.
    - A persistently abnormal biophysical score is associated with absence of end-diastolic flow
- **Prevention:**
  - All women should be encouraged to stop smoking since it is the commonest risk factor
  - Even passive smoking is harmful – husbands should be persuaded to stop.
  - Early aspirin treatment before 17 weeks (100-150mg) for patients with previous IUGR babies (possible role of placental thrombosis)
- **Labour And Delivery**
  - In the preterm failure to delilver poses the risk of chronic hypoxia while delivery exposes the neonate to the risks of prematurity
  - Most fetuses follow a decompesation cascade:
    - Absent end-diastolic flow→decelerativeCTG → reversed end diastolic flow → fetal death
  - IUGR fetus is more likely to become more hypoxic in labour
  - With AEDF or reversed EDF, delivery should be by caesarean section

From Kaplan lecture notes

Symmetric IUGR

- All ultrasound parameters (HC, BPD, AC, FL) are smaller than expected.
- Etiology is **decreased growth potential**, i.e., aneuploidy, early intrauterine infection, gross anatomic anomaly.
- Workup should include detailed sonogram, karyotype, and screen for fetal infections.
- **Antepartum tests are usually normal.**

Asymmetric IUGR

- Ultrasound parameters show **head sparing**, but **abdomen is small.**
- Etiology is **decreased placental perfusion** due to chronic maternal diseases (hypertension, diabetes, SLE, cardiovascular disease) or abnormal placentation (abruption and infarction).
- Amniotic fluid index is often decreased, especially if uteroplacental insufficiency is severe.
- **Monitoring** is with serial sonograms, non-stress test, amniotic fluid index, biophysical profile, and umbilical artery Dopplers.

➢ Intra uterine fetal death IUFD

- The term IUFD (Intra uterine fetal death) embraces before the 28th week of pregnancy (delayed) miscarriage) and those occuring later which result in macerated stillbirth.
- **Maceration** is a destructive process which first reveals itself by blistering and peeling of the fetal skin. This appears between 12 and 24 hours after fetal death. The ligaments are softened and the vertebral column is liable to sag. The skull bones overlap each other at the sutures because of the shrinkage of the brain (Spalding's sign). It takes several days for Spalding's sign to appear after intrauterine death, usually a week or more.

▪ Causes:

1. One of the commonest is pre-eclampsia
   - ❖ Hypertensive spasm of the utero placental vessels which results into reduced oxygen supply to the fetus.
2. Chronic hypertension
3. Chronic nephritis
   - ❖ Fetus dies from placental infarction and hypoxia even before the age of viability
4. Hyperpyrexia – a body temperature over 39.40C can kill the fetus directly
5. Diabetes in pregnancy
6. Fetal malformation
7. Placental insufficiency
8. Idiopathic

▪ Management:

- Conservative – await spontaneous labour
- Induction of labour
  - Prostin E2 (Vaginal pessary)
  - IV Nalador
  - Oxytocin
- Exclude coagulation disorder
  - generallyhypofibrinogenaemia does not set in until after about 4 weeks after the IUFD.

Causes of Fetal Death Based on Trimester

" from First Aid"

T1 (1–13 WEEKS)

1. Chromosomal abnormalities.
2. Environmental factors (eg, medications, smoking, toxins).
3. Maternal anatomic defects (eg, müllerian defects).
4. Endocrine factors (eg, progesterone insufficiency, thyroid dysfunction,diabetes).
5. Unknown.

T2 (14–27 WEEKS)

1. Anticardiolipin antibodies.
2. Antiphospholipid antibodies.
3. Chromosomal abnormalities.
4. Anatomic defects of uterus and cervix.
5. Erythroblastosis.
6. Placental pathological conditions (eg, circumvallate placentation, placenta previa).

T3 (28 WEEKS–TERM)

_ Anticardiolipin antibodies.

1. Placental pathological conditions (eg, circumvallate placentation, placentaprevia, abruptio placentae).
2. Infections (eg, toxoplasmosis, CMV,

- **Management from first aid**
  - D&E may be used if fetal death occurs in T2. D&E has ↓ maternalmortality compared to PGE2 labor induction, but also has the risk ofuterine perforation.
  - Labor induction if fetal death occurs in T3. Induction of labor withvaginal misoprostol is safe and effective even in patients with a prior cesareandelivery with a low transverse uterine scar.
  - Every attempt should be made to avoid a hysterotomy.
  - The patient should be encouraged to seek counseling due to emotionalstress caused by diagnosis of fetal death and length of time between diagnosisand delivery.

- **Monitoring for infections and coagulation profile if no evidence of problems we can wait for 2-3 weeks but if there is any problem deliver.**
- **Examine the fetus for malformations take blood from the cord + fetal tissue + placental tissue**

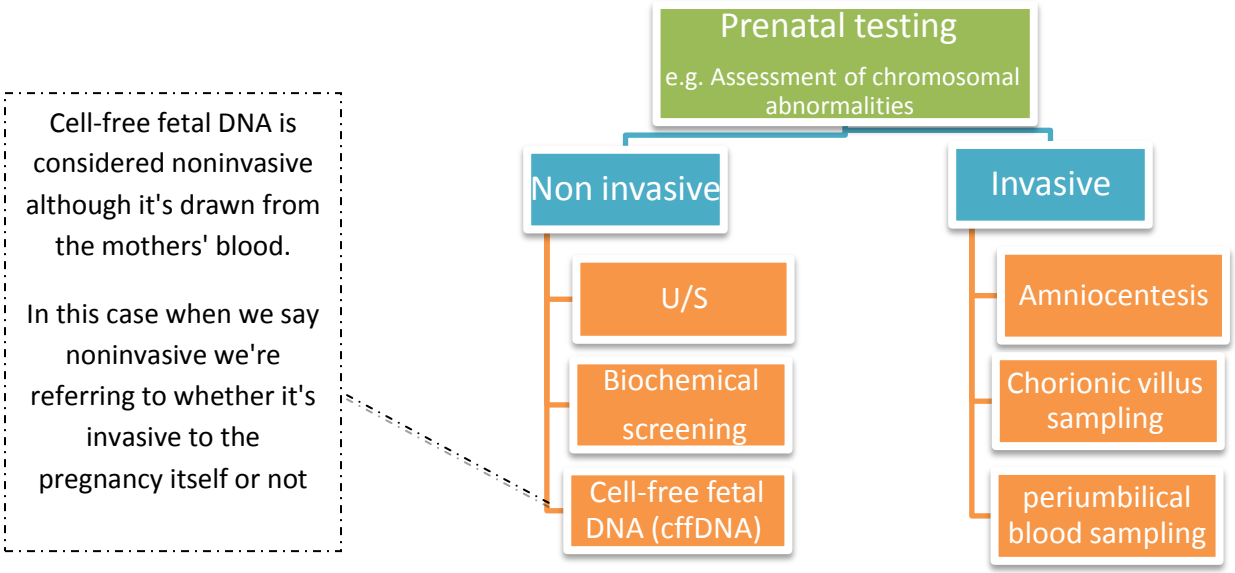
Prenatal Testing

Everything written below was mentioned by the doctor (Slides are not available for this lecture)

Prenatal Testing

Testing for fetal wellbeing during pregnancy.

E.g. chromosomal abnormalities, structure of the baby.



Cell-free fetal DNA is considered noninvasive although it's drawn from the mothers' blood.

In this case when we say noninvasive we're referring to whether it's invasive to the pregnancy itself or not

Methods available for screening for chromosomal abnormality

1- **Age of the mother :**

An old method in which screening was by the age of the mother only for e.g. if the mother is 35 she would be told that te incidence is high (This method is highly nonspecific and the false positive rate was very high

2- **Biochemical markers** : PAPPA&βHCG biochemical markers(Explained below)

Biochemical markers were developed in order to be more specific than the first method. However, they were not very specific

3- **U/S :**

Ultra sound measurement Nuchal translucency (N.T) + others(Explained below)

4- **Fetal DNA:**Free fetal DNA in maternal blood (Explained below)

The ideal screening is to do both U/S Nuchal translucency with PAPPA&βHCG biochemical markers to lower the false positive rate.(The doctor mentioned that biochemical markers are not done at KKUH)

General Facts:(The doctor said the facts are important and usually they get it in MCQs)

- The general incidence of Down is 1:1000
- The risk by maternal age:
  - at the age of 35 ----------1:365
  - at the age of 40----------1:109
  - at the age of 45----------1:32
- Risk of recurrence is 1% (0.75% higher than maternal age related risk
- In case of parental aneuploidy : 30% risk of Trisomy in offspring. (e.g. When themother or the father is a carrier of an abnormal chromosome)

1- Ultrasound screeningfor chromosomal abnormality

Nuchal translucency (N.T):(The most important way in screening and the most commonly used, it's also the easiest)

Skin fold thickness behind the fetal cervical spine.

- Timing: **11-13** $^{+6days}$ weeks of pregnancy. (timing is very imp because if you miss the time it might return to normal at later weeks)
- 75-80% of trisomy 21
- 5-10% normal karyotype(but could be associated with cardiac defects, diaphragmatic hernia, Exomphalos)

Which means it might be seen although the fetus has no chromosomal abnormalities. That's why when it is seen we move to the next step to confirm with amniocentesis or cffDNA

Markers other than N.T :

- Nasal bone: If you can see the nasal bone it means the fetus is okay if there is a short nasal bone the risk of chromosomal abnormalities is very high.
- Frontomaxillary angle : the same idea as nasal bone (usually people with Down syndrome or other chromosomal abnormalities have flat face)
- Blood flow in ductusvenosus (If you are an expert you can measure) → If abnormal there is high risk of chromosomal abnormality.

If the doctor would like to screen using one of the three points mentioned. The screening should be at the same gestational age for N.T screening (11-13 weeks)

2- Biochemical Screening

So if you find low PAPPA and normal nuchal translucency you still label the patient as high risk because she can develop IUGR ..etc

1st trimester At week 10 of pregnancy	
PAPPA: Low level is associated with chromosomally abnormal fetus. Low levels are also associated with IUGR , preeclampsia and abortion	From the mother blood
βHCG: If you find it too high for the gestational age , the risk is very high to develop chromosomally abnormal baby	

2nd trimester
Triple & quadruple Test

3- Amniocentesis

Obtaining a sample of amniotic fluid surrounding the fetus during pregnancy

Indications	
Diagnostic (at 11- 20 weeks) usually its done at 16weeks , the earlier it's done the more complications it causes)	**Therapeutic** (At anytime)
1. Chromosomal analysis (Down syndrome) 2. Spina bifida (Alpha fetoprotein) 3. Inherited diseases (muscular dystrophy) 4. Bilirubin level in isoimmunization 5. Fetal lung maturation (L/S ratio)	1. Reduce maternal stress in polyhydramnios 2. Mainly in twin-twin transfusion or if abnormality associated

Complications

1- Abdominal cramps
2- Leak or rupture membrane
3- Risk of abortion 1: 200
4- Bleeding
5- Infection e.g. chorioamniotis .
6- Injury by the needle.
7- If done therapeutically, the amount of fluid will be reduced and the fetus will be at risk of developing Club foot.

If you suspect the mother is having an infection for e.g. toxoplasma and you want to know if the fetus is infected or not you can do amniosentesis (if she's in first trimester so pregnancy can be terminated).

4- Chorionic Villus Sampling (CVS)

Sampling is done to the cyto-trophoblasts (**not syncitiotrophoblast**)

Indications for CVS	
Fetal karyotyping	**Genetic testing**
1. Advanced maternal age 2. Abnormal first trimester biochemical screen 3. Ultrasound findings 4. Personal and family history of trisomy 5. Abnormal parental karyotype	1. Family history of single gene disorder 2. Cystic fibrosis 3. Duchenne muscular dystrophy 4. Osteogenesisimperfecta

- Done between 10-14 weeks of pregnancy
- Risk of abortion : 1:100 (higher than amniosentesis)
- If the mother is Rh – if willing to do any procedure should be given anti D
- False rate : 1 % (because of the mixture in the placenta between maternal and fetal blood, so it can be the mothers' blood)

5- **periumbilical blood sampling**

Obtaining blood from umbilical cord during pregnancy.

It is done at the insertion of the umbilical cord into placenta to prevent bleeding .

Indications	Complications
1- Genetic testing. 2- If the patient needed screening but presented too late for nuchal translucency or amniocentesis or the patient is oligohydromnious and can't do amniocentesis . 3- If you're suspecting that the fetus is infected, it can be performed to confirm the infection 4- Low fetal hg or blood level (fetal anemia). So blood transfusion can be done through umbilical cord .	1-Fetal bradycardia. 2-Intrauterine death 3-Umbilical artery spasm **Due to its complications:** 1- It is performed in the delivery room 2- Dexamethasone is given for maturation of lung of the fetus. 3- Neonatal intensive care is prepared to take the baby in case of delivary 4- OR is prepaired for C-section

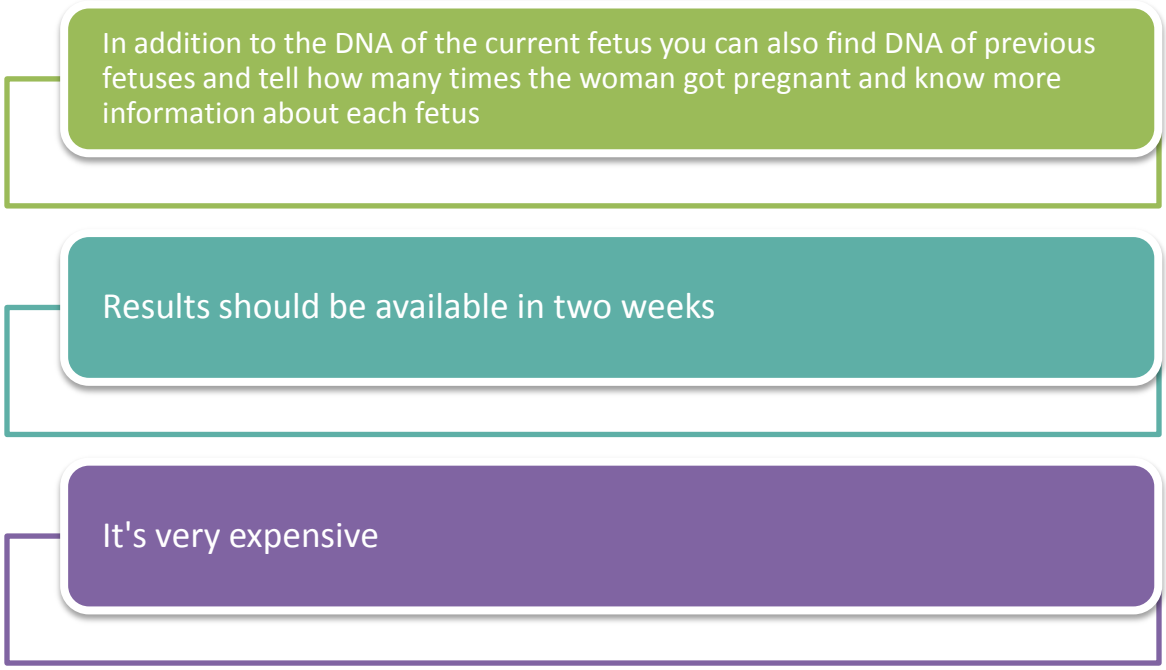
Noninvasive prenatal testing:

6- Cell-free fetal DNA

Free fetal DNA in maternal blood

- A sample of maternal blood ,where DNA of the fetus is separated from
- Allows to know the age of the fetus , blood group, any type of abnormal genes and sex.
- Can be done as early as 10 weeks .

Indications	
Detect chromosomal Abnormalities	Know the sex of the fetus
Know fetus blood group	Myotonic dystrophy
Beta thalassemia	Autosomal recessive disorders
Autosomal dominant disorders	Huntington disease
Cystic fibrosis	



An Alternative for all previous testing mentioned above!

Preimplantation genetic diagnosis:

Diagnosis of an embryo by IVF to know whether it's a carrier orhave any abnormal gene or chromosome before implanting it in the mother.

Multiple pregnancy.

Objectives:

-Incidence
-Diagnosis of multiple pregnancy
-Mechanism of twinning &Zygosity
-Complication of multiple pregnancy
-Causes of perinatal mortality & morbidity
-Twin to twin transfusion
-Antenatal management of multiple pregnancy
-Assessment of chorionicity by ultrasound
-Management of labour in multiple pregnancy

- WHAT THE DOCTOR MENTIONED
- The sentences in red were already red in the doctor's slides

Incidence of multiple pregnancy

- The natural rate of twinning is 1:90
- Slightly higher in blacks than whites
- In USA the incidence is 3%
- The incidence is increasing due to Assisted reproduction technique(ART)and ovulation induction
- The incidence of monozygotic twins is constant and is 4:1000 pregnancies
- The incidence of dizygotic twins increase with age, parity, weight(obesity) , height, and is higher in some families

  Dizygotic twins are higher in families with history of twins

Diagnosis of multiple pregnancy

Suspected if:

- Large for date uterine size

  For example: if the patient comesto the clinic and you calculate the gestational age according to the last menstrual period and you find that she is 16 weeks and when you do the examination the findings tell you that she is 20 weeks.
- Multiple fetal heart rates are detected
- Multiple fetal parts are felt
- HCG & maternal serum alpha-fetoprotein is elevated for gestational age
- Pregnancy with ART

Confirmed by ultrasound

Zygosity

Dizygotic: The commonest

- Diamniotic/Dichorionic
- 70-80% of all twins
- Fertilization of two ova
- Each fetus will be surrounded by amnion &chorion(each fetus has its own placenta)

Monozygotic:

- 20-30% of all twins
- Result from cleavage of a single fertilized ova
- The timing of cleavage determines placentation

1- **Dichorionic/diamniotic monozygotic twins:** This type behave like dizygotic

- Cleavage in the first 3 days after fertilization
- Each fetus will be surrounded by amnion &chorion(each fetus has its own placenta)like dizygotic twins
- Has the lowest mortality rate of monozygotic twins <10% of all monozygotic twins

2-Monochorionic/diamniotic:

- Cleavage between day 4 and 8 after fertilization
- Share single placenta but separate amniotic sac
- The mortality is 25%

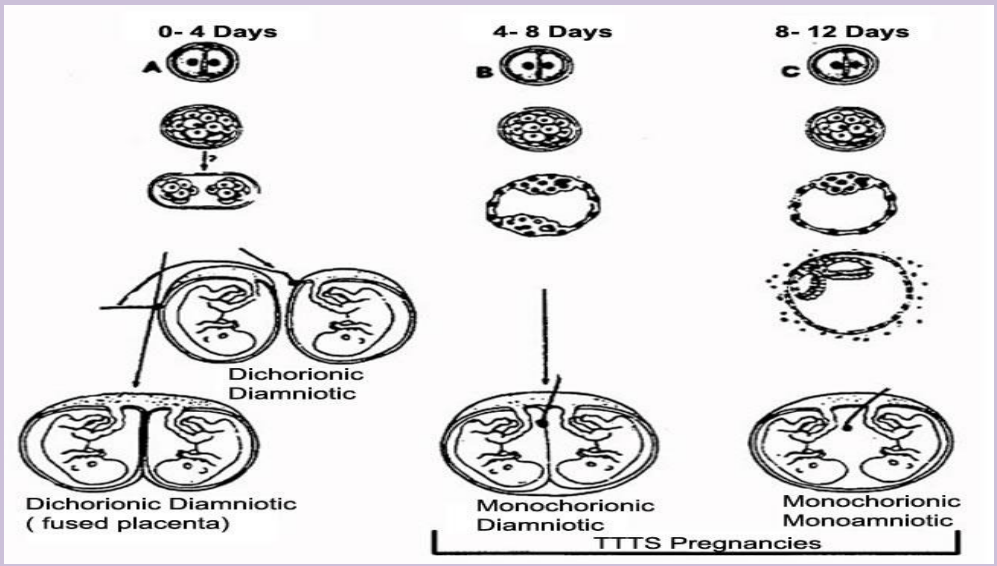
3-Monochorionic/monoamniotic: The worst type

- < 1% of cases
- Cleavage after the 8th day (day 9-12)
- Share single placenta & single sac
- Mortality is 50-60%, usually before 32 weeks

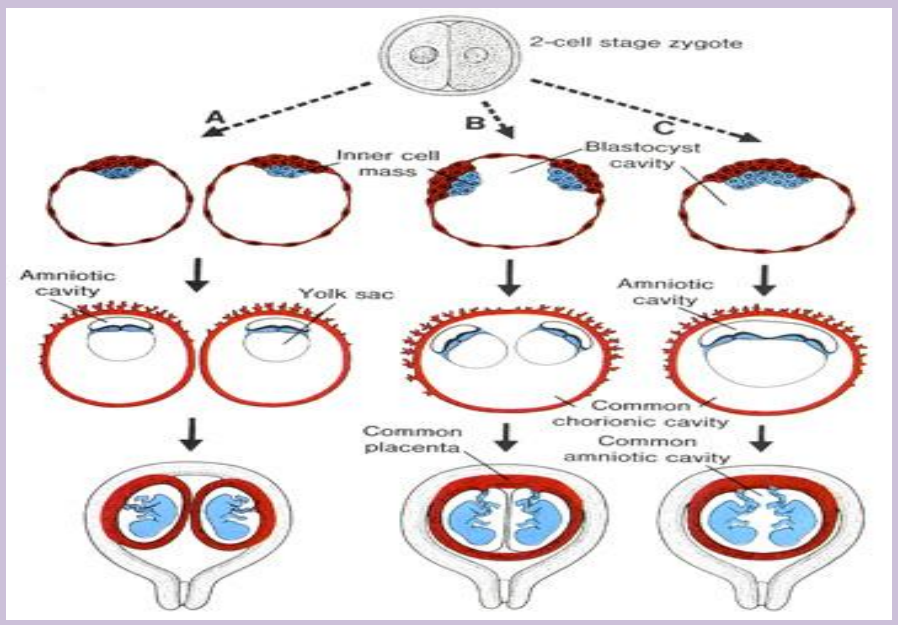
4-Conjoined twins:

- Cleavage after day 12
- Incidence is 1: 70,000deliveries
- The fetuses may fuse in a number of ways, most commonly chestand/or abdomen

Mechanism of twining



Monozygotic twins



Complications of multiple pregnancy

- High perinatal mortality & morbidity (3-4 times higher than singleton pregnancy)
- Abortion(<50% of twins diagnosed in the first trimester result in live
  - Nausea & vomiting"Hyperemesis gravidarum"

    Because of the high level of HCG

    They come to the emergency very dehydrated + high ketone and some time the sever vomiting cause lower GI bleeding
  - IUGR(Intrauterine growth restriction)

    The weight should be less than 2.5 at term delivery
- Preterm labour (50%)(twins delver at 37 weeks, triples at 33 weeks, Quadruplets at 29 weeks extreme prematurity)
- PET (3 times higher than singleton)
- Polyhydramnios (in 10%)
- Congenital anomalies
  - Postpartum hemorrhage

    Many mechanism one of them : OVER STRETCHING UTERINE→ UTERINE ATONY

    Normally contraction of the uterine muscle compresses the vessels and reduces flow and when the uterus is not contracted, the mother's blood vessels continue to pump "bleeding"
- Placental abruption, placenta previacaused by abnormal placentation site
-Discordant twin growth (more than 20%discrepacy in fetal weights)

In multiple pregnancy do more frequent ultrasound after 24 weeks because it may at any time discordant growth happens and when it happenes they are at higher risk that the placenta is feeding only one fetus so you have to observe closely and deliver her before intrauterine fetal death happens

-Malpresentation, cord prolapse, Operative delivery

Causes of perinatal mortality & morbidity

- Prematurity (Respiratory distress syndrome)
- Birth trauma
- Cerebral hemorrhage
- Birth asphyxia
- Congenital anomalies
- Still birth

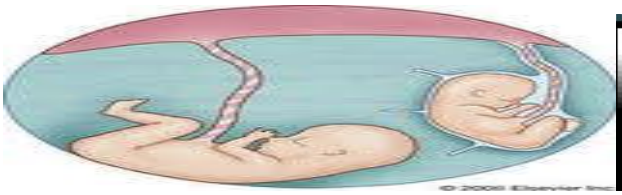
The second twin carry risk more than first twin

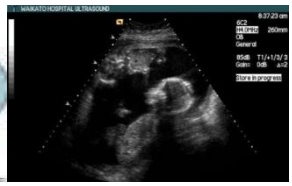
Twin-twin transfusion (TTN)

- Occur in 20-25% of monochorionic twins
- One fetus donate blood to the other due to vascular anastomosis
- The recipient fetus will have heart failure"too much fluid:, polyhydramnios, and hydrops
- The donor will have IUGR &oligohydramnios

  if the gestational age close to the maturity give dexamethasone and deliver them

-





Management:

-includes amnio-reduction of the receipient twin

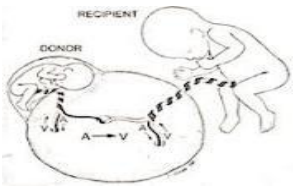
-intra-uterine blood transfusion for the donor twin

-selective fetal reduction

This happens when patients go to IVF center and they give her injection to stimulate the ovulation. in result to stimulation one ova gives 6 or 7 , instead of placing 3 , which is the maximum number , they place 6 because they think only one or two will be implanted surprisingly all the 6 get implanted and they decided to kill some of them

*لم يجيز ها العلماء

-fetoscopic laser ablation of placental anastomosis



Antenatal management of multiple pregnancy

Adequate nutrition (300 additional calories per day per fetus)

Prevent anemia

- More frequent antenatal visits

Normally in single pregnancy antenatal visits should be every month until 28 weeks then every 2 weeks until 36 weeks then every week , while in multiple pregnancy must be more frequent.

Ultrasound:

- Assess chorionicity at 9-10 weeks
- Nuchal translucency at 12-13+ weeks

  Measure the thickness of the skin at the back of baby's neck. "screening test for down syndrome"
- Assessment of fetal growth & fetal wellbeing every 3-4 weeks from 23 weeks onward
- Multifetal reduction may offered for high order multiple gestation in the first trimester

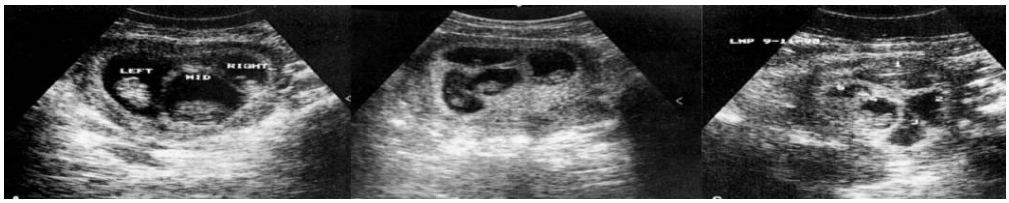
Preterm labour risk:

Serial cervical length assessment

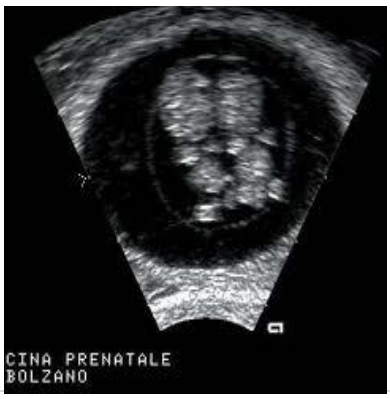
Steroids for fetal lung maturation

Assessment of chorionicity by ultrasoundNot important

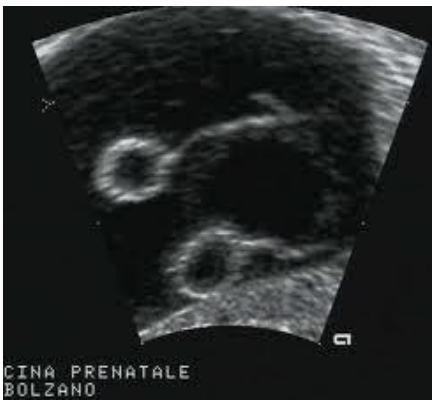
Multiple gestational sacs in first trimester



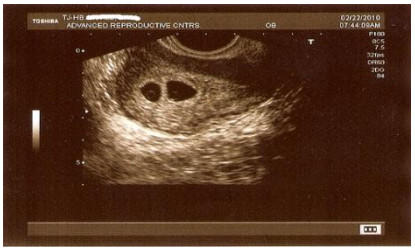
Conjoined twins



2 yolk sacs



2 gestational sacs

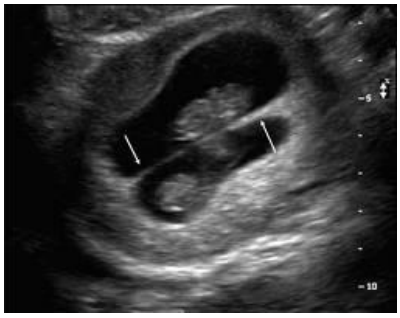


T sign

Monochorionic twin

Twin Peak Sign (Lambda)

Dichorionic twins



Management of labour in multiple pregnancyimportant

-Controversial

-Depends on presentation ex: breech→ CS, gestational ageex: preterm +rupture membrane +twins → CS , presence of fetal complications, experience of the obstetrician

-usually if the first fetus is cephalic→normal delivery

patient with assisted reproduction and prolonged infertility it is better to do CS because the second twin is at risk to change the position

-Non vertex first twin→ cesarean section

-Locked twins: Breech-vertex twins→cesarean section

- Active management of third stage to prevent PPH

Pre-requisite for intra-partum management of multiple pregnancy

- Secondary or tertiary center
- Well-functioning large-bore IV line
- Availability of emergency C/S –anesthesia- blood bank
- Continuous simultaneous fetal heart rates monitoring
- Availability of NICU beds- paediatrician
- Imaging technique (ultrasound)

- **Recommended books:**

- -Essentials of obstetrics & gynecology (Hacker and Moore's) P 160-172
- -Current diagnosis & treatment –Obstetrics & gynecology (p301-3100)

- **TRY TO READ IT FOM THE BOOK**

Summary and MCQ

-The incidence of Multiple pregnancy is increasing due to Assisted reproduction technique(ART)and ovulation induction

-The incidence of dizygotic twins increase with age, parity, weight (obesity) , height, and is higher in some families

-**Suspected if:**Large for date uterine size , Multiple fetal heart rates are detected ,Multiple fetal parts are felt ,HCG & maternal serum alpha-fetoprotein is elevated for gestational age,Pregnancy with ART**Confirmed by ultrasound**

-There are two types :

1-Dizygotic:•The commonest •Fertilization of two ova • Each fetus will be surrounded by amnion &chorion (each fetus has its own placenta)

2-Monozygotic:•Result from cleavage of a single fertilized ova•The timing of cleavage determines placentation

Tming of cleavage	first 3 days after fertilization	between day 4 and 8 after fertilization	-Cleavage after the 8th day (day 9-12)	Cleavage after day 12
placentation	Dichorionic/diamniotic monozygotic	Monochorionic/diamniotic	Monochorionic/monoamniotic	Conjoined twins
	-behave like dizygotic -Each fetus will be surrounded by amnion &chorion -Has the lowest mortality rate of monozygotic twins	-Share single placenta but separate amniotic sac	-The worst type -Share single placenta & single sac	-most commonly chestand/or abdomen

Twin-twin transfusion•Occurs in 20-25% of monochorionic twins•One fetus donate blood to the other due to vascular anastomosis •recipient fetus will have heart failure hydrops•The donor will have IUGR &oligohydramnios

Management:

•amnio-reduction of the receipient twin•uterine blood transfusion for the donor twin •fetoscopic laser ablation of placental anastomosis

Management of labour in multiple pregnancy•Controversial•Depends on presentation ex: breech→ CS, gestational age ,presence of fetal complications, experience of the obstetrician•usually if the first fetus is cephalic →normal delivery

Q:Which of the following is known to be the commonest presentation in twins?

A. Breech, cephalic **B.** Cephalic, breech **C.** Cephalic, cephalic **D.** Breech, breech

E. Cephalic, transverse

Diabetes Mellitus (DM).

- What's diabetes ? Its metabolic disease characterized by ……..-
- The main effect of diabetes is the absence of insulin-
- Pregnant ladies are young generation , they usually present with no diabetes OR if they are in forties they will present with late onset or gestational diabetes
- Minority of them present with type 1 DM (juvenile-onset) -
- It's important to differentiate between these types : DM 1(juvenile-onset) , Gestational and DM 2 (late onset)
- To be pregnant in young age is better than in forties , so don't delay the pregnancy because after forties they have (DM ,HTN , chromosomal anomaly e.g. down syndrome)

Types:

Type1 D.M:

1. formerly known as juvenile-onset or IDDM
2. Absolute insulin deficiency
3. increased risk of chronic micro vascular disease at an early age
   - DM 1 happened due insulin deficiency so blood sugar will be high and these patients may suffer from micro vascular diseases (cardiac , renal ,retinopathy)
   - so if a women came to your clinic with DM1 , then she is at high risk of developing complication during her pregnancy for her and her baby.
   - you must to differentiate between DM1 and gestational in the exam , type 1 DM you have risk of chronic vascular disease at early stage.

Type2 D.M:

1. formerly known adult onset or NIDDM
2. Tissue resistance to insulin
3. lower incidence of micro vascular disease during reproductive age range .
   - NIDDM : non insulin dependent DM
   - e.g. a lady in forties has DM will be on oral hypoglycemic agents (metformin) , once she is pregnant she will shift to insulin.
   - when a pregnant women (24 weeks) came to your clinic un-booked and you found her blood glucose is high , what are you going to say gestational ,late onset DM or DM1 ????? Its gestational DM because its discovered in pregnancy regardless if she have DM before her pregnancy or not .

Gestational DM: (GDM):

1. Carbohydrate intolerance first occurs during pregnancy or first detected during pregnancy
2. 4-5% of pregnancies are complicated by DM .
3. 90% of DM in pregnancy , the cause GDM.
4. GDM will increase seven fold risk of type 2 DM .
   - DM is very common here in Ksa
   - 40% of general population have DM -
   - More than 45% of pregnant ladies they will have DM -
   - Usually the cause of diabetes in pregnancy is due to gestational (90%) and if the lady have DM in pregnancy she is at high risk to be diabetic later in life
   - Almost half of these ladies develop diabetes later on, even their babies

---

Pathogenesis of gestational DM:

1. Increased insulin resistance in second trimester and progresses as pregnancy advances due hormones (estrogen,progestrone ,cortisol, prolactin and human placental lactogen).
2. placental degradation of insulin.
   - A Lady in her 1st trimester(12-13 weeks) came to your clinic with high blood sugar and you start giving her insulin , are you going to keep her in the same dose in her 2nd and 3rd trimesters ??? No you must to increase the dose of insulin because of insulin resistance
   - Hormones of pregnancy (progesterone , estrogen , cortisol , HPL ..etc) resist the insulin , so as pregnancy advances you have to increase the dose
   - In normal lady (not diabetic) there is automatic increase of insulin level to resist these hormones.

Risk Factors for GDM:-

1. H/O GDM
2. family /h D.M
3. age > 25 Y
4. Previous macrosomic baby
5. PCO5
6. twin pregnancy (because of high levels of hormones that resist the insulin)
7. Racial (Asians , Hispanic , African – Caribbean)

Screening and diagnosis of GDM:

1 -UK: (nice)

- Whom: selective if +ve risk factors without regard to age. 10% missed
- How: at 24-28 weeks , 2 hours , 75 gm OGTT
- It is a Screening and diagnostic
- Fasting (5.1to6.90) , 2h(8.5to11)
- One reading is required to be abnormal.

*If you screen a lady in her 1st trimester and there wasn't DM this test not exclude DM ,you have to repeat at 24-28 weeks ????Because insulin resistance is increase

2-ACOG: American College Of Obstetricians And Gynecologists

Universal, more practical , sensitive , no screening if <25 y if no risk factor

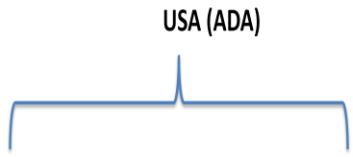
2 steps approach :

1st step :

- 50 gm oral glucose challenge → check serum glucose at I hour(no fasting required)
- ≥130mg/dl(7.2) → discover 90% of D.M Screening
- Do 3 hour.GTT glucose tolerance test or 75 gm.
- If abnormal fasting or any two abnormal→ diagnostic DM
- Follow up GTT can be done 32-34 w (to identify late onset DM)

Uk important	US and KSA important
its Screening and diagnostic at the same time 75 gm OGTT *In UK they screen only high risk group :obese , unexplained intrauterine death Previous macrosomic baby, shoulder dystocia , PCO , polyhydramnios (when mother blood glucose increase >> increase fetus blood glucose >>> increase fetus urination >> polyhydramnios .	They do screening first if the level is high they do diagnostic (challenge test). 1- give 50 mg of oral glucose for all pregnant ladies either they are fasting or not >>> screening 2-after one hour if the result is higher than (7.2) do diagnostic test by giving 75 or 100 gm or 3hour GTT >>> diagnostic *In KSA we screen all pregnant ladies because we are high risk population

The doctor didn't mention anything about this graph



ADA

ADA (AMERICAN DIABETES ASSOCIATION)

75

f

75 gm	100 gm
5.3	5.3
10	10
8.6	8.6
	7.8

Two abnormal readings are required to be abnormal

Pre pregnancy counseling (for types 1,2):

The rule of pre-pregnancy counseling: any patient with medical disorder should be controlled before pregnancy

1. high dose folic acid 5 mg (400 Microgram) up to 12/52. To prevent neural tube defect
2. evaluate renal function (24 h urine collection for protein ,creatinine clearance)
3. full history and examination , advise for diet , body weight, and exercise.
4. ophthamology referral
5. Echo (> 30 y , smoker, hypertensive)
6. cardiologist referral if suspected cardiac illness
7. monitor medications : ACEI (cause oligo hydraminos , renal failure, skull defects
8. Asprin if risk of preeclampsia . Preeclampsia =HTN +proteins in urine
9. HBA1C<6.1 if decreased less congenital anomalies (HBA1c in preg not sensitive)
   HBA1C ≥ 9.5 % carries >20% fetal major anomalies (advice women HBA1C >10% to avoid pregnancy
   What's the best test to determine the control of diabetes ? HB A1c
10. stop OHA and start insulin if required (a part from metformin).
    - Don't forget to do rubella test -
    - Why we are worried of diabetic women to get pregnant ? Because of the complication that's may affect her and her baby
    - Mother : increase the complication of retinopathy and nephropathy …etc
    - Fetus : abortion, congenital anomalies (sacral agenesis, cardiac anomalies).

Type 1 DM complications:

- FBS is low in pregnancy due increased renal clearance . in non diabetic increase in insulin to 50% to overcome the resistance
- insulin: requirement 3 times the normal dose

Mother complications:

1. hypoglycemia random blood sugar <3.9 mmol/l ,
2. nephropathy 5-10% of DM
3. chronic hyper tension
4. preeclampsia
5. preterm
6. rapid progression of microvascualr and atherosclerotic disease (IHD,HF,Cerebral ischemia),
7. DKA (Diabetic ketoacidosis):
    - Life threatening , can occur at lower blood glucose <200
    - Fetal mortality 10-30%
    - Maternal mortality is rare due to proper Rx.
    - Tx: rehydration.insulin.k and antibiotics

Pts with DKA present with:

1-abdominal pain 2-vomiting

3- acetone smell 4-infections

Rx 1-rehydration

2-oxygnation

3-pottasuim and bicarbonate

You have to differentiate between the complication in DM1 and the complication in gestations diabetes ?

1. Gestational diabetes appears after 20-24 weeks of pregnancy so the first trimester won't be effected by diabetes = no congenital anomalies in gestational diabetes ,Congenital anomalies happened only in non-controlled type1 DM.
2. In addition mother with DM1 or GDM >>>> she will usually have macrosomic fetus because of anabolic effect of insulin on cell growth
   Mother with DM1 >>>>>>>she will usually have IUFD or IUGR especially in uncontrolled diabetes because of vascular involvement that affect placental blood supply.

Fetal complications:

1. Miscarriage when Hba1c is high due congenital Anomaly for DM 1
2. congenital malformation:
   A. 30-50% of pn mortality (Hyper glycaemia is principle factor hypoglycemia, and hyperketonemia is suspected)
   B. 6-10% of diabetic mothers have major congenital anomaly
      - Cardiac (transposition of great vessels VSD , ASD, hypoplastic left ventricle , aortic anomalies , complex cardiac anomaly) it's the most common type of anomalies
      - CNS anomalies increase 10 fold .(NTD)
      - GIT malformation
      - genitourinary anomalies (poly cystic kidneys)
      - sacral agenesis (caudal regression) >>Rare: 400 times more frequent in DM
3. IUGR
4. IUFD 32-36 W in uncontrolled D.M

GDM Complications :

1. Preterm labor , B.P , c/s rate .
2. Recurrent G.dm , type 2 DM
3. Macrosomia , shoulder dystocia (fracture +palsy)
4. Neonatal hypoglycemia , birubin Level , later on obesity ,impaired GTT , intellectual
5. ↓ ca ↓ bloodsugar > Neonatal death
6. ↓ mg
7. 33% polycythemia : HCT > 65%
8. Chronic intrauterine hypoxia : increases erythropoietin production
9. Hyperbilirubinemia : neonatal jaundice (delay in fetal liver maturation in poor glycemic control)
10. RDS: fetal hyperinsulinemia : suppress production of surfactant.
11. Fetal cardiac septal hypertrophy and hypertrophic cardiomyopathy.

Macrosomia:
Causes of Macrosomia :
Glucose will pass to fetus by facilitated diffusion this will result in increased insulin production by fetus (act as growth f) > increase growth of cells
-wt 4-4.5 90^{th} percentile
-25- 42% of diabetic
-Shoulder dystocia increases 3 fold

Historical classification of White

it is still used by some experts :

A. Asymptomatic but abnormal GTT
B. onset ≥ 20 y duration < 10 y. no vascular complications
C. onset 10-19 y duration 10-19 y .no vascular complications
D. onset < 10 y duration ≥ 20 y vascular disease ,benign retinopathy, and leg artery calcification

- White classification = the relationship between the duration of time of having DM and its complications
- You don't need to know them in details only to know: if you have DM earlier you going to have more complication (retinopathy …)
- In other word : as years increase as complications increase.

Table 10-4. White Classification of Diabetes in Pregnancy

Class A1	GDM with normal FBS not requiring insulin
Class A2	GDM with elevated FBS requiring insulin
Class B	Overt DM onset after age 20 years and duration <10 years
Class C	Overt DM onset age 10–19 years or duration 10–19 years
Class D	Overt DM onset before age 10 years or duration ≥20 years
Class E	Overt DM with calcified pelvic vessels
Class F	Overt DM with nephropathy
Class R	Overt DM with proliferative retinopathy

Management :

1. Multidisciplinary (physician, midwife,obstr.nurse, nutrition consultant)
2. Referral urgently
3. Diet: CHO 40% Of total calories , vegetables , fruits of high fibers
   1800 kcal/day → 2400 kcal /d
4. Exercises : walking, yoga , swimming, upper arm ex(30 min /day)
5. glucose monitoring "glucometer" at home and to be reviewed every 1-2 weeks
6. Fasting . 1 h or 2 h after each meal (4times)

Target:

- UK fasting: 3.5-5.9
- 1h.p.p <7.8
- ACOG: F → 5.3
- 1h< 7.2
- 2 h<6.7

insulin:

1. **4 injections: 3 fast acting insulin before meals**

   1 long acting at bed time

   Fast acting :

   - standard soluble insulin, Humulin S (act rapid)
   - or fasting acting insulin analogue (novorapid,humalog)better onset 15 min ,peak 2-4 h,less hypoglycemia

NPH is insulin of choice (intermediate acting)

Neutral protamin Hagedorn , peak 6 h , last 12 h.

2. **2 injections (mixed long +short) >> increase neonatal complications**

Calculation and dose of initial insulin management :

- Don't more 60 u/day
- 0.7 u/kg (6-18 weeks)
- 0.8 u/kg (18-26)
- 0.9-1.1 (>26)
- ½ dose am (2/3 NPH , 1/3 novolog or humalog)
- ½ dose pm (1/2 NPH, ½ novolog)
- Eg: 60
  - 30 (20 NPH , 10 N)
  - 30 (15, 15)
- If steroids used (insulin)

Ante natal follow up :

1)1st trimester : control blood sugar, retinal, renal check up
2) 7-8 u/s for viability
3) 16 weeks : retinal Ex if abnormal 1st visit
4)20 W : U/S for heart and other structures .it's the best time to do ultrasound to detect cardiac anomalies
5)28 W : u/s for growth and A.F and retinal ex . If normal in 1st trimester
6) 32 U/S for growth
7) 36 u/s for growth
Discuss with pt mode of delivery and timing
8) 38 IOL Orc/s if wt > 4.5 kg
Maintain blood sugar 4-7 mmol/L during labor

Post delivery:

½ dose insulin

Modify life style , breast feeding , wt reduction , diet

-GDM : risk of DM 20-50% Within 10 y

GTT 6/52 POST Partum

At 32 weeks: do US and biophysical profile:

Us : looking for fetus growth (abdominal circumferences)

Biophysical vip :1-fetal movement

2- fetal tone

3- fetal breathing

4- amniotic fluid

5- CTG

if the baby is big or abnormal biophysical profile then terminate the pregnancy

Summary

Classification:

Table 10-3. Classification of Diabetes Mellitus by Pathophysiology

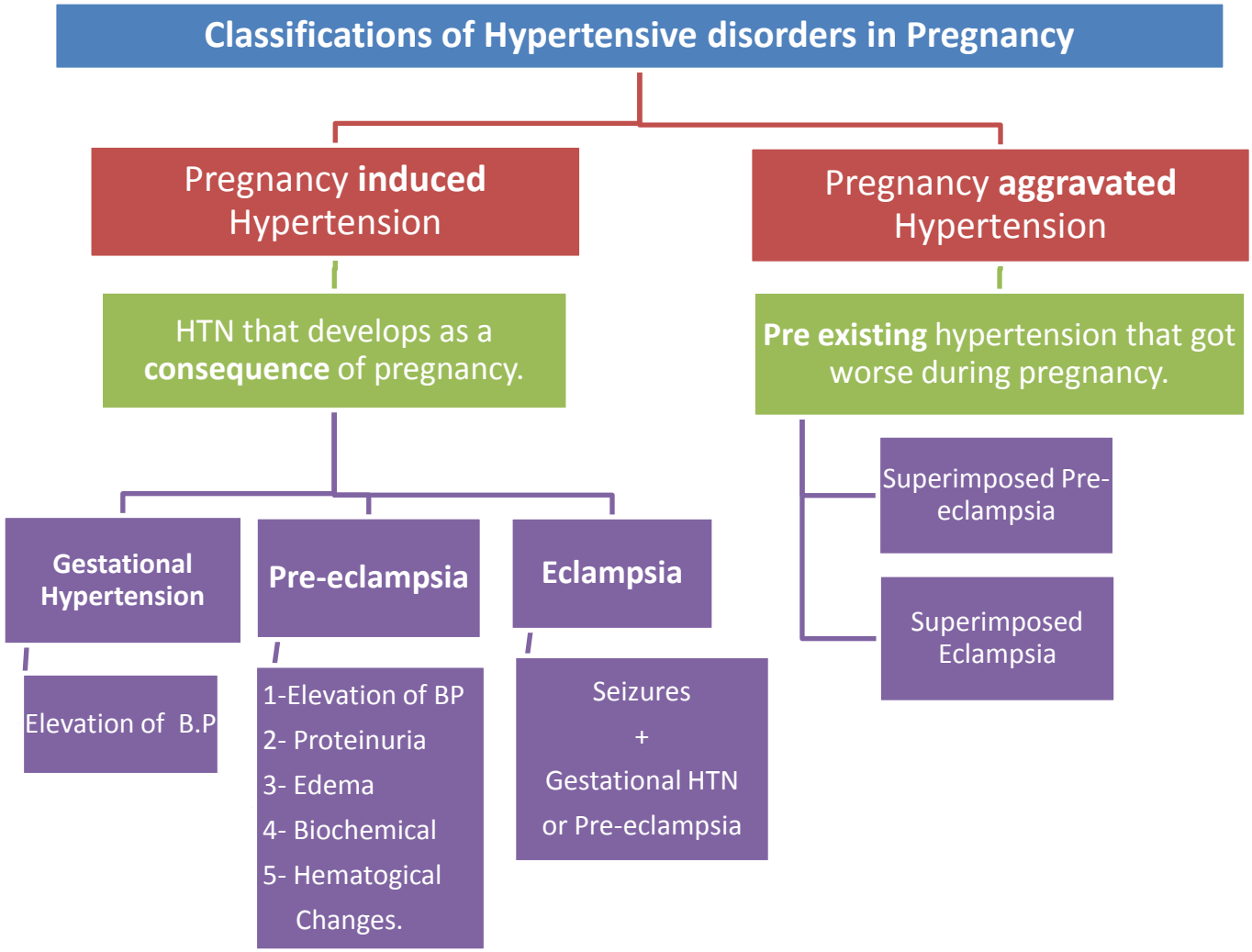
Gestational	Pregnancy onset	Insulin resistance
Type 1	Juvenile onset	Ketosis prone
Type 2	Adult onset	Insulin resistance

Gestational diabetes

Table 10-5. Gestational Diabetes

Questions	Criteria/Problems	Diag/Mgmt
1-hr 50g OGTT Screening test	<140 mg/dL	GDM ruled out
3-hr 100g OGTT Definitive diagnosis	≥2 values ↑	GDM diagnosed
Home glucose monitoring	Mean glucose values FBS >90; 1 hr pp >140	Start insulin
Fetal demise risk factors	1-Needs insulin 2-HTN 3-Previous demise	Starting 32 wk NST & AFI 2/wk
L&D problems	Arrest stage 1 or 2 Shoulder dystocia	CS if estimated fetal weight >4500 g
Post partum management	Prevent postpartum hemorrhage	FBS ≥126 mg/dL 2 hr 75 gm OGTT

Hypertensive disorders in Pregnancy.



If uncontrolled the patient may undergo:

HELLP Syndrome: Severe form of HTN in pregnancy.

- Hemolysis
- Low platelets
- Elevated liver enzyme

1. Pregnancy induced Hypertension :

HTN that develops as a consequence of pregnancy. Include 3 types:

a. Gestational hypertension:
Elevation of B.P
without Pathological edema, proteinuria, hematological or biochemical changes.
with or without fetal implication of HTN

b. Pre-eclampsia:
Elevation of BP with proteinuria and or pathological edema, biochemical and or hematogical changes.

- edema may not be present
- Edema here ➔ swelling of the face & upper extremities **not** the lower extremities!
- Pre eclampsia is further subdivided into :
  1. Mild
  2. Severe

  } further explanation is coming!

➔ No moderate category in pre-eclampsia.

c. Eclampsia: Convulsions
A complication of Gestational hypertension or Pre-eclampsia with clinical seizure!

2. Pregnancy aggravated HTN.

Pre existing hypertension that got worse during pregnancy. (already on hypertensive medication)

a. Superimposed pre-eclampsia
Patient with HTN getting worse during pregnancy + develop Pre eclampsia symptoms: (elevation of BP + proteinuria + pathological edema + biochemical changes and or hematogical changes)
Ex: Patient who has pre existing HTN getting worse by pregnancy & complicated by hematogical + biochemical changes ➔ super imposed pre eclampsia.

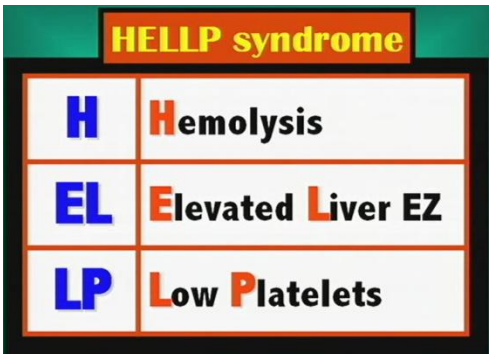
b. Superimposed eclampsia
Ex: Patient with pre existing HTN getting worse by pregnancy & complicated by hematogical + biochemical changes with seizure ➔ super imposed eclampsia.

3. HELLP Syndrome:

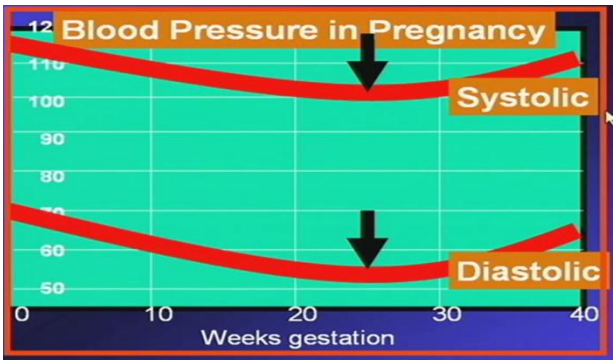
Sever form of HTN in pregnancy.

- **Hemolysis**
- **Low platelets**
- **Elevated liver enzyme**

Common MCQ's: components of HELLP syndrome



- **Physiological Changes in Blood pressure during pregnancy:**



- Slight drop in 1st trimester
- continuous through the 2nd trimester
- gradual rise to pre-pregnancy level in the 3rd trimester.

- HTN is never normal in pregnancy.

- Elevation of BP above pre-pregnancy

- The curve shows the systolic & diastolic BP changes , between 20 & 30 weeks there is a drop & continuous in the 2nd trimester & then rise again in the 3r trimester but to the pre pregnancy reading not more than that never.
- Patient with BP 120/70 in the booking appointment then she comes again in the second trimester with BP of 140/90 → this is abnormal, she has HTN.

- **Definition of Hypertension in Pregnancy:**

- **BP > 140/90 after 20 weeks gestation in the sitting position.**

Why after 20 weeks?

HTN before 20w ➔ this is a pre-pregnancy HTN not a pregnancy induce HTN.

Why sitting?

BP drop in lying down ➔ false reading.

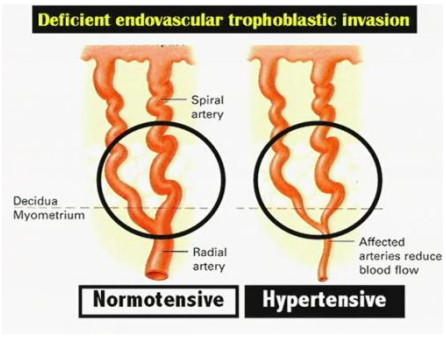
- Old definition : if there is an increase of 30 mmHg systolic, 15 mmHg diastolic ➔ diagnostic for pregnancy induce HTN➔ this is no longer used.
- Read BP twice, readings 6 hours apart, patient is well rested.

- **Risk factors for HTN in pregnancy :**
  - Nulliparity (HTN in pregnancy is called the disease of Primigravida)
  - Extremes of age (teenager or old age)
  - Multifetal pregnancy (twins, triplet)
  - Hydrops fetalis (Rh Disease)
  - Diabetes
  - Renal disease
  - Auto immune disorders (SLE, Thrombophilia, Antiphospholipid syndrome, Rheumatoid)

➔ first pregnancy, or if she has a 2nd or 3rd pregnancy but from a new husband ➔ first exposure to sperms)

* Molar pregnancy also a risk factor for the development of fulminating pre eclampsia but the doctor said I didn't include it here cause it usually diagnosed before 20 weeks of gestation .

- **Pathophysiology:**



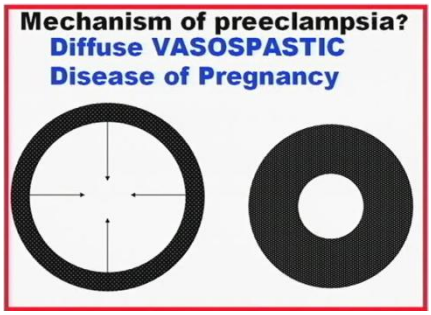
Vasospasms: Vascular constriction causes resistance to blood flow and accounts for the development of arterial HTN.

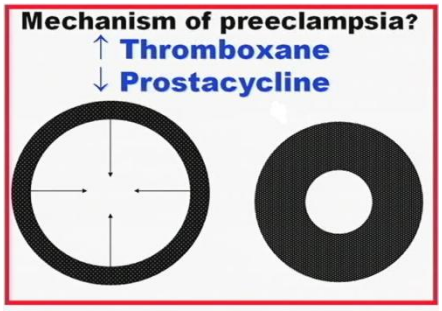
- Start at level of spiral vessel where the invasion of the trophoblasts to the endometrium take place at the time of implantation, this process start very early in pregnancy.

- Vasospasm is due to ↑production of Thromboxane ➔ (vasoconstriction)
  ↓ production of Prostocycline ➔ (vasodilatation)

➔ Management: Aspirin prophylaxis (thromboxane inhibitor) given early in pregnancy to protect from pre eclampsia & HTN.

- Dietary deficiency of Ca. ➔ not proven to be the cause but give Ca supplement.





- **Different scopes of the disease have maternal and fetal manifestations to different extents.** Depending on the **severity** of the disease & **duration** of the disease

A. Gestational HTN:

- May be asymptomatic ➔ (detected only by measuring BP)
- No edema
- No protein urea
- No hematological abnormalities
- No biochemical abnormalities

-/+ **Fetal involvement**

e.g. IUGR, oligohydramios Δ changes in fetal heart tracing and BPP depending on the duration of the diseases. placental abruption, fetal death

- mechanism of fetal involvement :

1. **Intra uterine growth restriction:**
   (vasospasm & reduction in placental blood flow➔ reduction in O2 & nutrition➔ IUGR)

2. **Oligohydramios** ➔ reduction in placental blood flow ➔ the first organ affected is the fetal Kidney (no adequate volume from the circulation ➔ making less urine ➔ less amniotic fluid production (the amniotic fluid is mainly the fetal urine) ➔ oligohydramios

3. **placental abruption** ➔ because of severe forms of pre-eclampsia ➔ **fetal death**

4. **Changes in fetal heart tracing** ➔due to reduce circulation & O2 ➔ asphyxia = hypoxia ➔ Changes in fetal heart rate : deceleration or reduce variability or others.
5. **Changes in Biophysical profile**:

- BPP = Biophysical profile ➔ **test to assess fetal well being**, composed of 4 component :

  1- Fetal breathing
  2- Fetal movement
  3- Fetal tone
  4- Amniotic fluid volume

  Score of 2 for each of these component, Total score out of 8.
  The baby get 2 or 0 for each component ➔ never get 1 ➔ this is a common mistake in exam

  In exam : they will gave us finding & ask to calculate the total score of BPP
  ➔ if they mention the tone is weak ➔ the baby get 0 not 1 !!

  Modified BPP :
  Same score together with ➔ **Non Stress Test**

  we do it to improve the score if baby is missing any of the 4 items above.
  score out of 2 also (Reactive=2, non reactive=0)

  Ex: if the baby is missing the breathing ➔ we do NST & the score will be out of 10

  BPP used a lots in hypertensive pregnant mothers to assess the fetal wellbeing

  pay attention : Fetal Heart Rate is **not** a component of BPP ➔ common mistake in exam

B. Pre-eclampsia:

1. Mild :
    - BP < 160/110.
    - Edema of the face and upper extremities.
    - Proteinuria > 300 mg /24 hours urine collection ➔ ("trace" up to "+1" in urine dipstick)

2. Severe :
    - BP > 160/110
    - Proteinuria 4-5 gm /24 hours (+ 2 or more urine dipstick)
    - Headache
    - Visual disturbance ➔ spots, lightning
    - Epigastric pain ➔ (usually pregnant have heart burn but if sudden severe this is an alarm) ➔ Epigastric pain is due to tension & edema in the liver capsule.
    - Oliguria ➔ because of intra vascular volume depletion ➔ important in administration of drugs➔ if the drug is not excreted through urine adequately she may become toxic.
    - Pulmonary edema
    - ↓ Platelets count ➔ coagulopathy + consumption of factors ➔ patient present with DIC specially if she has abruption of placenta➔ platelet is first indicator of ongoing process.
    - ↑ LFTS Liver Function Test
    - ↑ KFTS kidney function test
    - Fetal Involvement as previously mentioned. Depend on duration & severity of the illness

C. Eclampsia:

- Generalized tonic, clonic seizures
- You must Roll out epilepsy

➔ The patient may have epilepsy before & the first presentation is now during pregnancy!

➔ Hx, gestational age, lab test, no proteinuria, no edema ➔ this may not eclampsia but epilepsy!

- Eclampsia is considered as a severe form ➔ indication of terminating the pregnancy!

D. Pregnancy aggravated HTN with:

➔ you have to worn the pt. about the symptoms of pre eclampsia & eclampsia

* Super-imposed pre-eclampsia
* Super-imposed eclampsia with fetal involvement

E. HELLP syndrome:

• Severe form with rapid deterioration.
• Active aggressive management regardless of gestation age.
forget about the fetal age you have no time you must save the mother life!

Management:

• Prevention : ANC antenatal care
• Aspirin (reverse the process of thromboxane)
• Diet (Ca supplement)
• BP control in HTN patients
• Eclampsia & HELLP ➔ need admission

Investigations:

• **CBC** :

➔ platelet count ➔ indicate severity of the disease
➔ Hg ➔ if drop from 12 to 10 after 2 hour ➔ Hemolysis

➔ Hematocrit ➔ hemoconcentrated & intravascular volume depleted

• **U/A urine analysis**, 24 hour urine collection ➔ proteinuria
• **LFTS** ➔ elevated in severe disease (alkaline phosphatase normally rise in pregnancy)
• **KFTS** ➔ Na, K, creatinine, uric acid (uric acid normally elevated but to certain limit)
• **Coagulation profile**

- PTT + PT ➔ Prolonged
- D dimmer (fibrinogen + fibrinogen degradation products) ➔
- fibrinogen is low
- fibrinogen degradation products is high
- Coagulation factors ➔ being consumed
- micro-emboli & micro-thrombi.

Treatment depends on:

• Severity of the condition
• Fetal maturity

➔ Use them in combination if :
• Disease is mild & fetal is immature ➔ you have time to wait
• Disease is mild & fetal mature ➔ deliver
• Disease is severe & fetal is immature ➔ terminate the pregnancy (get the baby out not abortion) regardless of the fetal age because you are exposing the mother to dangers.

- Treatment in mild cases with prematurity :

  - conservative management is recommended
  - close fetal and maternal monitoring
  - administration of steroids.

- In severe cases:

Stabilization and delivery regardless of the fetal age is indicated

➔ Anti hypertensive + fluid + Magnesium sulfate + deliver

- Mode of delivery depends on how much time you have:

➔ cervix, parity, head fetus, rapture membrane, gestational age, deceleration

➔ Induction of labour or C.S.

* depending on factors above our decisions in clinical exam scenarios *

- **BASIC RULES in management:**
  - **A.** Use antihypertensive drugs to prevent maternal CVA cerebrovascular accident
  - **B.** Use Mg SO4 for seizure prevention
  - **C.** Continuous fetal monitoring
  - **D.** Measure Input & output

➔ **Use Antihypertensive to prevent maternal CVA cerebrovascular accident**

- If Diastolic BP of 105 ➔ start antihypertensive to save the mother from CVA

1. commonly used is I.V. **hydralazine** ➔ drug of choice
2. **Aldomet** is used in cases of **chronic HTN**
3. Lasix should **not** be used ➔ they cause intravascular depletion ➔ renal failure
4. ACE inhibitors are contraindicated ➔ fetal problem

➔ **Use Mg SO4 for seizure prevention** ➔ Intrapartum + 24 hours postpartum

❖ **Dosage :** ➔ (this is the only dose you need to memorize it)

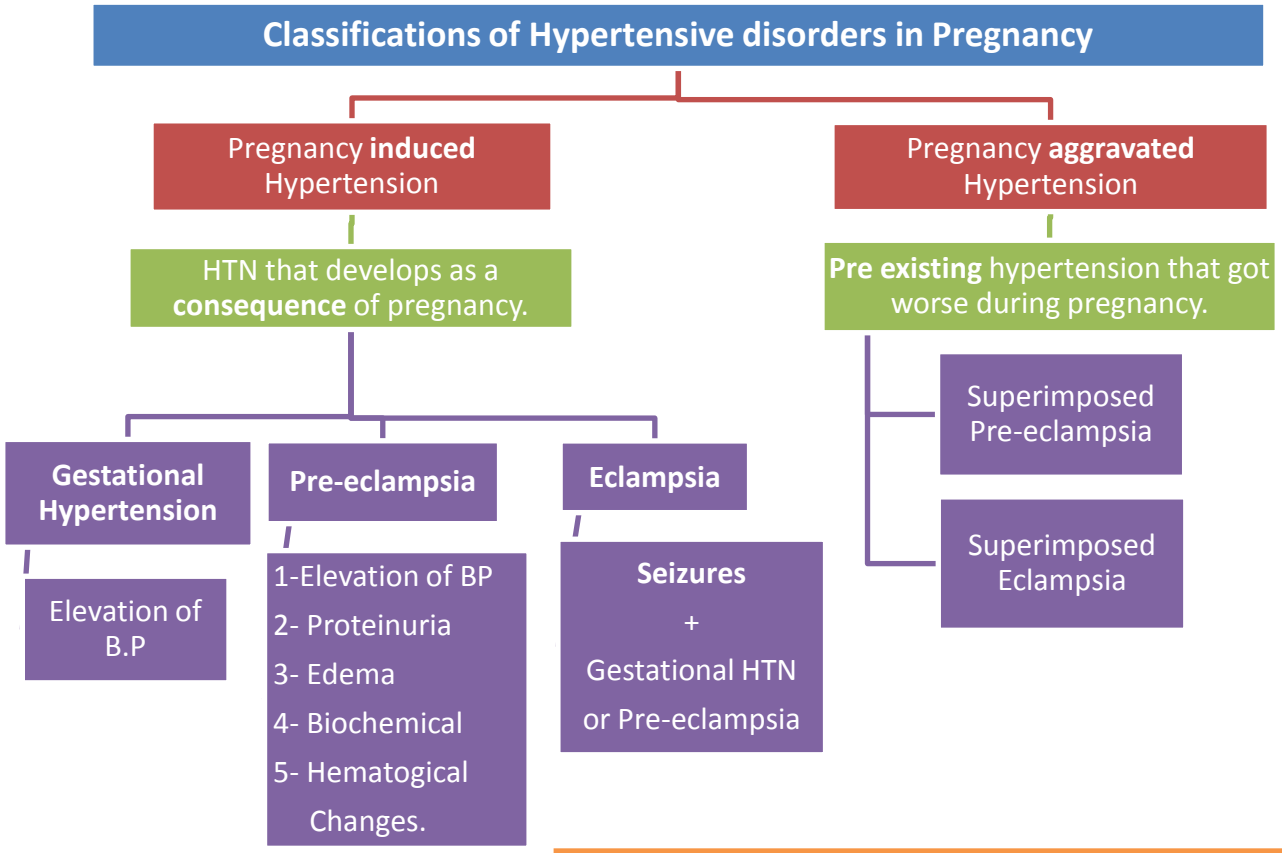
- 4 gm IV load over 30 minutes (prophylactic)
- followed by 2 gm/ hour Intrapartum infusion (maintenance) until the patient delivers.
- once she deliver reduce the dose to 1 g/hour for a period of 24 hours
- intramuscular injection is painful + with different doses.

❖ **Monitoring of patients on Mg SO4 :**

- Mg levels ➔ draw blood level every 4-6 hours to prevent toxicity
- U/O volume ➔ Urine output Less than 30-40 cc/ hour ➔ Mg level are high➔ check your infusion rate
- Reflexes ➔ CNS Depression ➔ over-dose
- Respiratory rate ➔ respiratory arrest

❖ **Anti dote of Mg SO4** ➔ **Calcium gluconate** injection.

- **Summery :**



Hypertension in Pregnancy:
BP > 140/90 after 20 weeks gestation in the sitting position.
HELLP Syndrome:
Hemolysis
Low platelets
Elevated liver enzyme
Investigations:
• CBC (platelet, Hg, Hematocrit)
• Urine analysis
• LFTS
• KFTS
• Coagulation profile

Fetal involvement :
IUGR, oligohydramios, changes in FHR and BPP, placental abruption, fetal death.
Basic rules in management:
1. Antihypertensive drugs to prevent maternal CVA ➔ I.V. **hydralazine** is a drug of choice
2. Mg SO4 for Seizure prevention
   - dose : 4 gm IV over 30 min ➔ 2 gm/h until delivery ➔ 1 g/hour for 24 hours postpartum.
   - antidote: **Calcium gluconate**
3. Continuous fetal monitoring.
4. Measure Input & output.

Treatment depends on:
• Severity of the condition + Fetal maturity

Physiological changes in BP in pregnancy:
- Slight drop in 1st trimester ➔ continuous through the 2nd trimester
- Gradual rise to pre-pregnancy level in the 3rd trimester.

Risk factors for HTN in pregnancy Nulliparity, Extremes of age, Multifetal pregnancy, Hydrops fetalis, DM, Renal disease
Pathophysiology : Increase production of Thromboxane + Dietary deficiency of Ca

Cardiac Disease in Pregnancy.

Objectives

- To understand the normal physiological changes of CVS in pregnancy.
- Symptoms and signs suggestive of CVS disease.
- When to investigate for cardiac disease.
- Types and grades of CVS disease.
- Effect of pregnancy on CVS disease and effect of cardiac disease on pregnancy.
- Pre-pregnancy counseling.
- Management of CVS disease in pregnancy, labor and purperium.

Hemodynamic Changes During Pregnancy

- Starts around 5-8 weeks of pregnancy.
- Peak at late second trimester; 20-24 weeks.
- Symptoms ad signs due to these changes include: fatigue, dyspnea, decreased exercise capacity, peripheral edema, physiologic systolic murmur and 3rd heart sound.

1) Blood volume

- Increase 40-50% up to 32 weeks.
- Plasma volume increases (50%) more then RBC mass (20%) resulting in physiologic anemia.

2) Cardiac output

- Rises 30-50% (max 20 weeks) by increased blood volume, reduced systemic vascular resistance and increase maternal heart rate by 10-15 beats per minute.
- Stroke volume increase in 1st and 2nd trimester and decrease in the 3rd trimester.

3) Slight decrease in BP

- Diastolic reduced more than systolic. (Reduced blood pressure is normal as long as the patient doesn't have any symptoms)

4) Labor and delivery

- Each uterine contraction result in displacement of 300-500 cc of blood to the general circulation →increase stroke volume and cardiac output by about 50%.
- Blood pressure & heart rate increase due to pain and anxiety.
- Blood loss during delivery may compromise the hemodynamic state.

5) Postpartum

- Relieve of vena cava compression by the gravid uterus →increase venous return →increase cardiac output 10-20 % → diuresis. (That's how women lose the excess blood volume they gained during pregnancy. You see patients in post-partum complaining of edema,it will gradually resolve with increase diuretics after delivery)

6) Changes due to epidural anesthesia

- Peripheral vasodilation → decrease cardiac output & blood pressure. Therefore patients need pre-hydration to prevent drop in blood pressure.

Symptoms And Signs Of Cardiac Disease In Pregnancy

There is overlap with the common **normal/physiologic**symptoms of pregnancy	Symptoms that merit a cardiac evaluation in pregnancy
❖ Fatigue ❖ Dyspnea ❖ Orthopnea ❖ Palpitation ❖ Edema(lower limb in up to 50% of women) ❖ Systolic flow murmur ❖ 3rd heart sound	❖ Progressive limitation of physical activity ❖ Chest pain ❖ Syncope

Evaluation

- History and physical exam.
- ECG.
- Chest radiogram.
- Echocardiogram.

NYHA functional classificationof cardiac diseases in pregnancy

NYHA Class	Symptoms
I	Asymptomatic.Cardiac disease, but no symptoms and no limitation in ordinary physical activity. E.g. shortness of breath when walking, climbing stairs etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity. E.g. walking short distances (20–100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while *at rest*. Mostly bedbound patients.

Management before conception

- Should be informed about the added risk of pregnancy on her self & the fetus.
- Class III and IV →mortality rate up to 7% and morbidity 30% →should be cautioned against pregnancy.
- **Factors that predict the woman chance of having adverse cardiac or neonatal complication:**

1) A prior cardiac event.

2) Cyanosis or poor functional class.

3) Valvular or outflow tract obstruction.

4) Myocardial dysfunction (LVEF < 40% cardiomyopathy).[LVEF: Left Ventricular Ejection Fraction]

So if a woman is class III or IV or has one of these criteria, she should be advised not to get pregnant.

Management after conception

- Cardiac assessment as early as possible (by cardiologist).
- Termination of pregnancy if there is a serious threat to maternal health.
- Close follow up by both obstetrician and cardiologist.
- Observe for signs and symptoms of heart failure.

Antibiotic prophylaxis for endocarditis(usually during labor)

- American Heart Association published a consensus statement that there is no need for antibiotics prophylaxis(to prevent bacterial endocarditisin patient with cardiac lesions) for vaginal delivery nor cesarean section as the risk of bacteremia is low 1-5%.
- IV antibiotics is optional if bacteremia is suspected or for high-risk patients (prosthetic cardiac valve, previous bacterial endocarditis, complex cyanotic congenital heart disease, surgical pulmonary shunts or conduits, ventricular septal defect, patent ductusarteriosus).
- Ampicillin 2 gm + Gentamicin 1.5 mg/kg within 30 minutes of procedure, followed by Ampicillin 1 gmafter 6 hours

Specific Cardiac Conditions

1) Cardiomyopathy(CMP)

- Look for symptoms and signs of congestive heart failure (CHF).
- Heart failure is often refractory to treatment.
- Serious conditionwith 5-year survival rate of 50%.

Pregnant women particularly at risk for developing cardiomyopathy:

-History of preeclampsia or hypertension

2) Peripartum cardiomyopathy

- Dilated CMP occurs in late pregnancy or first 6 months post partum
- Incidence 1:1300-15000
- Unknown cause(Women at risk are those with a history of preeclampsia, hypertension, and poorly nourished)
- Mortality 25-50% due to CHF, thrombo-emoblism or arrhythmia
- Need intensive monitoring and treatment during pregnancy and labor by cardiologist and OB

3) Septal defects:atrial septal defect (ASD),ventricular septal defect (VSD)

- Usually tolerate pregnancy well.
- ASD most common congenital lesion.
- ASD can cause atrial flutter. Treated after pregnancy by catheter ablation.
- Rarely uncorrected lesions lead to Lt to Rt shunt, pulmonary hypertension and CHF.
- Fetalechocardiography: incidence of VSD 4%.

4) Patent ductusarteriosus

- Well tolerated in pregnancy unless there is pulmonary hypertension.

5) Mitral regurgitation (MR)

- Usually well tolerated in pregnancy except in patients with atrial fibrillation or severe hypertension.
- Patients with severe MR should be advised surgical correction before pregnancy.

6) Mitral prolapse

- Most common congenital defect.
- Rarely have any implications on maternal fetal health.

7) Mitral Stenosis

- Moderate to severe disease often show deterioration in 3rd trimester or labor→ increased blood volume & heart rate →pulmonary edema.
- Atrial fibrillation →Cardiac failure.
- Normal vaginal delivery with Swanz-Ganz catheter monitoring in severe/moderate cases.
- Needs good pain relief in labor to reduce maternal heart rate and increase diastole.
- Can't tolerate the 2nd stage because of decreased preload with pushing therefore require instrumental delivery to shorten the 2nd stage.
- Post partum auto-transfusion can result in pulmonary edema → requires aggressive diuresis.

8) Aortic Regurgitation

- Generally well tolerated.
- Severe disease should have surgical repair before pregnancy.

9) Aortic stenosis

- Mild and moderate: well tolerated in pregnancy.
- Severe: deteriorate in 2nd or 3rd trimester with dyspnea, angina, syncope or CHF
- May require balloon valvoplasty in pregnancy.
- Monitoring with Swan-GanzCatheter in labor.
- No epidural. (Because it reduces blood pressure and these patients already have hypotension)
- Instrumental delivery to shorten the second stage.
- Mortality 17%. Any hypotension can causesudden death.
- Postpartum blood loss →reduce preload and volume resuscitation is necessary.

10) Congenital Lesions

A-Tetrology of Fallot (Rt to Lt shunt &cyanosis)

- Rt ventricular outflow obstruction.
- Ventricular septal defect (VSD).
- Rt Vent hypertrophy.
- Overriding Aorta.

Complications:

- Heart failure 40%.
- Spontaneous abortions & preterm labor.
- Intrauterine growth restriction (IUGR).
- Shunt worsens in labor & postpartum.
- Requires invasive cardiac monitoring in labor.

B-Eisenmenger's Syndrome

- Communication between pulmonary & systemiccirculation (e.g. large VSD).
- Lt to Rt shunt→ pulmonary hypertension →Rt to LT shunt.
- Termination of pregnancy is advisable.
- Materanl mortality rate (MMR) is 50% post partum death 1 week after delivery up to 4-6 weeks.
- Fetal mortality rate (FMR) is 50%.
- IUGR 30%.
- Preterm delivery 85%.

- Management during pregnancy: limitation of physical activity, oxygen and pulmonary vasodilators.
- Risk of death is greatest during labor & early postpartum.
- Requires central hemodynamic monitoring in labor with Swanz-Ganz catheter & instrumental delivery.

C-Coarctation of the Aorta

- Surgical correction in pregnancy only if dissection occurs.
- They have fixed cardiac output therefore maintain demand of pregnancy by increasing heart rate.

D-Marfan's Syndrome

- Congenital weakness of the connective tissue.
- They usually have aortic root dilatation, mitral valve prolapse and aneurisms.
- Sever cases complications in pregnancy: aortic dissection or rupture.
- Aortic valve replacement before pregnancy.
- Avoid hypertension by using B-blockers from 2nd trimester to avoid tachycardia.
- Delivery method is controversial, Caesarean section (CS) v.s.spontaneous vaginal delivery (SVD).

E-Idiopathic hyprtrophicsubaortic stenosis

- Lt Vent outflow tract obstruction.
- Worsen in the late 2nd and 3rd trimester.
- Lt ventricular failure.
- Supraventricular arrhythmias.

F-Ebstein'sanomaly

- Malformation of the Tricuspid valve.
- Surgical correction before pregnancy.

G-Congenital atrioventricular block

- With pacemaker can tolerate pregnancy well.

11) Arrhythmias

- Premature atria/ventricular complexes: no adverse outcome in pregnancy.
- Atrial fibrillation/flutter: severe problem butrare in pregnancy.
- Treated by digoxin & B-blockers.
- Serious arrhythmias should be treated before pregnancy.

12) Ischemic heart disease

- Uncommon in pregnancy.
- 67% occurs in 3rd trimester.
- If myocardial infarction (MI) occurs before 24 weeks →termination of pregnancy.
- If delivery occurs within 2 weeks of MI: mortality rate is up to 50%.

CVS Drugs InPregnancy(Just have an idea about it, you don't have to memorize it.The doctor only mentioned the drugs colored in red)

Class B: No risk in controlled animal studies

Anticoagulants	Antihypertensives	Antiarrhythmic	Diuretics	Antihyperlipidemic
1.Enoxaparin (Lovenox) for patients with recurrent fetal loss 2.Dalteparin (Fragmin) 3.Danaparoid (Orgaran) 4.Heparin	1.Methyldopa (Aldomet)most widely used 2.Acebutolol (1st trimester only) 3.Pindolol (1st trimester only)B-blockers are advisable in 1st trimester only	1.Encainide 2.Sotalol (Betapace) – 1st trimester only	1.Torsemide (Demadex) 2.Amiloride	1.Cholestyramine 2.Colestipol

Class C: Small risk in controlled animal studies

Antiplatelet Medications	Antiarrhythmic	Diuretics	Diuretics	Antihypertensive
1.Clopidogrel (Plavix) 2.Dipyridamole (Persantine) 3.Ticlopidine	1.Atropine 2.Digoxin 3.Disopyramide (Norpace) 4.Lidocaine 5.Procainamide 6.Quinidine 7.Amiodarone Neonatal Hypothyroidism Intrauterine Growth Retardation Cardiac disturbance	1.Acetazolamide (Diamox) 2.Furosemide (Lasix) 3.Mannitol	1.Niacin 2.Gemfibrozil (Lopid)	1.Hydralazine 2.Diazoxide 3.Clonidine 4.Nitroprusside (Nipride) 5.Prazosin 6.Reserpine 7.All Calcium Channel Blockers: Nifedipine XL (is a drug of choice for severe Hypertension in Pregnancy) Avoid other Calcium Channel Blockers in pregnancy 8.Most Beta Blockers (1st trimester only) Labetolol (drug of choice for severe Hypertension in Pregnancy) Metoprolol Nadolol Propranolol Timolol Esmolol (Class C in all trimesters)

Class D: Strong evidence of risk to the human fetus

Anticoagulants	Antihypertensive	Diuretics
1.Coumadin (Warfarin) causes congenital warfarin syndrome 2.Dicumarol	1.ACE Inhibitors 2.Angiotensin II Antagonists 3.Most Beta Blockers (second and third trimester) ❖ Associated with Intrauterine Growth Retardation ❖ Metoprolol ❖ Nadolol ❖ Propranolol ❖ Timolol ❖ Acebutolol (second and third trimester) ❖ Pindolol (second and third trimester) ❖ Atenolol	1.Ethacrynic Acid 2.Triamterene (Class B per manufacturer) 3.Bumetanide (Bumex) 4.Hydrochlorothiazide 5.Spironolactone

CVS Drugs In Pregnancy

Class B:

- In general B-blockers are advisable (and safest) in 1st trimester only.
- The anticoagulant Enoxaparin (Lovenox) is for patients with recurrent fetal loss.
- Methyldopa is the most widely used b-blocker.

Class C:

- Nifedipine XL (Ca channel blocker) is the drug of choice for severe Hypertension in Pregnancy. Avoid other calcium channel blockers in pregnancy.
- Labetolol (B-blocker) is the drug of choice for severe Hypertension in Pregnancy.
- Esmolol(B-blocker) is classified as class C in all trimesters.

Class D:

- Coumadin (Warfarin) causes congenital warfarin syndrome.
- ACE Inhibitors are contraindicated.

Cardiac Diseases In Pregnancy

Cardiomyopathy	❖ Heart failure is often refractory to treatment ❖ Serious condition with 5 year survival rate of 50%
Peripartum cardiomyopathy	❖ Mortality due to CHF, thrombo-emoblism or arrhythmia ❖ Needs intensive monitoring and treatment
Septal defects	❖ Well tolerated ❖ ASD can cause atrial flutter. Treated after pregnancy by catheter ablation
Patent ductusarteriosus	❖ Well tolerated unless there is pulmonary hypertension
Mitral regurgitation	❖ Severe MR should be advised surgical correction before pregnancy
Mitral prolapse	❖ Rarely have any implications on maternal fetal health
Mitral Stenosis	❖ Moderate/severe disease deteriorate in 3rd trimester or labor ❖ Normal vaginal delivery with SG-catheter monitoring in mod/severe cases ❖ Instrumental delivery to shorten the 2nd stage
Aortic Regurgitation	❖ Severe disease should have surgical repair before pregnancy
Aortic stenosis	❖ Severe deteriorate in 2nd or 3rd trimester ❖ Monitoring with SG-Catheter in labor ❖ No epidural ❖ Instrumental delivery to shorten the 2nd stage
Tetrology of Fallot	❖ Complications: Spontaneous abortions & preterm labor
Eisenmenger's Syndrome	❖ Termination of pregnancy is advisable ❖ Central hemodynamic monitoring in labor with Swanz-Ganz catheter ❖ Instrumental delivery
Coarctation of the Aorta	❖ Surgical correction in pregnancy only if dissection occurs
Marfan's Syndrome	❖ Aortic valve replacement before pregnancy ❖ Delivery method is controversial
Idiopathic hyprtrophicsubaortic stenosis	❖ Worsen in the late 2nd and 3rd trimester
Ebstein's anomaly	❖ Surgical correction before pregnancy
Congenital atrioventricular block	❖ Pacemaker can tolerate pregnancy well
Arrhythmias	❖ Premature atria/ventricular complexes: no adverse outcomes
Ischemic heart disease	❖ MI before 24 weeks → termination of pregnancy

Anemia and Thyroid Diseases in Pregnancy

Anemia

❖ PHYSIOLOGICAL CHANGES IN PREGNANCY:

1. Blood vol. ↑ 50%

2. Plasma vol.↑disprop. to red cell mass plasma volume is more than RBC so hemodilutional effect

3. HCT ↓

CBC is important in order to diagnose

❖ DEFINITION:

Hb< 12-g/dl in non pregnant

In pregnancy , definition of anaemia varies : <11 g/dl ~~in 1st trimester

<10.5 g/dl ~~in 2nd and 3rd trimester

physiological changes in pregnancy maximum effect is in 2nd trimester

Postpartum anaemia is defined as Hb<10 g/dl

❖ PREVALENCE:

Anaemia affects ~24.8% of the worlds population

Iron deficiency is the most common cause.

Iron difficiencyanaemia:

commonest deficiency state in the world as one third of the population is affected by IDA

- The commonest cause of anemia in pregnancy
- The fetus is always saved on the expense of maternal Fe stores

IDA:

- Hemoglobin <10
- MCV < 80
- RDW>15%

Diagnosis :

Clinical Symptoms & Signs:

Usually non-specific, unless severe:

- Fatigue..most common.
- Pallor, weakness, headache, palpitation, dizzines, dyspnea and irritability.
- Poor concentration and hair loss(because of depleted iron storage). mi

Investigations:

1. **CBC with red cell indices:** is screening (routine) test we did it for all patient we don't wait for the patient to reveal symptoms, once she experience symptoms so she is in the severe form of anemia

lowHb

↓MCV & MCH& MCHC=Microcytic Hypochromic red cells- -

-

2. **Smear to check for sickle cell**
3. **S. Ferritin, S. Fe & TIBC:** it is not a routine test however you have to consider it if you suspect IDA in the CBC picture

S.ferritn fall below 30 ug/l indicates early iron depletion -
It is best single indicator of storage iron. -
Patient we check her CBC even if she is not anemic we chick -
Ferritin is low because first iron stores become depleted and later on hemoglobin will be low

-

If there is IDA in the mother can the fetus acquire it as well?

No, because there is active transport of iron across placenta

However the complication of IDA could be IUGR and preterm birth

Management of Iron Deficiency:

1. **Dietry advice**
2. **Oral iron supplements** every patient coming to antenatal clinic should be provided iron supplements as a prophylactic usually but if she is IDA we increase dose to her as we always give it with folic acid as folic acid known to prevent NTD

Recommended dose for treatment = 100 – 200 mg daily (We stick to this dose to not get -
the undesirable side effects)
e.g ferrous fumurate, ferrous sulphate and ferrous gluconate, combined iron and folic acid -
preperations
+- Vitamin C to enhance absorption as it is preferable to be taken on empty stomach -
(There are substances which interfere with absorption of iron as: tea – Ca(milk-Yogurt) -
don't take it with supplement)

3. **Parenteral Iron Therapy**

- When there is absolute non-compliance, or intolerance to oral iron, or proven mal-absorption

- IM / IV

Most serious side effect of IV is anaphylactic reaction, IM is painful and can cause skin staining try to avoid it

We have to treat esp. if the patient is bleeding in delivery like post partum hemorrhage she may not tolerate the bleeding

SICKLE CELL ANAEMIA

It is an inherited disorderit is recessive autosomal so you have to get the tow gene affected to show the disease but even if she is a carrier we have to counsel her

Most of patient will come already diagnose because it is the disease of childhood

The red cell contains Hb(s) instead of normal Hb. which causes the sickling.

SS = disease S = carrier

The disease is common in the Southern region, e.g. Gizan, Asir, Najran

❖ Complications in Pregnancy:

-↑ Maternal mortality + morbidity

HTN

Pre-eclampsia

Eclampsia

-↑ Abortion

-↑ Perinatal mortality and morbidity

prematurity(PROM-PREM.LABOUR), IUGR, IUFD

❖ PATHOPHYSIOLOGY:

Sickling of red cells with Hbs→sickle cell crisis.

Ischemia and infarcts of different organs. →
Pain →

We have to see these patient more than normal pregnant women in order to prevent complications as they are more prone to have HTN , Premature labour, pre-eclampsia , abrptio placenta , post partum hemorrhage

❖ Diagnosis:

Hx and Physical + Lab:

Screening by sickling test

Diagnosis by Hb electrophoresis after all she diagnosed we have to check for other forms of abnormal hemoglobin because she could have a combine disease

Screening is peripheral blood test used to detect presence or absence of Hemoglobin S but can't differentiate between trait and disease we have to do diagnostic test which is hemoglobin electrophoresis

SICKLING →1. Bone △s

2. Renal medullary damage

3. Hepatosplenomegally

4. Ventricular hypertrophy

5. Pulmonary infarctions

6. CVS

7. Leg ulcers

8. Sepsis

The acute crisis are severe in pregnancy

→Serious complications specially pulmonary

- **Management:**
  She should avoid or pay attention to it dehydration and fever to avoid end organ failure or any crisis and she should come to the hospital

- Team approach Haematologist + Obstetrician
- R/O other causes of pain and fever and ↓Hb.
- Early Rx of infections : e. g. UTI

  pneumonia

  as they can cause acute crisis
  - IV hydration + analgesia
  - Bld transfusion
  - ? Prophylactic transfusion
  - Close fetal monitoring NST

    BPP

    U/S for grow

    \+ early delivery

❖ MODE OF DELIVERY

Vaginal delivery is encouraged

C/S is ↑ due to pelvic deformity

Continuous fetal monitoring due to impaired placental function

People don't like to leave them more than 40 wks because the higher the mortality and the higher the rate to get placenta abruption so a lot of people if she didn't go into labor before 40 wks they may induce it

In delivery get her hydrated , oxygenated and cross matched

NB:

Patient education and genetic counseling is important

Partner Hb electrophoresis

Prenatal diagnosis

✶ Sickle cell trait carries no risks

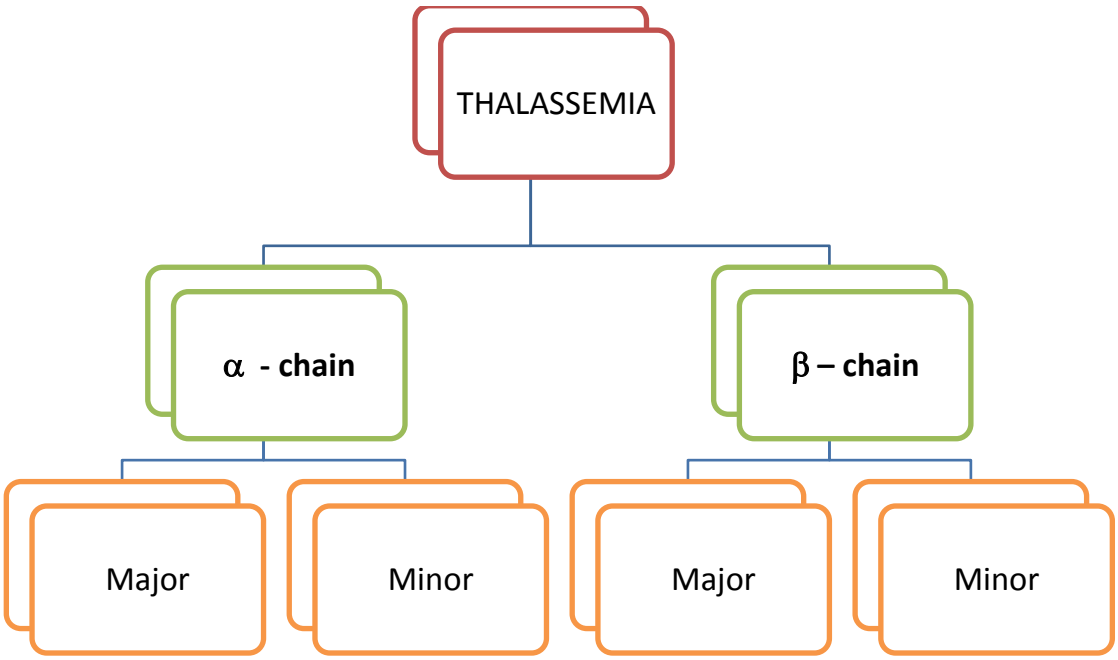
With SA trait

The only risk is increased UTI's in pregnancy

With SS disease increased risk of IUGR, fetal death , and preterm delivery

THALASSEMIAS

They are impaired production rate of one or more peptide chains that are normal components of globin →ineffective erythopoieses and hemolysis → anemia



aimesalaht fo ycanagerp no tceffa ACS sa reves sa ton

ti taert ot deeen ew dna ADI rof pse meht retinom ot tnaw ew

- **Other anemia's:**

IV Anaemia from acute or chronic Bl. Loss

V Anemia with chronic disease

VI Megaloblastic anemia Folic acid , B12

Thyroid disease in pregnancy

❖ introduction

Pregnancy has significant impact on the normal maternal physiology .

There is increase in maternal production of thyroid- binding globulin by the liver because of estrogen stimulation .

There is increase in total T4 and T3 but there is no significant change in free thyroid hormone FT4 FT3 .

The renal clearance of iodine increase in pregnancy .

Which hormone has affect on all (T3 – T4 – TSH)?

HCG

I. Maternal hyperthyroidism

It occurs I in 500 pregnancies .

Majority are diagnosed before pregnancy .

Graves disease comprises 90% it is an autoimmune disease with circulating thyroid stimulating antibodies

They had to be counseled regard 2 things: treatment and monitoring for complication in pregnancy and adjusting dose your aim is the lowest dose of that treatment which bring the maximum benefit which is upper normal level

Rare causes:

Toxic nodules.

Hashimotosthyroiditis .

Multiple nodular goiter

❖ Clinical presentation

Maternal tachycardia.

Weight loss.

Heat intolerance .

Heart murmurs .

Clinical diagnosis is difficult since all these symptoms are well recognized in normal pregnancy .

Thyroid function test will confirm the diagnosis.

Uncontrolled hyperthyroidism will cause.

Maternal cardiac arrhythmias like AF .

Vomiting ,diarrhoea ,abdominal pain and psychosis .

In case of autoimmune disease thyroid stimulating antibodies will cross the placenta and cause fetal goiter

hgiH si nixoryht dna wol si HST

When we start treatment?When we see low level of TSH

Other complications.

Fetal growth restrictions .

Stillbirth .

Fetal tachycardia ..

Premature labor.

- **Treatment**

Drug therapy to maintain FT4 and FT3 in the normal range .

Treatment is usually medical with carbimazole , the lowest dose should be used as high dose may cross the placenta that will leads to hypothyroidism

Occasionally beta- blockers and surgical treatment can be undertaken

Radioactive iodine is contraindicated.

II. Maternal hypothyroidismit is common in our society not only in pregnancy

The commonest worldwide cause is iodine deficiency

Maternal iodine deficiency is associated with the development of cretinism in the newborn due to congenital hypothyroidism .other cause is over treating hyperthyroidism .

Women treated with radioactive iodine will frequently use thyroxine supplements

❖ Symptoms:

Bradycardia .

Weight gain .

Heat intolerance .

Hair loss.

Constipation.

All is confusing with normal pregnancy symptoms

thyroid function test is not routinely done on pregnancy only when we suspect however we do offer them selectively to special patient esp. if they got Hx of thyroid diseases in the past – family Hx – previous Hx of infertility and miscarriages

❖ Treatment

bythyroxine replacement therapy.

SUMMARY 1

ANEMIAS	INVESTIGATION	MANAGEMENT
IDA	CBC + S.Fe, TIBC, and S.Ferretin which is the best indicator of iron storage Low hemoglobin, S.Fe, S.Ferretin Hypochromic microcytic High TIBC, RDW>15%	Iron tablets
SCA	**Screening** by sickling test **Diagnostic** by hemoglobin electrophoresis	In general try to avoid dehydration and fever to prevent end organ failure or any crisis and she should come to the hospitaland treat every complain accordingly

THYROID DISEASE	INVESTIGATION	MANAGEMENT
HYPER – THYRODISM	THYROID FUNCTION TEST High free T level Low TSH	Medical with carbimazole
HYPO – THYRODISM	THYRIOD FUNCTION TEST Low free T level High TSH	Thyroxine replacement therapy.

SUMMARY 2

Anemia

Iron deficiency anemia	Sickle cell anemia	Thalassima
Most common type of anemia in the pregnancy No major effect on fetus health **Diagnosis of anemia :** Pregnant women first trimester <11 Second and third<10.5 Non pregnant women :<12 **Investigation :** 1- CBC Low hemoglobin Low MCV High RDW 2-smear check for sickle 3-serum ferritin Rx: 1-diet 2-oral iron +folic acid + - vit C 3-parental :IM . I.V	Autosomal recessive Fetus monitoring should be appreciated because of sever outcome SS: sickle cell disease SA: trait **Its Complications during pregnancy :** 1-increase mortality and morbidity 2-increase abortions 3-PROM , IUFD,IUGR **Investigations:** Electrophoresis **Complications of disease:** 1-renal medullary damage 2-hepatosplenomegaly 3-venticular hypotrophy 4-bone infarction 5-pulmonary infarction 6-leg ulcer 7-sepsis **Rx**: 1-infection ?? Antibiotic 2-dehydration 3-fetal monitoring **Delivery :** Usually cesarean section because of small pelvis	**No serious impact on pregnancy**

Thyroid disease

Hyperthyroidism	Hypothyroidism
1-graves disease 2-toxic nodules 3- multinodular goiter	Most common than hyperthyroidism 1-iodine deficiency 2- hashimoto thyroiditis
Symptoms : Everything in the body is hyperactive Tachycardia , weight loss, heat intolerance	**Symptoms :** Bradycardia , weight gain , hair loss , cold intolerance
Investigations: Low TSH and high T3, T4	**Investigations:** High TSH and low T3 ,T4
Complications: 1-fetal tachycardia 2-still birth 3-premature labor 4-IUGR	**Rx:** Thyroxin replacement
Rx: 1- carbimazole 2-propylthiouracil	

Urinary disease in pregnancy.

Is UTI more common in men or women? Why?

More common in women because they have short urethra

Pregnancy is a risk factor for UTI

Anatomic Changes in Pregnancy:

- **Kidneys**: ↑ in length, weight, and pelves size (physiologic hydronephrosis); Rt> Lt
    - Why the kidney enlarged in pregnancy? Because the kidney is working more due to toxins removing from the fetus and the mother together
- **Ureters**: dilated or hydroureter (Rt> Lt), urinary stasis.
    - Why the ureter dilated in pregnancy? Because there is more progesterone secreting in the body, and this will lead to muscle relaxation, so the ureter will dilate and there will be urine stasis, other cause the veins become large (renal vein), this will compress the ureter and dilate it
    - Why the right ureter dilated more than the left ureter? Because the uterus will leans toward the right side because there is the sigmoid colon in the left side, so the uterus will be compress between the uterus and the bony pelvis.
- **Mechanism**: hormonal (progesterone)or mechanical(pressure from the uterus)
- **Consequences**: ↑ risk of urinary tract infections (the ureter dilation and urinary stasis: increase the risk of UTI)

Physiologic Changes in Pregnancy

- 40-50% ↑in renal blood flow and glomerular filtration rate (GFR) →creatinine clearance
    - blood test in normal pregnant lady: low creatinine (it's normal)
    - blood test in mild HTN pregnant lady: normal creatinine (it's not normal, should be low)
- ↓serum level of creatinine, urea, uric acid by 25%
- Fluid volumes: ↑ extracellular volume (intravascular 50% & interstitial component)
    - There will be physiological anemia, and lower limb edema because the electrolytes and the cell won't change but the fluid will increase.
- Na &Ka levels maintained
- Chronic loss of renal HCO3 →↑ risk of metabolic acidosis
    - Pregnant lady will get metabolic acidosis easily, specially who diabetic type1, any loss of fluid or vomiting can go on DKA.

Urinary Excretion of Nutrients

- **Glucosuria**: ↑ filtered tubular glucose and ↓tubular reabsorptive capacity, consequence: ↑ risk of UTI(it's normal if there is small amount of glucose in the urine, so it will be good environment for the bacteria " it is a risk factor")
- **Protienuria**: abnormal
- **Aminoaciduria**: ↑ risk of UTI
- **Water-soluble vitamins:**folate and B12

Urinary Tract Infections in Pregnancy

- Common medical complication of pregnancy (2-10%)
- **Pathphysiology:** ascending infection from vagina, rectumand anus
- Most common causative organisms: gram –ve enteric bacteria (e.g: E.Coli 60-80%, Proteus, K. Pnemoniae, Pseudomonas, and GBS)
    - Most common organism is E.Coli
    - Other common organisms are Proteus, K. Pnemoniae, Pseudomonas, and GBS
- Lactobacilli cause no UTI(it's normal flora)(MCQ)

Risk Factors for UTI's in Pregnancy

1. **Mechanical obstruction:**ureteropelvic junction, urethral or ureteric stenosis, & calculi.
2. **Functional obstruction:**pregnancy&vesicoureteral reflux(because of slow peristalsis).
3. **Systemic diseases:** DM, sickle cell trait/disease, gout, cystic renal disease

Classification of UTI's

Clinical:

- Asymptomatic (8%)(Third of them will be symptomatic with more serious complication)
- Symptomatic (1-2%)

Anatomical:

- Lower tract dis: asymptomatic bacteriuria and acute cystitis
  - Symptoms of lower UTI: urgency, frequency, dysuria, suprapubic discomfort.
- Upper tract dis: acute pyelonephritis
  - Chills, fever, flank pain

1- Asymptomatic Bacteriuria (ABU): (bacteria in the bladder but there is no symptoms)

- Incidence in pregnancy: 8%
- **Consequences**: acute pyelonephritis (30%)
- **Clinical presentation:** asymptomatic if there are symptoms **(**urgency, frequency, dysuria, suprapubic discomfort)
- **Diagnosis**: culture(MSU) (MSU is one of the investigation in the booking)
- Lactobacillus (normal vaginal contaminated)
- **Management**: outpatient Abx (amoxil(penicillin), 1st generation cephalosporin, nitrofurantoin)
- length: 3-10 days (non pregnant: 3 days)(the duration is not important)

2- Acute Cystitis

- Incidence in pregnancy: 1-2%
- **Consequences:** acute pyelonephritis (30%)
- **Clinical presentation:**urgency, frequency, dysuria, suprapubic discomfort
- **Diagnosis**: symptoms and culture (MSU) (we diagnose it depend on the symptoms and we confirm it with MSU)
- **Management**: outpatient Abx, analgesics
- Length: 7-10 days (never 3 days in pregnant lady)→reculture (to make sure no further infection)

3- Acute Pyelonephritis

- Incidence in pregnancy: 1-2%
- **Consequences:**sepsis, adult respiratory syndrome, anemia, renal failure, pretermlabour.
- **Clinical presentation:**fever, chills, CVA tenderness (costovertebral angle), flank pain
- **Diagnosis**: symptoms, physical examination and lab: culture of urine and blood(we do blood culture before the antibiotics to detect septicemia)
- **Management:** Inpatient:1- Admission 2- Antipyretic agents 3- Abx (i.v. ampicillin or cephalosporin then p.o)
  - Length: 7-14 days →reculture
  - Also we give her steroid for lung maturity of the fetus, we have to time it very well because if we give her early, the situation will be worse.

Type of UTI recurrences

1. **Relapse**:same organism within 2-3 wks(short time)
   2nd ry to perineal colonization or inadequate treatment.
2. **Reinfection**:new organism within 12 wks(long time)
   2nd ry to recurrent bladder bacteriuria.
3. **Superinfection**:new organism while on Rx (RX= therapy)(Superinfection is more common in immunocompromisedpt : HIV, sickle cell anemia and renal stones, patient on chemo therapy)

Prevention:

Prenatal screening for ASB (specially who are high risk like diabetic patient)

Summary:

Pregnancy is a risk factor of UTI because :

1- anatomical changes:
   - increase the size of the kidney(physiologic hydronephrosis)
   - dilated ureter and urine stasis

2- physiological changes:
   - increase GFR>>creatinine clearance
   - Chronic loss of renal HCO3
   - Increase the fluid, Glucosuria, Aminoaciduria

Pathphysiology: ascending infection from vagina, rectum and anus

Cause: Most common organism is E.Coli

Risk Factors for UTI's in Pregnancy:

1. Mechanical obstruction:ureteropelvic junction, urethral or ureteric stenosis, & calculi.
2. Functional obstruction:pregnancy&vesicoureteral reflux
3. Systemic diseases: DM, sickle cell trait/disease, gout, cystic renal disease

Classification of UTI's:

Clinical:

- Asymptomatic (8%)
- Symptomatic (1-2%)

Anatomical:

- Lower tract dis: asymptomatic bacteriuria and acute cystitis
- Upper tract dis: acute pyelonephritis

Asymptomatic Bacteriuria	Acute Cystitis	Acute Pyelonephritis
Consequences: acute pyelonephritis (30%) Clinical presentation: asymptomatic if there are symptoms (urgency, frequency, dysuria, suprapubicdiscomfort) Diagnosis: culture(MSU) Management: outpatient Abx (amoxil(penicillin), 1st generation cephalosporin, nitrofurantoin) Length of treatment : 3-10 days	Consequences: acute pyelonephritis (30%) Clinical presentation: urgency, frequency, dysuria, suprapubic discomfort Diagnosis: symptoms and culture (MSU) Management: outpatient Abx Length: 7-10 days (never 3 days in pregnant lady)→reculture (to make sure no further infection)	Consequences:sepsis, adult respiratory syndrome, anemia, renal failure, preterm labour. Clinical presentation:fever, chills, CVA tenderness, flank pain Diagnosis: symptoms, physical examination and lab: culture of urine and blood Management: Inpatient:Admission,Antipyreticagents,Abx (i.v. ampicillin or cephalosporin then p.o), steroid for premature labour. Length: 7-14 days →recultur

Perinatal Infections.

*Pregnancy is an immunosuppressive state due to hormonal and immunological changes. This state has advantages and disadvantages. The advantage is to not reject the fetus; i.e. accept the placento-fetal allograft, since the fetus is regarded as a foreign body. The disadvantage is making the mother more susceptible to various types of infections.

*It is important to screen the mother during the first antenatal visit (booking) for various organisms (ex. TORCH, Hepatitis and HIV).

*TORCH is an acronym for a group of five infectious diseases:

Toxoplasmosis
Other (syphilis)
Rubella
Cytomegalovirus (CMV)
Herpes simplex virus (HSV)

Infections that Affects the Fetus:

1. Genital Herpes Simplex Virus
2. Varicella Zoster
3. Syphilis
4. Rubella
5. Toxoplasmosis
6. Parvovirus
7. Cytomegalovirus
8. Human Immuno-deficiency virus
9. Chlamydia trachomatis
10. Hepatitis B
11. Group B Streptococcus
12. Listeriosis
13. Gonorrhea

General Principles of Prenatal Infections:

All viruses and most bacteria can pass through the placenta
The fetus does not make IgM until beyond 20 weeks gestation "That is why we say the defect is likely to happen in the first and early second trimester"
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** (as mentioned above why)
***All infections** can cause **abortion, IUGR, premature labor, severe neonatal sepsis, or long term carrier states** (like mental retardation) "Even if you don't remember what cretin infection can cause, you could mention these sequela"
Absence of fetal IgM at birth does not mean that infection did not occur unless the baby is 1 year old "because the immunity of the baby takes time to mature"

1-Genital Herpes Simplex Virus

"HSV is a DNA virus. Once the virus is transmitted it is there for live. After the initial infection, the herpes simplex viruses can hide within nerve cells and later launch new attacks. The recurrence of the disease is linked to stress and pregnancy. The classification of genital HSV infection includes: primary and recurrent infection"

*Herpes Simplex Type II "which is usually associated with genital sores"

*Risk of vertical transmission & **though the birth canal** "direct contact with the virus shed from infected sites"

***Primary infection** (first occurrence of HSV infection) **makes more damage than secondary attack** "symptoms of the primary infection include fever, malaise, myalgia and lymphadenopathy. Infants of women with the primary infection occurring during the pregnancy are at greatest risk."

*Primary Herpes infection in the late third trimester is far more dangerous than earlier infection

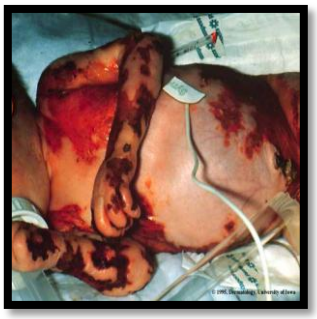
*Patients with outbreak during pregnancy should take acyclovir prophylaxis from 36 weeks until delivery "To decrease the need for cesarean birth"

***If lesion is present, cesarean section is the optimal mode of delivery**

"Women with primary infection in late trimester should have C/S. However, a woman with recurrent infection (who has a history and is seropositive for the HSV) first asses if she has genital lesions and give prophylaxis at 36 weeks if she is symptomatic, and if at labor she has lesions you do a C/S and if not you deliver vaginally"

*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



Congenital Herpes

2-Varicella Zoster (Chicken Pox)

***Vertical transmission through placenta**

*Infection before 20 weeks can lead to **abortion, limb hypoplasia, skin scarring, IUGR, neurological abnormality and hydrops fetalis**

*If infection near term, may lead to **postnatal infection which can be mild or fulminating leading to death**

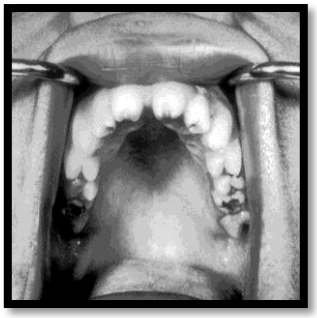
***Varicella Zoster immunoglobulin (VZIG)** should be given to pregnant mothers within 72 hours of exposure and to infants of mothers who develop chicken pox within 5 days before delivery or 2-3 days after delivery

Congenital Chicken Pox

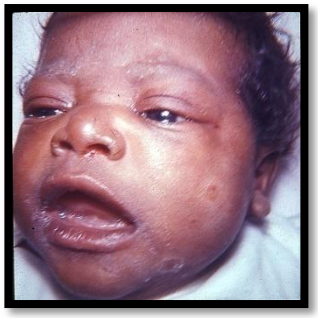
3-Syphilis (Treponema Pallidum)

*Infection to fetus is vertical in patients with **primary and secondary syphilis**
*Can lead to **abortion, still birth, or congenital syphilis** (maculopapular rash, hepato-spleenomegaly, lymphadenopathy, jaundice, 8th nerve deafness, saber shins, Hutchinson's teeth, saddle nose)
*Diagnosis by IGM antitreponemal antibodies
*Treatment is Penicillin
*Latent Syphilis may not transmit the disease

Saddle Nose



Hutchinson's teeth

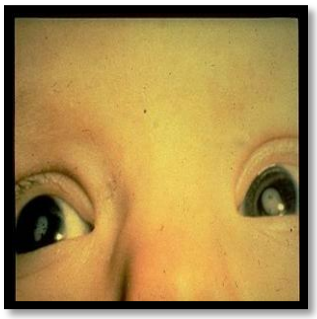


Maculopapular rash

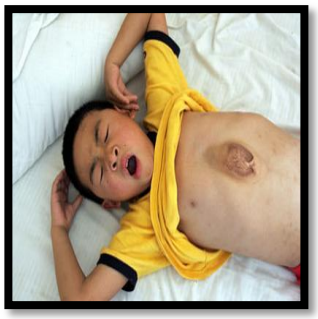
4-Rubella also known as German measles

(is very imprtant, and always comes in exames)

*Mainly first trimester infection can lead to **congenital rubella** (deafness, cardiac abnormality, cataract, microcephaly, mental retardation)
*No treatment
*Prevention is by **vaccination (childhood or post-natal)**
"it is contraindicated during pregnancy"
***Vaccine is live attenuated so, 3 months contraception is advised after vaccination**



Congenital Cataract



Congenital Heart Disease

5-Toxoplasmosa gondii

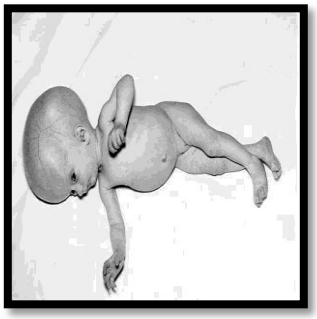
*Vertical transmission **through placenta**

***Mostly third trimester infection** that lead to severe neonatal manifestation

*Can lead to hydrocephaly, microcephaly, intracranial calcifications, jaundice, fever, seizers, chorioretinitis

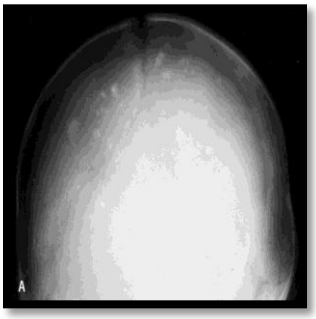
***If IgM titer is rising, spiramycin or pyrimethamine and sulphonamide are the treatment**

Hydrocephalus



Toxoplasma in Retina

Intracranial Calcification



6-Parvovirus B19

*Causes erythema infectious

*Vertical transmission can lead to **hydrops fetalis, hemolytic anemia, myocarditis, abortion, death**

*If less than 20 weeks and the fetus survive the infection, the fetus may be healthy

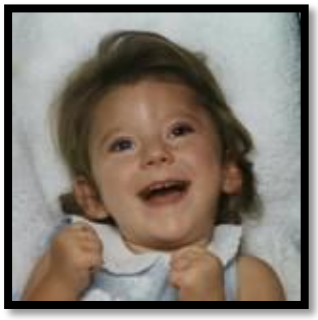
7-Cytomegalovirus

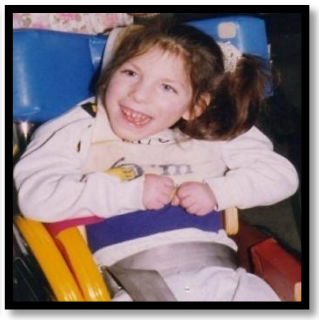
*In utero infection cause less than 1% of newborn infections

*Less than 10% of these infections will result in clinical illness

*Affected infants have **30% mortality; they may develop mental retardation, hearing loss, cerebral calcifications, hepato-spleenomegaly, thrombocytopenia, jaundice, chorioretinitis, and interstitial pneumonitis**

*10% of affected infants have no sequela





Mental Retardation

8-Human Immunodeficiency Virus (HIV)

*25% 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 (ROM)
***Cesarean section lower the transmission rate by two third in patients with no therapy**
***If ROM cesarean section within 4 hours is advised to protect the fetus**
*AZT (Zidovudine) that decrease the viral load during ante-partum, intra-partum, and neonatal period can reduce the risk of fetal infection by two thirds in mildly symptomatic ladies
***Avoidance of breast feeding reduce the risk of transmission by half**
*Special care during labor and in the operating room should be taken and needle brick prophylaxis when handling the infected patient
*Newborn is given I.V AZT

9-Chlamydia Trachomatis

*Infection is **through the birth canal**
*40% of infants will develop conjunctivitis, 10% will develop pneumonia
***Treatment is by erythromycin or azithromycin**

10-Hepatitis B (is very imoprtant)

*Transmission is vertical especially in the third trimester in acute infection
***HBsAg positive indicate chronic disease and risk of transmission to the fetus**
***HBeAg indicate high infectivity**
***The baby should be given Hepatitis B immunoglobulin at birth and an active immunization and repeated at 3, 6 months**
*Cesarean section or breast feeding is unlikely to alter the incidence of neonatal infection

11-Group B Sterptoccoci (GBS)

*5-20% of ladies carry GBS in vagina
*Infection through birth canal
*It is associated with **PROM**
*Can lead to **neonatal meningitis, pneumonia, sepsis**
*Intrapartum prophylaxis is indicated for carriers

12-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 amoxicillin or erythromycin

13-Gonorrhea

*Infection **through birth canal**
*Can lead to **conjunctivitis, arthritis, meningitis**
*Treatment by Penicillin and probenicid, or erythromycin

14-H1N1

*Same risk like any other viral infection
***Chemoprophylaxis (Tamiflu) for 10 days**
*Risk of abortion, preterm birth, pneumonia
*Infants risk of neural tube defects, seizers, encephalopathy, cerebral palsy, neonatal death

Rh isoimmunisation, ABO Incompatibility

Rh ISOIMMUNIZATION

Is an immunological disorder occurs in pregnant a Rh-ve mother carring Rh+ve fetus
It affects 1 in 250 live births in Europe and North America, it is much less frequent in other parts of the world such as Asia, where the Rh-negative blood group is uncommon.

-Pathophysiology:

The Rh antigen is limited to the red cell surface (Rh compex, C,D,E,c,e)

-fetomaternal HAEMORRHAGE (fmH):

Rh isoimmunization can only take place if fetal red cells cross the placental barrier into the maternal circulation.

- The placenta is subjected to maximal trauma during delivery
- After abortion with a gestational age above 14 weeks
- APH / trauma
- External version
- Amniocentesis
- Complicated and difficult deliveries
- Caesarean section

-THE NATURAL HISTORY OF Rh IsoiMMUNIZATION:

Rhesus antibodies are humoral antibodies or free antibody
IgM – large, unable to cross the placenta
IgG – small, able to cross the placeta and
Attach itself to Rh positive red cells
leading to haemolytic anaemia

-IMMUNE RESPONSES ARE:

A- Primary – first response to an antigen appears after several weeks and is IgM.
B- Secondary When exposed for the 2nd time a primed, antibody will appear within a few days and its IgG.
Generally, the quantity of antigen required to produce a secondary immune response is very much smaller than that required to initiate the primary immune response.

-Pathophysiology:

The first pregnancy is usually unaffected by RHD because FMH's of sufficient magnitude to induce primary immunization do not usually take place until delivery.
Only about 5% of all Rh-negative mothers form antibodies.
Vast majority of FMH's after delivery are small but about 0.2% of mothers have larger bleeds of 30 ml or more.
The risk of Rh immunization is proportional to the size of the FMH.

ABO INCOMPATIBILITY

When the mother and the baby are ABO incompatible such as an O mother and an A baby any fetal red cell (Group A) entering the maternal circulation (Group O) is destroyed, in an exactly similar way to that occurring in an ABO incompatible blood transfusion.

PREGNANCY: -

FMH does occur during pregnancy but is much less common than following delivery. Most of the bleeds occur in the last trimester when the placenta is degenerating and the barrier may become a little more pervious.

-The prevention of RHD:

D-positive FMH's can be neutralized by passively administered anti-D antibody (Rh immunoglobulin)" in every pregnancy ".

- At 28 weeks
-Post delivery
-After abortion
-APH / trauma
-External version
-Amniocentesis

-FAILURE RATE:

About 1% of Rh-ve women become immunized after D-positive pregnancies despite treatment with Rh immunoglobulin

Those already primed, even though overt antibody is undetectable by present techniques.

Large FMH's before delivery e.g. epileptic or eclamptic patients.

Extreme sensitivity to the D-antigen: thus small bleeds will produce primary response.

Large FMHs after delivery more than the amount that can be taken care of by standard dose of immunoglobulin.

Failure to give the immunoglobulin – patients who slip through the net.

MANAGEMENT OF ISOIMMUNIZATION:-

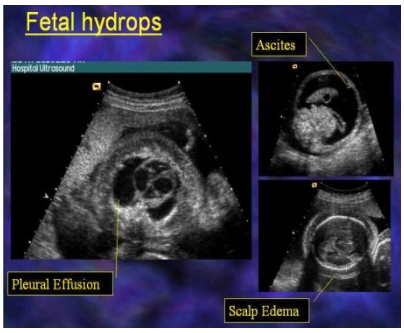
Pregnancies complicated by clinically relevant isoimmunization are managed in centers with fetal medicine units and regional blood transfusion.

1-Maternal blood group and antibody quantification
2-Paternal blood group genotyping
3-Fetal blood group genotyping
4-Ultrasound assessment
5-Amniotic fluid spectrophotometry
6-Fetal blood sampling
7-Fetal blood transfusion

MGT:-

Maternal blood group and antibody quantification at booking and 28 weeks if initial is –ve
Paternal blood group genotyping
Anti-D titer is not serous if below 1:16 and should be repeated every 2-4 weeks
If titer is above1:16 invasive testing:

- -Ultrasound assessment
- -Amniotic fluid spectrophotometry
- -Fetal blood sampling
- -Fetal blood transfusion



-ULTRASOUND ASSESSMENT:

The severely anemic fetus on scan will have: skin edema, ascites, pleural or pericardial effusions, cardiomegaly and an edematous placenta (Hydrops).
Middle cerebral artery blood flow is increased

-AMNIOCENTESIS AND AMNIOTIC FLUID ANALYSIS:

When fetal heamolysis occurs the amniotic fluid becomes bright yellow from the bilirubin
Amniotic fluid bilirubin concentration can be quantified by spectrophotometry by assessing the change in optical density at 450nm (△ OD 450)
Amniocentesis is started after 24 weeks under ultrasound guidance

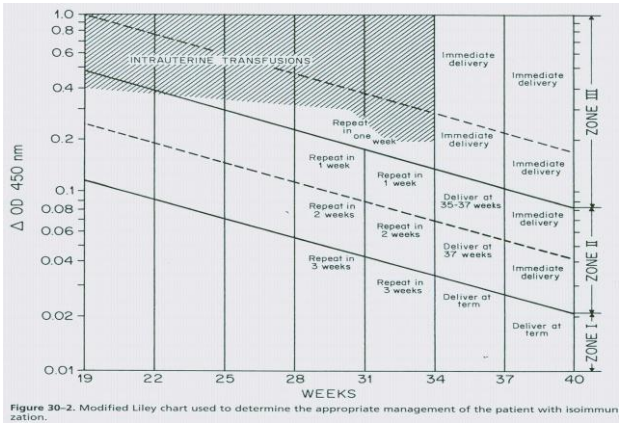


Figure 30–2. Modified Liley chart used to determine the appropriate management of the patient with isoimmunization.

COMPLICATIONS FOLLOWING INTRAUTERINE TRANSFUSION:-

- 1- Premature labour
- 2- Pre-labour ruptured membrane
- 3- Fetal haemorrhage
- 4- Fetal bradycardia
- 5- Failure to obtain a sample
- 6- Increase in maternal Iso immunization by inducing feto-maternal haemorrhage

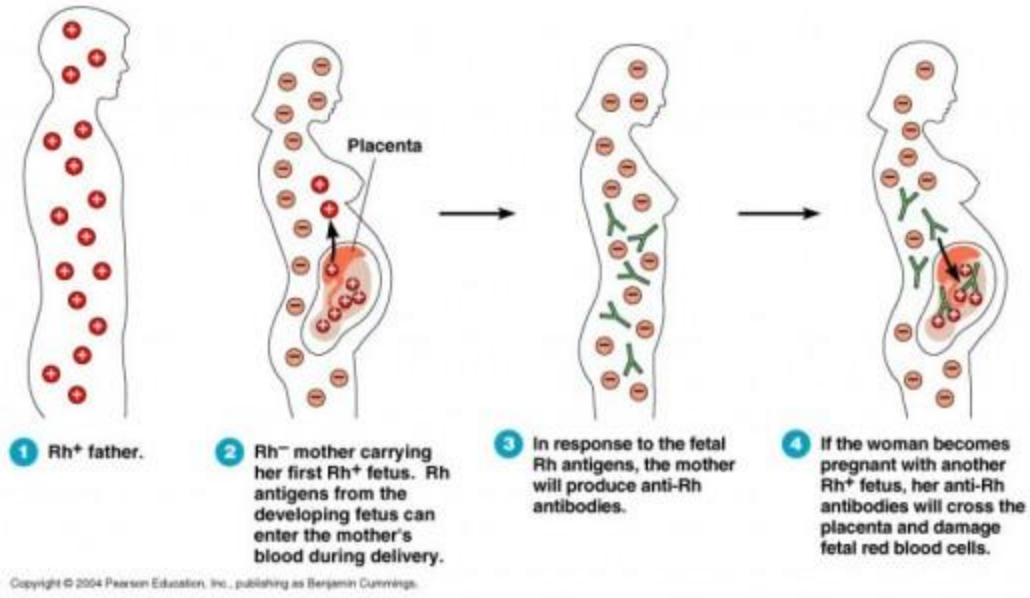
CTG: -

- 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 anaemia

Irregular Antibodies:-

- 2% other than Rh D
- Kell, Duffy, Kidd….ext

In summary


Placenta
1 Rh+ father.
2 Rh− mother carrying her first Rh+ fetus. Rh antigens from the developing fetus can enter the mother's blood during delivery.
3 In response to the fetal Rh antigens, the mother will produce anti-Rh antibodies.
4 If the woman becomes pregnant with another Rh+ fetus, her anti-Rh antibodies will cross the placenta and damage fetal red blood cells.
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Puerperium& Puerperal Sepsis

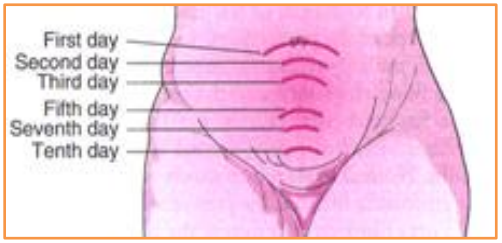
- **Puerperium:**
  - ✓ It is the time from the third stage of labor (delivery of the placenta) until reproductive organs return to their original non-pregnant condition.
  - ✓ It is a time of physiological and mental adjustment to the new environment with the arrival of a new baby.
  - ✓ All the physiological changes of pregnancy is reversed and the pelvic organs return to their previous state and endocrine influence of the placenta is removed

PUER= child
PERIUM= giving birth

- **Objectives of medical & nursing care during the puerperium:**
  - ✓ Monitor physiological changes of puerperium.
  - ✓ To diagnose and treats any postnatal complications.
  - ✓ To establish infant feeding.(encouraging mothers to choose breast-feeding)
  - ✓ To give the mother emotional support.(b/c of the big changes at the hormonal levels, and there are emotional and psychological changes happen in the puerperium too)
  - ✓ To advise about contraception.

THE PELVIC ORGANS

1. Uterine involution:



- After delivery, uterine fundus palpable at level of umbilicus
- 10-14 days later, disappears behind thesymphysis pubis
- This process is aided by oxytocinduring breastfeeding
- Delay in involution(doesn't go to its normal size) can happen if there is an infection, accessory lobe of placenta or retained products of placenta

2.The cervix:

The cervix before delivery will be firm and pink, but after delivery it will become fluffy and soft, and with time it will be firm but it will never close completely like before!

- After delivery, it becomesflaccid and curtain like
- Few days after →original form &consistency
- External os dilated (one finger (weeks—months))
- Internal os is closed to lessthan one finger by the 2nd week of the puerperium.

3.The vagina:

- 1st few days of puerperium,the vaginal wall becomes smooth, soft and edematous
- Slight distention return to normal capacity in few days
- Episiotomy and tears of vagina and perineum heal well
  (The vagina after delivery is very friable, edematous and congested, so it will be difficult to suture a small tear (you can just pack it) but if it's a big tear, you can suture it. And we advise the pregnant women if they would like to do perennial repair, they should never do it after delivery immediately b/c they may bleed a lot, so after delivery,they have to wait 3 to 4 months before doing any surgery in the vagina)
- Healing is impaired in presence ofhematoma or infection

4. Endometrium cavity:

- Decidua is cast off as a result of ischemia →lochial flow(bleeding after the birth) there are normal variations between the women, and normal variations during the puerperium, and normal variations between baby and other baby from the same lady.

- Lochia= blood, leucocytes, shreds of decidua and organisms.
- Initially; dusky red, fades after one week, clears within 4 weeks of delivery.
- New endometrium grows from basal layer of decidua.

1. Bladder & Urethra:

- Within 2-3 weeks →hydroureter and calycial dilatation of pregnancy is much less evident.
  It's important to know that b/c if the woman does US immediately after delivery, you may say "she is having stones" especially in the right side, but it's not! b/c this is normal!
- Complete return to normal → 6-8 weeks
- Diuresis during first day

2. Blood

(After delivery, large amount of blood will go form Intra-vascular to Extra-vascular space, so the women may become buffy and this is normal, but with time the body will get rid of it, that's why the women loss lots of weight in the first week.)

- ↓ Plasma volume
- Blood clotting factors and platelet count rise after delivery
- Fibrinolytic activity (which occurs during pregnancy) is reversed within 30 min. of placental deliver

Complications of the puerperium

Complications of the puerperium are serious.

Sometimes fatal disorders may arise during the puerperium

1. Thrombosis & Embolism

It is one of the main causes of maternal death.

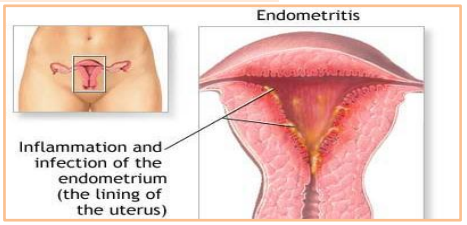
(It is the commonest cause in developed countries)

2.Puerperal infection (IMP)

Postpartum fever is defined as a temperature greater than 38.0°C on any 2 of the first 10 days following delivery exclusive of the first 24 hours

- **Causes:**

A- Endometritis -Inflammation and infection of the endometrial lining- (It is the commonest cause) -



- Local spread of colonized bacteria to the uterus affect 5-7%
- # 6 cause of maternal death(WHO)
- **Risk factors:**
  - ✓Prolong rupture of membrane(which means more than 24 h)
  - ✓C/S-Caesarean section- (now, we give prophylactic antibiotics for every C/S to prevent infection)
  - ✓Fever before delivery
  - ✓Prolong labor/ multiple examination(multiple vaginal Ex)
  - ✓Manual removal of placenta(if you remove the placenta manually, you may introduce infection)
  - ✓Others like diabeticmother, obese mother, using internal electronic fetal monitoring, or inserting any other instruments
- **Symptoms:**
  - ✓Fever and chills
    (If a woman comes to you after delivery with fever, you have to give her broad-spectrum antibioticuntil you exclude it)
  - ✓Abdominal pain
  - ✓Foul-smellinglochia
- **Diagnosis:**
  - ✓Symptoms and signs
- **Organisms:**
  - ✓Multi-organism
- **Dx/ Investigations:**
  - ✓Full ClinicalExamination
  - ✓MSU(Mid stream urine)
  - ✓Cervical & HVS
  - ✓Sputum C/S (if possible)
  - ✓Blood culture
- **Management:**
  - ✓After investigation, start antibiotics –broad spectrum antibiotics-

POSTPARTUM FEVER

Definition: Fever ≥100.4° F (38°C) on ≥2occasions ≥6 hours apart, excluding first 24 hours post-partum

PP fever- Day 2–3: Endometritis

Most common cause of postpartum fever.

Risk Factors: Emergency cesarean section after prolonged membrane rupture and prolonged labor.

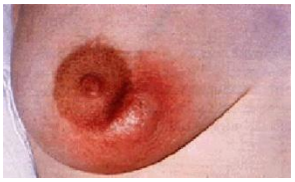
Clinical Findings: Moderate-to-high fever with exquisite uterine tenderness. Peritoneal signs should be absent and peristalsis should be present.

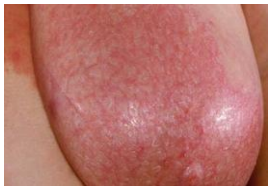
Management: Multiple-agent intravenous antibiotics (e.g., gentamycin and clindamycin) to cover polymicrobial genital tract flora.

B-Mastitis(Breast infection)

- **Acute intramammary mastitis**=>due to failure of milk withdrawal from a lobule
  Usually before starting breast-feeding, the breast will become engorged, hot, (she will start to shiver but with low grade temperature) quite painful and tender but there is no redness and no sign of cellulitis.
  Rx.→(supportive) encouragebreast-feeding, cold compress, if there is no improvement within 24 hrs =>antibiotics

- **Infective mastitis**=> May be due to staph. Aureus
  Rx.→Antibiotics according to sensitivity.





- **Breast abscess formation**=> Rare but preventable
  Rx.→ Surgical drainage if established.

  → Antibiotics, only if early.

PP fever - Day 7–21: Infectious Mastitis

Risk Factors:

Lactational nipple trauma leading to nipple cracking and allowing Staphylococcus aureus bacteria to enter breast ducts and lobes.

Clinical Findings:

Fever of variable degree with localized, unilateral breast tenderness, erythema, and edema.

Management: Oral cloxacillin. Breast feeding can be continued. Ultrasound imaging is needed to rule out an abscess if lactational mastitis does not respond to antibiotics.

- Other causes:

✓ Urinarytractinfection(it is common b/c pregnancy itself increases the risk of having infection, and sometimes during delivery, the women do not empty their bladder very well, and we sometimes insert Foley catheter and those will increase the risk of having infection).
✓ Deepveinthrombosis (DVT)
✓ Respiratoryinfection
✓ Surgicalwoundse.g. C.S.orepisiotomy, which is NOT common.
✓ Other non-obstetricscauses

3-Secondary postpartum hemorrhage:

Excessive blood loss from genital tract more than 24 hrand within 6 weeks of delivery

- Causes:
  - ✓ Retained placental fragments
  - ✓ Infection(late infection is usually endometritis and is caused especially by{chlamydia} and it can cause 2ry PPH)

~ Usually within few days after delivery(Commonest between 8-14 days)

- Management:
  - ✓ Mild bleeding →observe
  - ✓ IV fluid /blood + oxytocic drug
  - ✓ Evacuation of uterus under GA(general anesthesia)if:
    - USS suggests presence ofretained placental tissue
    - Heavy bleeding persists & the uterus is larger than expected and tender; the cervix is open.
    - The infection is treated appropriately.

4-Puerperial mental disorders:

- Types:

1-**Postnatal Blues**[Care of self and infant maintained]:it is NOT a disease (not pathology)

- ✓ Anxiety and depression
- ✓ Usually at 3rd and 4th day
- ✓ Self limiting(encourage her to not stay alone, to go out and change air, take a shower, and support her and her family)

2- **Puerperal Depression**[Care of self and infant neglected]:it's a disease and it needs treatment!

- ✓ Pre exiting depression
- ✓ Very traumatic delivery

3-**Puerperal Psychosis**[May express ideation to harm self and/or infant]:

- ✓ Uncommon, however serious
- ✓ Due to endocrine changes in puerperium, or are an uncovering of an underlying psychotic tendency at avulnerable stage.
- ✓ Psychiatrist opinion is needed hence risk of suicide and safety of baby are paramount consideration.
- ✓ **Warning signs:** Confusion, restlessness, extreme wakefulness, hallucination and delirium

➢ Treatment:

According to severity:

- ✓ If mild (Postnatal blues)→Observe, discuss, mild sedatives.
- ✓ If severe(Puerperal Depression and Puerperal psychosis)→ heavy sedation + transfer to psychiatric ward.

CONSUPMTIVE COAGULOPATHY (DIC)

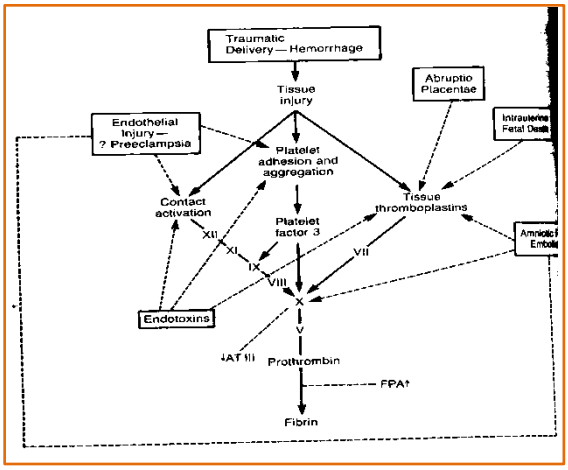
A complication of an identifiable, underlying pathological process against which treatment must be directed to the cause.

➢ Pregnancy Hypercoagulability:

- ✓ ↑Coagulation factors I (fibrinogen), VII, IX, X
- ✓ ↑Plasminogen; ↓ plasmin activity
- ✓ ↑fibrinopeptide A, b-thromboglobulin, platelet factor 4, and fibrinogen

➢ Pathological Activation of Coagulation Mechanisms:

- ✓ Extrinsic pathway activation by thromboplastin from tissue destruction.
- ✓ Intrinsic pathway activation by collagen and other tissue components.
- ✓ Direct activation of factor X by proteases.
- ✓ Induction of procoagulant activity in lymphocytes, neutrophils or platelets by stimulation with bacterial toxins.



Disseminated Intravascular Coagulation (DIC; Rare)

Risk Factors:
Abruptio placenta (most common), severe preeclampsia,amniotic fluid embo-lism, and prolonged retention of a dead fetus.

Clinical Findings: Generalized oozing or bleeding from IV sites or lacerations in the presence of a contracted uterus.

Management: Removal of pregnancy tissues from the uterus, intensive care unit (ICU) support, and selective blood-product replacement.

➢ Significance of Consumptive Coagulopathy:

- ✓ Bleeding.
- ✓ Circulatory obstruction→organ hypoperfusion and ischemic tissue damage.
- ✓ Renal failure.
- ✓ ARDS**[acute (adult) respiratory distress syndrome]**.
- ✓ Microangiopathichemolysis.

➢ Causes:

- ✓ **Abruptio placentae(most common cause in obstetrics)which may be revealed (blood comes out through the vagina) or concealed (no visible blood loss)**
- ✓ **SeverHemorrhage**(Postpartum hge)
- ✓ Fetal Death and Delayed Delivery >2wks
- ✓ Amniotic Fluid Embolus(very rare, fatal condition and the survival rate is less than 30%)
- ✓ Septicemia
- ✓ Acute fatty liver syndrome

➢ Treatment:

- ✓ Identify and treat source of coagulopathy
- ✓ Correct coagulopathy
  - FFP, cryoprecipitate, platelets

(You have to replace the blood and the factors)

Fetal Death and Delayed Delivery

- ✓ Spontaneous labour usually in 2 weeks post fetal death.
- ✓ Maternal coagulation problems <1 month post fetal death.
- ✓ If retained longer, develop coagulopathy.
- ✓ Consumptive coagulopathy mediated by **thromboplastin from dead fetus**.

➢ Treatment:

- ✓ Correct coagulation defects and delivery.

Amniotic Fluid Embolus

- ✓ Complex condition characterized by abrupt onset of hypotension, hypoxia and consumptive coagulopathy.
- ✓ 1 in 8000 to 1 in 30 000 pregnancies.(so it's rare)
- ✓ "Anaphylactoid syndrome of pregnancy".

➢ Pathophysiology:

- ✓ Brief pulmonary and systemic hypertension→transient, profound oxygen desaturation (neurological injury in survivors) → secondary phase: lung injury and coagulopathy.

➢ Diagnosis:is clinical.

➢ Management: Supportive.

➢ Prognosis:

- ✓ 60% maternal mortality; profound neurological impairment is the rule in survivors.
- ✓ Fetal: outcome poor; related to arrest-to-delivery time interval; 70% neonatal survival; with half of survivors having neurological impairment.

Septicemia

- ✓ Due to **septic abortion**, **antepartum pyelonephritis**, **puerperal infection.**
- ✓ Endotoxin activates extrinsic clotting mechanism through **TNF (tumor necrosis factor).**
- ✓ Treat the cause.

Abortion

➢ Coagulation defects from:

- ✓ Sepsis (Clostridium perfringens highest at Parkland) during instrumental termination of pregnancy.
- ✓ Thromboplastin released from placenta, fetus, decidua or all three (prolonged retention of dead fetus).

Some aspects in Neonatal Management

Contents:

1. Delivery Room Resuscitation:

Perinatal Physiology: Apnea = inability to breath, it has 2 types :

1. **Primary Apnea**: due to brief hypoxia ➔ Recovers with stimulation and oxygen supplement.
2. **Secondary Apnea**: with prolonged hypoxia ➔ Requires assisted ventilation and oxygen.

➔ Quickly stimulate the baby with simple stimulation "wiping" if he is not responding ➔ directly start assisted ventilation with O_2.

❖ **Goals of Resuscitation:**

1) Minimizing Immediate Heat Loss ➔ Dry the baby + put him under radiant warmer
   Then continue Resuscitation as usual ABC airway, breathing, circulation:
2) Establishing Normal Respiration and Lung Expansion
   Air way : clear it by suction of the fluid from mouth then nose & don't introduce the suction tube deeply ➔ this will cause vagal nerve stimulation ➔ bradycardia.
   - Clear the airway + position the baby + assess breathing & adequate lung expansion + skin color
3) Increasing Arterial PO2 ➔ check the arterial O_2 concentration by pulse Oximetry
4) Supporting Adequate Cardiac Output ➔ by pulse rate + pulse volume + color of the baby : pink ➔ good circulation , if cyanosed : abnormal may be the baby has bradycardia

❖ **Steps for effective resuscitation:**

Preparation ➔ pediatric team should be present, & informed a head of time about neonatal risks

1. identify high risk deliveries :
   - fetal distress "asses through CTG", fetal disease or serious conditions like: meconium, prematurity, post- maturity abnormal fetal weight "small or large", major anomalies "chest cage abnormality", hydrops, multiple gestation, cord prolapse, abruptio placentae. Neonatal team should know about the fetal condition before to be prepared, Nitric oxide for baby expected to have pulmonary HTN.
     Resuscitation team ➔ one doctor & one nurse for each baby. Many teams in multiple gestation.
   - Labor & delivery conditions: "maternal condition"
     APH "Antepartum hemorrhage", abnormal presentation "face, breech" , difficult labor.

2. No pediatric team required personnel for evaluation **in low risk baby**
   such for diabetic mother, the team can come after the delivery.
   - Neonatal conditions : unexpected congenital anomalies, respiratory distress, unanticipated neonatal depression.
   - Maternal conditions:
     signs of maternal infection, maternal illness e.g. DM, isoimmunization, PET, renal, endocrine, pulmonary, or cardiac disease.
     ➔ if any problem suddenly develop we call the resuscitation team, but no need for them to come from the beginning in low risk situations.

❖ **Necessary Equipment:**

1) Radiant warmer
2) Oxygen source
3) Self inflating bag with reservoir or anesthesia bag
4) Face mask with appropriate size
5) Suction tubes with different sizes 5, 6 for small baby, 8 for large, 10 for meconium "thick"
6) Stethoscope
7) Emergency box: laryngoscope, batteries, ET. endotracheal Tubes, drugs:
   - epinephrine (1:10000)
   - NaHco3 (4.2%) half concentration cause higher can cause intraventricular hemorrhage.
   - Naloxon ➔ antidote for opioids (pethidine or any narcotic) if the mother take it within 4h of labour, the antidote should be given to the baby to prevent respiratory depression.
   - Albumin, and NaCl 0.9% ➔ volume expander, but if large blood ➔ need Packed RBC.
8) Umbilical catheterization tray ➔ inserting catheter through umbilical vein is faster than peripheral veins in giving volume expanders, Na bicarbonate, epinephrine & others.
9) Syringes, needles, t-connectors, and stopcocks
10) Transport incubator with batteries
11) Pulse Oximetry ➔ to check oxygenation.

Begin a process of evaluation, decision, and action :

Ex: Evaluate the color "cyanosed body & limbs but with no bradycardia + with good breathing movement" ➔ no need for ventilator or pressure only free flow O_2 will improve the color.

But if the baby is "apneic no respiratory movement + cyanosed" ➔ he need positive pressure ventilation.

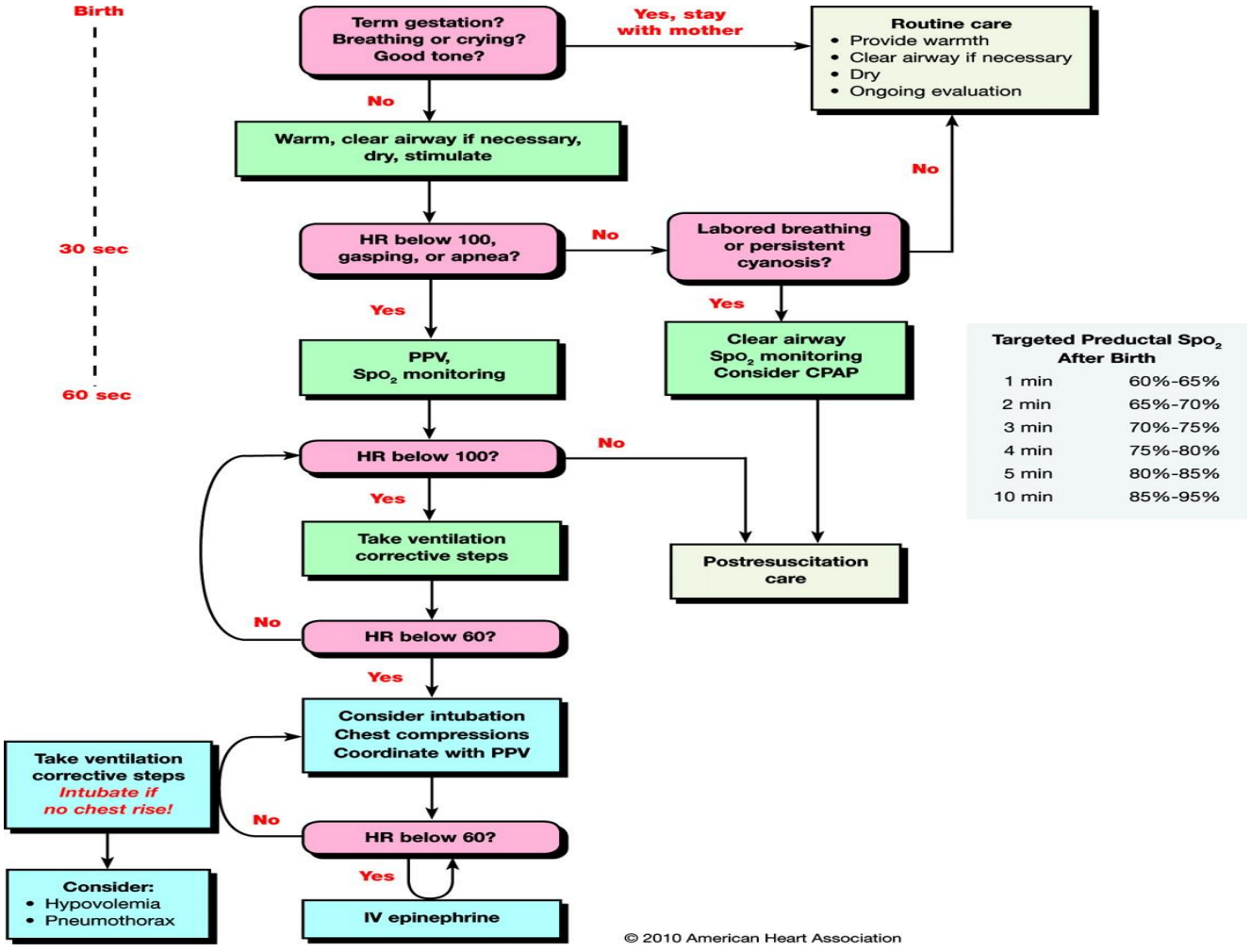
- Place on warm table
- Dry & discard the wet linens extra warming
- Positioning
- Suction the mouth, oropharynx, and nares (avoid deep pharyngeal suction) ➔ vagal nerve stimulation ➔ bradycardia.

Resuscitation algorithm :-

For term is different than preterm.

Time is critical ➔ 30 sec needed for each step in resuscitation.

1. Warm, clear, dry, stimulate + observe if healthy skin to skin contact with mother
2. Asses HR + breathing (positive pressure ventilation + O_2 + endotracheal tube)
3. Cardiac massage + positive pressure ventilation (HR :120 + ventilation : 30-60) ➔ 3:1 ratio 3 cardiac compress to 1 ventilation
4. HR is not responding & below 60 : give epinephrine, correct acidosis if present.
5. Check if there is a problem preventing the success if resuscitation : Pneumothorax "chest bulge from one side" Transillumination test, Pleural effusion, Hypovolemia "the best volume expander is normal saline"
6. Preductal saturation after birth: patent ductus arteriosus between pulmonary & aorta ➔ Preductal means the right upper limb only, the rest of the limbs & the umbilical cord is post ductal , the only part supplied by 3 ductal is the right hand ➔ fix the saturation monitor on the right hand & connect it to the machine ➔ normally O_2 saturation 60-65 in the first minute "physiological cyanosis" ➔ will reach above 90 after 10 minutes of age. But if saturation is not getting better give O_2 & observe.



2. APGAR score

Virginia Apgar devised the Apgar score in 1952 as a simple and replicable method to quickly and summarily assess the health of newborn children immediately after birth.

The five criteria of the Apgar score: out of 10

	Score of 0	Score of 1	Score of 2	Component of Acronym
Appearance/Complexion	blue or pale all over	blue at extremities body pink (acrocyanosis)	no cyanosis body and extremities pink	**A**ppearance
Pulse rate	absent	<100	>100	**P**ulse
Reflex irritability Reaction to suction	no response to stimulation	grimace/feeble cry when stimulated	cry or pull away when stimulated	**G**rimace
Activity Tone & movement	flat + full term ➔ asphyxia none Preterm + no tone ➔ normal	some flexion	flexed arms and legs that resist extension Flexion & spasticity	**A**ctivity
Respiratory Effort	absent	weak, irregular, gasping	strong, lusty cry	**R**espiration

- Gasping ➔ Prolonged hypoxia ➔ Metabolic acidosis (شهيق قوي كأنه مخنوق)
  Hypoxia cause accumulation of Lactic acidosis ➔ depression of brain & respiratory centre.
- Grunting ➔ Breathing against closed glottis (the sound of weak cough مثل الكحة بصوت ضعيف)
- Don't depend only on the first minute APGAR assessment give him a chance ➔ every 5 minute assess the score & resuscitate (until reach 20 minutes here stop).

3. Management of Premature Infant

Prematurity:

1- Definition: baby born more than 20 weeks and less than 37 weeks. Wight more than 500 - 2449 gm.

* we are not resuscitating babies 20 weeks or less. (we usually resuscitate 23 w & above)

2 - Features: Shiny Thin Red skin, extended arms and legs, little subcutaneous fat, sparse hair few palmar and sole creases, poorly developed ear cartilage, genitalia boys few rugae undescended testicles girls gaping labia prominent clitoris, small labia.

Premature infants:

the premature infant is quickly transferred from the warm fluid medium of the liquor amnii with its practically constant temperature to the variable circumstances of an extra uterine life; he loses the preparatory transition-time of the third trimester of intrauterine existence.

* Need special environment resembling the mother uterus.
* Hypotensive baby due to poor vascular tone "immaturity".

Problems of prematurity: (Temperature ➔ ABC)

- Thermoregulation (hypothermia ➔ incubator + humidity + Vaseline)
- Respiratory distress (surfactant)
- Apnea (control breathing centre is absence)
- Sepsis (thin skin, immature immunity)
- poor sucking & swallowing reflex

Delivery room Management of premature infants:

- Resuscitation
- Temperature management
- Ventilation support and maintain normal saturation (89-95%).
- Surfactant administration for infants 1kg or less.
- Counseling the mother
- Transportation to NICU

Premature infants management in NICU:

- Ventilation
- Monitoring blood pressure
- Prevention of insensible water loss
- Treating sepsis
- Monitoring acid –base status
- Prevention of intra ventricular hemorrhage
- Nutrition, entral and parenteral ➔ the best : mother breast milk only!
- Fluid, electrolytes, glucose

4. Management of Infant Diabetic Mother

Fetal-neonatal complications are directly related to inadequate glycemic control during key periods of pregnancy.

1) Poor peri-conceptional and **early first trimester glycemic** control are related to spontaneous abortions, early growth delay, and major congenital malformations.
2) **During the second trimester**, it is predictive of Pregnancy Induced Hypertension (PIH), preterm labor and delivery and minor congenital anomalies**.**
3) **During the third trimester** of pregnancy it is predictive of macrosomia, birth trauma, fetal dystocia, maternal trauma and high cesarean delivery rate .It is also associated with complications linked to fetal **hyperinsulinism** such as neonatal hypoglycemia, respiratory distress, cardiac Asymmetric Septal Hypertrophy (ASH) ,and to decreased fetal oxygenation and its acute or chronic complications such as neonatal polycythemia ,or thrombocytopenia.
4) Finally, hyperglycemia **in labor** aggravates the risk of neonatal hypoglycemia

Major congenital malformations:

High sugar concentration is toxic to cultured cell growth ,which may explain the early growth delay highly predictive of congenital malformations of complicated diabetic pregnancies .Maternal magnesium (Mg) depletion (lost in urine with polyuria) may contribute to malformation**.**

Mg is necessary for parathyroid gland & Ca ➔ fetal hypomagnesaemia & hypocalcaemia in the baby ➔ convulsion, spasticity, jitteriness.

All malformations are more frequent in IDM's, but some, such as **caudal regression syndrome are highly specific to maternal diabetes**.

- **Small left colon syndrome:** transient
- **Macrosomia:** After 20 weeks gestation maternal hyperglycemia leads to fetal hyperglycemia and hyperinsulinemia, which in turn lead to enhanced growth and macrosomia .
- **Intrauterine growth restriction:** A small subgroup of IDMs delivered to mothers with advanced diabetic class, with significant vascular disease, may be affected by growth restriction.
- **Fetal and neonatal hypoxia:** Poorly controlled diabetes may lead to both decreased oxygen supply to the fetus, and increased oxygen consumption by the feto-placental Unit.

Increased affinity of glycosylated hemoglobin **(HbA1c)** to O2 may be contributory to decreased O2 maternal-fetal transfer In the presence of excess fuels or of **hyperinsulinemia**, the placental metabolic and oxygen consumption rates increase, depriving the fetus of sufficient oxygen

Chronic fetal hypoxemia may lead to:

wide range of clinical consequences, from "sudden" intrauterine death, to mild neonatal depression at birth .It also leads to increased production of fetal erythropoietin and increased rates of polycythemia.

Management of IDM

- **Neonatal Hypoglycemia (NH):**
- **Disorders of mineral metabolism in IDM's:**
- **Decreased bone density in IDM's:** Decreased bone density has been reported in IDM's and appears to be due to increased bone resorption, rather than to decreased bone formation.
- **Neonatal hypocalcemia and hypomagnesemia:** In a recent past, Risk factors of NHC in IDM's are birth asphyxia and prematurity .Mg deficiency plays also an important role in the pathogenesis ,maternal glycosuria, accompanied by urinary Mg loss In turn, maternal Mg deficiency leads to fetal Mg deficiency Mg is necessary for the appropriate secretion of PTH, as well as in its action upon its target cells .
- **Prematurity and Respiratory Distress Syndrome (RDS):**

Neonatal Management of the IDM :

- **Delivery room management** (difficult labor, birth truma, glucose level)
- **Nursery management**.:

a. Vital signs examination and monitoring, at least hourly for the next following 4-6 hours, time during which signs and symptoms of complications such as hypoglycemia or RDS may develop
b. Complete physical examination
c. Screening for and management of NH *neonatal hypoglycemia. The definition of NH is highly controversial. Many reports have arbitrarily defined NH as being a serum, plasma, or whole Blood Glucose value below 30-50 mg/dl Lucas et al., who found a threshold value of 47 mg/dl (2.5 mmol/ l) to be more predictive of lower Bayley scores. ➔ less than this dangerous to brain + delay development.

Management of hypoglycemia:

Oral mother "if not responding" ➔ IV bolus of dextrose "if not responding ➔ increase to high concentration dextrose & use UVC umbilical vein cord for high conc.

2 ml/kg dextrose 10% in water (D10W), intravenously) and/or starting a continuous infusion of glucose (D10W at 80–100 ml/kg per day). The goal is to maintain plasma glucose concentrations in symptomatic infants between 40 and 50 mg/dl.

Asymptomatic infants: A pragmatic approach was developed

there is little or no evidence to indicate that asymptomatic NH in the first days

5) **Premature vs. Dysmature:**

Premature:

- Less than 37 weeks
- Incomplete organ system development

Dysmature:

post maturity syndrome a syndrome due to placental insufficiency that causes chronic stress and hypoxia, seen in fetuses and neonates in post term pregnancies, characterized by decreased subcutaneous fat, skin desquamation, and long fingernails, often with yellow meconium staining of the nails, skin, and vernix.

➔ poor intrauterine nutrition

Definitions:

1. Of, relating to, or characteristic of faulty embryologic development, often leading to structural and/or functional abnormalities.
2. Relating to or characteristic of an infant whose birth weight is inappropriately low for its gestational age

A complex of symptoms occurring in an infant, such as a relative absence of subcutaneous fat, skin wrinkling, prominent fingernails and toenails, and a meconium staining of the skin and the placental membranes, that is associated with post maturity or placental insufficiency.

* There are lots of things from “Major congenital malformations” and below,

the doctor didn’t comment on it because of shortage of time.

Dysmenorrhoea, Premenstrual Syndrome & Endometriosis.

Dysmenorrhea

(Painful menstruation)

Kaplan:

- Primary dysmenorrhea is the most common gyne complaint among adolescent females. While secondary is more common among women in fourth and fifth decades of life.

Primary dysmenorrhoea:

- No pelvic pathology.
- The pain is associated with bleeding in the first and second day.

Secondary dysmenorrhoea:

- Secondary to pelvic pathology as endometriosis, chronic pelvic infection or endometrial polyps
- The pain starts few days before menstruation, continues for the duration of menses and may persist for days after.

Incidence:

80% of patients attend family planning clinic have dysmenorrhoea and was severe in 18% of them (Robinson et al., 1992)

Epidemiology:

- Long time smoker six time more than non-smokers
- Age is inversely associated with dysmenorrhoea
- Less common in parous women.

Pirimary Dysmenorrhea

Aetiology:

- Uterine hyperactivity: abnormal (increased) uterine hyperactivity leading to uterine eschemia. (and ischemia cause pain)
- Hyperalgesic substances e.g. prostaglandin E.

Causes:

- Increased uterotonic prostaglandins PGF_2a (found to be high in patient with dysmenorrhoea cause increase in contraction> ischemia> pain)
- Leucotrines produced by endometrium stimulates myometrial activity
- Vasopressin is a vasoconstrictor substance which stimulates uterine contraction. Circulating vasopressin levels was found to be higher on the first day of menstruation in women with dysmenorrhoea. (the same, cause increase in contraction> ischemia> pain)

Treatment of primary dysmenorrhoea

Medical treatment

- Reassurance and simple analgesic(sometime is enough specially for primary)
- NSAIDs are useful first line treatment with 80-90% improvement, particularly the mefenamic acid derivatives.
- If contraception is also required OCCP is appropriate.
- Oxytocin antagonist for future. (nothing currently)

Surgical treatment(rarely as a last choice)

- Used as last resort
- Laparoscopic uterosacral nerve ablation LUNA
- Hysterectomy (with bilateral ovarian oophorectomy)
- Cervical dilatation has no beneficial effect

Secondary Dysmenorrhea

Aetiology

- Endometriosis and adenomyosis(The commonest cause)
- Chronic PID
- Congenital or acquired uterine abnormalities

Investigations:

- USS (Very IMP for gyne, if she have endometriosis, endometrial polyps, all appear in US)
- HSG (Radiological modality, injecting contrast to view)
- Hysteroscopy
- laparoscopy

Treatment of secondary dysmenorrhea:(that of the cause), e.g.

- Endometriosis
- Adenomyosis
- Uterine abnormalities

Premenstrual Tension Syndrome

Recurring cyclical disorder in the luteal phase of the menstrual cycle, involving behavioral, psychological and physical changes resulting in loss of work or social impairment (Ried and Yen 1981). PMT may occur after hysterectomy with conservation of functioning ovaries

Diagnosis:(Should be severe enough to effect work or social life)

The American psychiatric association (APA) criteria for diagnosis are:

A. Symptoms are temporarily related to menstruation
B. The diagnosis requires at least 5 of the following symptoms, and one of the symptoms must be one of the first 4:

1. Affective labiality sudden onset of being sad, tearful, irritable or angry
2. Anxiety or tension
3. Depressed mode, feeling of hopelessness
4. Decreased interest in usual activities
5. Easy fatigability or marked lack of energy
6. Difficulty in concentration
7. Changes in appetite (food craving or over eating)
8. Insomnia
9. Feeling of being overwhelmed or out of control
10. Physical symptoms (bloating, breast tenderness, headache, edema, joint or muscular pain and weight gain.

A. The symptom interfere with work, usual activities or relationship
B. The symptoms are not an exacerbation of another psychiatric disorder

Prevalence:

Difficult to ascertain; 40% reported mild symptoms, of them 2-10% the symptoms interfere with their work or life style.

Etiology list of biological theories:

- Estrogen excess
- Progesterone deficiency
- Hyperprolactinemia
- Hypoglycemia
- Vit. B deficiency
- Increased aldosteron activity

Treatment of PMS

A. Non pharmacological treatment:

- Reassurance and support
- Relaxation and stress management
- Reflexology therapy that reduce somatic and psychological PMS symptoms
- Increase aerobic exercise ? By altering endorphins
- Well balanced diet with low sodium and fat contents
- Restriction of alcohol, chocolate, caffeine and dairy products
- Supplementation with vitamin B_6, E, magnesium and calcium
- Evening primrose oil
- Women on estrogen replacement therapy does not develop symptoms of PMS unless progesterone is added

B. Medical treatment:

- Pharmacological suppression of the hypothalamopituitary ovarian axis should offer a logical approach to therapy (to stop cyclical ovarian activity)
- Ovarian suppression using OCCP is beneficial in some patients but cause exacerbation of symptoms in others
- Danazol for breast symptoms
- GnRH agonist: it improve symptoms in some women & can be used as a treatment
- Diuretics in patients complaining of bloating, edema and weight gain
- NSAIDs: reduce many of the somatic symptoms as dysmenorrhoea
- For emotional and psychological manifestations serotoninergic antidepressent offer good first line approach. Fluoxetine (Prozac)
- Anoxiolytic as alprazola (Xanox) also offer some help.

C. Surgical treatment:(For really severe cases)

- Reserved only to patients with severe symptoms not responding to medical treatment
- Hysterectomy
- Bilateral oophorectomy (balance between symptoms relief and hypoestrogenic state and complications and the coast of HRT.

Endometriosis

- Endometriosis means the presence of endometrial tissue (glands and stroma) in abnormal sites, that is outside the normal uterine cavity. This ectopic endometrium responds to the ovarian hormones as the normal endometrium.

PREVALENCEL:

- The prevalence is about 5-10% in adult women and 20-40% in the infertile women.

THEORIES OF AETIOLOGY:Disease of theories!

- Endometriosis is explained by more than one theory because not all cases arise in same way. However, the true cause is unknown. (one theory is retrograde menstruation, few spots, some blood does not go out through the cervix and vagina out, it goes up to the tubes due to construction or whatever, that is why the ovaries are the most common site for ectopic endometriosis, or it goes to Pouch of Douglas which another common site. This is the closest theory)

CLASSIFICATION:

1-internal endometriosis:

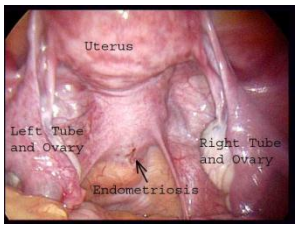
known as adenomyosis, which is endometriosis of the myometrium.

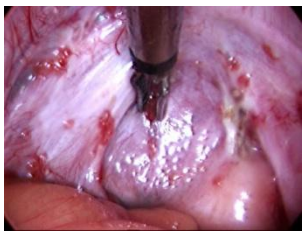
2- external endometriosis:

The commonest site ofextrauterine-endometriosis is the ovary (75% of cases) and the next common site is theperitoneumof Douglas pouch.

SITES:

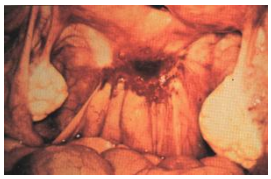
A. Pelvic Endometriosis:





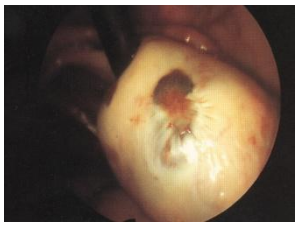
- Uterine body, in the myometrium or perimetrium
- cervix
- tubes
- ovaries
- pelvic peritoneum
- theuterosacral and round ligaments
- rectovaginal septum
- urinary bladder and ureters
- rectum and sigmoid colon
- vagina
- vulva.

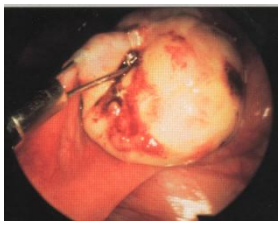
- Adhesions may obliterate the rectovaginal pouch fixing the uterus in retroversion, or there may be adhesion of the pelvic colon to the posterior surface of the uterus and the Pouch of Douglas Adhesions causing problems to the pt like infertility.



Ovarian Endometriosis:

The ovary is a common site for endometriosis and may present either with superficial lesions or the more classic endometrioma or endometriotic cyst (chocolate cyst) Looks like chocolate because it's collection of blood, and the color change to look like chocolate because it's not a fresh blood.

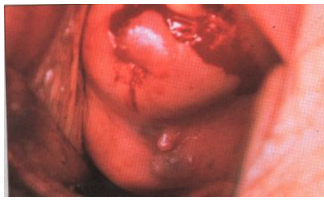




B.Extrapelvic Endometriosis:

- Umbilicus
- abdominal scar as after caesarean section
- abdominalvisceraas gallbladder or appendix.
- Actually endometriosis can occur anywhere in the bodyeven in the limbs. Like in eyes and the pt tear blood, or lung and the pt cough blood, limb or scar.

Extrapelvic Endometriosis*:* Endometrium has been identified in almost every organ in the body
- The picture is for Endometriosis involving the posterior fornixDr. Meshaal had many pt having endometriosis in the P fornix.



Posterior fornix

Risk factors:

- Hyperoestrinism.
- Delayed marriage and infertility. sometime we advise the pt to get pregnant so the tissue get atrophied, and to prevent recurrence, this is why delayed marriage & infertility could be a risk factor.
- Cervical obstruction
- Hysterosalpingographyand curettage. That is why it's done for the pt 2-3 days after menstrual cycle.

Presentaation:

GYN Train: (Kaplan)

Endometriosis

- Chronic pelvic pain
- Painful intercourse
- Painful bowel movement

- Dysmenorrhoea (2ry):progressive (crescendo dysmenorrhoea).
- Chronic pelvic pain and backache
- Dyspareunia
- Infertility.
- acute abdominal pain.
- Dysuria (on the bladder), dyschezia(on the rectum), cyclic haematuria and rectalbleeding during menstruation.

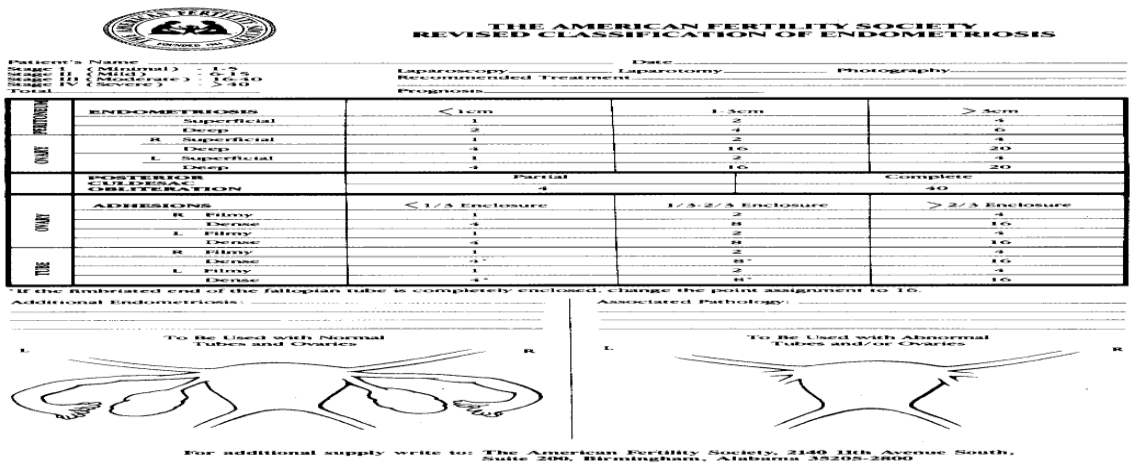
INVESTIGATIONS:(remember US always but it not definitive, like you can see hemorrhage on US and it looks like chocolate cysts)

The gold standard for diagnosis of pelvic endometriosis is ***laparoscopy***. Visual inspection by laparoscopy has increased the awareness of the multiple, subtle and typical appearances of peritoneal endometriosis

* (you don't have to know the staging neither the staging. It's important for doctors to know the response to treatment) the schedule is not being done by the general OB/GYNs, maybe it specialize clinics.

The laparoscope is used to classify the disease into 4 stages - Stage I (minimal); stage II (mild); stage III (moderate); and stage IV (severe). This is the classification of the American Fertility Society and is done before starting therapy and to follow the response to treatment.

The American Fertility Society Revised Classification Of Endometriosis:



THE AMERICAN FERTILITY SOCIETY
REVISED CLASSIFICATION OF ENDOMETRIOSIS

Patient's Name | Date
Stage I (Minimal) : 1-5
Stage II (Mild) : 6-15
Stage III (Moderate) : 16-40
Stage IV (Severe) : 41-80
Total
Laparoscopy | Laparotomy | Photography
Recommended Treatment
Prognosis

PERITONEUM	ENDOMETRIOSIS	≤ 1cm	1-3cm	> 3cm
	Superficial	1	2	4
	Deep	2	4	6
OVARY	R Superficial	1	2	4
	Deep	4	16	20
	L Superficial	1	2	4
	Deep	4	16	20
	POSTERIOR CULDESAC OBLITERATION	Partial		Complete
		4		40
	ADHESIONS	≤ 1/3 Enclosure	1/3-2/3 Enclosure	> 2/3 Enclosure
OVARY	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4	8	16
TUBE	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4	8	16

If the fimbriated end of the fallopian tube is completely enclosed, change the point assignment to 16.

Additional Endometriosis | Associated Pathology

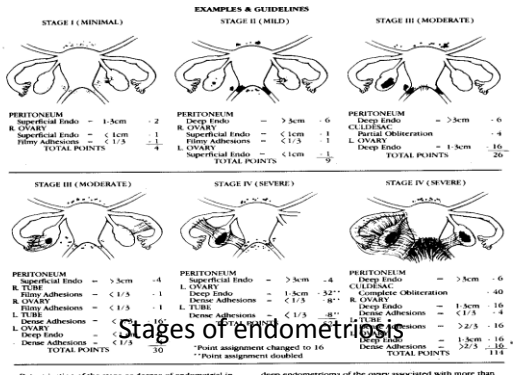
To Be Used with Normal Tubes and Ovaries | To Be Used with Abnormal Tubes and/or Ovaries

L R | L R

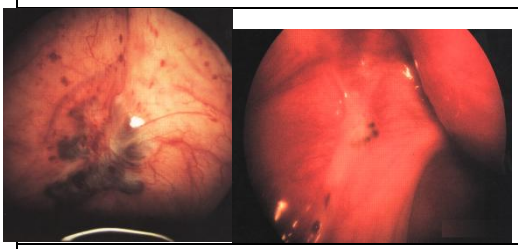
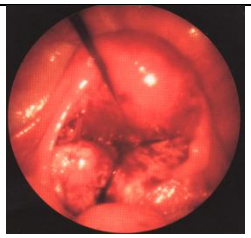
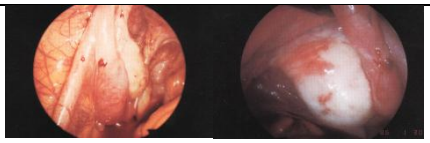
For additional supply write to: The American Fertility Society, 2140 11th Avenue South, Suite 200, Birmingham, Alabama 35205-2800

American Fertility Society Classification (1979):

- Stage I (Mild) 1-5.
- Stage II (Moderate) 6-15.
- Stage III (Severe) 16-30.
- Stage IV (Extensive) 31-54



Stages of endometriosis

Classical Lesions		
		
Classical lesions:"Powder-burn", puckered black. Some pthave frozen pelvic, when you open the pt up, everything is adhisions to each other, you can't see anything neither operate.	Bilateral ovarian endometriosis, adherent to each other and posterior uterine wall "kissing ovaries"	Tubal endometriosis

Subtle lesions: (pictures in the slides)

- Vesicular
- Sacular
- Haemorrhagic
- Papular.
- Nodular.
- Discolored: Yellow, brown, White and Blue.
- Peritoneal defects
- Cribriform peritoneum.
- Subovarian adhesions.

Other investigations:

- Ultrasonography
- Serum CA-125 (not very specific to endometriosis, could be high my menstrual cycle or cancers, but you may use it for treatment follow up)
- Cystoscopy, proctoscopy orsigmoidoscopy may be needed to diagnose endometriosis of the bladder or bowel.
- Magnetic resonance imaging. (for extra-pelvic)

Management:

1. No Treatment:

Small symptomless lesions require no treatment, but the patient is kept under observation and examined every 6 months. Sometimes, the lesions become inactive after a time.

II. Nonhormonal Treatment:

- Indicated for small lesions with mild symptoms.
- Analgesics are given, for pain.
- Prostaglandin inhibitors are given for pain and menorrhagia.Because the condition improves as a result of pregnancy, young women are encouraged to conceive. During pregnancy the ectopic endometrium is changed intodecidua followed by atrophy of the glands.

III. Hormonal Treatment:

Indications: (in non surgical cases, but conditions like chocolate cyst managed by surgery)

- Severe symptoms with small pelvic lesions, lesions more than 2 cm in diameter respond poorly to hormone therapy. (for smalls lesions)
- Recurrence of symptoms after conservative surgery.
- May be given for a short time (6-12 weeks) before surgery to make dissection easier. (like what we do with neoadjuvant chemotherapy)
- After conservative surgery to allow any residual lesion to regress.
- When operation is contraindicated or refused by the patient.

1.Pseudopregnancy: (Is one of the best to do)

Aim of treatment:Ovulation and menstruation are inhibited for 9 months (6-18 months) using a combined oral contraceptive or a progestogen alone to avoid the oestrogenic side effects. The endometrium will undergo atrophy during the pseudopregnancy state.

2. Pseudomenopause:

Aim of treatment: The hormone cause amenorrhoea and endometrial atrophy. It included:

A-Danazol

B-Gestrinone

C-A gonadotrophin releasing hormone analogue

D- Gossypole

IV. Surgical Treatment:

It is indicated for large lesion when hormonal therapy fails.

Surgery is conservativ or radical

1. Conservative Surgery:

In young patients below 40 years the aim of operation is to remove all areas of endometriosis leaving behind healthy ovarian tissue

2. *Radical Surgery:*

When the patient is above 40 years the treatment is total abdominal hysterectomyand bilateral salpingo-oophorectomy.

V. Radiological Treatment:

- Induction of artificial menopause by external pelvic radiation cures the conditionby causing atrophy of endometrial tissue.
- It is applied only in patients above 40 inwhom operation cannot be done as in case of wide spread pelvic endometriosis(frozen excise surgically. or endometriosis of the rectovaginal septum which is difficult to excise surgically.)

Adenomyosis

It is uterine endometriosis in which endometrial glands and stroma are found within the myometrium (the diagnosis is hard, the uterus is just enlarge, no typical presentation. The defined diagnosis is by histopathology)

PATHOLOGY:

- Adenomyosis may be diffuse or localized. In the diffuse type the uterus is slightly symmetrically enlarged and firm. It rarely exceeds the size of 12 weeks pregnancy. Occasionally, there is a localized area of endometriosis causing irregular enlargement of the uterus. On cut section the myometrium is thickened and shows a whorled appearance like that of a myoma but without a capsule.
- The presence of endometrial glands leads to proliferation of muscle and connective tissue fibres.
- Small darkbrown spots are seen between the musclefibres which are endometrial glands distended with blood.
- Sometimes the endometrial glands do not contain blood as the lesion arises from the basal endometrium which does not always respond to ovarian hormones due to lack of progesterone receptors.
- The cavity of the uterus is enlarged and the endometrium is thick and hyperplastic

- asymptomatic
- Menorrhagia
- Dysmenorrhoea. It is a special typeofdysmenorrhea whichisprogressive (crescenddysmenorrhoea).However, special type of dysmenorrhoemaybe absent in some case of adenomyosis.

INVESTIGATIONS:

- Ultrasonography
- Magnetic resonance imaging. It can give accurate diagnosis.
- Histological examination of the uterus after hysterectomy is the only sure diagnostic method.

Presentation:

- CLINICAL PICTURE
- Age. Most cases are seen in patients aged 40-50 years.
- Parity. Most of the cases (80%) are parous women.
- Social and economic state. More common among the lower classes.
- Associated lesions. Fibroids (in 50% of cases), endometriosis in other sites (10%)and endometrial hyperplasia

Kaplan:

- The most common presentation is diffuse involvement of the myometrium.
- In most cases, the diagnosis is made clinically by identifying an enlarged, symmetric, tender uterus in the absence of pregnancy.

Treatment:

- Medical treatment: Analgesics for dysmenorrhoea. Antiprostaglandins improve both dysmenorrhoeaandmenorrhagia .
- Severe menorrhagia is treated by dilatation and curettage.
- Gonadotrophin releasing hormone analogues lead to amenorrhoea and decrease in uterine size. However, the effect is temporary and the uterus returns to its original size with the same symptoms after cessation of therapy.
- **Hysterectomy is the definite treatment.**

Summery

- Dysmenorrhea is two types, primary and secondary. **Primary**does not have pelvic pathology while **secondary** is secondary to pelvic pathology as endometriosis, chronic pelvic infection or endometrial polyps.
- Secondary dysmenorrhea most common causes are ensometriosis and adenomyosis.
- Premenstrual tension syndrome symptoms are temporarily related to menstruations.
- Endometriosis means the presence of endometrial tissue (glands and stroma) in abnormal sites, that is outside the normal uterine cavity. This ectopic endometrium responds to the ovarian hormones as the normal endometrium.
- Endometriosis is two types, internal endometriosis which is known as adenomyosis, and external endometriosis.
- The commonest site for external endometriosis is the ovary (75% of cases) and the next common site is the peritoneum of Douglas pouch.
- In endometriosis, patient could present with dysmenorrhoea (2ry):progressive (crescendo dysmenorrhoea).
- The gold standard for diagnosis of pelvic endometriosis is laparoscopy.
- Adenomyyosis is uterine endometriosis in which endometrial glands and stroma are found within the myometrium.
- Magnetic resonance imaging can give accurate diagnosis of adenomyyosis.
- Hysterectomy is the definite treatment of adenomyyosis.

1-Dysmenorrhea

1ry

-painful menstruation in the first and second days of the cycle .

-no pathology underplaying.

2ndry

Painful menstruation because of diseases

Causes:

1-Endometriosis 2-adenomyosis

3-PID 4-POLYPS

5-uterine anomalies

Adenomyosis

Endomatrium tissue inside the myomatruim of the uterus .

Clinical presentation :

1-symmtrical enlargement of the uterus

2-tender uterus

3- absence of the pregnancy

4-cresendo dysmenorrhea

investigation:

1-us

2-MRI (accurate)

3-histology after hysterectomy (definite)

Endometriosis

Endomatruim tissue outside uterine cavity

Clinical presentation :

1-painful menstruation- pelvic pain

2-dysparenunia – infertility

3- abdominal pain

4-dysuria-dyschiza

Site:

-intrapelvic: ovaries (most common site)

-extrapelvic: anywhere

Risk factors:

1-hyperestinism

2-delayed marriage

3- cervical obstruction

4- HSG

investigation :

1-laproscopy: (best) powder burn , kissing ovaries

2-US: chocolate cyst (endometrioma)

3-serum CA-125

4-MRI: for extra pelvic tissues

Management :

1-**medical :**

a-hormonal: pseudopregnancy(OCP for 9 months) and pseudomenpause(GnRH analogue) leuprolid

b-non hormonal : prostaglandin antagonists

2-surgical :

a-conservative: if patint < 40 years

b-radical: if patient >40 years

3- radiological : if wide adhesion and patint >40 years

2-pre-menstrual tension syndrome

Recurrent changes during leutal phase of menstrual cycle combined with 1-behaviral 2-physilogical 3- psychological changes result in loss of work or interfere with social activates.

Clinical presentation :

1-brest tenderness

2-psychatric , mood changes

3- GI symptoms.

Management :

1-non medical : reassurance

2- medical :

A-inhibit ovulation: OCP

B-brest tenderness: Danazole

C-inhibit the P-H-overian axis :GnRH agonist

D- Dysamenrhea:NASID

E-antidepressnt : Fluoxtine

3- surgical : Bilateral Oophorectomy , hysterectomy.

Amenorrhea.

Definitions:

- What is 1ry amenorrhea?
  - Lack of the onset of menses by the age of 16 years in a female ♀ with 2ry sexual characteristics(A girl who has normal pubertal development)
    Or
  - By the age of 14 years in ♀ without 2ry sexual development
- What is 2ry amenorrhea?
  - Cessation of menses for a period of 6 months in a female ♀ who previously had initiation of menses(A girl who has no pubertal development)
    Or
  - For three previous cycle intervals

1ry amenorrhea Classification

Classified into for types depending on whether there is secondary sexual characteristics manifested by:

- Breast development
- Uterus

a) Breast absent uterus present

(No breast development but normal uterus, and gonads didn't form normally)

GONADAL DYSGENESIS:

1. Turner syndrome 45XO
   Streak gonads (type of gonadal dysgenesis, ovaries didn't't form normally)

- Features:
  - 1ry amenorrhea
  - No breast development
  - Normal female ♀ genital organs (external /internal)
  - Streak gonads (ovaries are replaced by white nonfunctioning tissue)(Ovaries didn't develop normally so they don't function normally)
  - Short stature
  - Webbed neck (Short broad neck) with a low hair line
  - Cubitusvulgus(Wide carrying anglewith the arm extended at the side and the palm facing forward, the forearm and hand are held at greater than 15 degrees)
  - Shield chest / Widely spaced nipples
  - High arched palate
  - Short 4th metacarpal
  - Coarctation of the aorta or VSD(Do an echocardiogram)
  - Horse shoe kidney or single kidney
  - Lymphedema

  (They might also have other congenital malformations so once the patient is diagnosed we have to look for other things)

- Variations of Turner's syndrome⇨(When it's a mosaic turner case they might not have all the previous features)

2. Mosaicism XO/XX(Two cell lines)⇨ not always short
(Mosiac type of Turner's syndrome, they don't always have the typical manifestation of turner syndrome)

- They will have menses, get pregnant then⇨ develop premature menopause

3. Structural abnormalities of the X chromosome

- Deletion of the short arm of the X chromosome⇨ Short stature
- Deletion of the long arm⇨ normal HT, 2ry Amenorrhea, streak gonads

4. Pure gonadal dysgenesis 46XX
(Normal chromosomal configuration 46XX, it's just that the gonads didn't develop normally)

- Mutation in an autosomal gene ⇨ Accelerated germ cell loss ⇨ Streak gonads
- Female♀genetalia , normal Mullerian structures
- Rarely Turner's Stigmata

5. Pure gonadal dysgenesis 46 XY
(If the patient is an XY patient with streak gonads we have to do gonadectomy due to the risk of malignancy)

- Female ♀ genitalia
- Streak gonads ⇨ ↑ risk of malignancy
- Normal Mullerian structures
(Testis secretes the mullerian inhibiting factor which will inhibit the development of the mullerian duct, but if there is dysgenesis of the testis and it doesn't function normally → no secretion of mullerian inhibiting factor → even if XY uterus will develop and there will be female external genetalia)

6. 17-α hydroxylase deficiency (rare)

- ↓Ovarian synthesis of estrogens ⇨ 1ry Amenorrhea
- Sexual immaturity (No breasts)
- ↓Cortisol⇨ ↑ ACTH
- ↑ Na ↓K ↑ BP
- ↑ Progesterone as it is not converted to cortisol

7. Galactosaemia (rare)

- Galactosaemia is toxic to oocytes

HYPOTHALAMIC FAILURE:

8. Isolated GnRH deficiency (Kallman's Syndrome)(Congenital hypothalamic failure)

- Anosmia &HypogonadotropicHypogonadism
- X linked → Mutation in the KAL gene
- More common in male ♂>Female ♀
- Midline defects ⇨ Cleft lip & Palate
- Somatic defects ⇨ color blindness, renal agenesis, retinitis pigmentosa, neurosensory deafness
- Lack 2ry sexual characteristics & the ability to smell (anosmia)
- Height & bone age appropriate for age

9. Hypogonadotropic Amenorrhea
   - Hypogonadotrophic means low FSH & LH (no breast development)
   - Such as in kallman's syndrome (but one is congenital the other is acquired)

- Occurs with CNS tumors ⇨↓GnRH pulses ⇨↓ LH & FSH ⇨↓ estradiol
- Hypothalamic Lesions ⇨Craniopharyngioma, granuloma, aqueduct stenosis, & the sequelae of encephalitis (Following encephalitis)
- CNS tumor ⇨ interfere with the –ve feedback of Dopamine on Prolactin ⇨ ↑ Prolactin
- Other causes of HypoGonadotropic Amenorrhea ⇨ hypothyroidism, PraderWilli& Laurence Moon Biedl syndromes

10. Anorexia Nervosa, Malnutrition, Excessive Exercise & Chronic Illness
    (All these can have an effect on the hypothalamus; the patient is normal, congenitally has no problem or tumors in the CNS but has other problems)

- Functional GnRH deficiency
- May present with or without Breast development(Depends when the problem started)
- Physical stress delay menarche
- Each year of athletic training before menarche delayed menarche 5 months
- Osteoporosis could occur with prolonged periods of Amenorrhea, low body Weight (Due to low estrogen)

b) Breast present, uterus present

1. HYPOTHALAMIC CAUSES
   - CNS lesions (tumors)
   - Stress, Excessive exercise & low body Weight
2. PITUITARY CAUSES
   - Hyperprolactinemia
   - Hypothyroidism ⇨ ↑ TRH ⇨ ↑ prolactin
3. OVARIAN CAUSES
   - PCO (polycystic ovarian syndrome)
4. OUTFLOW TRACT OBSTRUCTION
   - Imperforate hymen
   - Transverse vaginal septum

c) Breast present, uterus absent

(Normal breast development but no uterus)

1. Testicular feminization/ Androgen insensitivity
   - XY Karotype⇨ produce MIF (testis is there) ⇨Mullerian structures are absent
   - Complete/ Partial absence of androgen receptors
   - X linked recessive or dominant
   - Female external genitalia with Short blind vagina(No uterus)
   - Testosterone ⇨ normal male ♂ range
     No androgen receptors in their body → high level of androgen in the body (they look normal but no hair growth & no uterus → no menstruation)

   - Breast development due to peripheral conversion of androgens(testosterone) to estrogens
   - Sexual hair is absent due to absence of androgen receptors
   - Gonadectomy after puberty ⇨Due to the ↑ risk of malignancy (gonadoblastoma, dysgerminoma

2. 5 αreductase deficiency

- Autosomal recessive
- Formation of the male ♂ external genitalia requires

5α REDUCTASE

Testosterone ⇨⇨⇨⇨Dihydrotestosterone

There are androgen receptors but the enzyme that converts testosterone is absent	Active compound which acts on the external genetalia to give a normal male genetalia

- Formation of the internal wollfiane structures respond directly to testosterone
- External genitalia female ♀ with mild masculinization
  Ambiguous genetalia (depends on the level of the hormone deficiency)
- Absent uterus(Due to testosterone secretion)
- At puberty ⇨↑ testosterone secretion ⇨virilization

3. Mullerian Agenesis/ Mayer – Rokitansky-Kuster-Huser syndrome

- Etiology?Not known
- Failure of mullerian duct development ⇨ absence of the upper vagina, cervix & uterus (uterine remnants may be found)
- The ovaries & fallopian tubes are present(Absence of uterus cervix and upper vagina)
- Normal 46XX ♀ with normal external genitalia(Normal female with developmental problems)
- Patient present with 1ry amenorrhea
  - Normal ovaries but no uterus to menstruate
  - Ovaries secrete estrogen → normal breast development
- 47% have associated urinary tract anomalies(Such as absent kidney)
- 12% skeletal anomalies

  Treatment:

  ⇨ Psychological counseling

  ⇨Surgical

  - Vaginoplasty(due to short vagina)
  - Excision of uterine remnant (if it hasfunctioning endometrium)
  - Vaginal dilators

d) Breast absent, uterus absent

- The least common presentation of 1ry Amen
- All Patients are 46 XY
- Testosterone ⇨↓ or Normal
- FSH/LH ⇨ ↑

1. 17-20 Desmolase Deficiency

- The enzyme required for the synthesis of Androgens ⇨↓ Androgens ⇨↓ estrogen
- The testes produce MIF therefore no Mullerian structuresNo uterus or breast (no formation of androgen or estrogen)
- Male ♀ external genitalia (Doesn't require the presence of estrogen)
- Insufficient estrogens for breast development

2. 17 α Hydroxylase Deficiency
   - Similar to 17-20 desmolase deficiency
   - Cortisol synthesis also ↓⇨ ↑ BP, hypernatraemia&hypokalaemia
3. Agonadism
   - Degeneration of the testes (in utero) after the production of the MIF

   Similar to dysgenesis but the problem occurred after the testis started secreting MIF

Investigations and treatment of 1ry amenorrhea:

We do History & Physical examination to place the Patient in one of the four categories

a) ***Breast absent uterus present***

We begin by checking the gonadotropins

1. Hypogonadotropic↓↓**FSH&LH** → CNS/Hypothalamic Disorder:
   - Kallman's syndrome(Congenital defect, isolated GnRH deficiency)
     →Treatment: Estrogen Progesterone replacement (HRT)
     GnRH deficiency (it is hard to replace GnRH because in the body it is secreted in pulses so giving it continuously will not stimulate the production of FSH&LH)
     →Normal treatment for kallman's syndrome is hormone replacement but if a patient wants to get pregnant gonadotropins in the form of injections to induce ovulation
     → Occurs once treatment is established:
     - Breast development
     - Menses
     - Improve bone Density (Without the treatment → osteoporosis)
   - ↓Decreased weight (anorexia), ↑ increased exercise/stress(Excessive → such as Olympic trainers >7-8 hours a day training)
     Treatment:
     → Psychiatric help
     → Treat the cause
     → Estrogen Progesterone replacement (HRT)
   - ↑ TSH with Normal prolactin→ Hypothyroidism
     →Treatment: Give Thyroxin
   - Normal TSH with ↑ increased or normal prolactin→ CT/MRI of the head → CNS tumors (pituitary adenoma → macro or micro)
     → Treat Accordingly

2. Hypergonadotropic↑↑ **FSH&LH**
   - Gonadal Dysgenesis:
     (Gonads aren't secreting estrogen or androgen → elevated FSH trying to compensate for that loss)
     - Investigate by Karyotyping
       - XO → Turner's Syndrome
         →Treatment: Estrogen Progesterone replacement (HRT)
       - XY →Treatment: Gonadectomy then → Estrogen Progesterone replacement (HRT)
         - Agonadectomy is done because testosterone converts into estrogen which increases the risk of malignancy

- Hormone replacement therapy HRT (we give them progesterone, because the uterus is present & estrogen can't be given alone or endometrial hyperplasia might develop)

- XX →↑ Na ↓ K (measure electrolytes), ↑ Progesterone →17a Hydroxylase deficiency

Treatment:

→ Replace the hormone deficiency

→ Estrogen Progesterone replacement (HRT)

Occurs in all the previous three karyotypes once treatment is established:

- Breast development
- Menses
- Improve bone Density (Without the treatment → osteoporosis)

b) Breast present, uterus present

We begin by doing a hormonal profile

1. ↑ TSH with Normal prolactin → Hypothyroidism
   →Treatment: Give Thyroxin
2. Normal TSH with ↑ increased prolactin (hyperprolactenemia) → CT/MRI of the pituitary
   →Asses if there is a pituitary adenoma (macro or microadenoma→ treat according to case)
   → Medication: Bromocriptin or cabergoline
3. Normal TSH and Normal prolactin
   If both normal do a Progesterone challenge test → we give the patient progesterone (Primolut N 5mg a twice a day for 5 days), after stopping one of two will occur:

- + Positive Progesterone Challenge(after stopping the progesterone if she has normal outflow tract and normal estrogen level menstruation will occur)→amenorrhea occurred due to Anovulatory cycle (No progesterone in the body → no period)
  →Treatment: Progestin D16-25 (given in the second half of the cycle)
- - Negative Progesterone Challenge (causes)
  - ↑ FSH→Ovarian failure(Premature)→ Karyotyping (To make sure there is no XY or we will have to do gonadectomy)
    →Treatment: Estrogen Progesterone replacement (HRT)
  - Outflow tract obstruction
    → Treatment is surgical
    → She might have imperforated hymen or Transverse vaginal septum(open the membrane and she will menstruate)
  - ↓ FSH →Hypothalamo/pituitary axis failure → MRI/CT of the head → CNS Tumor
    →Treatment: Estrogen Progesterone replacement (HRT)

c) Breast present, uterus absent

- No Uterus but normal secondary sexual characteristics
- We begin by checking the gonadotropins

1. ↑ Testosterone → in the normal male range

- Karyotyping → XY (most likely)Testicular feminization/androgen insensitivity
  - U/S Pelvis (To make sure they don't have a uterus)
  - U/S MRI gonad →Gonadectomy

Treatment:

→Estrogen Progesterone replacement (HRT)
→Gonadectomy
→ Surgical: Vaginoplasty or vaginal Dilators (to create a vagina)

2. ↓ Testosterone → in the normal female range

- Karyotyping → XX Mullerian agenesis
  - U/S Pelvis and Kidney
  - IVP

Treatment:

→ No Need for Gonadectomy
→Estrogen Progesterone replacement (HRT)
→ Surgical: Vaginoplasty or vaginal Dilators (to create a vagina)

d) Breast absent, uterus absent

All are 46 XY

- Physical exam, U/S and MRI to lock for gonads

Treatment:

→Estrogen Progesterone replacement (HRT)
→Gonadectomy

2ry amenorrhea

Definition:

Cessation of menses for a period of 6 months or 3consecutive menstrual cycles in a Female♀ who previously had initiation of menses

Prevalence of Amenorrhea: 1.8-3%

Classification of 2ry amenorrhea:

- Hypergonadotropic(High FSH&LH) →CNS / Hypothalamic
- Hypogonadotrpic(Low FSH&LH) → Pituitary
- Euogonadotrpic(Normal FSH&LH) →Ovarian
- Anatomic defects →Outflow obstruction Uterine, Cervix, Vaginal]
- Hperprolactinemia

a) HypogonadotropicAmenorrrhea

1. Stress→↑ β-endorphins →↓GnRH→↓FSH &↓LH →↓Estrogens

2. Exercise (Excessive strenuous exercise) Runners & Ballet dancers]
   - Mechanism is similar to stress

   Treatment:

   →↓ In training intensity to a level where regular menses resumes

   → HRT ⇨ Cyclic estrogen / progesterone

   - Premarin 1.25 mg continuously
   - Medroxyprogestrone acetate 5 mg /D for 12 D each cycle

   ⇨ OCP ⇨ better compliance

3. Weight loss "Anorexia nervosa"
   - More frequent in adolescent & young adults
   - 0.5-1% of women aged 15 –30 years
   - To diagnose their body weight has to be15% < Ideal body Weight

   Treatment:

   → Psychiatric treatment:

   → Meanwhile ⇨ HRT

   → Long term follow up → due to Frequent relapses after attaining ideal body Weight

4. Functional "Non of the above causes"(not related to the previous causes)

   No LH pulses or Persistent pulse frequency of "luteal phase " → due to 2ry to neurotransmitter abnormality of the CNS (? ↑Opioid activity)

   Treatment: HRT / ovulation induction (If they want to get pregnant)

- Is it of any concern if these young women become amenorrheic?
  No menses due to Hypoestrogenism which is the main concern→ can lead to osteoperosis
- Why is it more worrying in the menopausal women?
  During adolescence estrogen plays a critical role in determining PEAK BONE DENSITY, which is reached, in the 2nd decade of life(If there is amenorrhea she will be deprived of reaching the peak bone density)
- Is there any evidence of its effect on the bones?
  - Amenorrheic Athletes ⇨↓↓ Bone Mineral Density (BMD) in lumbar spines, femur, tibia
  - Athletes with menstrual irregularities ⇨↓ BMD < athletes with regular cycles
  - Anorexia nervosa Patients ⇨↓ BMD (0.64) < Normal controls (0.72)
  - Anorexia nervosa Patients may have osteoporotic fractures

5. Shehan's Syndrome

- Pituitary failure ⇨ following severe post partum hemorrhage
- Deficiency of all pituitary hormones
- ↓FSH & LH ⇨ Failure of ovarian follicular development ⇨↓ estrogen ⇨ Amenorrhea
- Treatment⇨ HRT
  ⇨hMG for ovulation induction(If the patient wants to get pregnant).

b) EuogonadotrpicAmenorrrhea

Polycystic ovary syndrome (PCO)

- Amenorrhea → due to anovulatory cycles
  Enlarged polycystic ovaries(Usually described as necklace appearance on ultrasound as there is multiple cyst in the ovaries)
  Treatment:
  → Cyclic progestogen
  → Oral contraceptive pills (OCP)

  } They protect the Endometrium, regulate the cycle, and ↓menorrhagia
- Infertility(Not ovulating)
  Treatment:
  →Ovulation Induction by oral therapy: Clomid→ Ovulation 70% & Pregnancy 40%
  (If it fails we go to injection (gonadtropins injections → stronger)
  →hMG
  →Surgical Treatment: Ovarian Drilling (through laproscopy, holes are made in the ovary wall)→ Ovulation 92% & Pregnancy 70%
- Hyperinsulinemia / Obesity
  Treatment→ Glucophage and weight loss

- Hyperandrogenism / hirsutism
  Either chemical or clinical (androgen level is normal but high free testosterone which results in hirsutism)

Treatment:

→ Oral contraceptive pills (OCP) →↓Decreases Ovarian androgens &↑ increases SHBG (Increase Sex hormone binding globulin therefore decrease the level of free testosterone in the body → decrease hirsutism)

→Antiandrogens

- Sprinolactone
- Cyproterone acetate
- Flutamide

} Bind to androgen receptors&↓Androgens + ↓5αreductase activity

- ↑ LH(High LH in relative to FSH)
- Acyclic estrogen production / unopposed by progesterone →↑ risk of endometrial hyperplasia/Ca(The patient is recommended to use cyclic Progestogen to prevent the development of endometrial hyperplasia)
- Inheritable disorder with a complex inheritance pattern(Can run in families but has no specific gene for it)

c) HypergonadotropicAmenorrrhea

(Increased FSH&LH)

Premature Ovarian Failure (POF)

- 2ry Amenorrhea (menstruating previously)
- ↑ FSH & LH
- ↓Estrogen
- Before the age of 40 years
- Incidence is 1%
- Causes:
  - Unknown / autoimmune / genetic factors
  - Associated autoimmune disease 39% (Can also have hypothyroidism or other endocrine function might be affected)
- Pathological Characteristics:
  1. Ovarian sclerosis & lack of follicles
  2. Resistant ovary syndrome(There are follicles but it doesn't respond to FSH)
- Management:

  → Rule out other autoimmune diseases ⇨ Rheumatic factor, ANA, Antithyroid Antibodies, Antichromosomal Antibodies, glucose, cortisol, Ca , Phosphorus, TSH

  → HRT ⇨ to prevent osteoporosis

  → Spontaneous pregnancy can occur in women with POF on HRT 8%(Sporadic ovulation might occur (doesn't occur regularly)

  →hMG/HCG glucocorticoids have been claimed to give better pregnancy rates (If the patient wants to get pregnant)

d) Hperprolactinemia

- The most common pituitary cause of 2ry Amenorrhea
- Causes
  - Pituitary adenoma
    Treatment:

    →Macroadenoma⇨> 10 mm

    ⇨ Respond to medical treatment⇨ Dopamine agonist (bromocriptin) ⇨↓ size of the tumor &↓ prolactin level

    ⇨ Patient not responding to medical treatment or not tolerating it ⇨ Surgery/ Irradiation

    →Microadenoma⇨< 10mm

    ⇨Remain stable in size⇨ Treatment⇨Bromocriptin⇨↓ prolactin level ⇨ Normalize the menstrual cycle

  - Loss of inhibition by dopamine→ Hypothalamic or pituitary stalk lesions
    →Treatment: Surgical excision

  - Hypothyroidism
    → Treatment: L-Thyroxin → If still amenorrheic after treatment →Parlodel + Thyroxin

  - PCOS
  - Medications⇨phenothiazines , haloperidol monoamineoxidase inhibitors, TCA, H2 receptors blockers
    →Treatment: If no substitute for the medications that cause hyperprolactinemia

    ⇨ HRT

  - Idiopathic
    →Treatment: Dopamine agonist ⇨Bromocriptin or Pergolide

    - Side effects of dopamine agonists
      -Postural hypotension

      -Nausea

      -Headache

      -Nasal stuffiness

      Starting with a low dose & gradually ↑ it helps to avoid the side effects

  Symptoms:

- Galactorrhea⇨ 1/3 of Patients
- Amenorrhea/ HyperprolactinemiaPt⇨ at risk of osteoporosis due to ↓ estrogen

e) Anatomicical causes(Doctor didn't mention it)

- Uncommon cause of 2ry Amenorrhea
- Asherman's Syndrome ⇨ History of D/C for RPOC after abortion/puerperium or previous uterine infection
- Intrauterine Adhesions
- Normal hormones
- -veprogesteronechallenge test
- Diagnosis⇨ HSG / HYSTROSCOPY
- Treatment⇨Hystroscopic resection of the adhesions followed by estrogen therapy.

Summary

Primary amenorrhea

	Causes	Investigation	Rx
Breast - Uterus +	1-**hypothalamus –pituitary axis** Isolated GnRH deficiency (kallman's syndrome) - a b-Hypogonadotropica c-anorxia nervosa, exercise , malnutrition. d-galactosaemia 2-**gondal dysgenesis** : a- turner syndrome 45XO b- Mosaicism XO- XX c-Pure gonadal dysgenesis 46 XX d-Pure gonadal dysgenesis 46 Xy 3- **17 a hydroxylase deficiency**	Hypogonadotropic :if FSH and LH are low that's mean the problem in the hypothalamus pituitary axis not in the ovary. Hypergonadotropic: if FSH and LH are high that means the problem in the ovaries.	HRT hormonal replacement therapy XO>> HRT XY>> gonadectomy
Breast + Uterus +	**1-hypthalamus** : CNS tumors , stress or exercise . **2- pituitary :** hyperprolactinemia , hypothyroidism **3-ovaries**:PCO **4-outflow obstruction :**imperforated hymen ,septum.	-High FSH +normal prolactin = hypothyroidism -Normal FSH+high prolactin = hyperprolactinemia Normal FSH+normal prolactin = do progesterone challenge test	>>>>thyroxine >>>>> bromocriptine
Breast + Uterus -	1-androgen insensitivity XY 2- 5 a reductase deficiency 3-mullirian agenesis 46 XX	>>> high testosterone level >>>low testosterone level	>>>gonadectomy >>>HRT

Breast - Uterus – All are XY	1-17,20-desmolase deficiencies 2-17 hydroxylase deficiency 3-Agonadism	HRT +gonadectomy

Secondary amenorrhea

	Causes	Rx
Hypogonadotropic low GnRH	1-exercise 2-stress 3-anorxia nervosa 4-functional (no LH pulses) 5-shehan syndrome	HRT
Hypergonadotropic high GnRH	Overian failure	HRT
Euogonadotropic normal GnRH	PCO	1-progestrone 2-OCP 3-Treat the symptoms
Hyperprolactinemia	It's the most common cause of 2ry amenorrhea 1-adenoma 2-pco 3-hypothyrodisim 4-medication : dopamine	>10 mm bromocriptine if doesn't respond >>surgery <10mm bromocriptine >>>>L.thyroxine

Abnormal Uterine Bleeding, Menorrhagia.

Normal menstrual cycle:

To say this is a normal cycle you need to ask about these parameters: 1) Duration of the flow 2) Amount of the blood loss "In clinical setting it is a descriptive variable either heavy or light" 3) Pattern "Regular vs. Irregular" It is normally 28 days +/- a week.

The average adult menstrual cycle lasts 28 to 35 days

The first day of menses represents the first day of the cycle (day 1)

Approximately 14 to 21 days in the follicular phase

14 days in the luteal phase (Fixed)

How to know when the ovulation will happen?

You need to have some basic info about the patient menstrual cycle: 1-LMP 2-Pattern of the cycle 3-Expexted date of the next cycle. Then, you subtract 14 days from the expected date. Let's take an example of a woman whose LMP is August 20th. Her cycles are regular with 40 days. Assuming that August and September are 30 days long. Her expected date for the next cycle is October 1st, and the patient will ovulate on 16th of September.

Some Definitions and Facts

-**Abnormal uterine bleeding:** change in the frequency of menses, the duration of flow (>7days), and the amount of blood loss (>80ml). Present in ~10-20% of women >30 y. old

-**Menorrhagia:** heavy or prolonged, but regular bleeding

-**Metrorrhagia:** irregular (and heavy) bleeding, intermenstrual bleeding, spotting, or breakthrough bleeding

-**Menometrorrhagia:** prolonged bleeding at irregular intervals

-**Polymenorrhea:** menstrual interval <21 days

-**Oligomenorrhea:** menstrual interval >35 days

-**Dysfunctional Uterine Bleeding:** excessive uterine bleeding with no demonstrable organic cause (idiopathic); most often endocrinologic in origin

History (Every point is important)

1-Suggestive Symptoms of Heavy Bleeding

-Pads (# of pads/day, using maxi size, using 2 together)

-Presence of clots, socking clothes and /or bed

-Symptoms of anemia

2-Bleeding Pattern

-Regular or not

-Postcoital bleeding

-Intermenstrual bleeding

3-Other Symptoms

-Dysmenorrhea

-Chronic abdominal pain

-Symptoms of hyperandrogenism, hyperprolactenemia , hypothyrodism

4-Past Medical History

-Bleeding tendency

-Medication history

Examination

-**Vital signs** (are important to know if the patient is stable)
-Weight, height and BMI
-General exam: signs of anemia, hairsuitesm
-Abdominal exam (masses, scars and tenderness)
-Pelvic exam: masses, uterine size, tenderness
-Pap smear, endometrial biopsy

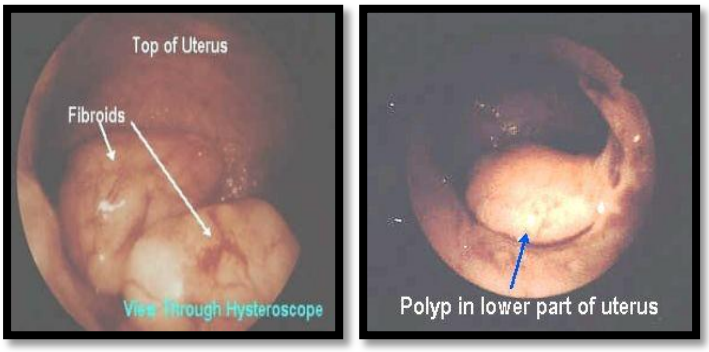
Systemic Causes of AUB

-Disorders of blood coagulation:
- Von Willebrand disease **(adolescence)**
- Prothrombin deficiency
- Carriers of hemophilia
- Factor XI deficiency
- Platelet deficiency (leukemia, severe sepsis, ITP, hypersplenism)

-Hypothyroidism, rarely hyperthyroidism
-Renal failure
-Cirrhosis (hypoprothrombopenia, decreased clotting factors)

Reproductive Tract Disease

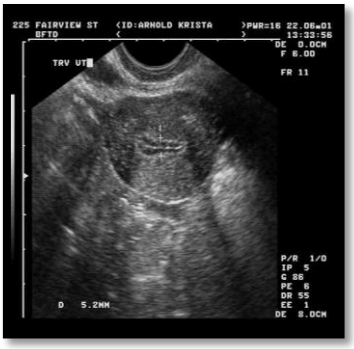
-Anatomic uterine abnormalities: sub mucous myomas (fibroids), endometrial polyps "You really need to read about uterine fibroids and polyps, the doctor mentioned that we will get a separate lecture about them"



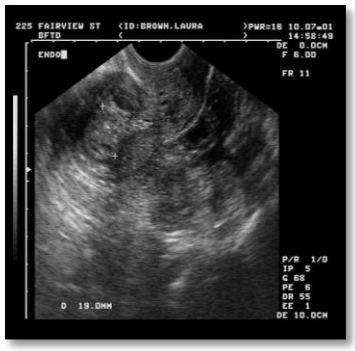
-Adenomyosis "It is simply endometrial tissue growing inside the muscular layer of the uterus" "you should differentiate it from endometriosis, which is endometrial tissue outside the uterus"
-Premalignant lesions: endometrial hyperplasia
-Malignancies: endometrial, cervical, vaginal, vulvar & oviductal Ca, estrogen-producing ovarian tumors (granulosa-theca cell)
-Infection of the upper genital tract endometritis "mainly postpartum"
-Cervical lesions: erosions, polyps, cervicitis (may cause esp. postcoital spotting)
-Traumatic vaginal lesions
-Severe vaginal infections
-Foreign bodies
-Oral and injectable steroids, tranquilizers, antiseizure medications
-Other drugs with estrogenic activity: digoxin, marijuana, ginseng

Investigations

-**CBC** "To know if the patient is anemic"
-Beta HCG "In a sexually active woman, you have to rule out pregnancy"
-**TSH** "Esp. Hypothyroidism"
-Prolactin "Usually it causes amenorrhea, however it may also causes AUB"
-Coagulation studies (Women with systemic disease)
-Von Willebrand disease (Adolescent girls)
-**U/S** is a very important test in Gynecology (uterine size, endometrial thickness, fibroids, polyps)



TV U/S showing the endometrial thickness



This is a Uterine Fibroid

-Day 21, Progesterone, (Luteal phase serum progesterone)
-Daily basal temp
"The goal of both of these tests is to know if ovulation has happened or not"
-Premenstrual sampling of the endometrium (office biopsy, D&C, hysteroscopic biopsy)
Indications for endometrial biopsy in AUB:
1-Every post menopausal woman who is bleeding
2-Women who are older than 40 with Irregular bleeding
3-Women who are younger than 40 but who have high risk for endometrial cancer (ex. PCOS)
-Hysteroscopy (remember: D&C misses the diagnosis in 10-25% of women; ~25% of women with presumptive Dx of DUB have uterine lesions on hysteroscopy)

Dysfunctional Uterine Bleeding

-Diagnosis of exclusion
-Caused alterations in prostaglandin synthesis
-Ovulatory (in up to 10%)
Short or inadequate corpus luteal phase
Often results in menorrhagia and intermenstrual bleeding (BTB)

-Anovulatory "more common, an example is PCOS"
Secondary to alterations in neuroendocrine function
Hypoestrogenic state or chronic unopposed estrogen

Treatment Modalities

-Treatment of DUB

Medical treatment

1-Hormonal Estrogens, Progestins (systemic or Progesterone releasing IUCD) Combined OCs
2-NSAIDs (esp. in ovulatory DUB)
3-Antifibrinolytic agents "Are agents given also in trauma and Post Partum Hemorrhage"
4-Low-dose danazol
5-GnRH agonists

Surgical

1-D & C
2-Endometrial ablation
3-Hysterectomy

-Treatment of uterine fibroid

Medical-same as DUB

UAE uterine artery embolization

Surgical

1-Myomectomy (laparoscopy hysteroscopy or laparotomy)
2-Hysterectomy

Always remember

1-Stabelize the patient first
2-Get IV access
3-Blood group and x-match
4-Treat anemia

Cases "Answers are from doctor"

Case # 1:

14 years old female presents with "heavy periods" never been sexually active generally healthy?

A-What is your DDx?

Von Willebrand disease -DUB

B-What is your treatment?

In the case of DUB, since the patient is young and still her body is adapting to cycles, you reassure the patient.

Case # 2:

38 years old woman with a history of heavy, infrequent (two per year), menses since menarche at age 12

- Spontaneous pulmonary embolism six years ago
  - O/E - Wt. = 150 kg. Ht. = 145 cm
  - Hirsutism involving upper lip, chin, midline chest and abdomen - negative speculum exam, bimanual limited by BMI.

A-What is your DDx?

PCOS-Hypothyroidism

B-What is your most likely diagnosis?

Most likely it is PCOS (High BMI, Hirsutism)

Case # 3:

48 years old obese pt. with oligomenorrhagia---> presents with 6 wk. history of constant bleeding --> now very heavy

- O/E ; Wt = 150kg, vitals stable, pelvic ; non-contributory except bleeding + + +
- Hgb =7, MCV=85

A-What is your most likely diagnosis?

Uterine Fibroids

B-Outline your immediate investigations and treatment

Order CBC, Hormonal Profile, and you might do a biopsy. The treatment is the same as mentioned in the lecture.

Case # 4:

43 years old lady known type2 DM and uterine fibroid presented with heavy regular vaginal bleeding.

A-What is your management?

You start with the medical options; however you have to inform the patient about all available treatments for fibroids.

B- Which investigation you need to do?

U/S

C-Do u need to do endometrial biopsy?

No, since the patient is having a regular bleeding and has a low chance of cancer

PCO + Hirsutism.

Polycystic Ovary Syndrome

❖ Definition:

- ACOG and NIH (1990): Hyperandrogenism(with or without)andchronic anovulation excluding other causes.
- Stein and Levanthal (1935): Association of amenorrhea with polycystic ovaries and variably: hirsutism and/or obesity

❖ Epidemiology:

- Prevalence: 4-6% females. (From observation, it's high in our country due to our lifestyle that causes obesity)
- Probably same world wide
- No difference between blacks and whites
- 75% of women w/ irregularity or infertility

❖ Pathophysiology:Not clear

- "Vicious cycle" PCO > anovulation > hormones disturbance > insulin resistance > obesity. ± hyperandrogenism
- Abnormal gonadotropin secretion
- Excess LH and low, tonic FSH
- Hypersecretion of androgens
- Disrupts follicle maturation. This is the main defect! Ovaries produce follicles but at mid-antral stage of development, they get arrested. That's why we see small cysts in the ovaries.
- Substrate for peripheral aromatization
- Negative feedback on pituitary
- Decreased FSH secreation
- Insulin resistance, Elevated insulin levels

❖ PCO:

- Usually in obese woman
  FSH: LH ratio, in the proliferative(follicular) phase of the cycle will be reversed. That's why we test FSH/LH in this phase (2^{nd}-3^{rd} day of period) because FSH and LH normally go to baseline and if there is any abnormality, it will appear. IMP!!
- ↑ Oestrogen
- Hirsutism±
- Raised level of circulating insulin due to insulin resistance
- Raised blood sugar

❖ Diagnosis:

1. History: Irregular cycle, Oligonorrhoea(scanty period),Infertility, galactorrhoea, recurrent abortions(seen in the recurrent fetal loss clinic).
2. Ex.: Usually obese but it can happen in thin patients ± Hirsutism, acanthosis, acne.
3. Investigations:
   - Hormones - ↑ LH, FSH may be normal, ↑ Oestrogen(because the small follicles still produce it), Free testosterone may be ↑ or normal
   - Ultrasound - multiple small cysts at the periplery of the ovary looks like necklace. Nucleus appearance
   - Laparoscopy – thick, enlarged non-active ovaries

❖ Treatment:

- Weight reduction. Always the 1st line!! Especially if they are young.
- Induction of ovulation either through injections or Clomid (clomiphene).
- Metformin to decrease insulin resistance, however some pts will not benefit from it if they didn't follow a diet.
- Laparoscopic ovarian diathermy. Invasive, not done now, reserved as a laaast option.
- IVF
- Ovarian wedge resection, contraindicated to be performed now because it is associated with severe adhesions and can jeopardize the infertility forever since a part of the ovary is resected.

❖ Associated disorders:

- Diabetes
- Hyperlidpidemia (LDL, Triglycerides)
- Obesity
- Hypertension
- CAD?
- Incr in Risk Factors, but not mortality
- Endometrial CA due to the unopposed estrogen secretion
- Ovarian CA?
- +/- Breast CA
- NO increase in Osteoporosis
- Eating disorders
- Psychiatric disease

That's why in the clinic, there's a package and we test for everything.

In the OSCE, a picture of an ultrasound that shows the nucleus appearance of the ovaries in PCO came last year. Students were asked to identify it and mention some of the associated risks and only few were able to answer so focus!

Hirsutism

❖ Definition: Abnormal hair growth in abnormal areas.

❖ Causes:

- Pathological - PCO, adrenal cortex tumors, Cushion syndrome, medications.
- Constitutional – certain races.

❖ Site: Face, chest, anterior abdominal wall. Excessive hair in arms and legs doesn't count as hirsutism.

❖ Investigations:

- Free testosterone level, ATCH (for adrenal tumors), FSH, LH (for PCO), DHEA.
- Radiology – US for the ovaries and adrenals,..etc

❖ Treatment:

- Difficult α needs reassurance
- Hair removal by different methods
- Diane, contains Cypreterone acetate – anti-androgen. YOU MUST GIVE CONTRACEPTIVE PILLS WITH IT because it can cause teratogenicity.
- Treatment will take long time.

Summary

❖ PCO:

- Hyperandrogenism+ chronic anovulation.
- Usually in obese woman.
- **Dx:**Irregular cycles and infertility, ↑ LH, FSH may be normal, ↑ Oestrogen, free testosterone may be ↑ or normal±Hirsutism.
- Ultrasound - multiple small cysts at the periplery of the ovary looks like necklace (Nucleus appearance).
- **Rx:**Weight reduction, Metformin, Clomid for ovulation induction

❖ Hirsutism:

- Site: Face, chest, anterior abdominal wall
- Rx: Very difficult,Diane (MUST GIVE CONTRACEPTIVE PILLS WITH IT because it can cause teratogenicity).

Infertility.

Fertility - Subfertility – Sterility

Sterility:Absolute and irreversible inability to conceive.
Infertility: Diminished capacity to conceive and bearchild.
Clinically inability to conceive despite regular unprotected sexual intercourse over 12 months
Pregnancy rate:

57%	3 months
72%	6 months
85%	12 months
93%	24 months

- 7% conceive in the second year justifies starting investigation for infertility after one year.
- Female fertility decline after the age of 35 and decline more rapidly after age of 40.
- If women 35 years of age, the investigation should be not be delayed.
- Primary – No previous pregnancy
- Secondary – Previous pregnancy whatever theoutcome

Prevalence: 10 – 15%

- ⅓in the female
- ⅓ in the male
- ⅓ in the couple combined

Disorders:important

- Involving each of the major physical events that are necessary to produce a pregnancy.
- Production of healthy eggs.
- Production of a healthy sperm.
- Transportation of the sperm to the site of fertilization.
- Transportation of the egg and zygote to the uterus for implantation.
- Successful implantation in a receptive endometrium
- Presence of other factors.

Causes of infertility

For pregnancy to occur there must be fertile sperm and egg, a means of bringing them together and a receptive endometrium to allow the resulting embryo to implant. A defect at any of these stages can lead to subfertility.

Commonest causes in the female:

- Ovulatory factor
- Tubal factor
- Endometriosis
- Failure of implantation
- Uterine factor

No oocyte production and oocyte abnormalities:

- Failure to ovulate
- The disorders are grouped into three general categories:
  - Hypothalamus
  - Pituitary
  - Ovarian Dysfunction

Anovulatory Infertility:

- Hypergonadotrophic hypogondism – failure of the ovary to respond to gonadotrophic stimulation(very high FSH) by the pituitary gland result from premature ovarian failure and exhaustion of the ovarian follicle pool.
- Resistant ovary syndrome – elevated gonadotropin in the presence of good reserve follicle due to abnormalities of FSH receptors.
- Hypogonadotrophic hypogonadism
  - Pituitary dysfunction:failure of pituitary gland to produce gonadotropin will lead to lack of ovarian stimulation due to destruction by:
    - Pituitary tumor (adenoma)
    - Pituitary inflammation (TB - Ischemia as in Sheehan's Syndrome)
    - Pituitary damaged by radiation or surgery
    - Hypothalamicdysfunction: if pulsatile secretion of GnRH is slowed orstops (easy to treat: only give GnRH) secondary to:
      - Excessive exercise.
      - Psychological distress.
      - Anorexia nervosa.

To differentiate use biopsy:

- Hypergonadotrophic hypogonadism: no follicles
- Resistant Ovary Syndrome: follicles are present.

However this is not necessary since the management is the same.

Ovarian Dysfunction: Polycystic ovary syndrome (PCO)

Endocrine disorder: hyperprolactinemia

Hypothyroidism

Tubal infertility:(anything that causes adhesions or blockage of tubes)

- BID – Chlamydia trachomatis
- Pelvic infection or abscess from appendicitis
- Septic abortion
- Pelvic surgery.Ectopic pregnancy.
- T.B.
- Crohn's disease
- IUCD

Endometriosis: Severe form can lead to tubal damage due to adhesion formation caused by endometrial deposit

Uterine Factors:

- Sub mucous fibroid – occlude tubes
- Congenital uterine abnormalities
- Intrauterine adhesion due to excessive curettageAsherman's syndrome

Unexplained infertility:

Complete of routine investigation fail to reveal cause in 15-30% of cases does not indicate absence of a cause but rather inability to identify it. The result of IVF shown there may be undiagnosed problems of oocytes or embryo quality or of implantation failure neitherof which can easily be tested unless IVF is undertaken.

Anovulation:

- History: Irregular Menses
- Basal body temperature is flat (no mid cycle rise), Progesterone levels are low and Biopsy of endometrium shows proliferative changes (no evidence of progesterone)
- Correctable causes: hypothyroidism and hyperprolactinemia.

Tubal Disease:

- HSG, if normal no further testing
- If abnormal consider Laparoscopy to diagnose and treat tubal disease: tuboplasty OR Salpingectomy and IVF

Source: Kaplan.

Definition of unexplained infertility:

No pregnancy with:

- Normal semen analysis
- Confirmed ovulation
- Patent oviducts

Management:

- Wait for 3 years, 60% of couples achieve pregnancy.
- Ovulation induction and IUI and see if that works.
- If however it doesn't, we try IVF.

IVF indications:

- Severe oligozoospermia
- Irreparable tubes
- Unexplained pregnancy

Source: Kaplan

History and Examination:

Personal & Social History(this was explained as a history directed to the male, though it is not in the proper place in this lecture.)

- Age – female partners
- Occupation especially the male – exposure to high temperature
- Chemical and radiation can affect sperm production
- Works away from home – affect frequency of sexual intercourse around the time of ovulation
- Smoking-Alcohol

Menstrual History:

- Age of menarche and regularity of periods
- Irregularmenstrualcycle,oligomenorrhiaand amenorrhoea are all suggestive of anovulation
- Amenorrhoea – menopausal symptoms
- Weight loss or gain. (PCOS patients have anovulation when there is weight gain).
- Symptomsof hyperprolactinemia and hypothyroidism.

Obstetric History:

- Enquire about previous pregnancies, outcome.
- Breast-feeding and any sustained galactorrhoea. (Hyperprolactinemia causes anovulation)
- Difficulties or treatment required prior to achieving a previous pregnancy. If the patient has had difficulties in a previous pregnancy and was successful using IVF. The next pregnancy → go directly to IVF without trying anything else.

Contraception:

- Use of contraception pills and long acting progesterone followed by amenorrhoea
- Use of long acting progesterone contraception followed by delay in the resumption of ovulation
- IUCD ↑risk of infection – young nulliparous leading to tubal disease (not given to young patients)

Sexual History:

- Frequency of sexual intercourse.
- Ejaculatory dysfunction.

Other important points

- Folicacid to prevent neural tube defects
- Rubellavaccine to prevent congenital rubella syndrome
- Family history – Diabetes, endometriosis, PCO

Examination:

Assessment of body mass index, obesity andunder weight cause anovulation

Investigation: The aim to assess:

- Ovulation
- Tubal patency
- Uterine factors

Under the influence of estrogens, cervical mucus becomes abundant, clear, and stretchable, somewhat like egg white. The stretchability of the mucus is described by its spinnbarkeit, from the German word for the ability to be spun. Only such mucus appears to be able to be penetrated by sperm. After ovulation, the character of cervical mucus changes, and under the influence of Progesterone it becomes thick, scant, and tacky. Sperm typically cannot penetrate it. Wikipedia.

Assessment of Ovulation: important

- History of regular period.
- ↑Level of progesterone in serum approximately 8 days after LH surge (Mid luteal phase) indication of ovulation.
- BBT basal body temperature rises at ovulation.
- Endometrialhistology (secretory phase is an indication of ovulation).
- Cervicalmucus (mucus becomes 'Spinnbarkeit' at ovulation).
- LHdetectionkits (to advise when is the best time to have intercourse).

Tubal Patency Tests:

- HSG.
- Laparoscopy and dye test.
- Falloposcopy – assessment of tubal patency and mucosa.
- Ultrasoundscanandhydrotubation.

Assessment Of The Uterus:

- HSG.
- Hysteroscopy.
- TVU with injection of N/S (Hysterosonography).
- Postcoital test: provides information concerning the ability of the sperm to penetrate and survive in the cervical mucus. Maybe the problem is only in the thick mucus.

Management of Anovulatory Infertility:

- Patients with ovarian failure and resistant ovary syndrome will not respond to ovulation induction and they offered oocyte donation.
- Normalization of body weight in underweight and obese patients can help to regain ovulation without the need for medical intervention.
- Medical treatment of prolactinoma.
- Ovulation induction in patients with hypogonadotrophic hypogonadism with pulsatile GnRH or by gonadotophin.
- Ovulation induction in PCOS patients achieved by weight normalization in obese patients

Ovulation induction medication: (under hospital observation)

- Clomiphene therapy (ant estrogen that feeds back to pituitary to produce more FSH)
- Gonadotropin therapy
- Risk of multiple pregnancy &OHSS(ovarian hyperstimulation syndrome is the most serious consequence of induction of ovulation)

Surgical methods are either ovarian drilling or wedge resection the theory was that the thick tunica albugenia prevented the release of the ovum. Not used anymore.

Disadvantages: tubal damage and adhesion from destruction of the ovarian stroma and reduction of ovarian reserve

Advantages: No risk of multiple pregnancy and OHSS

Management Of Tubal Infertility: Can be treated with tubal surgery, IVF and embryo transfer (IVF-ET) or selection salpingography. Although tubal surgery is no longer recommended for severe tubal disease since the introduction of IVF-ET, it still has a place in less severe forms of the disorder.

Management Of Endometriosis-Related Infertility

- Depends on the severity of the condition and the presence of any other infertility factors. The medical methods are inappropriate in an infertile patient either induce anovulation or teratogenicity
- Conservative surgical treatment of minimal or mild endometriosis may improve natural concption rates postoperayively .Diagnostic laparoscopy and diathermy to endometriosis can be delivered at the same session
- Sever endometriosis for IVF-ET.

Management of Unexplained Infertility:

Conservative management, ovulation induction with or without intauterine insemination, and IVE-ET are the main approaches to managing unexplained infertility. It provides information about fertilization and egg and embryo quality. Owing to its high cost, IVF-ET is usually seen as a last resort in unexplained infertility.

Management Of Uterine Factor Infertility:

- Myomectomy either laparoscopically or by laparotomy
- Entry into the uterine cavity should be avoided if possible, and adhesion barriers and and microsurgical technique to reduce the risk of adhesions
- Hysteroscopy:Resection of submucous fibroids depending on the size of the fibroid and its degree of protrusion into the uterine cavity
- Risk of haemorrhage uterine perforation and endometrial scarring leading to intrauterine adhesions

Male Infertility:

Testes: Under Gnrh

- Steroidogenesis Leydig cells between seminiferous tubule. Testosterone – (LH)
- Spermatogenesis. Sertoli cells (inhibin) – (FSH)
- Both lead to production of healthy spermatozoa.

Cryptoorchidism: Infection – orchitis – mumps

Other Factors That May Cause Male Infertility:

Occupation – excess heat – radiation –toxic - Lifestyle – smoking - alcohol - Drugs (salfasalyasin)- Ejaculation – disorders – Retrograde ejaculation (semen goes into urine)- premature ejaculation – impotence - congenital abnormalities - chromosomal anomalies - traumatic causes - coital abnormalities – vascular - hormonal - inflammatory - immunological – environmental.

Examination:

- General Health.
- Presence of 20 sexual characteristics.
- Genital Examination.
    - Epididymis.
    - Testes.

Semen Analysis: If abnormal repeat after 4-6 weeks because sperm count varies through time.

Obtain 2-3 days abstinence and examine within 2 hours.

Overweight men generally have a low sperm count. (Kaplan)

Investigation:

- Hormonal
  - Testosterone.
  - FSH.
- Chromosome Karyotype.
- Semen Analysis:have to know it by heart.

Volume	2–6ml
Liquefa	within 30 min
Density	20-250 million/ml
Motility	> 50% progressive movement
Morphology	>30% of sperms are of normal morphology

Azoospermia:

Obstructive - Non obstructive.

Principle Of Management: Deal with the couple together

Aim Of Investigation:

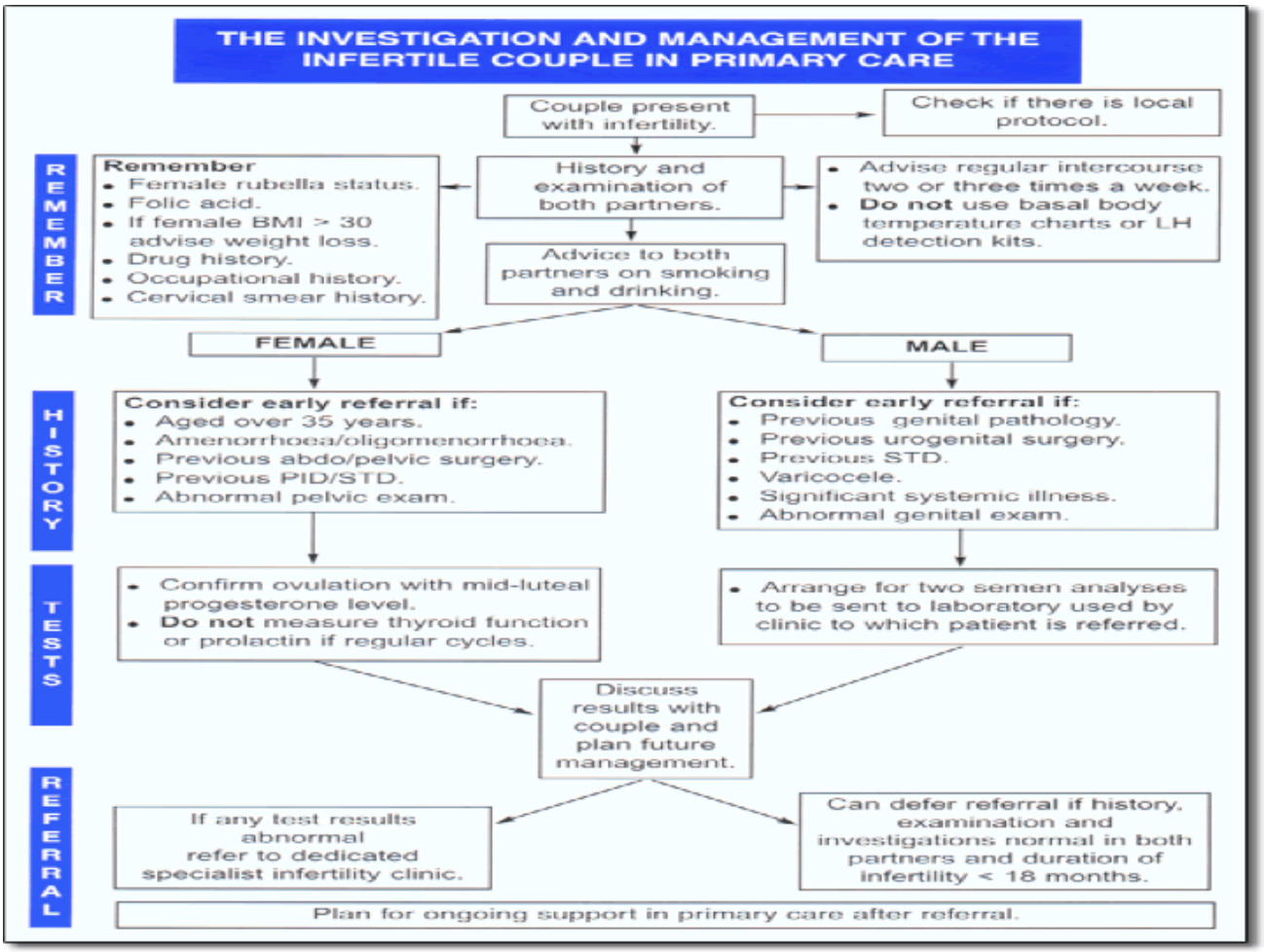
- To give an explanation of the cause.
- To form basis for treatment.
- Prognosis.

Assisted Conception A.R.T

AIH- IUI Artificial Insemination By Husband– Intra Uterine Insemination
ZIFT Zygote Intrafallopian Transfer
GIFT Gamete Intrafallopian Transfer
IVF In Vitro Fertilization
ICSI Intra-Cytoplasmic Sperm Injection

Kaplan Summary:

Question	Answer	Management
Criteria of Infertility	12 months of unprotected intercourse	
1. Anovulation	BBT is flat, Progesterone levels are low and biopsy shows proliferative tissue.	Clomiphene or HSG
2. Male Factor	Semen analysis. Repeat if abnormal.	IUI, ICSI or AID
3. Tubal disease	HSG	Laparoscopy repair or IVF
Unexplained	1, 2 & 3: no problem	Clomiphene + IUI
	If that doesn't work	IVF



Genital Prolapse.

Anatomy

- The pelvic floor closes the outlet of the pelvis.

- It is made up of a number of muscular and fascial structures.
  The most important: the **LEVATOR ANI muscles**.

- These structures are pierced by:
  - Urethra
  - Vagina
  - Rectum

  } passing through the exterior of the body

- These structures are **supported** in place by:
  - ligaments
  - condensation of fascia

- Genital tract is supported by:
  - Pelvic organs:
    bladder, urethra, bowels, anal canal, rectum
  - Ligaments:
    1) broad ligament 2) round ligament 3) uterosacral ligaments
    4) cardinal (transverse) ligaments 5) pubocervical ligaments
    Number (3) & (4) are the **most important** for uterus support... If they fail, prolapse occur.

Etiology

A relaxed vaginal outlet is usually a sequel to mere **OVERSTRETCHING** of the perineal supporting tissues (ligaments) as a **result of previous parturition.**
"Excessive stretching during pregnancy, labor, difficult vaginal delivery (especially with forceps or vaccum)"

- Lack of hormone (as in **later in life**) → causes muscular atony + loss of elastic tissue
- Delivery and pelvic surgery → damage to perineal or pelvic nerves → denervation
- Pelvic surgery = iatrogenic factor (eg. of surgery: hysterectomy)

"**Increased intraabdominal pressure** resulting from:
- chronic cough - ascites- repeated lifting of heavy weights
- habitual straining as a result of constipation
may predispose to prolapse"

- Obesity, genetics can be risk factors

Types of Genital Prolapse (Pelvic Organ Prolapse: POP)

1-Cystocele
2-Urethrocele
3-Rectocele
4- Enterocele
5- Uterocele (uterine prolapse)
6- Vault prolapse

1-Cystocele:

- As a result of defect in the pubo-cervical facial plane which supports the bladder anteriorly
- It causes the bladder to sag down below and beyond the uterus. (Bladder bulges into vagina)

2-Urethrocele:

When the defective fascia involves the urethra (urethra bulges into vagina)

3-Rectocele:

Due to attenuation in the pararectal facea → permits the rectum to bulge into vagina

4-Enterocele:

- Perintoneal hernial sec. along the anterior surface of the rectum.
- Often contains loops of small intestine
- In other words: hernia → peritoneum herniates to posterior fornix of vagina, it often contains bowels

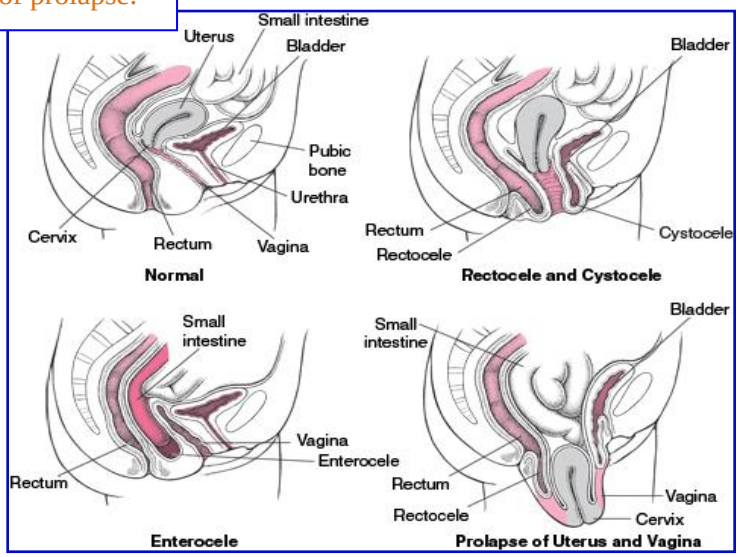
5-Uterocele (uterine prolapse):

- Uterus bulges (sags down) to vagina, might extend down to introitus or even below and protrude outside.
- **Grades** of prolapse:
  - 0 : in place
  - 1 : a slight prolapse into vagina (level of ischial spines)
  - 2 : comes to vaginal canal midway (between ischial spines and introitus)
  - 3 : passes midway and reaches introitus
  - 4 : completely out (called: complete **procidentia.** "Represents failure of all vaginal support")

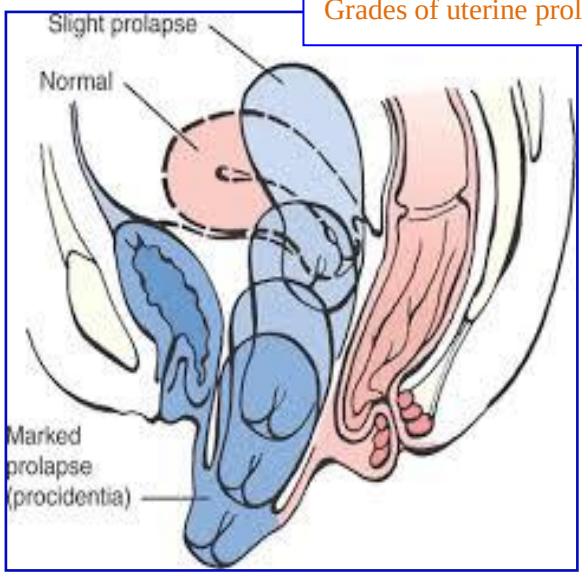
6-Vault prolapse:

- prolapse of the top of the vagina, this happens when the patient had a **hysterectomy** before, so there's no uterus to prolapse and instead the **vagina will prolapse**.

s of prolapse:



Grades of uterine prolpse



Diagnosis of POP

Symptoms:

- Majority are **asymptomatic** (don't treat)
- Pressure and heaviness in the vaginal region
- Sensation of "everything dropping out"
- Bearing down discomfort in the lower abdomen
- Backache

Other associated problems
(coming from adjacent organs)**:**

- Fecal incontinence (e.g. with complete perineal laceration) and often with loose of stools.
- Difficulty in emptying the bladder with marked cyctocele
- Cystitis, due to residual urine → frequency of micturition & ascending UTI
- Urinary incontinence: stress incontinence
- Difficulty of defection and constipation with rectocele → hemorrhoids

Complaint of lump/mass protruding through → marked prolapse

Signs / Examination:

- BMI and vitals
- **Abdominal examination**: lumps or tumors that could be a factor, also check bladder (incontinence, retention)
- **Inspection:** [lithotomy position]
  - External inspection:
    - Is the organ seen bulging?
    - Gaping introitus
    - Perineal scars
    - Visible cystocele and rectocele / urethral
    - Uterine complete prolapse→ Cervical Ulceration (contact)= **Decubitus ulcer**
  - Inspection by speculum

  [position: lateral position, limbs bent and one leg is pulled]:

  In this case we use **SIMS SPECULUM** (single blade) to visualize the walls of vagina.

  Then ask patient to bear down (strain down) to check for protrusion.

- Assess the degree of the prolapse
  (how much the organ is displaced, as mentioned in uterine prolapse & as seen in pic of uterine prolapse in previous page)
- "Rectal-Vaginal examination is often useful to demonstrate a rectocele and distinguish it from an enterocele"
- The doctor mentioned that we can see a cystocele or urethrocele by ultrasound

Treatment

Considerations before treatment:

- Degree of prolapse
- Associated symptoms
- Age (menopausal or premenopausal)
- Future plans for reproduction

Let the patient choose

We try to treat conservatively, unless it does not work

Treatment options:

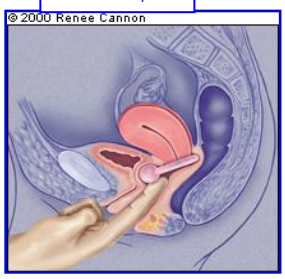
- Conservative measures:

Pelvic floor exercise (Keigel's exercise) → physiotherapy

Pessaries for intravaginal support:

* Can be considered when patient is not a candidate for surgery (unfit, pregnant, post partum).
* They're good to allow cubital ulcers to heal before surgery
* Should be removed, cleaned then re-inserted every 6-12 weeks.
* If neglected, they can cause ulceration and irritation, leading to fistulas, impaction, bleeding, infection.

Pessary



- Pharmacological treatment: no drugs available

- Surgery:

Surgery is "repair":

* Anterior vaginal wall repair is called: anterior repair or **anterior colporrhaphy**
* Posterior repair: also called **posterior colporrhaphy**
* Perineal repair: **perineorrhaphy**

For uterine prolapse surgical repair:

- Hysterectomy + repair, if patient completed her family
- plication of fascia: Uterosacropexy, done laparoscopically (we're not expected to know all details)

Return the uterus to its normal position

- Vaginal vault repair: colpopexy

Counsel the patient about:

Recurrence rate 30 % after surgery

Complications of surgery

For more info regarding this topic + diagrams of surgical repair,

visit ACOG website (American Congress of Ob/Gynecologists):

Summary

- **Causes of pelvic organ prolapse:**
  - Overstretching of the tissues as a result of parturition
  - Lack of hormones later in life
  - Delivery and pelvic surgery (eg: hysterectomy)
  - Obesity
  - Genetics
  - Increased intra-abdominal pressure (chronic cough, constipation... etc)

- Types of POP:
  - Cystocele: Bladder bulges into vagina
  - Urethrocele: urethra bulges into vagina
  - Rectocele: rectum to bulge into vagina
  - Enterocele: peritoneum herniates to posterior fornix of vagina, often contains bowels
  - Uterocele (uterine prolapse): Uterus bulges (sags down) to vagina, might extend.
  - Vault prolapse: of the top of the vagina, when the patient had a hysterectomy

- Grades of uterine prolapse:
  4 grades of prolapse
  (grade 4 is complete procidentia which means uterus is completely out, failure of support)

- Symptoms of pelvic organ prolapse:
  - Majority: asymptomatic [no need to treat]
  - Pressure and heaviness in the vaginal region,
  - Sensation of "everything dropping out"
  - Discomfort in the lower abdomen , Backache
  - Other associated problems (from adjacent organs: rectum & bladder)
  - Complaint of lump/mass protruding through → marked prolapse

- Examination:
  - With complete prolapse, check for decubitus ulcers
  - Use Sim's speculum to check for prolapse

- Treatment:
  - Conservative: Keigel's exercise, pessaries
  - Surgical: Repair
    - Anterior or posterior colporrhaphy
    - Perineorrhaphy
    - For uterine prolapse: uterosacropexy (if we want to reserve uterus... if patient completed her family: hysterectomy + repair
- Recurrence rate: 30%

Lower Genital tract infections + PID.

What is normal?

- The normal vaginal flora is predominately aerobic organisms
- The most common is the H+ peroxide producing lactobacilli
- The normal PH is <4.5
- Normal vaginal secretions ↑ in the middle of the cycle because of ↑ in the amount of cervical mucus .
- -it is clear or white. It may become stretchy and slippery during ovulation or OC
- Complains could be abnormality in the amount, smell or color.

Making the Diagnosis:

❖ Symptoms:
- discharge, odor, irritation, or itch
- discharge
- Clear, white, green, gray, yellow
- Consistency – thin, thick, or curd like

❖ Signs:
- excoriations
- erythema
- discharge

-Normal vaginal secretion is called leucorrhoea.

When exposed to air it might change in color "mustard like"

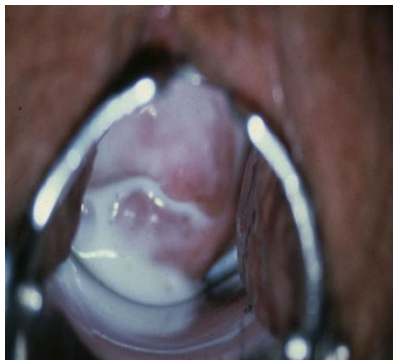
99.999% of my clinic complaints are abnormal bleeding or abnormal discharge.

Vaginal Complaints:

- Most common reason for gyn visits
- 10 million office visits annually
- PE and laboratory data are recommended
- 3 most common etiologies are: **vaginal candidiasis / bacterial vaginosis / Trichomoniasis**

Bacterial Vaginosis (BV):

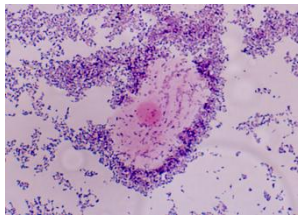
- Most common cause of vaginitis in premenopausal women
- It is caused by alteration of the normal flora, with over-growth of anaerobic bacteria
- It is triggered by ↑ PHof the vagina (intercourse, douches)
- Recurrences are common
- 50% are asymptomatic
- Itching and inflammation are uncommon
- It is not a STD,doesn't require STDs screening



BV

Complications:

- Increases risk for:
  - **Preterm labor** in pregnant women
  - Endometritis and postpartum fever
  - Post-hysterectomy vaginal-cuff cellulitis
  - Postabortal infection
  - Acquiring other STDs, especially HIV



clue cells
(very characteristic for BV)

Diagnosis:

1. Fishy odor (especially after intercourse)Bec semen is alkaline so similar effect to KOH
2. Gray secretions
3. Presence of clue cells
4. PH >4.5
5. +ve whiff test (adding KOH to the vaginal secretions will give a fishy odor)

Treatment:

1. Flagyl 500mg Po Bid for one week (95% cure)
2. Flagyl 2g PO x1 (84% cure)
3. Flagyl gel PV
4. Clindamycin cream PV
5. Clindamycin PO

- Treatment of the partner is not recommended

Candidiasis:

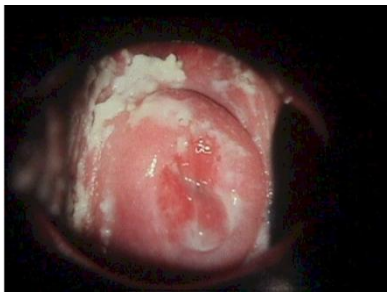
- 75% of women will have at least once during their life
- 45% of women will have two or more episodes/year
- 15% have chronic infection
- Rare before menarche, but 50% will have it by age 25
- Less common in postmenopausal women, unless taking estrogen
- It is not an STD
- 90% of yeast infections are secondary to Candida Albican
- Other species (glabrata, tropicalis) tend to be resistant to treatment

It doesn't affect the fetus or anything **unlike BV**, so if patient doesn't complain **leave it**.

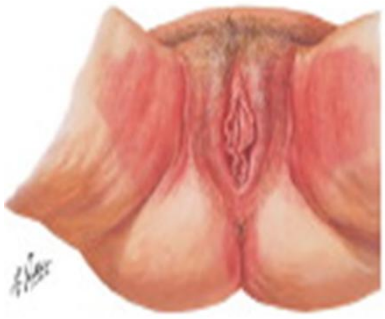
Predisposing factors:

1. Antibiotics: disrupting the normal flora by ↓ lactobacilli
2. Pregnancy (↓ cell-mediated immunity)
3. Diabetes
4. OCP
5. Disinfecting the vagina (As some women wash it with detol or soap ……)

Diagnosis:



1. Vulvar pruritisand burning
2. The discharge vary from watery to thick cottage cheese discharge
3. Vaginal soreness and dysparunea
4. Splash dysuria
5. O/E: erythema and edema of the labia and vulva
6. The vagina may be erythematous with adherent whitish discharge
7. Cervix is normal
8. PH< 4.5
9. budding yeast or mycelia on microscopy



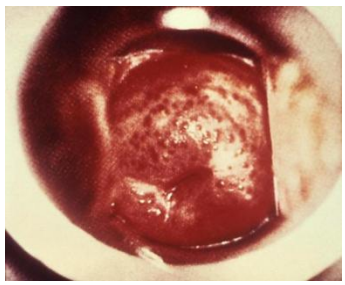
Treatment:

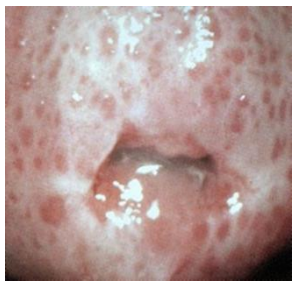
1. Topical Azole drugs (80-90% effective)
2. Fluconazole is equally effective (Diflucan 150mg PO x1), but symptoms will not disappear for 2-3 days
3. 1% hydrocortisone cream may be used as an adjuvant treatment for vulvar irritation
4. Chronic infections may need long-term treatment (6 months) with weekly Fluconazole

TrichomonasVaginalis

- It is an anaerobic parasite, that exists only in trophozite form
- 3rd most common vaginitis
- 60% of patients also have BV
- 70% of males will contract the disease with single exposure
- Virtually always sexually transmitted
- Patients should be tested for other STDs (HIV, Syphilis, hepatitis)

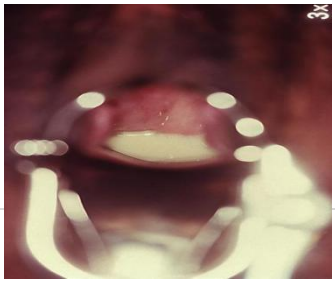
Diagnosis:

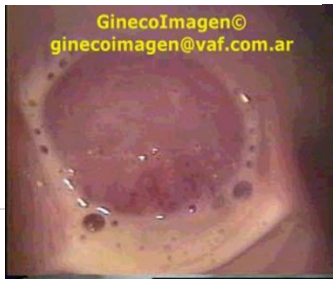




Strawberry cervix

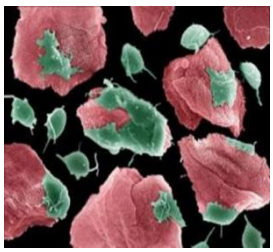
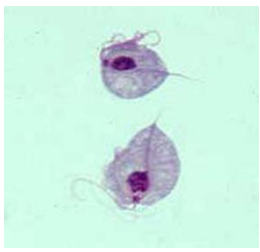
1. Profuse, purulent malodorous discharge
2. It may be accompanied by vulvar pruritis
3. Secretions may exudate from the vagina
4. If severe → patchy vaginal edema and strawberry cervix
5. PH >5
6. Microscopy: **motile trichomands**and ↑ leukocytes
7. Clue cells may if BV is present
8. Whiff test may be +ve





Wet mount:

- Trichomonads seen only in 50 – 70%
- Elevated pH
- Can increase leukocytes
- Paps

Tricomonads

Treatment:

1. Falgyl PO (single or multi dose)
2. Flagyl gel is not effective
3. The partner should be treated
4. If refractory to treatment
    - Retreat with 7 day course
    - If fails again, try 2gm dose daily x 3 – 5 days
    - Assure compliance with partner/culture

Other causes of Vaginitis:

- Atrophic vaginitis (in post menopausal women or anything that lowers estrogen like oophorectomy "surgical menopause")
    - High vaginal pH, thin epithelium, d/c
    - Parabasal cells on wet mount
    - Topical estrogen cream

Atrophic vaginitis is not an infection, it is menopause symptoms.

- Atypical manifestations: HSV, HPV
- Noninfectious vulvovaginitis
    - Irritants/allergens
    - Lichens syndromes (sclerosus, simplex chronicus, planus)(Skin condition, you treat it with steroids)

Herpes Simplex Virus

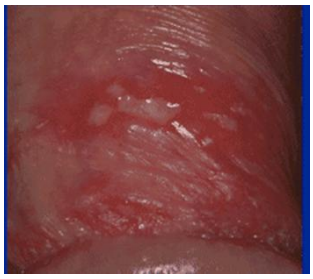
- The "silent epidemic"
- > 45 million in the US
- > 1 million newly diagnosed annually
- The most common STD in US, and likely the world
- Almost 25% of Americans have HSV2 antibodies by the age of 30
- **HSV – 1 :** Mostly oro-labial, but increasing cause of genital herpes
- **HSV – 2**
    - Almost entirely genital
    - > 95% of recurrent genital lesion

Primary Herpes – Classic Symptoms:

- Systemic – fever, myalgia, malaise

  Can have meningitis, encephalitis, or hepatitis
- Local – clusters of small, painful blisters that ulcerate and crust outside of mucous membranes

  Itching, dysuria, vaginal discharge, inguinal adenopathy, bleeding from cervicitis
- New lesions form for about 10 days after initial infection, but can last up to 3 weeks
- Shedding of virus lasts 2 – 10 days

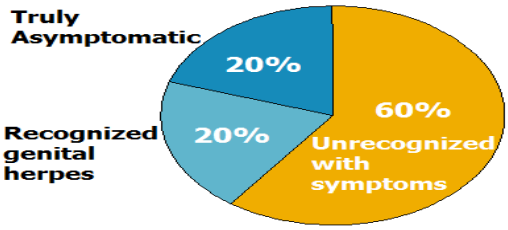


Genital Herpes
If it was active, we can't let her deliver vaginally!

Recurrent herpes:

- Reactivation of virus
- Mild, self-limited
- Localized, lasting 6-7 days
- Shedding: 4-5 days
- Prodrome: 1-2 days

The Clinical Spectrum of HSV - 2 positive people:



Diagnosis:

- Viral isolation (culture)
  - High specificity, low sensitivity
    - 50% for primary infxn
    - 20% for recurrent infxn
- Direct detection of virus (Tzcank smears, PCR)
- Serology(doesn't tell if it was active or not !)
  - Newer tests that are specific for type of virus (HerpesSelect 2, herpes glycoprotein for IgG, ELISA)

Management Goals:

- Relieve symptoms
- Heal lesions
- Reduce frequency of recurrent episodes
- Reduce viral transmission
- Patient support and counseling

Oral Antiviral Therapy:

- Valacyclovir (Valtrex)
- Famciclovir (Famvir)
- Acyclovir (Zovirax)

Cervicitis:

- My cause abnormalvaginal discharge ,postcoital bleeding or irregular bleeding
- Neisseria Gonorrhea and Chlamydia Trachomatis infect only the glandular epithelium and are responsible for mucopurulentendocervisitis (MPC)
- Ecto-cervix epithelium is continuous with the vaginal epithelium, so Trichomonas, HSV and Candida may cause Ecto-cervix inflammation
- Tests for Gonorrhea (culture on Thayer- martin media) and Chlamydia (ELISA, direct IFA) should be performed

Treatment:

Depends whether acute or chronic.

But usually:

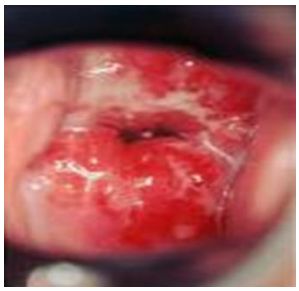
-**Chlamydia**: doxy twice daily for 7 – 10 days

-**Gonorrhea**: cephalosporin

Table 15.2 Treatment Regimens for Gonococcal and Chlamydial Infections

***Neisseria gonorrhoeae* endocervicitis**
Ceftriaxone 125 mg intramuscularly (single dose), or *Ofloxacin* 400 mg orally (single dose), or *Cefixime* 400 mg orally (single dose), or *Ciprofloxacin* 500 mg orally (single dose)
***Chlamydia trachomatis* endocervicitis**
Doxycycline 100 mg orally b.i.d. for 7 days, or *Azithromycin* 1 gram orally (single dose), or *Ofloxacin* 300 mg orally b.i.d. for 7 days, or *Erythromycin* base 500 mg orally 4 times a day for 7 days, or *Erythromycin ethylsuccinate* 800 mg orally 4 times a day for 7 days

Morbidity and Mortality Weekly Report. Centers for Disease Control and Prevention. *MMWR* 1993;42 51–57.



What is bad about STDs is that they cause **tubal factor infertility**☹

Pelvic Inflammatory Disease (PID):

- Ascending infection, ? Up to the peritoneal cavity
- Organisms**: Chlamydia, N Gonorrhea**
- Less often: H Influenza, group A Strept, Pneumococci, E-coli(also TB which is more in south are)
- acute PID 1-2% of young sexually active women each year
- 85% because of STD- 15% occur after procedures that break cervical mucous barrier(like infertility procedures)

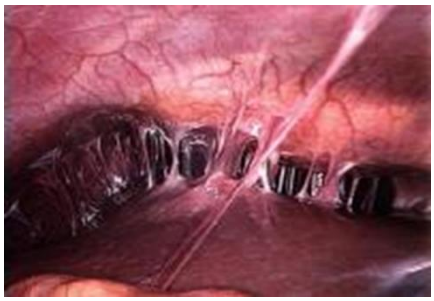
Diagnosis: difficult because of wide variation of signs and symptoms

- Clinical triad: pelvic pain (90%) ,cervical motion & adnexal tenderness
- fever
- Cervical motion tenderness indicate peritoneal inflammation
- Patients may or may not have mucopurulent discharge
- leukocytosis

Cervical motion tenderness: during digital exam when we move the cervix they jump bec of pain

Differential diagnosis:

- acute appendicitis
- Endometriosis
- torsion/rupture adnexal mass
- ectopic pregnancy
- lower genital tract infection
- 75% associatedendo-cervical infection & coexisting purulent vaginal discharge.
- **Fitz-Hugh-Curtis syndrome** :
  - 1-10%
  - perihepatic inflammation & adhesion
  - s/s ; RUQ pain, pleuritic pain, tenderness at RUQ on palpation of the liver
  - mistaken dx ; acute cholecystitis, pneumonia



Fitz-Hugh-Curtis syndrome

Table 15.3. Clinical Criteria for the Diagnosis of Pelvic Inflammatory Disease

Symptoms
None necessary
Signs
Pelvic organ tenderness Leukorrhea and/or mucopurulent endocervicitis
Additional criteria to increase the specificity of the diagnosis
Endometrial biopsy showing endometritis Elevated C-reactive protein or erythrocyte sedimentation rate Temperature higher than 38°C Leukocytosis Positive test for gonorrhea or chlamydia
Elaborate criteria
Ultrasound documenting tuboovarian abscess Laparoscopy visually confirming salpingitis

1 major 2 minor
Abdominal pain + 2

-Also if we find bilateral abscess then it is PID

Risk factors:

- Sexual behavior
- others
  - IUD user (multifilament string"not used anymore")
  - surgical procedure
  - previous acute PID
  - Reinfection → untreated male partners 80%
- Decrease risk

- barrier method
- OC

Sequelae:

- Infertility~20%
- Ectopic pregnancy ~6fold increase
- Chronic pelvic pain
- TOA~ 10%(tubo-ovarian abscess)
- Mortality -acute 1%
  -after rupture TOA ~10%

Medications:

- Empirical ABx cover wide range of bacteria
- Treatment start as soon as culture & diagnosis is confirmed/suspected
  - failure rate, OPD oral ATB → 10-20%
  - failure rate, IPD iv ATB → 5-10%
- reevaluate 48-72 hrs of initial OPD therapy

Table 15.4 CDC Guidelines for Treatment of PID

Outpatient treatment

Regimen A:

Cefoxitin 2 g intramuscularly, plus *probenecid,* 1 g orally concurrently, or *ceftriaxone* 250 mg intramuscularly, or equivalent cephalosporin
<*PLUS*>
Doxycycline 100 mg orally 2 times daily for 14 days

Regimen B:

Ofloxacin 400 mg orally 2 times daily for 14 days
<*PLUS*>
Clindamycin 450 mg orally 4 times daily, or *metronidazole* 500 mg orally 2 times daily for 14 days

Inpatient treatment

Regimen A:

Cefoxitin 2 g intravenously every 6 hours, or
Cefotetan 2 g intravenously every 12 hours,
<*PLUS*>
Doxycycline 100 mg intravenously or orally every 12 hours

Regimen B:

Clindamycin 900 mg intravenously every 8 hours
<*PLUS*>
Gentamicin loading dose intravenously or intramuscularly (2 mg/kg of body weight) followed by a maintenance dose (1.5 mg/kg) every 8 hours

Morbidity and Mortality Weekly Report. Centers for Disease Control and Prevention. *MMWR* 1993;42 78–80.

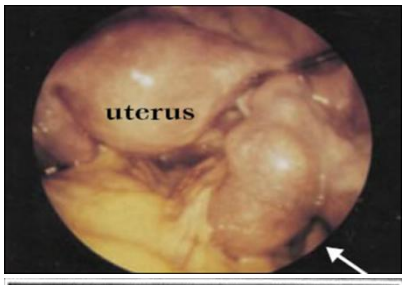
Criteria for hospitalization:

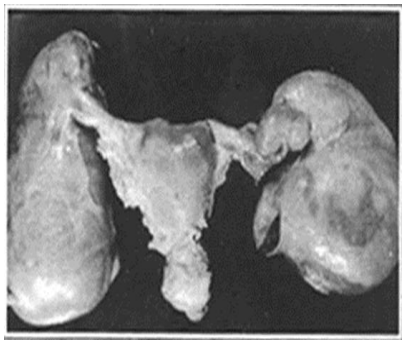
TABLE 28.3.
Criteria for Hospitalization of Patients With Acute Pelvic Inflammatory Disease

The following criteria for hospitalization are based on observational data and theoretical concerns:

- Surgical emergencies such as appendicitis cannot be excluded.
- The patient is pregnant.
- The patient does not respond clinically to oral antimicrobial therapy.
- The patient is unable to follow or tolerate an outpatient oral regimen.
- The patient has severe illness, nausea and vomiting, or high fever.
- The patient has a tuboovarian abscess

Tubo-ovarian Abscess





- End-stage PID
- Causes agglutination of pelvic organs (tubes, ovaries and bowel)
- 75% of patients respond to IV antibiotics
- Drainage may be necessary

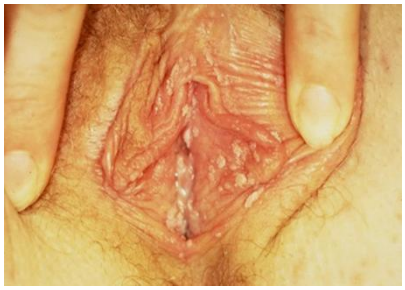
Genital Warts (highly infectious)

- Condylomaaccuminata secondary to HPV infection (usually 6&11), these are non-oncogenic types
- Usually at areas affected by coitus (posterior fourchette)
- 75% of partners are infected when exposed
- Recurrences after treatment are secondary to reactivation of subclinical infection

Table 15.5 Treatment Options for External Genital and Perianal Warts

Modality (%)	Efficacy (%)	Recurrence risk
Cryotherapy	63–88	21–39
Podophyllin 10–25%	32–79	27–65
Podofilox 0.5%*	45–88	33–60
Triochloroacetic acid 80–90%	81	36
Electrodesiccation or cautery	94	22
Laser†	43–93	29–95
Interferon	44–61	0–67

*May be self-applied by patients at home.
†Expensive, reserve for patients who have not responded to other regimens.



Summery

	BV	Candidiasis	trichomoniasis
Presentation &Dx	-Fishy odor -gray discharge -clue cells -+ whiff test -PH >4.5	-Asymptomatic -Itching -Cheesy discharge -budding yeast or mycelia on microscopy -PH< 4.5	-Itching -frothy discharge - strawberry cervix -tricomands. -PH >5 (might be accompanied with BV)
Management	Metronidazole (flagyl) PO	If symptomatic topical azole	Metronidazole (flagyl) PO + treat the partner

MCQ:

Which of the following is a sexually transmitted infection ?

A) HPV

B) chlymedia

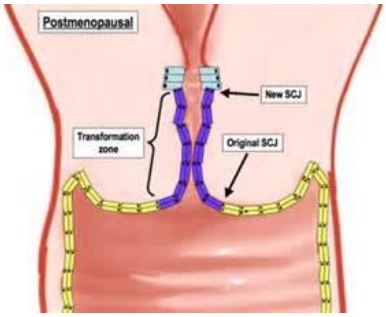
C) toxoplasmosis

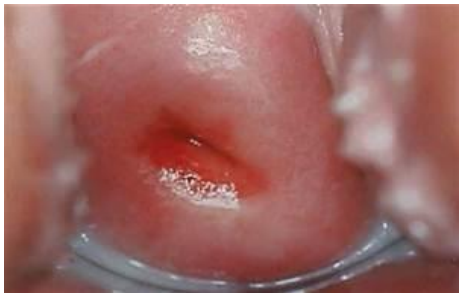
Cervical Intraepithelial Lesion.

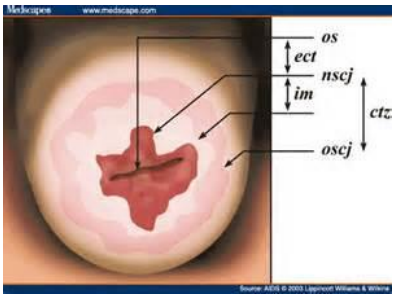
Anatomy :

- The ectocervix ..surface of the cervix that is visualized on vaginal speculum examination is covered in squamous epithelium, and the endocervix, including the cervical canal, is covered with glandular epithelium.

- Transformation zone :
  zone on the cervix at which squamous epithelium and columnar epithelium meet; changes location in response to a woman's hormonal status.







- SCJ is a dynamic point that change in response to :
  - Puberty
  - Pregnancy
  - Menopause
  - Hormonal stimulation
- In neonate it located on the exocervix, at menarche, the production of estrogen causes the vaginal epithelium filled with glycogen.
- Lactobacilli " vaginal normal flora " act on glycogen to lower the PH, stimulate the subcolumnar reserve cells to undergo metaplasia.

HPV :

- Human Papilloma virus – sexually transmitted virus .
- (HPV) is divided into two classes:
  1. Oncogenic.
  2. Nononcogenic.
- Infection with oncogenic (or high-risk) HPV usually is a necessary but not sufficient factor for the development of squamous cervical neoplasia. Therefore, only a small fraction of women infected with HPV will develop significant cervical abnormalities and cancer.
- HPV 16 has the highest carcinogenic potential and accounts for approximately 55–60% of all cases of cervical cancer worldwide.
- HPV 18 is the next most carcinogenic genotype and is responsible for 10–15% of cases of cervical cancer.
- Approximately 10 other genotypes are associated with the remainder of cases of cervical cancer.
- The current model of cervical carcinogenesis posits that HPV infection results in either transient or persistent infection.
- Most HPV infection is transient and poses little risk of progression.
- Only a small fraction of infections are persistent, but persistent infection at 1 year and 2 years strongly predicts subsequent risk of cervical intraepithelial neoplasia (CIN) 3 or cancer regardless of age.
- Two low-risk types (6 and 11) cause benign disease of the HPV infection "anogenital warts and condyloma" .

Risk factors :

- Cofactors that increase the likelihood of persistence infection include:
  - Cigarette smoking and HPV infection have synergistic effects on the development of CIN and cervical cancer.
  - Compromised immune system.
  - Human immunodeficiency virus (HIV) infection
  - Oral contraceptives" why ?because of estrogen " — Long-term use of oral contraceptives has been implicated as a cofactor that increases the risk of cervical carcinoma in women who are. The excess risk of cervical cancer declines after discontinuation of oral contraceptives, and by 10 years, returns to the baseline risk in nonusers
  - Herpes simplex virus and Chlamydia "sexually transmitted virus". Infection with chlamydia, herpes simplex virus, or other sexually transmitted infections may be a surrogate marker of exposure to HPV rather than a causal factor itself . Alternatively, these infections may modulate host immunity, thereby facilitating persistence of oncogenic HPV
  - The risk of transmission of HPV correlates with the lifetime number of sex partners, but the prevalence of HPV infection is substantial (4 to 20 percent) even in those with one partner.
    - In the United States (US), up to 50 percent of sexually active young women will have positive HPV tests within 36 months of first sexual activity, and up to 57 percent of sexually active female adolescents are infected with HPV at any one point in time
    - Human papillomavirus infection is most common in teenagers and women in their early 20s, with a decrease in prevalence as women age.
    - Most young women, especially those younger than 21 years, have an effective immune response that clears the infection in an average of 8 months or reduces the viral load in 85–90% of women to undetectable levels in an average of 8–24 months

Screening :

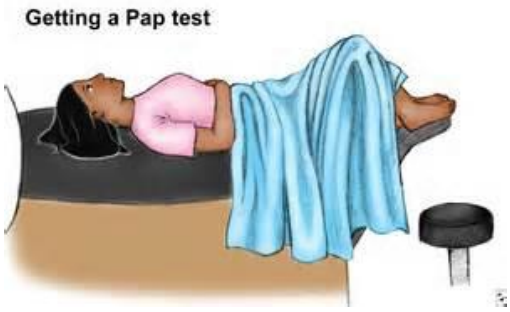
- Most cervical cancer occurs in women who were either never screened or were inadequately screened.
- Estimates suggest that 50% of the women in whom cervical cancer is diagnosed never had cervical cytology testing, and another 10% have not been screened within the 5 years before diagnosis.
- Thus, approximately 60% of diagnoses of cervical cancer are a result of inadequate screening

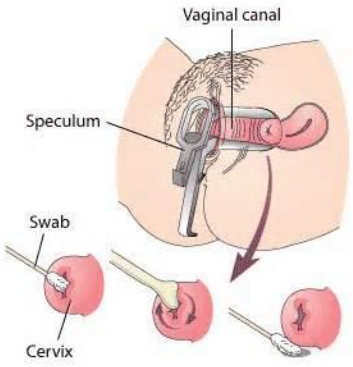
When to screen :" imp as MCQ "

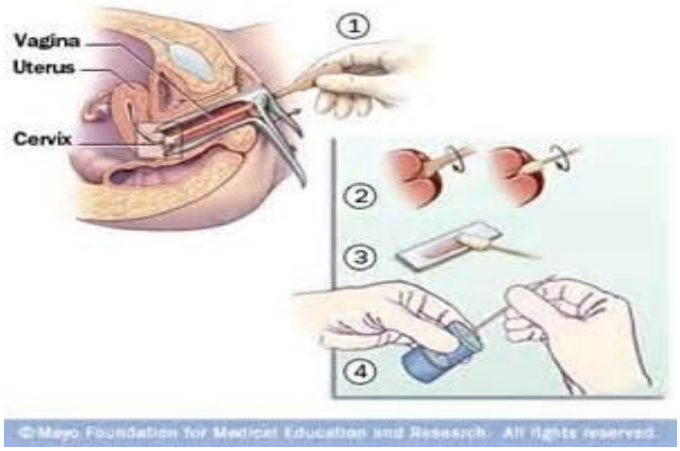
- Cervical cancer screening should begin at age 21 years
- 21-29yrs…. PAP every 3years.
- 30-65yrs…. PAP +HPV every 5years "preferable "
  PAP every 3years."acceptable "

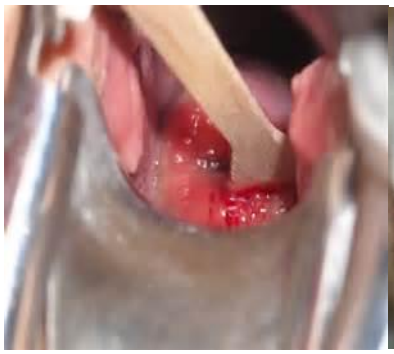
- Above 65… no screening"if the patient does not have any abnormal PAP smear " " if she has previous event , we have to screen her till 20 years after the diagnosis "
- Vaccinated women should continue age specific screening protocol.

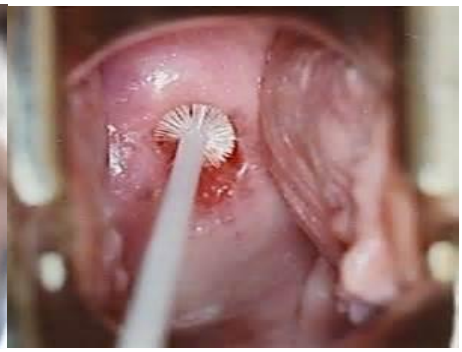
PAP… Cytology :







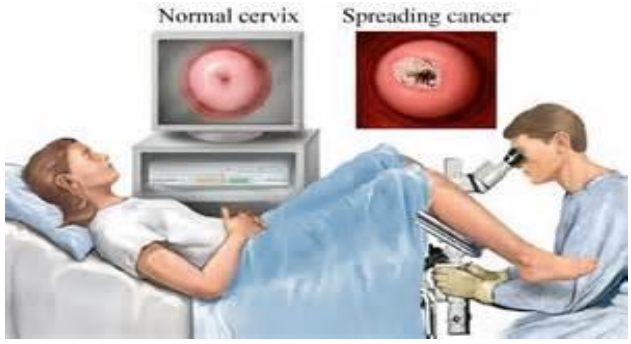




PAP… interpretation :

- Bethesda system classification
  Squamous cell abnormalities
  - ASC " abnormal sequamous cells":
    - ASC-US "atypical squamous cells of undetermined significance "
    - ASC-H"atypical squamous cells favoring high grade "
  - LSIL"low-grade squamous intraepithelial lesion" consistent with CIN 1
  - HSIL" high-grade squamous intraepithelial lesion" consistent with CIN2, CIN3, CIS
  - SCC"Squamous cell carcinoma"
  - Glandular cell abnormalities
  - Atypical glandular cell " it gives you a hint that check above , if there is any abnormalities in the endometrium "
    - Atypical endocervical cell
    - Atypical endometrial call
    - No otherwise specific
  - Atypical glandular cell favor neoplastic
    - Endocervical
    - No otherwise specific.
  - AIS
  - Adenocarcinoma

Colposcopy :



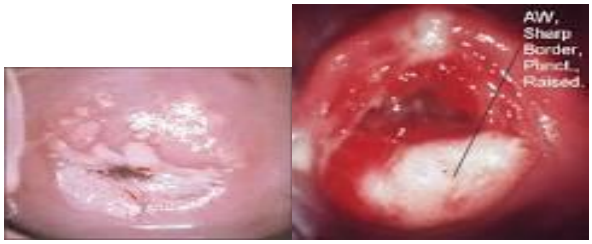
- Steroscopic binocular microscope of low magnification.
- 3% ascetic acid to remove adherent mucus & cellular debris.
- Green filter to accentuate the vascular changes.
- Original squamous epithelium appears gray & homogenous.
- The columnar epithelium appears red and grape like.
- TZ glands opening that are not covered by the squamous metaplasia and by the paler color of the metaplastic epithelium.“Normal colposcopic finding “

Who need colposcopy :

- Persistent atypical cells of undetermined significance (ASC-US) or ASC-US with positive high-risk human papillomavirus (HPV) subtypes
- ASC suggestive of high-grade lesion (ASC-H)
- Atypical glandular cells (AGC)
- Low-grade squamous intraepithelial lesions (LSIL)
- High-grade squamous intraepithelial lesion (HSIL)
- Suspicious for invasive cancer
- Malignant cells present

Abnormal finding on colposcopy :

LeucoplakiaAceto-white area



Mosaicism Punctation

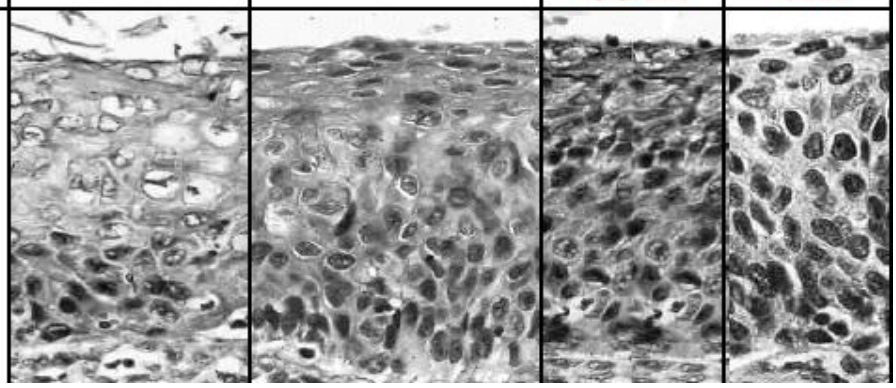
Biopsy and ECC :

- The most abnormally appearing areas are biopsied. Biopsies are relatively contraindicated in patients on anticoagulation medication, who have a known bleeding disorder, or who are pregnant.
- Endocervical curettage or sampling is performed in patients with ASC-H, HSIL, AGC, adenocarcinoma in situ (AIS), LSIL but no visible lesion, if ablative treatment is contemplated, and those with an unsatisfactory colposcopic examination

Histological definitions :

- CIN 1 is a low-grade lesion. It refers to mildly atypical cellular changes in the lower third of the epithelium. Human papillomavirus (HPV) cytopathic effect (koilocytoticatypia) is often present.
- CIN 2 is considered a high-grade lesion. It refers to moderately atypical cellular changes confined to the basal two-thirds of the epithelium (formerly called moderate dysplasia) with preservation of epithelial maturation.
- CIN 3 is a high-grade lesion. It refers to severely atypical cellular changes encompassing greater than two-thirds of the epithelial thickness and includes full-thickness lesions (previous terms were severe dysplasia or carcinoma in situ).

Biopsy… Histopathology :

<table>
<tr><td rowspan="2">LAST System[1]</td><td>Cytology</td><td>LSIL</td><td colspan="3">HSIL</td></tr>
<tr><td>Histology</td><td>LSIL</td><td colspan="2">p16 staining should be performed*</td><td>HSIL</td></tr>
<tr><td rowspan="2">Bethesda Classification System[2]</td><td>Cytology</td><td>LSIL</td><td colspan="3">HSIL</td></tr>
<tr><td>Histology</td><td>CIN 1</td><td colspan="2">CIN 2</td><td>CIN 3</td></tr>
<tr><td colspan="2">Previous terminology</td><td>Mild dysplasia</td><td colspan="2">Moderate dysplasia</td><td>Severe dysplasia</td><td>Carcinoma in-situ</td></tr>
<tr><td colspan="2">Histologic images</td><td colspan="5">  </td></tr>
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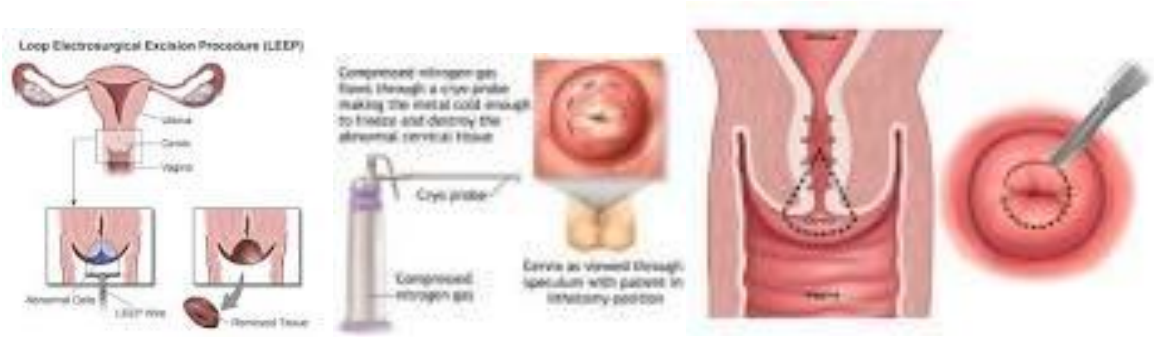
CIN 1 , 2 , 3 = They are not crossing the basement membrane .

CIN to cancer :

- The outcome of CIN 1 lesion depends upon the preceding cytology:
  - CIN 1 preceded by ASC-US or LSIL cytology –will be diagnosed with CIN 2,3 within 6 to 24 months of follow-up . No studies have reported invasive cervical cancer in this patient population within this follow-up period.
  - CIN 1 preceded by ASC-H or HSIL cytology, five-year risk of CIN 3+ of 15 percent
  - For CIN 2 lesions, 40 to 58 percent of lesions will regress if left untreated, while 22 percent progress to CIN 3, and 5 percent progress to invasive cancer
  - For CIN 3, the estimated spontaneous regression rate is 32 to 47 percent, with 12 to 40 percent progressing to invasive cancer if untreated

Management of CIN :

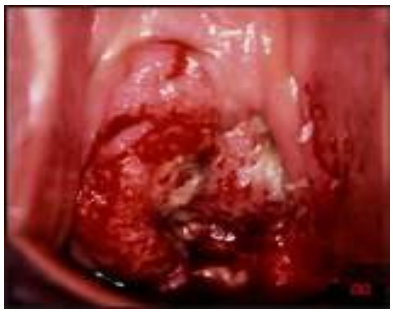
LEEP "office procedure" cryosurgery Cone biopsy

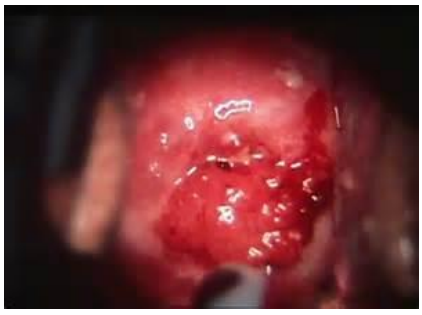


- Cone biopsy:
  - Diagnostic and theraputic
  - Under GA
  - Complications:
    - Bleeding
    - Infection
    - Cervical stenosis
    - Cervical incompetence
  - Cone for whom :
    - *Diagnostic* cone
      - Colposcopy is unsatisfactory
      - ECC shows High grade lesion
      - Discrepancy between PAP and biopsy
      - CIS or AIS on PAP
      - Biopsy confirm invasion
    - Cone as *therapeutic* :
      - CIN 3
      - Stage IAI

No PAP….NO colposcopy :

- Any patient **with grossly abnormal** cervix should have a punch **biopsy** regardless of any previous result.





Cervical cancer :

- 2008:
  - 530,000 new cases …275,000 deathes worldwide..
  - 8-6% in developing countries.
  - The tenth most common cause of death in developed countries 9 per 100,000 women
  - In the developing countries second most common type of cancer (17.8 per 100,000 women) and cause death 9.8 per 100,000 women.
- In USA:
  - Over 12,000 new cases anualy and 4000 cancer death
  - Third cause of death among gynecological cancer
  - With effective screening program and vaccination 75% decrease in incidence and mortality had noticed in the past 50years in the developed countries.

There are two main types of cervical cancer:

- squamous cell carcinoma and adenocarcinoma.
- Squamous cell carcinoma of the cervix is more prevalent than adenocarcinoma.
- Both types are found in sexually active women.
- Infection with specific high-risk strains of human papillomavirus (HPV) is central to the pathogenesis of cervical cancer Of the approximately 30 to 40 HPV genotypes that infect the mucosa of the genital tract, eight (types 16, 18, 45, 31, 33, 52, 58, and 35) are responsible for 95 percent of cervical cancers, and two (types 16 and 18) are responsible for about 70 percent of cervical cancer [5]. Two low-risk types (6 and 11) cause about 90 percent of benign anogenital warts.

How to evaluate :

- **Symptoms**
  - Abnormal vaginal bleeding
    - Poscoital
    - Intermenstrual
    - Postmenopausal.
  - Persistent vaginal discharge
  - Pelvic pain" or pelvic pressure "
  - Leg swealling
  - Urinary frequency
  - Constipation and PR bleeding.
- **Physical finding**
  - Normal because it is a microscopic disease when it is stage 1a1
  - Weight loss?
  - Enlarged inguinal or supraclavicular LN.
  - Lower limb edema.
  - Local exam….
  - Normal cervix
  - Lesion in endocervix
  - Ulcerative, exophytic, grannular or necrotic.
  - Friable cervix … bleeding to touch "very characteristic sign "
- **Clinical exam** " why ?becausecervical cancer is clinical staging disease not surgical staging – This is MCQ trick "
  - Rectovaginal exam is essential to determine the extent of the tissues invovlevement
  - Evaluate the vaginal fornices
  - Evaluate the pelvic side wall

Pattern of spread : very imp

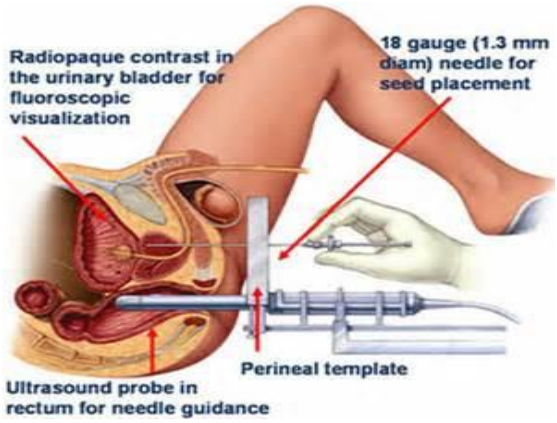
- **Direct invasion of**
  - Cervical stroma
  - Corpus
  - Vagina
  - Parametrium
- **Lymphatic spread**
  - Pelvic, inguinal& going up to supraclavicular
  - Paraaortic
- **Haematogenous**
  - Lung
  - Liver
  - Bone

Work up :

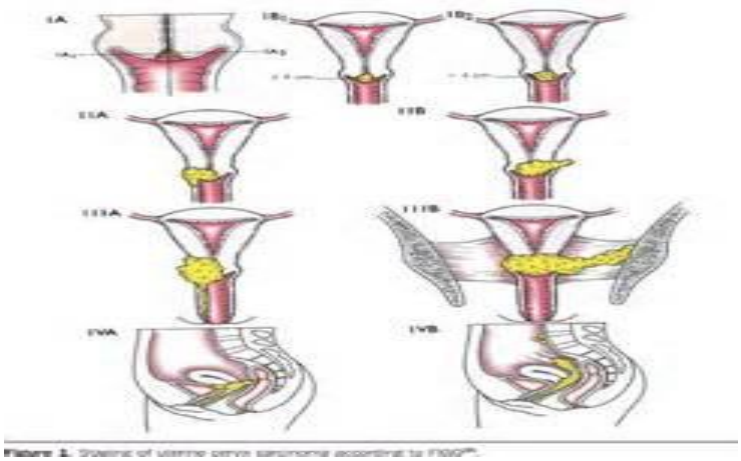
- **History … Examination**
  Ask about last PAP :
  - When
  - Result
  - Any specific management
- **Physical exam**
  - Biopsy for any gross lesion
  - PAP if no lesion seen
  - Sever bleeding… packing
  - LN assessment
- **Blood work**
  - CBC
    - Low HB in case of bleeding
  - KFT
    - High creatinin in case of ureteric obstruction
    - 30% in stage III disease.
    - 40% in stage IV disease.
  - Hypercalcemia indicate bone metastasis
  - LFT
    - Abnormal results indicate metastasis.
- **Images**
  - CXR
  - IVP
  - Abdominal CT
  - MRI pelvis

Cervical cancer stages :

- Clinical exam…underanasthesia?
  - Evaluation of the cervix
  - Upper and lower vagina
  - Rectovaginal exam to evaluate the parametria and pelvic side wall
- Cystoscopy
  - Bladder invasion
- Proctoscopy
  - Rectal invasion



Figo staging :



- **Stage I**
  - The carcinoma strictly cofined to the cervix" we know the depth of the invasion by doing cone biobsy "
    - IA microscopic disease… no gross lesion "manage by cone biobsy or hysterectomy "
      - IA1… invasion </=3mm extension </=7mm
      - IA2 …invasion >3mm but not more than 5mm, extension not more than 7mm.
    - IB macroscopic disease more than stage IA or visible lesion , "manage by radical hysterectomy " – radical means removal of parametrium .
      - IB1 visible lesion </= 4cm in greatest dimension
      - IB2 visible lesion > 4cm in greatest dimension

- **Stage II**
  - Extension beyond the cervix but not to the pelvic side wall or lower vagina
    - IIA…without parametrial invasion
      - IIA1 … clinically visible lesion </=4cm in greatest dimension.
      - IIA2… clinically visible lesion > 4cm in greatest dimension.
    - IIB… with parametrial invasion." Only we know it by rectovaginal exam "

- **Stage III**
  - Tumor invade pelvic side wall& or lower third of the vagina & or causing hydronephrosis or non – functioning kidney.
    - IIIA only lower third of the vagina
    - IIIB invading pelvic side wall & or causing hydronephrosis or non – functioning kidney.

- **Stage IV**
  - Tumor extended beyond the true pelvis or has invade the mucosa of the rectum or the bladder.
    - IVA.. Tumor invading adjacent organ
    - IV B.. Tumor invading distant organ.

Summary :(from Kaplan)

1-The most common etiology of cervical cancer is HPV .

2- Risk Factors. These include early age of intercourse, multiple sexual partners, cigarette smoking and immunosuppression .

3- Pap smear classification :

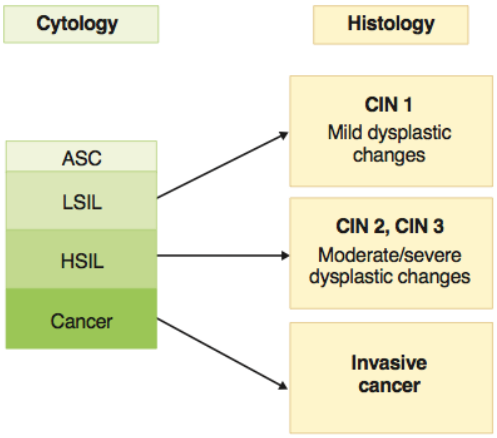


Figure II-4-4. Classification of Cervical Dysplasias

4-Invasive Cervical Cancer : is cervical neoplasia that has penetrated through the basement membrane .

5- Staging : is clinical based on pelvic examination .

6- Management :

- **Specific by stage:**

  Stage Ia1: Total simple hysterectomy, either vaginal or abdominal

  Stage Ia2: Modified radical hysterectomy

  Stage IB or IIA: Either radical hysterectomy with pelvic and paraaortic lymphadenectomy (if premenopausal) and peritoneal washings or pelvic radiation (if postmenopausal). In patients who can tolerate surgery, a radical hysterectomy is preferred; however, studies have demonstrated equal cure rates with radiation or surgical treatment.

  Stage IIB, III, or IV: Radiation therapy and chemotherapy for all ages.

Endometrial Neoplasm.

Endometrial neoplasm:

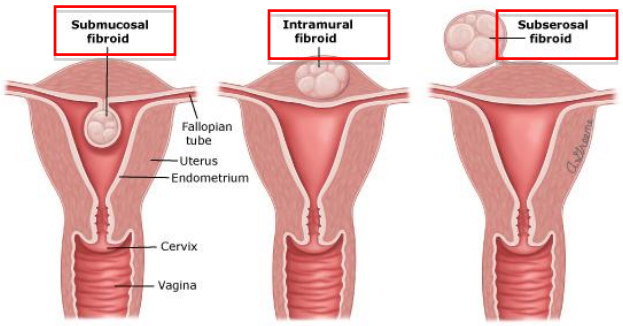
Neoplasms include both benign and malignant tumors (cancer).

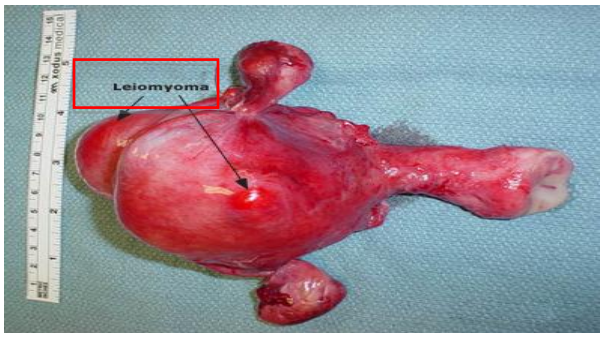
- Definitions
- Pathogenisis, Behavior & malignant potential.
- Clinical presentations
- Work – up
- Managements.

Leiomyomas

- Most common neoplasm of the uterus.
- Benign monoclonal tumors.
- Derived from the smooth muscle cell of the myometrium.
  (leiomyomas + fibroids + Myomas)→benign

Terminology & Location:(how to differentiate in images)



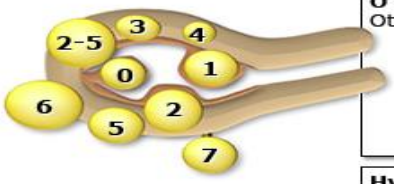


Figo staging:(not clinically applicable)

Polyp	Submucosal
Adenomyosis	Other
Leiomyoma	
Malignancy & hyperplasia	

Coagulopathy
Ovulatory dysfunction
Endometrial
Iatrogenic
Not yet classified

Leiomyoma subclassification system

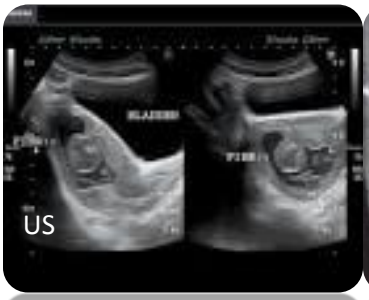


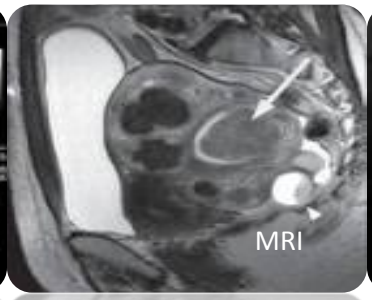
SM - Submucosal	0	Pedunculated intracavitary
	1	<50 percent intramural
	2	≥50 percent intramural
O - Other	3	Contacts endometrium; 100 percent intramural
	4	Intramural
	5	Subserosal ≥50 percent intramural
	6	Subserosal <50 percent intramural
	7	Subserosal pedunculated
	8	Other (specify, eg, cervical, parasitic)

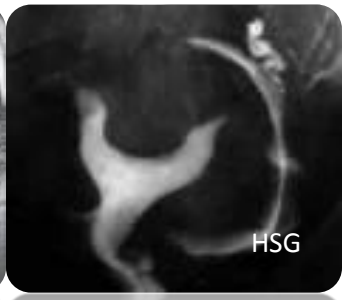
Hybrid leiomyomas (impact both endometrium and serosa)	Two numbers are listed separated by a hyphen. By convention, the first refers to the relationship with the endometrium while the second refers to the relationship to the serosa. One example is below.	
	2-5	Submucosal and subserosal, each with less than half the diameter in the endometrial and peritoneal cavities, respectively.

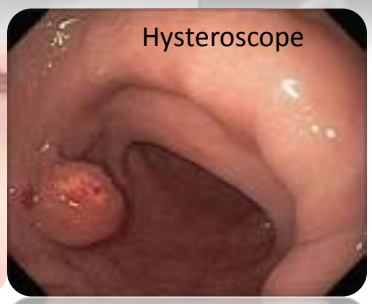
Submucosal:

- Submucosalmyomas (FIGO type 0,1,2)
  - These leiomyomas derive from myometrial cells just below the endometrium.
  - These neoplasms protrude into the uterine cavity.





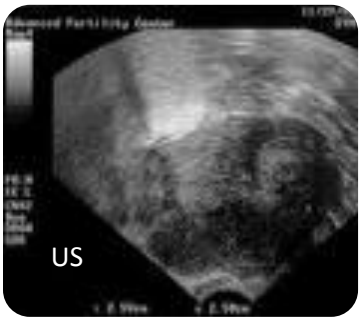


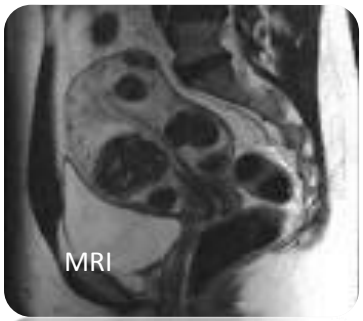



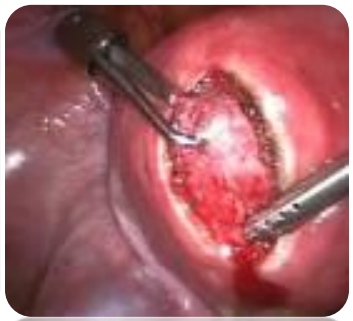
To differentiate between >50% and <50% intramural involvement histologically → when they cut the uterus it is not coming from the muscle itself, it is just pushing it.

Intramural:

- Intramural myomas (FIGO type 3,4,5) –
  - These leiomyomasdevelop from within the uterine wall.
  - They may enlarge sufficiently to distort the uterine cavity or serosal surface.
  - Some fibroids can be transmural and extend from the serosal to the mucosal surface.

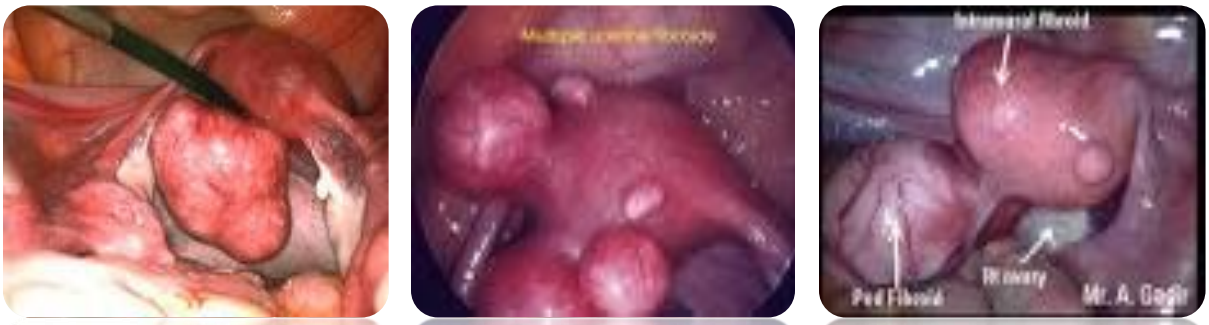







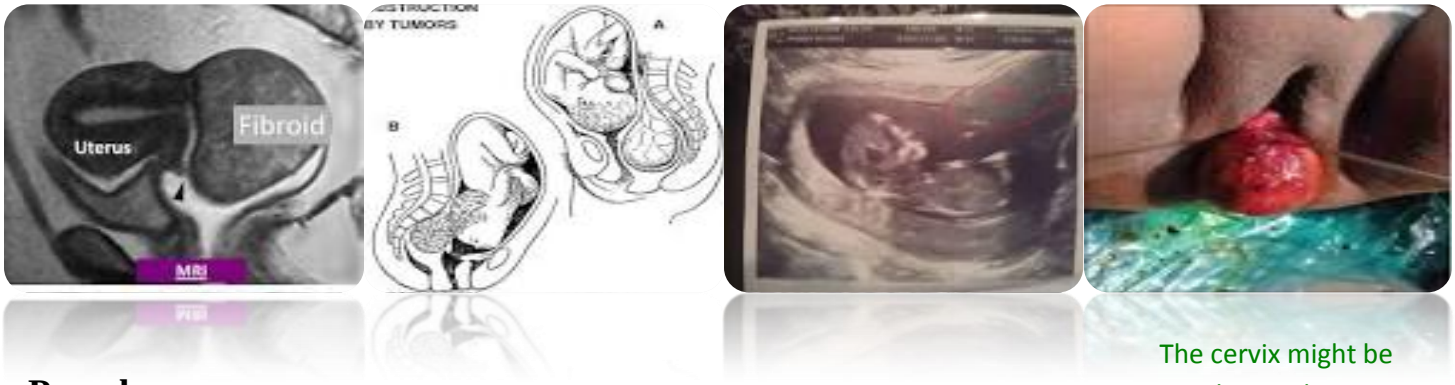
Subserosal:

- Subserosalmyomas (FIGO type 6,7) (the least type bothering the patient unless it is moving and causing irritation to underlying structures. Completely protrude outside the cavity)
  - These leiomyomas originate from the myometrium at the serosal surface of the uterus.
  - They can have a broad or pedunculated(with wide base or small base) base and may be intraligamentary (ie, extending between the folds of the broad ligament).



Cervical:

- Cervical myomas (FIGO type 8) (the least common type. One of the reasons of obstructed labour)
  - These leiomyomas are located in the cervix, rather than the uterine corpus.



The cervix might be mistaken with uterus

Prevelance:

- A hysterectomy study found myomas in 77 percent of uterine specimens
- The epidemiology of leiomyomas parallels the ontogeny and life cycle changes of the reproductive hormones estrogen and progesterone.
- Myomas are clinically apparent in approximately 12 to 25 percent of reproductive age women and noted on pathological examination in approximately 80 percent of surgically excised uteri .
- Most, but not all, women have shrinkage of leiomyomas at menopause.
  (Estrogen and progesterone does not cause fibroid, the initial reason for fibroid origination is not known, estrogen make the fibroid grow)

  (postmenopause = no hormone → fibroids do not grow)

Risk factors: The most important thing is the risk factors

- Race: (very important risk factor)
  - Two- to three-fold greater in black women than in white women
  - The natural history of leiomyomas also differs by race. Most white women with symptomatic fibroids are in their 30s or 40s; however, black women develop symptoms on average four to six years younger and may even present with disease in their 20s
  - Fibroids grow at a slower rate after age 35 in white women, but not in black women (e.g. if there are 100 women waiting in the waiting area, and halve of them are black and the other halve are white, and all of them will examine by US. 50% of black women will have fibroid while only 15% of white will have fibroid even if they are asymptomatic)
  - Compared with white women, black women experience more severe disease based on their symptoms and have more extensive disease at the time of hysterectomy
- Menstrual history and parity. (Any reason that make the woman exposemore to estrogen)(IMP in MCQs)
  - Early menarche (<10 years old) is associated with an increased risk of developing fibroids
  - Prenatal exposure to diethylstilbestrol is associated with an increased risk of fibroids
  - Paritydecreases the chance of fibroid formation (keeping the woman in aprolonged period of estrogen suppression)
  - Early age at first birth decreases risk and a longer interval since last birth increases risk
- Hormonal contraception
  - Use of low dose oral contraceptives (OCs) does not cause fibroids to grow, therefore administration of these drugs is not contraindicated in women with fibroids
  - Long acting progestin-only contraceptives (eg, depot medroxyprogesterone) protect against development of leiomyomas
  - Progestinworks on endometrium not myometrium and the fibroid does not originate from endometrium.
- Heredity
  - Studies imply a familial predisposition to leiomyomas in some women. There is also increasing evidence of specific susceptibility genes for fibroids.
- Ovulation induction agent
  - There are isolated reports of leiomyoma enlargement in women treated with clomiphene
- Obesity
  - Most studies show a relationship between fibroids and increasing body mass index. The relationship is complex and is likely modified by other factors, such as parity, and may be more related to change in body habitus as an adult.
- Diet, Caffeine, Alcohol & smoking
  - Beef and other reds meats (1.7-fold) is associated with an increased relative risk of fibroids and consumption of green vegetables (0.5-fold) and fruit (especially citrus fruit) with a decreased risk, There is increasing evidence that vitamin D deficiency or insufficiency is linked to fibroid risk
  - Consumption of alcohol, especially beer, appears to increase the risk of developing fibroids.
  - Caffeine consumption is not a risk factor.
  - Smoking decreases the risk of having fibroids.

Clinical manifestations:

- Heavy or prolonged menstrual bleeding:(it is important to know the definition of heavy periodmenorrhagia and polymenorrhagia)
  Ask the patient about clot passage, and how many time she change her pads)
    - Most common fibroid symptom, but does not mean that every patient with heavy bleeding has fibroid.
    - If the patient complains of postcoital bleeding (due to irritation of the cervix), think about polyp more.
    - Degree of uterine bleeding are determined by the location of the fibroid, size is of secondary importance.
    - Patient with submucus fibroid bleeds more than patient with intramural fidroid while patient with subserosalmay be silent and does not give any complain.
    - Local cause= more bleeding.
    - Submucosalmyomas that protrude into the uterine cavity (eg, types 0 and I)are most frequently related to significant menorrhagia
- Pelvic pressure and pain
    - Bulk-related symptoms
        - Urinary frequency, difficulty emptying the bladder, and, rarely, urinary obstruction can all occur with fibroids
        - Fibroids that place pressure on the rectum can result in constipation and difficulty in defecation.
        - Back pain may, on occasion, be related to the presence of myomas
        - Very large uteri may compress the vena cava and lead to increase in thromboembolic risk (very rare)
        - Alarming signe→ when the paitent come to the doctor and says: "doctor I can not defecate without putting something to stimulate and bring the stool out". This is due to compression not constipation.
    - Dysmenorrhea
    - Dysmenorrhea is also reported by many women with fibroids(in early reproductive age). This pain in many women appears to be correlated with heavy menstrual flow and/or passage of clots.(Black woman complaining of very bad Dysmenorrhea followed by heavy bleeding = alarming sign)
    - Dyspareunia (painful intercourse) especially if the fibroid tilting the uterus= alarming sign.
        - It is controversial
        - anterior or fundal fibroids are the most likely to be associated with deep dyspareunia.
        -

"Your clinical sense is your guidance"

- Leiomyoma degeneration or torsion"excruciating pain"
    - (A patient come to the doctor and tell her that she knows that she has fibroid but she can not tolerate the pain. The doctor should think about complications of fibroid, one of them degeneration or torsion especially if she has subserous fibroid)
    - Usually intramural does not cause sever pain.
    - The more sever pain comes with pedunculated fibroid.
    - Infrequently, fibroids cause acute pain from degeneration or torsion of a pedunculated tumor.

- Pain may be associated with a low grade fever, uterine tenderness on palpation, elevated white blood cell count, or peritoneal signs.
- The discomfort resulting from degenerating fibroids is self-limited, lasting from days to a few weeks, and usually responds to nonsteroidalantiinflammatory drugs.

❖ Reproductive dysfunction
- Leiomyomas that distort the uterine cavity (submucosalor intramural with an intracavitary component) result in difficulty conceiving a pregnancy and an increased risk of miscarriage.
- Adverse pregnancy outcomes (placental abruption, fetal growth restriction, malpresentation, and preterm labor and birth)
- Poor vascularity of the endometrium covering the fibroid making the implantation very poor.
- If the fibroid distorting the endometrial cavity, it could be a cause of defect in fertility. If the woman is lucky to pregnant she will be at risk of IUGR and preterm labour.

Diagnosis:

❖ Pelvic exam:
- Bimanual pelvic examination, an enlarged, mobile uterus with an irregular contour
- Infrequently, on speculum exam, a prolapsed submucosal fibroid may be visible at the external cervical os

❖ Imaging:
- Ultrasound
  - Transvaginal ultrasound has high sensitivity (95 to 100 percent) for detecting myomas in uterus less than 10 weeks' size
  - Most widely used modality due to its availability and cost-effectiveness
  - Saline infusionsonography (sonohysterography) improves characterization of the extent of protrusion into the endometrial cavity by submucousmyomas
  - What is the first deferential of a large uterus in a reproductive age group? Pregnancy.
  - Always think about normal things then think about the abnormal.
  - The gold standard step to detect submucous fibroids → Saline infusion sonohysterography.

❖ Diagnostic hystroscopy
- Office flexible hystroscope to diagnose submucosmyoma and extend of protrusion to endometrial cavity(without anesthesia)

❖ MRI
- Best modality for visualizing the size and location of all uterine myomas. Due to the expense of this modality, its use is best reserved for surgical planning for complicated procedures.
- MRI is not a gold standard in evaluation of myoma.
- A 20-year-old patient has a big fibroid and the doctor wants to take her for surgery. It is important to evaluate how big this fibroid? Where does it locate? How many fibroids? Is it in the endometrial cavity? When the doctor wants to take her to surgery, she will go for a uterine conservative surgery because the patient is young. In this case MRI is needed.

❖ HSG: (one of the infertility tool measures)
- Good technique for defining the contour of the endometrial cavity.
- If you don't have saline sonohysterography but you have HSG and MRI, which one you will use first? You use the cheaper one, which is HSG.

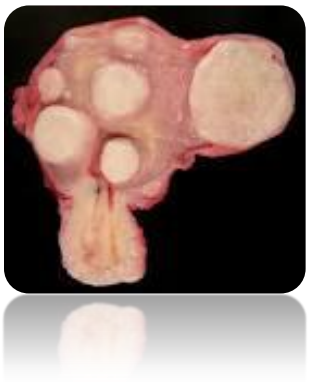
Differential diagnosis:

- Leiomyoma
- Uterine adenomyosis or adenomyoma
- Leiomyoma variant
- Adenomatoid tumors
- Pregnancy
- Hematometra
- Uterine sarcoma
- Uterine carcinosarcoma
- Endometrial carcinoma
- Metastatic disease (typically from another reproductive tract primary

  A 70-year-old patient came with bleeding and large uterus. It is not a fibroid (because it shrink in menopause woman). You have to think about sarcoma.

Pathology:

- Spherical, well circumscribed, white firm lesion with whorled appearance on cut section
- Does not have true capsule… pseudocapsule. (MCQ) (pseudocapsule surround it and there are lymphatic and vascular vessels. When they obstructed or dilated →red degeneration (bleeding inside the fibroid or surrounding the capsule)
- Degenerative changes as the tumor enlarge
  - Hyaline degeneration (Most common)
  - Cystic degeneration
  - Calcification (After menopause)
  - Fatty degeneration (Rare)
  - Red degeneration 5-10% during pregnancy."Due to high vascularity"



Management:(expectant, medical, surgical and interventional)

- Relief of symptoms is the major goal in management
- The type and timing of any intervention should be individualized, based upon the following factors:
  - Type and severity of symptoms
  - Size of the myoma(s)
  - Location of the myoma(s)
  - Patient age
  - Reproductive plans and obstetrical history
  - Do not manage 20-year-old patient even if she has a 10 cm fibroid for hysterectomy. It is important to preserve her uterus.You can go for myomectomy.
  - Do not tell a patient with 3 cm fibroid and bleeding that the cause of bleeding is the fibroid unless it is submucus fibroid.
  - 3 cm submucus fibroid → bleeding.
  - 3 cm intramural fibroid → no bleeding. If it is reach 10 or 20 cm it can cause bleeding + pelvic pressure symptoms.

- **Expectant:**
  - Can shrink substantially during the postpartum period. (Sudden drop in estrogen level the fibroid will shrink. patient with 8 cm fibroid with an exposure to estrogen during pregnancy it is expected to grow more. When the patient is going to CS for any reason do not be ahero and remove the fibroid, there will be too much bleeding. The causes are: 1st hypervascular status during pregnancy. 2nd thing the fibroid itself is vascular (bleeding surgery). If it is not complicated leave it and observe the patient postpartum it will shrink due to decrease in estrogen level.
  - Initial imaging study (usually an ultrasound) to confirm that a pelvic mass is a fibroid and not an ovarian mass.(As abase line then follow up)
  - Annual pelvic exams and, in patients with anemia or menorrhagia, check a complete blood count.(First thing, it is important to control the bleeding)
  - If symptoms or uterine size are increasing, we proceed with further evaluation and patient counseling regarding treatment options.
  - Rule out other causes of menorrhgea
    - Hypothyrodism.(Routine evaluation in all patients in reproductive age group, TSH)
    - Bleeding disorders.

- **Medical therapy:**
  - Gonadotropin-releasing hormone agonists
    - Most effective medical therapy for uterine myomas.
    - Work by initially increasing the release of gonadotropins, followed by desensitization and downregulation to a hypogonadotropic, hypogonadal state that clinically resembles menopause.
    - Most women will develop amenorrhea, improvement in anemia and a significant reduction (35 to 60 percent) in uterine size within three months of initiating this therapy.
- GnRh agonist side effect
  - Rapid resumption of menses and pretreatment uterine volume after discontinuation of GnRH agonists.
  - Hot flashes, sleep disturbance, vaginal dryness, myalgias and arthralgias, and possible impairment of mood and cognition [15]. Bone loss leading to osteoporosis after long-term (12+ months) use is the most serious complication and most often limits therapy. (To prevent osteoporosis → give low dose of combined oral contraceptive pills like Climenor Progyluton. It will not affect the fibroid growth and it will protects the bone + Vit D andCa++)
- Used as preoperative therapy.
  - GnRH agonists are approved for administration for three to six months prior to leiomyoma-related surgery in conjunction with iron supplementation to facilitate the procedure and enable correction of anemia.
  - Reduction in uterine size can facilitate subsequent surgery by reducing intraoperative blood a transverse (rather than vertical) abdominal incision, or a minimally-invasive procedure.
  - Shrink the fibroid size.
- Gonadotropin-releasing hormone antagonists
  - Similar clinical results have been achieved with GnRH antagonists, which compete with endogenous GnRH for pituitary binding sites.
  - The advantage of antagonists over agonists is the rapid onset of clinical effects without the characteristic initial flare-up observed with GnRH agonist treatment.

- **Interventional radiology:**
  - A patient does not want to go for surgery and she did not respond to medical treatment and she is still suffering → use interventional radiology.
  - Uterine artery embolization
    - minimally invasive option for management of leiomyoma-related symptoms, excellent technical and clinical success has been reported.
    - It is an effective option for women who wish to preserve their uterus and are not interested in optimizing future fertility. (Also for old women as alternative therapy).
    - UFE results in shrinkage of myomas of approximately 30 to 46 percent

Magnetic resonance guided focused ultrasound:MRI→ estimate the myometrium invasion , hint to plan for surgery.

- More recent option for the treatment of uterine leiomyomas in premenopausal women who have completed childbearing.
- This noninvasivethermoablative technique converges multiple waves of ultrasound energy on a small volume of tissue, which leads to its thermal destruction.

- **Surgical therapy:**
  - Myomectomy
    - Myomectomy is an option for women who have not completed childbearing or otherwise wish to retain their uterus.
    - Disadvantage of this procedure is the risk that more leiomyomas will develop from new clones of abnormal myocytes and bleeding.
    - Hysteroscopic myomectomy is the procedure of choice for removing intracavitarymyomas(submucus fibroid specifically)

  - Hystrectomy(definitive treatment of fibroid)
    - Women with acute hemorrhage who do not respond to other therapies
    - Women who have completed childbearing and have current or increased future risk of other diseases.
    - Women who have failed prior minimally invasive therapy for leiomyomas
    - Women who have completed childbearing and have significant symptoms, multiple leiomyomas, and a desire for a definitive end to symptomatology.

Endometrial cancer:

- Epidemiology:
  - Most common gynecological cancer in the developed countries, with an incidence of 12.9 per 100,000 women and a mortality rate of 2.4 per 100,000.
  - In developing countries, it is the second most common gynecologic malignancy, with an incidence of 5.9 per 100,000 and a mortality rate of 1.7 per 100,000.
  - The average age of diagnosis of uterine cancer in the US is 61 years old
  - From ages 50 to 70, women have a 1.4% risk of being diagnosed with uterine cancer
  - Women in the US have a 2.6% lifetime risk of developing uterine cancer

Histological types:

- **Two histologic categories:**
- Type I tumors: (85% of cases)
  - Type I tumors include tumors of endometrioid histology that are grade 1 or 2; these comprise approximately 80 percent of endometrial carcinomas. These tumors typically have a favorable prognosis, are estrogen-responsive, and may be preceded by an intraepithelial neoplasm (atypical and/or complex endometrial hyperplasia).
- Type II tumors:(non endometrioid tumor)(25%)(behave like ovarian cancer when it is papillary serous)
  - Account for 10 to 20 percent of endometrial carcinomas. They include grade 3 endometrioid tumors & serous, clear cell, mucinous, squamous, transitional cell, mesonephric, and undifferentiated. These tumors are often high-grade, have a poor prognosis, and are not clearly associated with estrogen stimulation. A precursor lesion is rarely identified.

Risk factors:The most important thing is the risk factors

- Type I … estrogen dependent tumor
  - Exogenous estrogen
    - Tamoxifen(All patients coming to you taking Tamoxifen for breast cancer in the first evaluation, give themMirenato prevent endometrial cancer)
    - Unopposed systemic estrogen therapy(progesterone has protective effect of any estrogen. To protect the endometrium from the effect of estrogen it is better to give progesterone in the second half of the cycle or give Mirena)
    - Postmenopausal estrogen therapy
    - Phytoestrogen
  - Endogenous estrogen
    - Chronic anovulation(PCOs)
    - Early menarche --- late menopause
    - Obesity (Fat → estrogen)
    - Estrogen secreting tumors(granulosa cell tumor)
- Family history and genetics predisposition
  - Lynch syndrome (hereditary nonpolyposis colorectal cancer)
    - Autosomal dominant caused by a germline mutation in one of several DNA mismatch repair genes
    - Develop the disease at a young age.
    - Accounts for two to five percent of all endometrial carcinomas.

        - Women with Lynch syndrome, the lifetime risk of endometrial carcinoma is 27 to 71 percent compared with 2.6 percent in the general population
        - Mean age of diagnosis of endometrial cancer 46-54yrs
- BRCA I mutation
    - BRCA1 mutation carriers reported a significant increase in the risk of uterine cancer (RR 2.65, 95% CI 1.69-4.16).
    - Data from a prospective series suggested that the risk of endometrial carcinoma was significantly elevated only for BRCA mutation carriers taking tamoxifen.

Associated factors:

- Nulliparity and infertility
    - The risk of endometrial carcinoma is inversely related to parity.
    - Nulliparity and infertility do not appear to independent risk factors for endometrial carcinoma; instead, the association is probably with the high frequency of anovulatory cycles in infertile women.
    - Data are inconsistent regarding whether ovulation induction for treatment of infertility is associated with an increased risk of endometrial carcinoma.
- Diabetes and hypertension: (DM does not cause endometrial cancer but it is associated with it)
    - Women with diabetes mellitus and hypertension are at increased risk for endometrial carcinoma.
    - Comorbid factors, primarily obesity, account for much of this risk, but some studies have found independent effects, as well.
    - The risk of developing endometrial carcinoma is higher in type 2 than type 1 diabetics. Diets high in carbohydrates and associated hyperinsulinemia, insulin resistance, and elevated levels of insulin-like growth factors may play a role in endometrial proliferation and development of endometrial carcinoma; this is an area of active investigation
- Breast cancer
    - A history of breast cancer is a risk factor for development of endometrial carcinoma, clearly in women treated with tamoxifen

Protective factors:

- Hormonal contraceptives
    - The use of estrogen-progestin oral contraceptives (OCs) (combined)decreases the risk of endometrial carcinoma by 50 percent or higher. Estrogen alone is causing cancer.
    - The benefit of hormonal contraceptives is likely due to the progestin(only) component, which suppresses endometrial proliferation.
    - Studies have found that progestin-only contraceptives provide endometrial protection against development of endometrial neoplasia
- Increasing age at last birth
    - Childbearing at an older age, independent of parity and other factors, was associated with a decreased risk of endometrial carcinoma. As an example, women who last gave birth at age 35 to 39 years had a 32 percent decrease in risk (95% CI 0.61-0.76).
- Smoking
    - Cigarette smoking is associated with a decreased risk of developing endometrial carcinoma in postmenopausal women
- Physical activity
- Coffee and tea.

Clinical presentation/manifestation:

- Abnormal uterine bleeding
  - Suspicion of the presence of endometrial neoplasia (neoplastic endometrial hyperplasia or carcinoma) depends upon symptoms, age, and the presence of risk factors.
  - Abnormal uterine bleeding is present in approximately 75 to 90 percent of women with endometrial carcinoma but only 10 % of patients with bleeding are diagnosed with endometrial cancer because the most common cause of postmenopausal bleeding is atrophy.
  - The amount of bleeding does not correlate with the risk of cancer.
  - A 45-year-old patient, infertile, she has PCO and started the treatment. She now has bleeding. Endometrial biopsy done for her→ cancer
  - Any patient >35-year-old presenting with abnormal bleeding → consider endometrial biopsy in the work up.
- Postmenopausal women
- Any bleeding, including spotting or staining. Three to 20 percent of women with postmenopausal bleeding are found to have endometrial carcinoma and another 5 to 15 percent have endometrial hyperplasia. (IMP)
- Age 45 to menopause
  - Any abnormal uterine bleeding, among cases of endometrial carcinoma, 19 percent occur in women aged 45 to 54 years compared with 6 percent in those aged 35 to 44 years.
- Younger than 45 years
  - Abnormal uterine bleeding that is persistent, occurs in the setting of a history of unopposed estrogen exposure (obesity, chronic anovulation) or failed medical management of the bleeding, or in women at high risk of endometrial cancer (Lynch syndrome)→you have to do endometrial biopsy.
- Abnormal PAP smear:
  - Adenocarcinoma – Adenocarcinoma is sometimes seen on cervical cytology. Since the malignant cells may arise from either the cervix or endometrium, further evaluation with cervical and endometrial biopsy is required.
  - Atypical glandular cells - Atypical glandular cells detected by cervical cytology should be investigated withan endometrial (and endocervical) biopsy to determine whether an endometrial neoplasm is the cause.
  - Endometrial cells – The presence of endometrial cells on cervical cytology is reported in the results in women ≥40 years of age. The appearance of normal endometrial cells on cytology in asymptomatic premenopausal women is rarely associated with pathology and no further work-up is required.
- Incidental finding on imaging
  - A thickened endometrial lining is sometimes found incidentally on ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) performed for another indication.
  - Endometrial thickening in postmenopausal woman not exceeding 3 mm.
- Incidental finding at hysterectomy
  - Endometrial carcinoma or hyperplasia is sometimes discovered incidentally when hysterectomy is performed for benign disease.
  - Prior to hysterectomy, all women with abnormal uterine bleeding should have endometrial sampling (biopsy) to rule out cancer.

Work up:

- Calm the patient, reassure her, treat her and protect her.
- Endometrial sampling
  - Office endometrial biopsy.
  - Can be performed without anasthesia.
- D&C in some women.
  - Cannot tolerate an office biopsy
  - Those with heavy bleeding (D&C is both a diagnostic and therapeutic procedure),
  - Hysteroscopy with D&C to ensure that focal lesions are identified and biopsied.
- Both give the same specificity and sensitivity.

What about screening:

- Routine screening is not advisable except for women known with Lynch syndrome and if they are still bleeding.

Management:

- Endometrial cancer is surgically staged disease while cervical cancer is clinically staged disease
- Further management depends on the stage
- Basic surgery include
  - Total hystrectomy
  - Bilateral salpengo-oherectomy
  - Bilateral pelvic lymphadenectomy
  - Para-aortic lymphadenectomy
  - Omentectomy and peritoneal washing in type II

Preoperative work up:

- In endometrial biopsy
  - Tumor histology type(endometrioid, papillary serous or carcinosarcoma)
  - Tumor grade
    - Risk of lymph node involvement
      - G1 3% Pelvic… 2% aortic
      - G2 9% pelvic…5% aortic
      - G3 18% pelvic… 11% aortic

FIGO stage:

- Stage I
  - Tumor confined to the uterus
    - IA.. Less than 50% myometrial invasion
    - IB … more than 50% myometrial invasion
- Stage II
  - Invading cervical stroma but does not extend beyond the uterus
- Stage III
  - Tumor extend beyond the uterus
    - IIIA… serosa of the uterus and or adnexa
    - IIIB …vagina or parametrial involvement
    - IIIc…lymph nodes
      - IIIc1 pelvic lymph nodes
      - IIIc2 para-aortic lymph nodes
- Stage IV
  - IVA… bladder or bowel mucosa(If it is not invading the mucosa it is not considered metastatic to the bowel)
  - IVB… abdominal metastasis or inguinal lymph node

Why we need to stage? For treatment and prognosis.

Endometrial cancer → if stage 1A (85% of patients in this stage) → treatment is surgical and the prognosis 5 years survival reaching up to 95%.

When to give radiation:

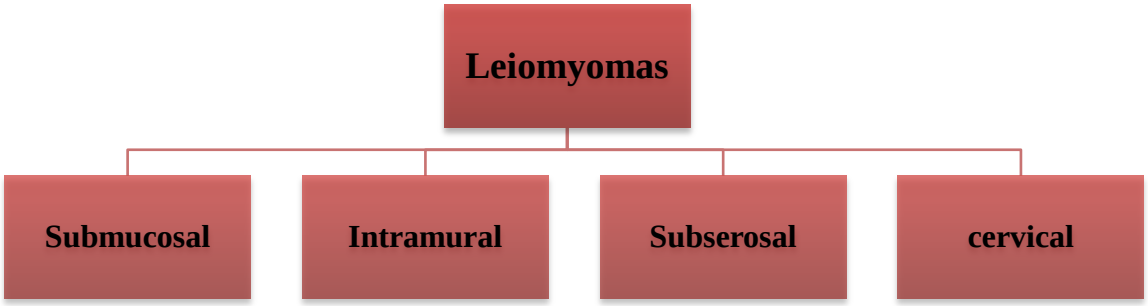
- Types of radiation
  - External beam radiotherapy
  - Brachytherapy (put a device through the vagina and give a radiation)

Summary:

-Endometrial neoplasm includes both benign and malignant.

*__Leiomyomas (__Benign) are the most common neoplasm of the uterus.

-**Types of leiomyoma**:



-**Risk factors include**: race (more in black women),menstrual history and parity, hormonal contraception, heredity, ovulation induction agent, obesity, diet, caffeine, alcohol and smoking.

- **Clinical manifestation**: Heavy or prolonged menstrual bleeding, Pelvic pressure and pain,Leiomyoma degeneration or torsion and Reproductive dysfunction.

-Diagnosis: pelvic exam, imaging, Diagnostic hysteroscopy, MRI and HSG.

-**Differential diagnosis**: leiomyoma, uterine adenomyosis or adenomyoma, leiomyoma variant, adenomatoid tumors, pregnancy, hematometra, uterine sarcoma, uterine carcinosarcoma, endometrial carcinoma, metastatic disease.

-**Pathology:**Spherical, well circumscribed, white firm lesion with whorled appearance on cut section, does not have true capsule (pseudocapsule).

-**Management:**expectant(Relief of symptoms is the major goal in management), medical, surgical and interventional.

***Endometrial cancer:**

-Histological types

Type I	Type II
Endometrioid tumors	Non endometrioid tumors
85% of cases	25% of cases
Estrogen dependent	Non estrogen dependent
Favorable prognosis	Poor prognosis

-Risk factors for type I: exogenous and endogenous exposure to estrogen, family history and genetics predisposition(Lynch syndrome) and BRCA I mutation.

-**Associated factors:**nulliparity and infertility, diabetes and hypertension and breast cancer.

-**Protective factors:** hormonal contraceptives, increasing age at last birth, smoking, physical activity, coffee and tea.

-Clinical presentation/ manifestation: abnormal uterine bleeding, postmenopausal bleeding,age 45 to menopause, younger than 45 years,abnormal PAP smear, incidental finding on imaging and incidental finding at hysterectomy.

-Work up:calm the patient, reassure her, treat her and protect her. Endometrial sampling or D&C(Cannot tolerate an office biopsy).

-Management: endometrial cancer is surgically staged disease. Basic surgery include: Total hysterectomy, bilateral salpengo-oherectomy, bilateral pelvic lymphadenectomy, para-aortic lymphadenectomy, omentectomy and peritoneal washing in type II.

Gestational TrophblasticNeoplasia (GTN)

History of GTN:

- The first record of gestational trophoblastic disease (GTD) probably dates to 400 BC, when Hippocrates described "dropsy of the uterus"
- In 1276, the attendants of Margaret Countess of Henneberg noticed that her abnormal delivery consisted of multiple hydropic vesicles

Definition:

- GTN defines a heterogeneous group of lesions that represent an aberrant fertilization event
- The pathogenesis is unique because the maternal tumor arises from fetal tissue
- It is the most curable gynecologic malignancy

In tumors in general the person's own cells invade tissues and metastasize whereas in GTN it is the embryo's cells responsible for the invasion and metastases.

Introduction:

Clinical spectrum that includes all neoplasms that derives from abnormal placental (trophoblastic) proliferation.

A. Benign disease:hydatidiform molar pregnancy (most common)

B. Malignant disease:

1. Invasive trophoblastic disease, choriocarcinoma, placental site trophoblastic tumors
2. 20% of patients with benign molar disease develop malignant disease

Classification:

A complete mole is more likely to become malignant than a partial mole

- Benign:
    1. Partial mole:
    2. Complete mole:
- Malignant:
    1. Persistent / Invasive GTD
    2. Choriocarcinoma
    3. Placental site trophoblastic tumors

Epidemiology:

- Less than 1 in 1000 pregnancies in most of the world, 2 in 1000 in Japan (differences in diet)
- Vitamin A deficiency in the rhesus monkey produces degeneration of the seminiferous epithelium with production of primitive spermatogonia and spermatocytes

Studied incidence in immigrants (Japanese) living in the USA and discovered that these immigrants have the same risk as the population they live in that means it is related to environment not genetics

Risk Factors:

- Women <15 years or ≥40 years of age getting pregnant
- Patients with previous history of molar pregnancy
- Possible other factors: deficiency of animal fat, Vitamin A and carotene, professional occupation, history of prior spontaneous abortion

Complete and Partial Moles:

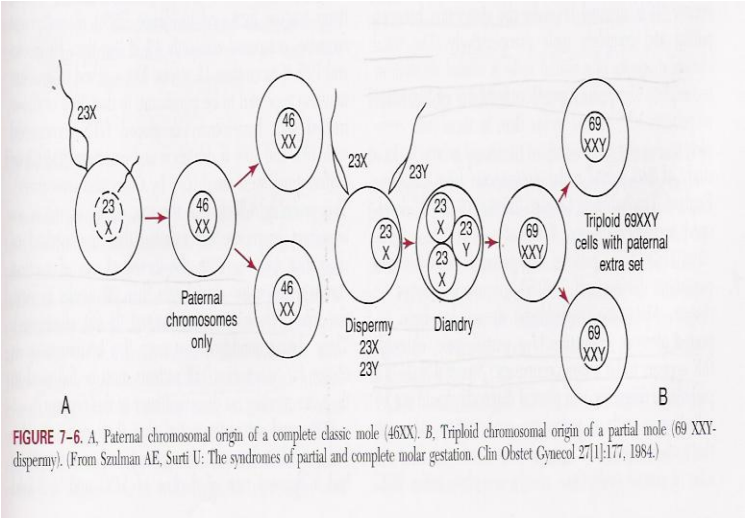


FIGURE 7-6. A, Paternal chromosomal origin of a complete classic mole (46XX). B, Triploid chromosomal origin of a partial mole (69 XXY-dispermy). (From Szulman AE, Surti U: The syndromes of partial and complete molar gestation. Clin Obstet Gynecol 27[1]:177, 1984.)

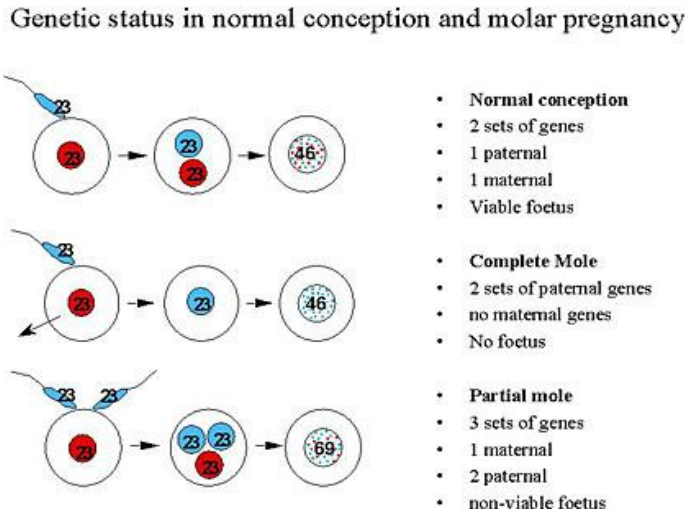


Table II-6-1. Benign Gestational Trophoblastic Neoplasia—H Mole

Complete	Incomplete
Empty egg	Normal egg
Paternal X's only	Maternal and paternal X's
46,XX (**di**ploidy)	69,XXY (**tri**ploidy)
Fetus absent	Fetus nonviable
20% → malignancy	10% → malignancy
No chemotherapy; serial β-hCG titers until (–); follow-up 1 year on oral contraceptive pill	

Even though in the complete mole the number of chromosomes might seem normal but they are all paternal unlike the incomplete (partial) mole

Symptoms and signs:

- Vaginal bleeding prior to 16 weeks' gestation is the **most common** symptom and passage of vesicles from the vagina.
- Patients with complete mole may have: first trimester pre-eclampsia, hyperthyroidism, hyperemesis, increased uterine size and theca-lutein cysts
- The **most common** sign is fundus larger than dates, absence of fetal heart tones, bilateral cystic enlargements of the ovary known as **theca-lutein cysts**.
- Patients with partial moles are diagnosed clinically as missed or incomplete abortion
- Excessive nausea/ emesis (because of high β-hCG levels)

One of the differential diagnosis to bleeding in the first trimester is molar pregnancy

TABLE 46.1 Features of Partial and Complete Hydatidiform Moles

Feature	Partial Mole	Complete Mole
Karyotype	Triploid	46,XX, rarely 46,XY
Pathology		
Fetus	Often present	Absent
Amnion, fetal RBCs	Usually present	Absent
Villous edema	Variable, focal	Diffuse
Trophoblastic proliferation	Focal, slight to moderate	Diffuse
Clinical presentation		
Diagnosis	Missed abortion	Molar gestation
Uterine size	Small or appropriate for gestational age	50% large for gestational age
Theca lutein cysts	Rare	>25% depending on diagnostic modality
Medical complications	Rare	Becoming rare with early diagnosis
Postmolar invasion and malignancy	<5%	15% and 4% respectively

RBCs, red blood cells.
(Table modified from *ACOG Practice Bulletin #53* June 2004. Updated information from Berkowitz RS, Goldstein DP. Gestational trophoblastic disease. In: Hoskins WJ, Perez CA, Young RC, eds. *Principles and Practice of Gynecologic Oncology*, 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2005:1057–1061.)

β-hCG Assays:

- The family of pituitary and placental glycoprotein hormones: HCG, FSH, LH and TSH, all have a common α-subunit and a distinct β-subunit
- Many β-hCG assays are available, some detect intact β-hCG and others are selective for individual fragments
- The competitive RIA using a polyclonal antibody recognizing all forms of β-hCG remains a gold-standard assay for use in the management of GTD
- The amount of hCG produced corresponds with tumor volume so that a serum hCG of 5 IU/L corresponds to approximately 10,000 to 100,000 viable tumor cells.

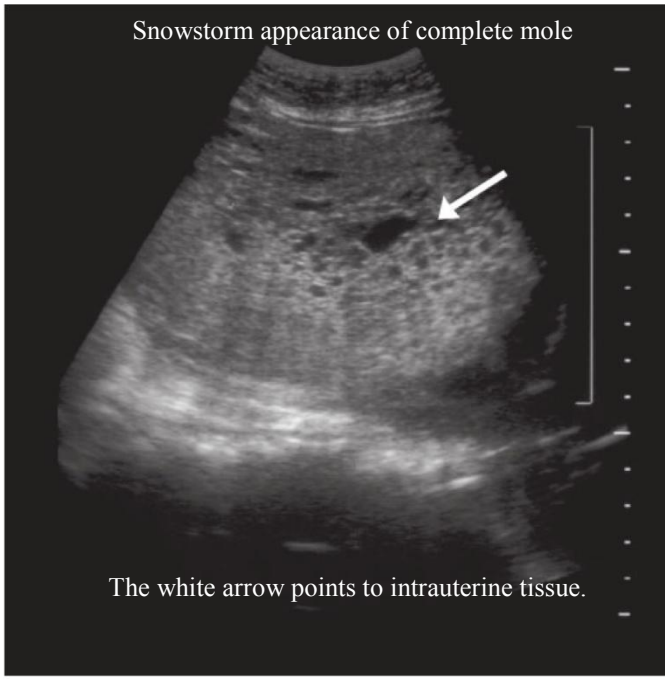
Diagnosis:

- In many patients the first evidence to suggest the presence of a hydatidiform mole is the passage of vesicular tissue
- A quantative pregnancy test (hCG levels) of greater than 100,000 IU/L, an enlarged uterus, and vaginal bleeding suggest a diagnosis of a hydatidiform mole
- Ultrasound is the **test of choice** will show multiple echoes (snowstorm)

Management:

- Evacuation curettage: the method of evacuation
- RH –ve patients should receive Rhogam
- IV oxytocin should be administered after a moderate amount of the tissue has been removed
- Complications may include: uterine perforation, hemorrhage, and trophblastic embolization
- Hysterectomy may be selected as a method of evacuation in patients who desire sterilization
- Baseline quantitative β-hCG titer
- Chest X-ray to rule out lung metastasis

Place the patient on effective contraception (oral contraceptive pills) for the duration of the follow-up period to ensure no confusion between rising b-hCG titers from recurrent disease and normal pregnancy.



Contraception:

- In a systemic review of the influence of Oral Contraceptive Pills (OCP) in the development of post-molar trophblasticneoplasia, two randomized controlled trials (RCT) where included for analysis.
- There was no clear evidence for an association between OCP use and the incidence of GTN was found.

Table 7–1. MANAGEMENT OF HYDATIDIFORM MOLE

1. β-hCG determination every 1–2 weeks until negative twice
   a. Then bimonthly for 1 year
   b. Contraception for 6–12 months
2. Physical examination, including pelvic every 2 weeks until remission
   a. Then every 3 months for 1 year
3. Chest film initially
   a. Repeat only if hCG titer plateaus or rises
4. Chemotherapy started immediately if:
   a. hCG titer rises or plateaus during follow-up
   b. Metastases are detected at any time

hCG, human chorionic gonadotropin.

Gestational TrophblasticNeoplasia (GTN):

- The hydatidiform mole precedes malignant disease in 50% of patients. There is an antecedent normal pregnancy in 25% of the patients and an abortion or ectopic pregnancy in the other 25%.
- In many patients the preceding pregnancy occurred years before.
- In other cases patients with GTN may have no localized disease in the uterus and have only metastatic disease.

The **most common site of distant metastasis** is the **lungs**.

Invasive Hydatidiform Mole:

- It is clinically identified by the combination of an abnormal uterine ultrasound scan and a persistent or rising β-hCG level after uterine evacuation of a molar pregnancy
- Pathologic confirmation of invasion is rarely required.

Choriocarcinoma:

- Highly malignant
- Greater risk of hemorrhage and metastases
- May arise from any type of pregnancy

Table 7–2. CLASSIFICATION OF GESTATIONAL TROPHOBLASTIC NEOPLASIA

Nonmetastic disease: no evidence of disease outside the uterus
Metastatic disease: any disease outside the uterus
Good prognosis metastatic disease
1. Short duration (last pregnancy <4 months)
2. Low pretreatment hCG titer (<100,000 IU/24 hr or <40,000 mIU/ml)
3. No metastasis to brain or liver
4. No significant prior chemotherapy

Poor prognosis metastatic disease
1. Long duration (last pregnancy >4 months)
2. High pretreatment hCG titer (>100,000 IU/24 hr or >40,000 mIU/ml)
3. Brain or liver metastasis
4. Significant prior chemotherapy
5. Term pregnancy

hCG, human chorionic gonadotropin.

NonmetastaticTrophblastic Disease:

- Disease is limited to the uterus
- Patients can be treated with single agent chemotherapy
- Treatment is 100% successful
- Single agent **methotrexate** or **actinomycin D** is the treatment of choice

Good Prognosis Metastatic Trophoblastic Neoplasia:

- Therapy can be the same as that described for nonmetastatic disease
- Methotrexate is considered by many to be the drug of choice
- If resistant to methotrexate occurs, patients are switched to actinomycin D

WHO Prognostic Scoring System:

- Patients who score between 0 and 6 receive low-risk chemotherapy
- Patients scoring **7 or more** are given high-risk treatment

Table 44–5. WHO Prognostic Scoring System as Modified by FIGO

Scores	0	1	2	4
Age (years)	<40	≥40	–	–
Antecedent pregnancy	Mole	Abortion	Term	
Interval (months) from index pregnancy	<4	4–<7	7–<13	≥13
Pretreatment serum human chorionic gonadotropin (IU/ L) level	$<10^3$	10^3–$<10^4$	10^4–$<10^5$	$\geq 10^5$
Largest tumor size (including uterus)	–	3–<5 cm	≥5 cm	–
Site of metastases	Lung	Spleen, kidney	Gastrointestinal	Brain, liver
Number of metastases identified	–	1-4	5-8	>8
Previous chemotherapy failed	–	–	Single drug	Two or more drugs

Work Up of Gestational TrophblasticNeoplasia:

Table II-6-3. Gestational Trophoblastic Neoplasia—Basic Approach

β-hCG titer	Baseline for future comparison
Chest x-ray	Lung metastasis is ruled out
Suction D&C	Empty uterus contents
Oral contraceptive pills for 1 year	Prevent confusion: recurrent disease and normal pregnancy

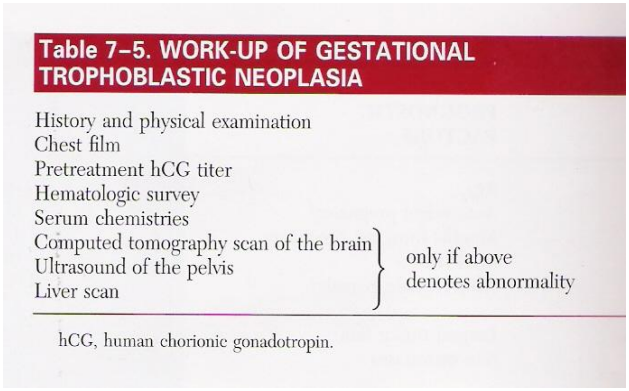


Table 7–5. WORK-UP OF GESTATIONAL TROPHOBLASTIC NEOPLASIA

History and physical examination
Chest film
Pretreatment hCG titer
Hematologic survey
Serum chemistries
Computed tomography scan of the brain
Ultrasound of the pelvis
Liver scan
} only if above denotes abnormality

hCG, human chorionic gonadotropin.

Chemotherapy:

GTN is Sensitive to chemotherapy

a. Single-agent chemotherapy (for treating nonmetastatic disease)

i. Methotrexate or actinomycin D

ii. Cure rate up to 100%

b. Combined chemotherapy for treatment of metastatic disease International Federation of Gynecology and Obstetrics (FIGO) score≥7

i. EMACO [**E**toposide, **M**ethotrexate, **A**ctinomycin D, **C**yclophosphate, **O**ncovin]

ii. Cure rate up to 80%-90%

c. Adjunctive radiotherapy is used for patients with brain metastasis

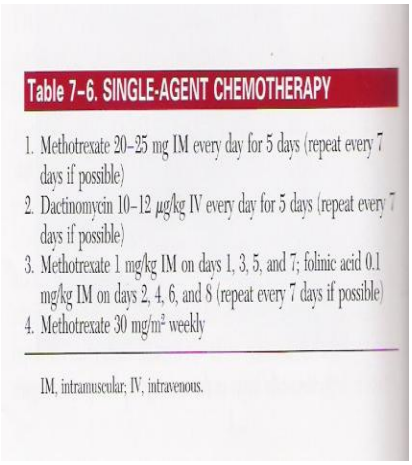


Table 7–6. SINGLE-AGENT CHEMOTHERAPY

1. Methotrexate 20–25 mg IM every day for 5 days (repeat every 7 days if possible)
2. Dactinomycin 10–12 μg/kg IV every day for 5 days (repeat every 7 days if possible)
3. Methotrexate 1 mg/kg IM on days 1, 3, 5, and 7; folinic acid 0.1 mg/kg IM on days 2, 4, 6, and 8 (repeat every 7 days if possible)
4. Methotrexate 30 mg/m² weekly

IM, intramuscular; IV, intravenous.

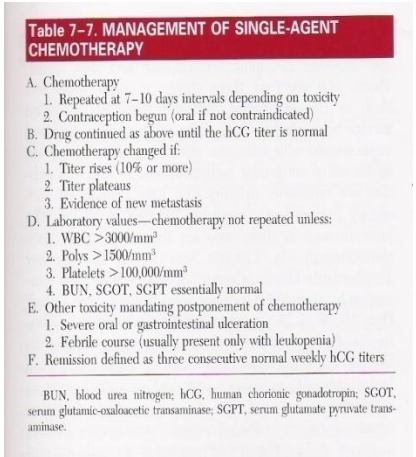


Table 7–7. MANAGEMENT OF SINGLE-AGENT CHEMOTHERAPY

A. Chemotherapy
   1. Repeated at 7–10 days intervals depending on toxicity
   2. Contraception begun (oral if not contraindicated)
B. Drug continued as above until the hCG titer is normal
C. Chemotherapy changed if:
   1. Titer rises (10% or more)
   2. Titer plateaus
   3. Evidence of new metastasis
D. Laboratory values—chemotherapy not repeated unless:
   1. WBC >3000/mm³
   2. Polys >1500/mm³
   3. Platelets >100,000/mm³
   4. BUN, SGOT, SGPT essentially normal
E. Other toxicity mandating postponement of chemotherapy
   1. Severe oral or gastrointestinal ulceration
   2. Febrile course (usually present only with leukopenia)
F. Remission defined as three consecutive normal weekly hCG titers

BUN, blood urea nitrogen; hCG, human chorionic gonadotropin; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamate pyruvate transaminase.

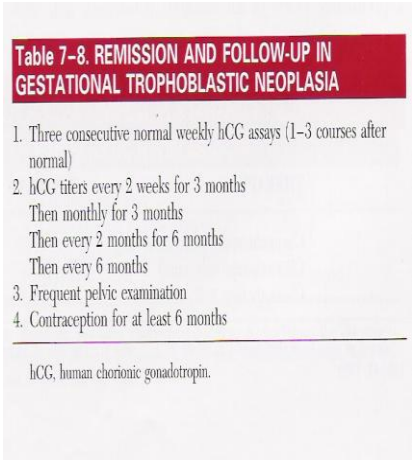


Table 7–8. REMISSION AND FOLLOW-UP IN GESTATIONAL TROPHOBLASTIC NEOPLASIA

1. Three consecutive normal weekly hCG assays (1–3 courses after normal)
2. hCG titers every 2 weeks for 3 months
   Then monthly for 3 months
   Then every 2 months for 6 months
   Then every 6 months
3. Frequent pelvic examination
4. Contraception for at least 6 months

hCG, human chorionic gonadotropin.

Drug-resistant disease:

CT of the chest and abdomen together with MRI of the brain and pelvis is often helpful and can detect deposits not previously seen.

Sites of metastasis: **lungs**, vagina, CNS, kidney, liver.

The role of repeat uterine evacuation in the management of persistent GTD:

- After a second uterine evacuation 68% of the patients (368 patients) had no further evidence of persistent disease and did not require chemotherapy
- Chemotherapy was more likely when the hCG level is ›1500 IU/L
- Third evacuation is not recommended

Poor Prognosis Metastatic Trophoblastic neoplasia:

- Multiple agent chemotherapy is recommended in this disease
- EMA-CO is considered the regimen of choice in most high-risk patients (Etoposide, Methotrexate, Actinomycin D, Cyclophosphamide, Vincristin)
- The overall survival rate for these patients is 80-85%
- Patients with cerebral or hepatic metastases are treated concurrently with radiotherapy for the whole brain or liver (for hemostasis)
- Surgery is not necessary in most patients, it may play a role in cases of tumor

Persistent Low HCG Levels:

- Pituitary HCG
- False +ve HCG results
- Quiescence GTD

Placental Site Trophoblastic Tumor:

- Rare tumors (account for 0.23% cases of GTD)
- It has a variety of clinical features and its course is unpredictable
- Can appear shortly after termination of pregnancy or years later
- Hysterectomy is considered optimal therapy and is usually adequate in most situations
- Chemotherapy can still play a major role

From Step Up

- Secretes small amounts of hCG
- Rarely metastatic
- Resistant to standard chemotherapy

Future childbearing:

- After treatment of GTN, molar pregnancies occur in only about 1-2% of subsequent pregnancies
- These patients should be evaluated with a first trimester ultrasonography
- Pregnancy outcome in women with history of molar gestation is similar to those with no such history
- Standard chemotherapy protocols have minimal impact on the subsequent ability to reproduce

Ovarian Tumors

❖ Ovarian Tumors

- It is estimated that 5-10% of women will undergo a surgical procedure for a suspected ovarian neoplasm during their lifetime
- The majority of these neoplasms are benign
- Age is the most important factor for determining the potential for malignant change; the older the woman, the higher chance for malignancy.

❖ Ddx Of Adnexal Mass(Adnexa = Ovary + fallopian tube)

Table 10–1. DIFFERENTIAL DIAGNOSIS OF ADNEXAL MASS

ORGAN	CYSTIC	SOLID
Ovary	Functional cyst Neoplastic cyst Benign Malignant Endometriosis	Neoplasm Benign Malignant
Fallopian tube	Tubo-ovarian abscess Hydrosalpinx Parovarian cyst	Tubo-ovarian abscess Ectopic pregnancy Neoplasm
Uterus	Intrauterine pregnancy in a bicornuate uterus	Pedunculated or interligamentous myoma
Bowel	Sigmoid or cecum distended with gas or feces	Diverticulitis Ileitis Appendicitis Colonic cancer
Miscellaneous	Distended bladder Pelvic kidney Urachal cyst	Abdominal wall hematoma or abscess Retroperitoneal neoplasm

❖ Functional/Physiological Cysts

- They are related to the process of ovulation.
- Normal cycle: follicular growth > ovulation > corpus luteum. (Anything can happen during this process)
- These cysts are benign and represent an exaggerated physiologic response of the ovary
- Corpus luteum, Follicular and Theca-lutein cysts
- They are the most common clinically detectable enlargement of the ovary occurring during the reproductive years
- They can reach a size as large as 10 cm in diameter
- The cysts usually resolve within a few days to 2 weeks since they are physiological. However, they can persist longer.
- We don't operate on them! In fact, operating on them might be considered as malpractice since there is a risk of taking out healthy tissue which might lead to adhesions >> jeopardizing fertility. 2
- Risk factor: pts with fertility problems on ovulation induction.

❖ Ovarian Neoplasms

- Unrelated to menstrual cycle.
- 20% of all ovarian neoplasms are malignant. Most are benign!
- Most of these neoplasms are asymptomatic unless they have subject to rupture (very rare) or torsion (pt present with severe ischemic pain + N/V).
- Because it is mostly asymptomatic and there is no screening test, pts mostly present AT A LATE STAGE!
- Abdominal distension/pressure is the presenting symptom,; they grow slowly and it might take months.
- They can be cystic or solid tumors.

➢ **Physical examination:** Solid or cystic? Fixed or mobile? Any ascites (indicative of Ca)?

- Solid fixed irregular masses are suspicious for CA
- Predictive value of the examination alone improves as the patient ages since risk of malignancy is high.

➢ **CA 125 and non-malignant gynecologic diseases:**

Disease	% CA 125 > 35
H mole	60%
Early PG	60%
Fibroids	40%
PID/TOA	35%
Dermoids	20%
Endometriosis	10-80%
Normal Controls	4%

- We use CA 125 mainly to monitor response of treatment and follow up for any recurrences.
- It is much useful for older pts since these conditions are less common among them.

➢ **CA 125 and menopausal status:**

- Premenopausal women: approx. 15% with elevated CA 125 and pelvic mass have malignancy
- Postmenopausal women: approx. 80% with elevated CA 125 and pelvic mass have malignancy

➢ **Ultrasound criteria:**

- Tumor volume
- Wall structure
- Septal structure.

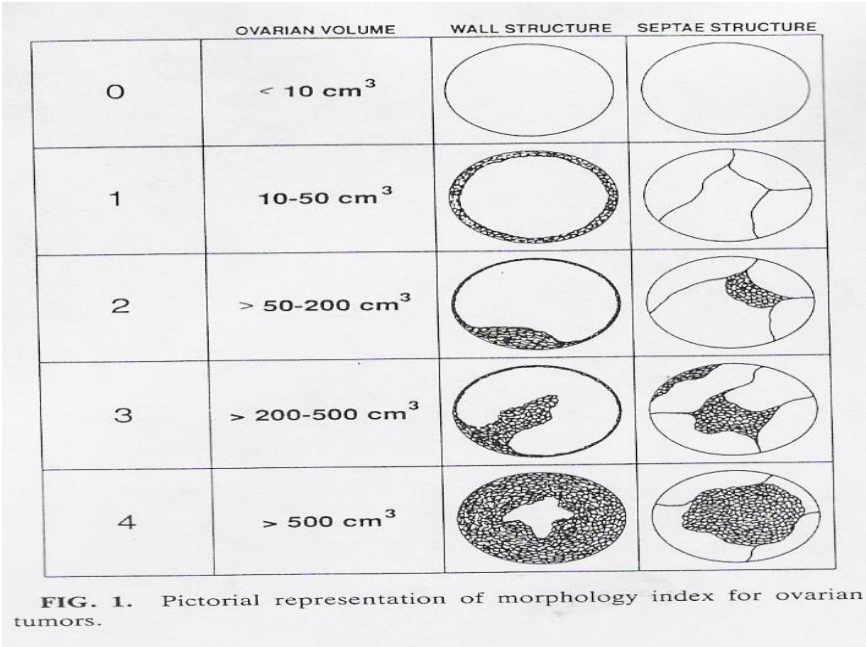
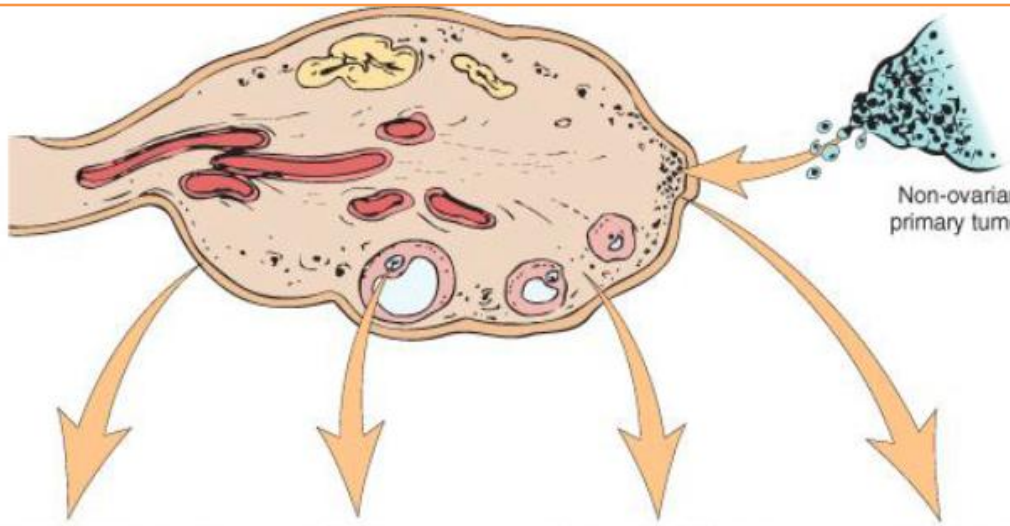
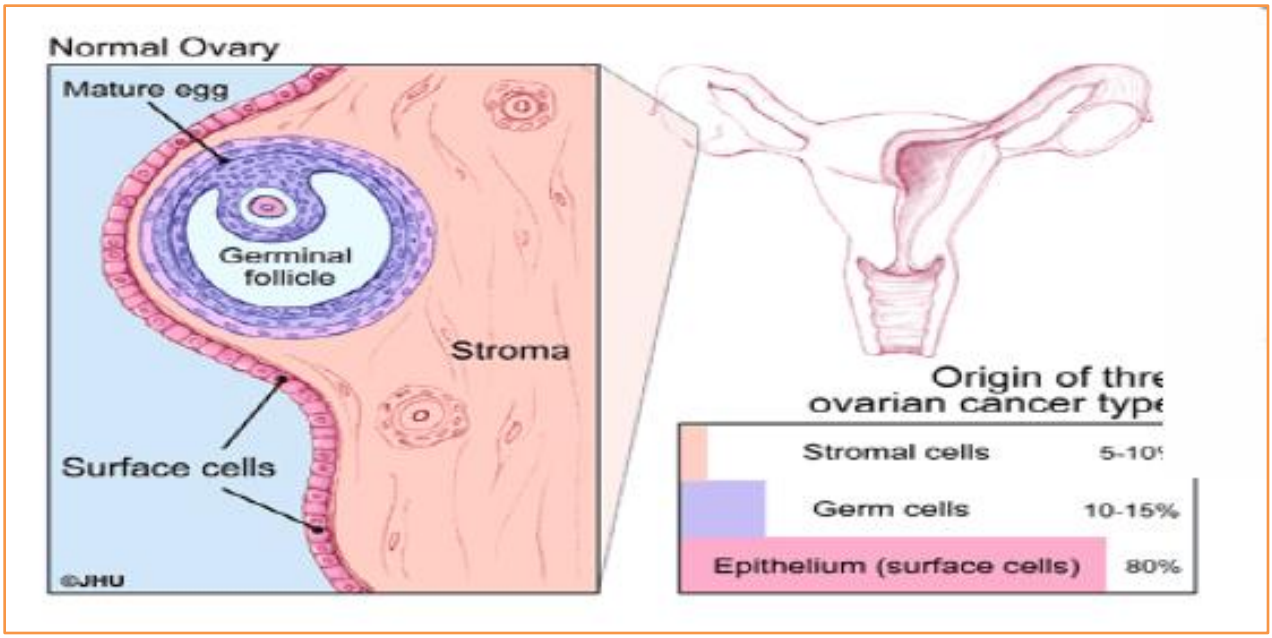


FIG. 1. Pictorial representation of morphology index for ovarian tumors.

❖ Origin Of Ovarian Neoplasms



ORIGIN	SURFACE EPITHELIAL CELLS (Surface epithelial–stromal cell tumors)	GERM CELL	SEX CORD–STROMA	METASTASIS TO OVARIES
Overall frequency	65%-70%	15%-20%	5%-10%	5%
Proportion of malignant ovarian tumors	90%	3%-5%	2%-3%	5%
Age group affected	20+ years	0-25+ years	All ages	Variable
Types	• Serous tumor • Mucinous tumor • Endometrioid tumor • Clear cell tumor • Brenner tumor • Cystadenofibroma	• Teratoma • Dysgerminoma • Endodermal sinus tumor • Choriocarcinoma	• Fibroma • Granulosa–theca cell tumor • Sertoli–Leydig cell tumor	



❖ Ovarian Neoplasms

➢ **Benign neoplasms:**

- The most common benign cystic neoplasms of the ovary are serous and mucinous cystadenomas and cystic teratomas (dermoids)
- Benign solid tumors of the ovary are usually connective tissue origin (Fibromas, Thecomas)
- Meigs' syndrome is an uncommon clinical entity in which a benign ovarian fibroma is seen with ascites and hydrothorax, thesedisappear after resection.

➢ **Ovarian Ca:**

- The lifetime risk for developing ovarian cancer is 1.6% in the general population
- Ovarian cancer accounts for 3.3% of all new cases of cancer.
- The fifth in cancer deaths among women.
- Accounts for more deaths than any other cancer of the female reproduction system due to the late presentation.
- Only 19% of ovarian cancers discovered at early stage.
- Most cases are diagnosed in the seventh decade of life.
- Mostly sporadic

➢ **Risk factors for ovarian Ca:**

- Nulliparous women: Women who have been pregnant have 50% decrease risk for developing ovarian cancer compared to nulliparous women AND Multiple pregnancies offer an increasingly protective effect
- HRT.
- Obesity: Studies have suggested that women who are obese at age of 18 are at increased risk of developing ovarian cancer before menopause.
- Hereditary: BRCA1+2 gene mutation.
- OCP: The use of OCP more than one year reduce the risk of ovarian cancer by 30%-50%

❖ Surface Epithelial Tumors

➢ **Histology:**

1. Serous(tubal)
2. Mucinous (endocx& intestinal)
3. Endometrioid
4. Transitional cell - Brenners.
5. Clear cell

➢ **It could be:**

Type	Gross	Microscope
Benign (Cystadenoma)	Cystic	Fine papillae, single layer covering (no stratification, no nuclearatypia, no stromal invasion).
Borderline	Cystic / solid foci	Papillary complexity, stratification, nuclear atypia, no stromal invasion
Malignant (Cystadenocarcinoma)	Solid& hemorrhage / necrosis	Papillary complexity, stratification, nucleatypiastromal invasion

1. Serous Cystadenomas

- Serous cystadenomasmore common than the mucinous type.
- 10% are bilateral.
- Usually they are smaller in size while mucinous very large
- Usually they are unilocular.
- There is always a chance of recurrence after surgery.

2. Mucinous Cystadenomas

- Less common 25%, very large
- Rarely malignant – 15%
- Multilocular (many small cysts)
- Usually large tumor
- Rarely bilateral – 5-20%
- Tall columnar, apical mucin

➢ **PseudomyxomaperitoneiIMP!**

- Ovarian mass associated with large amount of mucin ascites (gelatinous ascites)
- It is almost always appendicular in origin(we always check the appendix and we remove it surgically!)
- The treatment is surgical, but recurrence is usual
- Hard to treat, b/c the mucinous cells are implanted all over the peritoneal surfaces. They die from malnutrition

3. Brenners Epithelial Tumor

- Usually benign can be malignant.
- May coexist mucinous cystadenoma.
- Can be associated with endometrial cancer.

❖ Borderline Malignant Epithelial Ovarian Neoplasms

- Account for 15% of all epithelial ovarian cancers
- They occupy an intermediate position between the benign cystadenomas and the frankly malignant cystadenocarcinomas.
- The 10 year survival rate for stage I is over 95%
- Late recurrence may occur as many as 20 years after initial diagnosis
- The treatment is essentially surgical

➢ **Genetic causes:**

- Represent 5% of all ovarian cancer .
- Two syndrome are clearly identified:

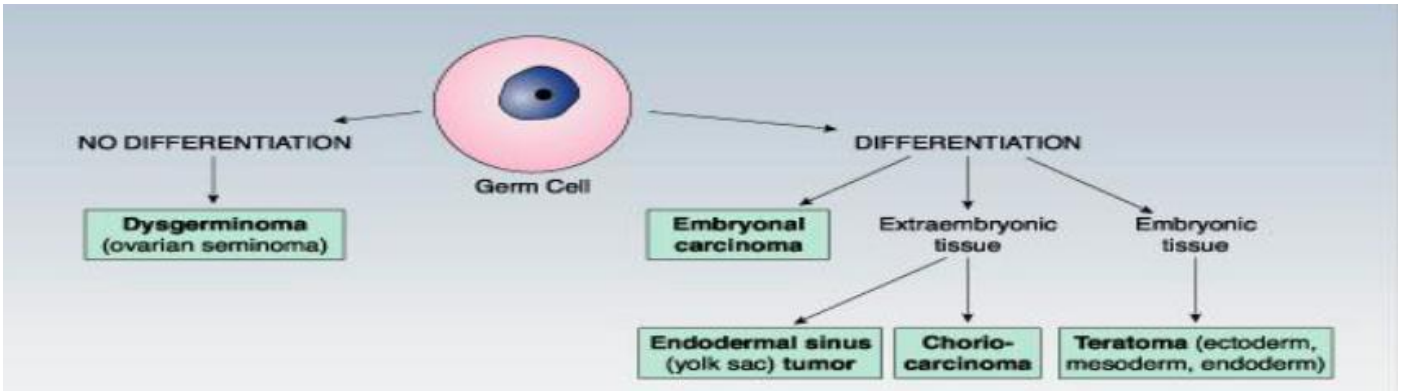
A. Breast/ovarian cancer syndrome (BRCA gene mutation).
B. Lynch ll syndrome or hereditary non polyposis colorectal cancer (colorectal, endometrial, stomach, small bowel, breast ,pancreas and ovarian cancer).

❖ Germ Cell Tumors

- Originate from germ cells.
- Mostly present at stage 1 due to alarming symptoms such as bleeding and pain.

➢ **Histology:**

1. Teratoma(most common. they usually twist, risk is higher during pregnancy).
    A. Benign cystic (dermoid cysts)
    B. Solid immature
    C. Monodermal – strumaovarii, carcinoid
2. Dysgerminoma
3. Yolk sac tumor (Endodermal sinus tumor).
4. Choricarcinoma
5. Mixed germ cell tumor



A. Dermoid Cyst (Benign Cystic Teratoma)

- They are rarely large, 15% are bilateral
- They are the most common neoplasms in the reproductive age
- They contain tissues from: ectoderm, endoderm, and mesoderm
- 1% of theses tumors may undergo malignant degeneration

B. Malignant Germ Tumors: Not common -only 3% of ovarian cancer.

1. Malignant Teratoma (Immature Teratoma)

- Primitive neuroepithelium with multiple neural tubes

2. Dysgerminoma

- 2% of all ovarian malignancies
- Most common malignant germ cell tumor
- Affects primarily younger females with the majority in the second and third decades.
- It is the most frequently encountered ovarian malignancy in pregnancy
- May result in gonadal dysgenesis
- An excellent prognosis. Highly radiosensitive.
- Tumor marker: LDH.

3. Endodermal Sinus Tumor (Yolk Sac Carcinoma)

- It is a highly malignant and clinically aggressive neoplasm
- Most frequently in children and young females
- 20% of malignant germ cell tumors
- Tumor marker: AFP.

4. Choriocarcinoma

- Non-gestational carcinoma.
- Tumor marker: hCG.

❖ Sex Cord – Stromal Tumors

1. Granulosa-Cell Tumor

- Hormonally active tumor. They produce estrogen. In older women, they will have unopposed estrogen > may lead to endometrial CA.
- The most common estrogenic ovarian neoplasm.
- The adult form in postmenopausal women 5%
- (Associated with endometrial hyperplasia and carcinoma)We always take a biopsy
- The juvenile type occurs in the first two decades (precocious sexual development)
- Late recurrence
- Tumor marker: Inhibin

2. Thecoma Fibroma

- Functional tumors producing estrogen
- It occur in postmenopausal women
- Endometrial hyperplasia or carcinoma
- Solid tumor
- May be associated with Meig's syndrome

3. Sertoli-Leydig cell tumors

- It occurs predominantly in young women.
- Commonly androgenic cause defeminization of women manifested as breast atrophy, amenorrhea, and loss of hair and hip fat , to virilization with hirsutism.

❖ Metastatic Ovarian Tumor

- About 3% of malignant tumors in the ovary are metastatic
- The most common primary site is the breast followed by the large intestine, stomach, and other genital tract organs

➢ Krukenberg tumor

- Is applied to the uniform enlargement of the ovaries
- (Bilaterally)
- The commonest primary site is the stomach followed by the colon.

❖ Staging

	Stage I (Growth is limited to ovaries)	**Stage II** (Extension to pelvis)	**Stage III** (Abdomen)	**Stage IV** (Metastasis)
A	Growth limited to 1 ovary, no ascites, no tumor on external surface, capsule intact	Extension and/or metastases to the uterus or tubes	Tumor grossly limited to pelvis, negative lymph nodes but histological proof of microscopic disease on abdominal peritoneal surfaces	Distant metastases; pleural effusion must have a positive cytology to be classified as stage IV; parenchymal liver metastases equals stage IV
B	Growth limited to both ovaries, no ascites, no tumor on external surface, capsule intact	Extension to other pelvic tissues	Confirmed implants outside of pelvis in the abdominal peritoneal surface; no implant exceeds 2 cm in diameter and lymph nodes are negative	
C	Tumor either stage Ia or Ib but with tumor on surface of one or both ovaries, ruptured capsule, ascites with malignant cells or positive peritoneal washings	Stage IIa or IIb but with tumor on surface of one or both ovaries, ruptured capsule, ascites with malignant cells or positive peritoneal washings	Abdominal implants larger than 2 cm in diameter and/or positive lymph nodes	

❖ Management Of Ovarian Neoplasia

1. Observation. (in physiological cyst)
2. Surgical intervention: Laparoscopy or laparotomy.
3. Cysstectomy.
4. Oopherectomy.

- The standard treatment for ovarian cancer start with staging and cytoreductive surgery.
- For post operative treatment, chemotherapy is indicated in all patients with ovarian cancer except those patients with stage 1 and low risk characteristics.

➢ **The 5-year survival rates are as follows:**

- Stage I - 73%
- Stage II - 45%
- Stage III - 21%
- Stage IV - Less than 5%

❖ Summary

1. The majority of ovarian neoplasms are benign.
2. **Physiological Cysts:** They are related to ovulation and are the most common clinically detectable enlargement of the ovary occurring during the reproductive years. Resolve by their own.
3. The most common benign cystic neoplasms of the ovary are serous and mucinous cystadenomas and cystic teratomas (dermoids)
4. **Serous Cystadenomas:** usually small andunilocular.
5. **Mucinous Cystadenoma:** very large and multilocular.
6. **Pseudomyxomaperitonei:**It is almost always appendicular in origin (we always check the appendix and we remove it surgically!)
7. **Dermoid Cyst (Benign Cystic Teratoma):** They are the most common neoplasms in the reproductive age. They contain tissues from: ectoderm, endoderm, and mesoderm
8. **Dysgerminoma:**

- Most common malignant germ cell tumor
- Affects primarily younger females.
- It is the most frequently encountered ovarian malignancy in pregnancy
- An excellent prognosis. Highly radiosensitive.
- Tumor marker: LDH.

9. **Endodermal Sinus Tumor (Yolk Sac Carcinoma)**

- It is a highly malignant and clinically aggressive neoplasm
- Most frequently in children and young females
- Tumor marker: AFP.

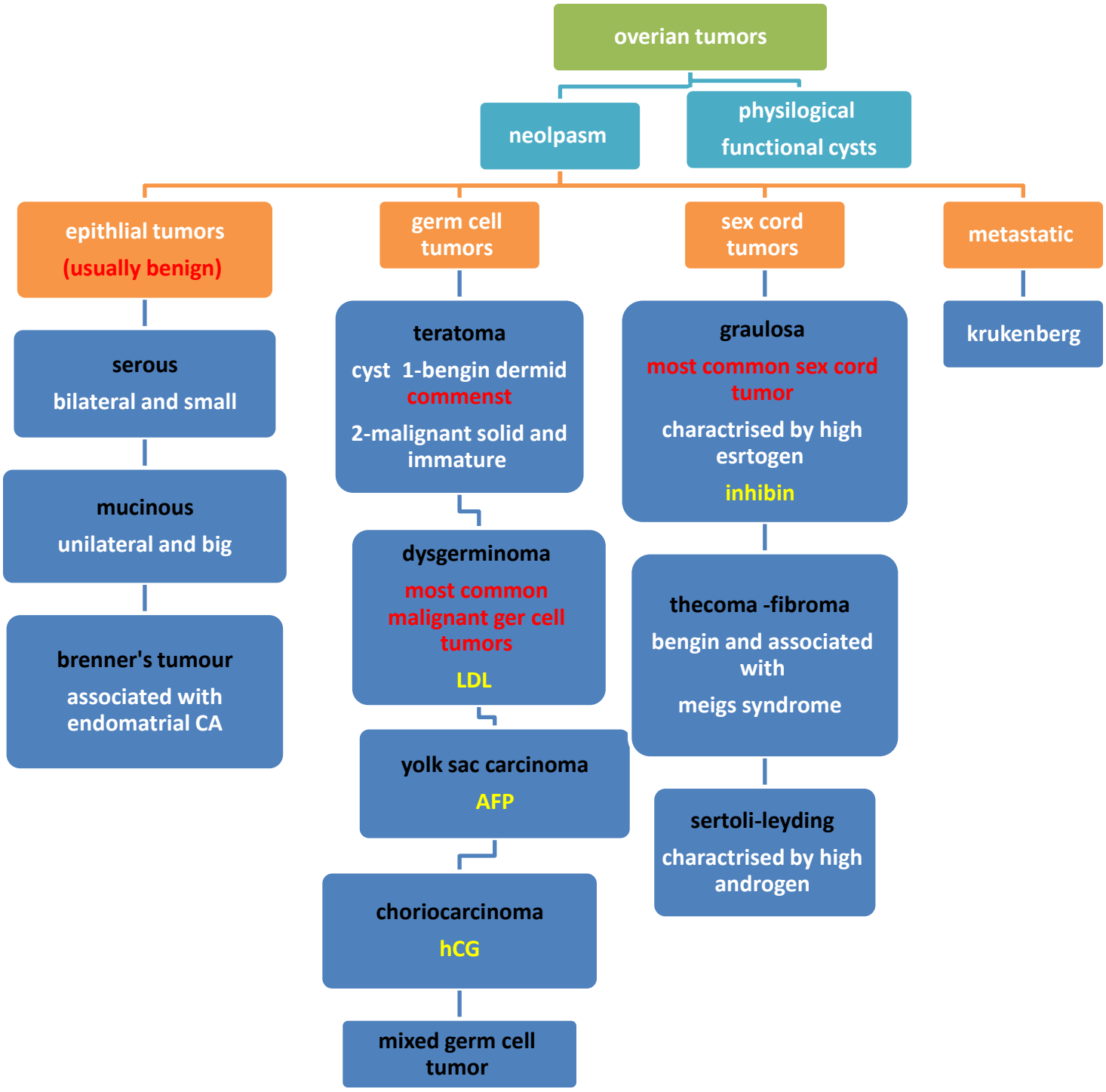
10. **Choriocarcinoma:** Tumor marker: hCG.
11. **Granulosa-Cell Tumor:**

- The most common estrogenic ovarian neoplasm.
- Associated with endometrial hyperplasia and carcinoma.
- Tumor marker:Inhibin

12. **Thecoma Fibroma:** May be associated with Meig's syndrome (an uncommon clinical entity in which a benign ovarian fibroma is seen with ascites and hydrothorax.)
13. **Sertoli-Leydig cell tumors:**

- Predominantly in young women.
- Commonly androgenic.

Summary 2


overian tumors
neolpasm
physilogical
functional cysts
epithlial tumors
(usually benign)
germ cell tumors
sex cord tumors
metastatic
serous
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associated with endomatrial CA
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meigs syndrome
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krukenberg

Meigs' syndrome, is the triad of

1- ascites**,**

2-pleural effusion **(hydrothorax)**

pseudomyxoma peritonei

1-overian tumor + mucinous ascites

2-usually appendicular in site

Menopause & Postmenopausal Bleeding

Definitions

word means cessation of menstruation.
(After 40 years of age, before that it's premature ovarian failure)

- **Climacteric**:
  The period of life when fertility and sexual activity decline (starts around 40 years)
  It is a wide term leading to:
  - Pre Menopause
  - Peri Menopause
  - Post Menopause
- **Perimenopause**:
  3-5 years period before menopause, with increase **frequent irregular** anovulatory bleeding, followed by **episodes of amenorrhea** and **intermittent** menopausal symptoms.
- **Menopause**:
  - The point in time at which menstrual cycles **permanently cease**.
  - It is a **retrospective diagnosis** after **12 months of amenorrhea** women **classified as being menopause**.
  - Mean age – 51 years.

Pathophysiology of Menopause

- The number of **primordial follicle** declines even before birth, but **declines dramatically just before menopause.**

- **Increase in FSH, LH** from about 10 years **before** menopause.
  (depleted follicles in ovaries → low progesterone & low/normal estrogen→ -ve feedback → ↑ FSH & LH)

- **Close to menopause**, there will be:
  - **Anovulation**
  - **Inadequate luteal phase** → decrease progesterone **but not estrogen level** → leading to **DUB** (dysfunctional uterine bleeding) & **endometrial hyperplasia** (heavy bleeding)

- **At menopause:**
  - Dramatic **decrease of estrogen** →menstruation ceases + symptoms of menopause start.
  - But still ovarian stroma produces small androstenedione and testosterone.
  - However, main **postmenopausal estrogen** is **estrone** produced by **peripheral fat** from adrenal androgen.

Symptoms of Menopause

- **Hot flushes** (cutaneous vasodilation), usually first symptom:
  - occur in **75% of women**
  - more severe after surgical menopause (removal of ovaries)
  - continue for 1 year
  - 25% continue more than 5 years
- **Urinary Symptoms**
  - UTI
  - urgency
  - frequency
  - nocturia
- **Psychological changes** (decreased level of central neurotransmitters):
  - Depression
  - Irritability
  - Anxiety
  - Insomnia
  - loss of concentration

So if patient after 40 and has psych. symptoms, suspect menopause and investigate it before referring her to psychiatry

- **Atrophic Changes [IMP]**
  - Vagina:
    - **vaginitis** due to thinning of epithelium. (causes bleeding)
    - ↑ PH and ↓ lubrication
    - dyspareunia to decrease vascularity and dryness
  - Decrease size of cervix and mucus
  - Retraction of squamocolumnar (SC) junction into the endocervical canal.
  - Decrease size of the uterus, shrinking of myoma (so if she has polyps, it will shrink with menopause, so don't treat if it is not causing problems like anemia)
  - Adenomyosis.
  - Decrease size of ovaries, become non palpable.
  - Pelvic floor relaxation → **prolapse** (procidentia)
  - **Urinary tract atrophy** →loss of urethral tone → **caruncle** (click here to read more)
    Also: Hypertonic Bladder - detrusor instability (incontinence)
  - Decrease size of breast
  - Breast benign cyst
- **Skin Collagen**
  ↓ collagen & thickness → ↓ elasticity of the skin
- **Reversal of premenstrual syndrome**

Late Effects of Menopause

- **Osteoporosis: [IMP]**
  - Bone mass reaches its peak at the end of 3rd decade of life
  - After 40 years, bone resorption exceeds bone formation by 0.5% per year.
  - This negative balance increases after menopause, to a loss of 5% of bone per year.
  - Predisposes to fractures by slight causes, eg: pelvic fracture → 50% may die
  - Considered a cause of death in old ladies
  - **Risk factors:**
    - Gender: more in women (male to female ratio is 1:3)
    - BMI (low BMI is more risky than obese)
    - Race:
      - High in white women
      - Moderate in Asian women
      - Lowest in Black women
    - Family History +ve
    - Life style: smoking, caffeine intake, alcohol, increase in protein diet, decrease in calcium and vit D intake
    - Steroid Medication :
      - **Exogenous medication** (so you should give prophylactic treatment with medication that could cause osteoporosis)
      - Cushing Syndrome

  - **Diagnosis:**
    DEXA (Dual Energy X-ray Absorptiometry),
    for assessment of bone densemetry, to demonstrate if bone density is above or below fracture threshold.

  - **Prevention:**
    - improve lifestyle
    - regular exercise
    - eliminate smoking & alcohol

  - **Medication**:
    - ERT (Estrogen Replacement Therapy),
      controversial because may cause breast cancer. Not used routinely.
    - **Bisphosphonate** (Fosamax): inhibits osteoclastic activity & minimal side effects.
    - **Raloxifene** (Evista): selective estrogen receptors modulator [SERM]:
      (it's a good drug, drug of choice if we fear breast cancer)
      - It binds with high affinity to estrogen receptors.
      - It has some **estrogen like effect** e.g. ↑ bone density, ↓LDL Cholesterol [cardioprotective]
      - Acts as **estrogen antagonist** on **endometrium and breast**.
    - Calcitonin: inhibit osteoclastic activity + analgesic effect of bone pain
    - Calcium Supplement & Vit D. [IMP]

- ## Cardiovascular Disease:

  - CVD is now the leading cause of death among post menopausal women:
    - **before menopause**: risk of heart attack is 1/3rd compared to men
    - **after menopause**: increase in women risk of heart attack, so it becomes equal to the risk of men at the age of 70 years

  - Because of effect of estrogen:
    - Before menopause:
      - Increase HDL
      - Decrease LDL
      - Decrease atherogenic plague formation by direct action on vascular endothelium.

  - After menopause:
    - HDL : LDL ratio become closer to male ratio

  - Observational Studies:
    - HRT decreases mortality by 30%.
    - But **recent epidemiological studies** do not show a beneficial effect of HRT on CHD, but there is **increase number of breast cancer** when compared with non users of HRT.

- ## Urogenital System:

  - Embryologically, female genital tract & lower urinary system develop in close proximity from primitive urogenital sinus.
  - The **urethra** and **vagina** have a high **concentration of estrogen receptors**
  - There is significant evidence to support the **use of estrogen in treatment of urogenital symptoms** such as recurrent UTI, vaginitis and dyspareunia.

- ## Alzheimer's Disease:

  - Prevalence of dementia: as high as 50% by age of 85 years.
  - Alzheimer's disease accounts for 60-65% of cases.
  - Observation studies: decrease risk of Alzheimer's by 1/3 among women taking HRT.
  - HRT has beneficial effect on brain function, but no randomized studies to confirm observational data.

Diagnosis and Investigations

- **The triad of:**
  - Amenorrhea (12 months)
  - Hot flushes
  - increase FSH > 15 i.u./L (i.u. = international unit)
  - Above 40 years of age

- **Before starting treatment, the following should be performed:**
  - breast self examination (to ensure no masses before treating)
  - mammogram
  - pelvic exam (pap smear) (to check for any cancers, because HRT will promote their growth)
  - Measurement of: weight, blood pressure

- **No indication to perform:**
  - Bone density
  - Endometrial Biopsy

  - However, **any bleeding** should be **investigated** before starting any treatment.

Treatment

- Estrogen:
  A minimum of 2mg of estradiol is needed to maintain bone mass and relieve symptoms of menopause.

- Women **with uterus** → add **progestin** in **last 10 days** to **prevent** endometrial **hyperplasia**

- Sequential Regimens → used in patient close to menopause
  (patient will have monthly bleeding, given if patient desires it so she can feel she's still young)
  - **Estrogen** in the **first half** of 28 day per pack
  - **Estrogen** & **progestin** in **2nd half** of 28 day pack

- Combined continuous therapy that has **progesterone** everyday (along with estrogen):
  - Useful for women who are few years **past menopause**, and who do not have vaginal bleeding

- There is evidence of increased **risk of endometrial cancer** with **sequential regimens** for more than 5 years.
- However, **combined** continuous regimens **decrease** risk of **cancer**.

Hormonal Replacement Therapy (HRT)

- **Benefits:**
  - Vagina: ↑ vaginal thickness of epithelium → ↓ dyspareunia & vaginitis.
  - Urinary tract: enhancing normal bladder function.
  - Osteoporosis: decrease fractures by more than 50%
  - CVS: decrease by 30% by observation studies, but recent studies shows no benefits.
  - Colon Cancer: decrease up to 50%

- **Confirmed Risk:**
  - Endometrial cancer (and breast cancer) eliminated by:
    1. Adding progesterone
    2. or: Using selective estrogen receptors modulators (SERMS).
  - Gall Bladder Disease (with estrogen replacement therapy):
    - ↑ triglyceride
    - ↑total cholesterol
    - increase risk of gall stone, and liver disease, so be careful if +ve history.
  - **Breast cancer** risk with long term HRT adds:
    - 2/1000 after using it for 5 years – 6/1000 after 10years
    - 12/1000 after 15 years
    - background risk 45/1000 between the age of 50 and 70

- **Contraindications to HRT:**
  - Undiagnosed vaginal bleeding
  - Acute liver disease, or chronic impaired liver functions
  - Acute vascular thrombosis
  - Breast Cancer

Postmenopausal Bleeding

- **Definition:**
  Vaginal **bleeding** that occurs after **12 months of amenorrhea** in middle aged women who are **not receiving replacement therapy**.
  It can never be dysfunctional or anovulatory in nature (because with loss of functional ovarian follicle, bleeding from normal ovulatory cycle is impossible).

- **Causes:**

- Upper reproductive tract causes:
    - Atrophic endometritis
    - Endometrial polyp, submucous fibroids
    - Endometrial hyperplasia
    - Endometrial cancer (important to be excluded)
    - Ovarian or tubal cancer

- Lower reproductive tract causes:
    - Vaginits (most common along with atrophic endometritis)
    - Vaginal or vulvar tumors
    - Varicose veins (from vulva)
    - Cervical polyp or cervical tumors

- GIT causes:
    - Hemorrhoids
    - Anal fissures
    - Colorectal cancer

- Anticoagulant medications

Endometrial cancer (as a cause of Postmenopausal Bleeding)

- The most common gynecological malignancy.
- Endometrial neoplasia can progress from simple hyperplasia to invasive cancer caused by **unopposed estrogen**.
- The **mechanism** of many endometrial cancers is prolonged estrogen stimulation of the endometrium, unopposed by progesterone.
- The **source** of estrogen may be:
    - **Exogenous** estrogen (E2) (Estrogen Replacement Therapy: ERT), without taking progesterone
- **Peripheral aromatization** of androstenedione to estrone, as in obesity or PCO
- Estrogen (E2) producing **tumor**, like granulosa cell ovarian tumor
- **Tamoxifen** stimulation of endometrium (tamoxifen is used for breast cancer)

- **Risk factors:**

- No pregnancy
- Prolonged reproductive life – late menopause
- Unopposed estrogen
- Triade od iabetes, hypertension and obesity
- Tamoxaifen (worst prognosis).

Diagnosis of Postmenopausal Bleeding

- **Upper Reproductive Tract Causes :**

  - Upper reproductive tract causes can be identified only by:
    tissue diagnosis obtained by endometrial evaluation .
    An ultrasound is done before that to check endometrial thickness

  - Endometrial Biopsy:
    Inaccurate for diagnosis of polyp, misses a sufficient number of hyperplasia.

  - **Hysterosonography**:
    - performed by infusion saline in the uterine cavity to identify endometrial **polyps**.
    - If endometrial thickness is more than10mm → indicates risk of hyperplasia→ **tissue** should be obtained for **histological studies**.

  - **Pipelle sampling** for endometrial **biopsy** (catheter that has suction and takes biopsy):
    - If +ve → refer to oncology (and perform hysterectomy)
    - If –ve → continue investigation (hysteroscopy) because it's a blind procedure

  - **Hysteroscopy [best facility, diagnostic]**:
    - performed at the time of D&C for polyp & operative resection.

  - Fractional dilation and curettage (D&C):
    - Done if no hysteroscopy
    - It's the good standard for evaluating post menopausal bleeding.
    - It is performed in 2 stages:
      1. Initially, **endocervical** canal is curetted obtaining the first specimen to rule out invasion of cervix by cancer.
      2. **Uterine cavity** is curetted; obtaining second specimen to assess endometrial neoplasia or malignancy.
    - If negative: still it's not diagnostic

  - Pap Smear:
    - has **poor sensitivity** for endometrial cancer. Only 40% cases are identified

  - MRI

- **Lower Reproductive Tract Causes :**
  - Pelvic Exam
  - Pap Smear & appropriate Biopsy
  - Colposcopy and cervical biopsy

- **GIT etiology:**
- Rectal exam
- Stool for occult blood
- Proctosigmoidoscopy

Treatment of Postmenopausal Bleeding

1. Investigate the cause, exclude cancer... Treat the cause.
2. Atrophic vaginitis, cervicitis and endometritis may need only **local estrogen preparations**\

Summary

- **Peri**menopause: 3-5 years period before menopause, increase **frequent irregular** anovulatory bleeding, **episodes of amenorrhea** and **intermittent** menopausal symptoms.

- **Menopause**: The point in time at which menstrual cycles **permanently cease**. It is a **retrospective diagnosis** after **12 months of amenorrhea**

  - 10 years before menopause: **increase in FSH, LH**
  - Close to menopause: Anovulation, Dysfunctional uterine bleeding (because **progesterone decreases** but **estrogen is still the same**)
  - Menopause: ovarian estrogen decreases... instead, peripheral fat tissues secrete **menopausal estrogen** called: **estrone**

- **Menopause symptoms:**

  - **Hot flushes**, usually first symptom, occur in 75% of women.
  - **Urinary Symptoms**: UTI, urgency, frequency, nocturia
  - **Psychological changes**

    Atrophic Changes [IMP]:
    - **vaginitis** due to thinning of epithelium. (causes bleeding), ↑ PH & ↓ lubrication, dyspareunia
    - Decrease size of cervix and mucus... Decrease size of the uterus, shrinking of myoma
    - Adenomyosis.
    - Pelvic floor relaxation → **prolapse** (procidentia)
    - **Urinary tract atrophy**
    - Breast: Decrease size, benign breast cysts
  - Skin Collagen decreases → skin is less elastic
  - **Reversal of premenstrual syndrome**

- **Late effects of menopause:**

  - **Osteoporosis [IMP]:**
    - Predisposes to fractures by slight causes, eg: pelvic fracture → 50% may die
    - Considered a cause of death in old ladies
    - **Diagnosis:** DEXA (Dual Energy X-ray Absorptiometry)
    - **Prevention:** lifestyle, exercise, quit smoking and alcohol
    - **Medications:**
      - Calcium Supplement & Vit D. [IMP]
      - ERT (Estrogen Replacement Therapy): Not used routinely.
      - **Bisphosphonate** (Fosamax): inhibits osteoclastic activity & **minimal side effects.**
      - **Raloxifene** (Evista): selective estrogen receptors modulator [SERM]:
        A good drug, drug of choice if breast cancer is feared....
        Has some **estrogen like effect** e.g. ↑ bone density, ↓LDL Cholesterol [cardioprotective] + it has **estrogen antagonist** on **endometrium and breast**.
      - Calcitonin: inhibit osteoclastic activity + analgesic effect of bone pain

  - **Cardiovascular Disease:** It is now the leading cause of death among post menopausal women:
    - After menopause: HDL : LDL ratio become closer to male ratio

  - **Urogenital system:** use of estrogen in treatment of urogenital symptoms such as recurrent UTI, vaginitis and dyspareunia.

- **Diagnosis of menopause:**
  Triad: Amenorrhea (12 months) , Hot flushes , increase FSH > 15 i.u./L [should be Above 40yrs]

- **Before starting treatment, the following should be performed:**
  - breast self examination (ensure no masses before treating) , mammogram , pelvic exam (pap smear) → check for any cancers before HRT
  - Measurement of: weight, blood pressure

  - **Any bleeding** should be **investigated** before starting any treatment.

- **Treatment of menopause:**
  - Sequential Regimens → used in patient close to menopause
    - **Estrogen** in the **first half** of 28 day per pack
    - **Estrogen** & **progestin** in **2nd half** of 28 day pack

  - Combined continuous therapy that has **progesterone** everyday (along with estrogen):
    Useful for women who are few years **past menopause**, and who do not have vaginal bleeding

  - **Increased** risk of **endometrial cancer** with **sequential regimens** for more than 5 years.
  - **Decreased** risk of **cancer** with **combined continuous regimen**

- **HRT (hormonal replacement therapy):**
  - Benefits for Vagina, Urinary tract, Osteoporosis, CVS (maybe) , Colon Cancer
  - Risks: Endometrial cancer, Gall Bladder Disease (increase risk of gall stone, and liver disease, so be careful if +ve history.) , **Breast cancer** risk with long term HRT.

- **Post menopausal bleeding**: Vaginal **bleeding** that occurs after **12 months of amenorrhea** in middle aged women who are **not receiving replacement therapy**.
  - Most common cause: atrophic vaginitis, atrophic endometritis
  - **Endometrial cancer should be excluded** when postmenopausal woman presents with bleeding
  - Endometrial neoplasia can progress from simple hyperplasia to invasive cancer caused by **unopposed estrogen**.

- **Diagnosis of Postmenopausal bleeding:**

  - **Upper Reproductive Tract Causes:**
    can be identified only by **tissue diagnosis** obtained by endometrial evaluation .

    - Endometrial Biopsy: Inaccurate
    - **Hysterosonography**
    - **Pipelle sampling** for endometrial **biopsy**: not diagnostic
    - **Hysteroscopy [best facility, diagnostic]**
    - Fractional dilation and curettage (D&C): Done if no hysteroscopy (but not diagnostic)
    - Pap Smear:
    - MRI
  - **Lower Reproductive Tract Causes :**
    Pelvic Exam, Pap Smear & appropriate Biopsy , Colposcopy and cervical biopsy

  - **GIT etiology:** Rectal exam , Stool for occult blood , Proctosigmoidoscopy

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- **Treatment:**
  - Investigate the cause, exclude cancer... Treat the cause.
  - Atrophic vaginitis, cervicitis and endometritis: → **local estrogen preparations.**
  - Malignant cervical, uterine or ovarian pathology will require **specific treatment.**

Contraception.

Types of Birth Control

A) Reversible:

1) Hormonal.(Oral – injection – implant)

2) Intrauterine Contraception Devices (IUCD).

3) Barrier Method.(E.g. coitus interruptus, simple and doesn't have side effects but it has high failure rate because sperm may enter the vagina if withdrawal isn't properly timed or if pre-ejaculation fluid contains sperm).

4) Natural Methods.

5) Spermicides.

B) Irreversible(Surgical Methods):

1) Tubal ligation. Laparoscopic sterilization: Rings – Clips – Bipolar diathermy –Lazer.

2) Vasectomy.

The Ideal contraception method should be:

- Acceptable –requires no user motivation so compliance not problem.
- Safe.
- Accessible.
- Less side effects.
- Low failure rate.
- Non-invasive.
- Rapid reversible.
- Prevention of STD.

Type 1 - Reversible Methods

1- Hormonal

A) Combined Oral Contraceptive pills:

- Combined Oral Contraceptive (COC) contains a mixture of estrogen and progesterone.
- Progesterone only contraception:
  - Pills - levonoregesterol.
  - Injectable - DMPA (Depot Medroxyprogesterone Acetate).
  - Subdermal implant.

- Pills are safe and effective when taken properly. They are over 99% effective.(Must be taken in 3 hours time window e.g. if the woman decided to take it at 8 p.m. she must take it everyday between 8-11 p.m.)

- Estrogen component of most modern COC:
  - ethinyloestradiol (EE) 20-50 ug.(Estrogen suppresses lactation and has risk of thrombosis)

- Progesterone Component:
  - Second generation (e.g. norethisterone and levonorgestrill)
  - Third generation (e.g. desogestrel and gestodene)
  - Third generation have higher affinity for progesterone receptors and lower affinity for the androgen receptor than second generation, i.e. they confer greater efficacy with few androgenic side effects.
  - Third generation also have fewer effects on carbohydrate and lipid metabolism.

Mechanism of action:(MCQs)

- Stop ovulation by inhibition pituitary FSH and LH secretion.
- Cervical mucus becomes scanty and viscous with law spinnbarkeit and thus inhibits sperm transport.(Spinnbarkeit is the stringy, elastic character of cervical mucus)
- Thins uterine lining(endometrium lining)and become unreceptive to implantation.
- Direct effect on fallopian tubes impairing sperm and ovum transport.

Combined oral contraceptive formulation is either:

- Fixed dose.
- Phasic: the dose of estrogen and progesterone changes once (biphasic) or twice (triphasic) in each day course. They are designed to mimic the cyclical variation in hormone levels.

Positive benefits of Oral Contraceptive pills (OCP):

- Prevent pregnancy.
- Less dysmenorroea and menorrhagia.
- Less incidence of carcinoma of the endometrium and ovary.
- Less incidence of benign breast disease.
- Less incidence of pelvic inflammatory disease (PID).(Can relief pain)
- Less incidence of ovarian cyst.
- Protective effect against rheumatoid arthritis, thyroid disease and duodenal ulceration.
- Less acne.

Side effect and risks:

- Weight gain – with pills containing Levonoregestrel(2nd generation)but not desogestral or gestodene(3rd generation).
- Carbohydrate metabolism: effect on insulin secretion.
- Lipid metabolism: effect on ratio of HDL/LDL.
- No protection from STDs.
- Cardiovascular effects – increase risks of thromboembolism by three to four fold in women with risk factors: congenital acquired throbophilias, obesity, advanced age and immobility
- Myocardial infarction and hemorrhagic stroke and increased with:
  - ↑Oestrogen dose.
  - Hypertension.
  - Smoking.
- Breast Cancer –long termoral contraceptive user before age 25 specially with more potent progesterone.
- Cervical cancer –↑ incidence due to ↓ immunity to antigenic causal factor, with greater sexual activity without benefits of Barrier contraception.

Contraindication:(we are taking about absolute contraindications)

- Arterial or venous thrombosis.
- Ischemic Heart disease.
- Focal migraine.
- Athcrogeniclipid disorder.

- Inherted or acquired throbophilias.
- Post-cerebral hemorrhage.
- Pulmonary hypertension
- Disease of Liver: Acute liver disease i.e. with
    - Abnormal LFT test
    - Adenoma or Carcinoma
    - Gallstones
    - Acute Hepatic prophyrias.
- Others:
    - Pregnancy.
    - Undiagnosed genital tract bleeding.
    - Estrogen dependent neoplasm e.g. Breast Cancer.

B) Progesterone only contraceptive (also called Mini Pill)

Mechanism of action:

- Supression FSH and LH secreation and inhibits ovulation.
- Cervical mucus modification,which inhibits sperms penetration.
- Endometrial modifications to prevent implantation.

Advantage of progesterone only contraception:

- Minimal impact on lipid profile and hypertension so can be used in cardiovascular disease.
- Used by lactating mother.
- Advantages of Depot Medroxyprogesterone Acetate (DMPA):
    - Protection against endometrial cancer.
    - Protection against Acute PID.
    - Protection against Vaginal candidiasis.
    - Protection against ovarian cancer, endometriosis and fibroids.
    - Relief dysmenorrhea and pre menstrual syndrome.
    - No daily pills to remember.
    - Given once every 3 months.
    - 99.7% effective preventingpregnancy.

Disadvantages of Progesterone only Contraception:

- Menstrual disturbance – ammenorrhoea with injection.
- Irregular prolonged spotting or bleeding with pills.
- May develop functional ovarian cyst due to luteinzation of unruptured ovarian follicle.

- Protect against intrauterine pregnancy but not ectopic because it modify tubal function - ↓ ovum transport.
- Acne, headaches, Breast tenderness and lose of libido (androgenic progesterone).

Sub dermal implants:

- Need trained personal for insertion and removal.
- Out patients procedure.
- 99.5% effectiveness rate.
- Nouser motivation i.e. compliance isn't a problem.
- Amennorhoea is common.

The Norplant System consists of 6 capsules implanted under the skin of the upper arm. They prevent pregnancy by secreting progesterone into the body. They may be left in place up to 5 years or surgically removed at any time

Failure of the Pill:

- If the patient forgets to take the pill.
- Gastroentroentritis.
- Drugs:
  - Anticonvulsant: phenytoin, phenobarbitone.(Affect the metabolism of the contraceptive)
  - Antibiotics.(Change intestinal flora → affects the metabolism. E.g. ampicillin and rifampin)

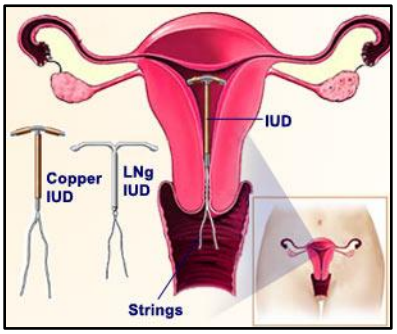
2- Intrauterine Contraception Devices

- Most commonly used reversible method of Contraceptionworldwide
- Effective > 97%.
- The newer devices have failure rate< 0.5%

Three Types of IUCD:

A) Inert:

- These are polytheneIUCD, bulkier than other types.
- More likely to cause heavy bleeding and pelvic actinomyosis.
- No longer available.



B) Copper bearing IUCD:

- Consist of a plastic frame with copper wire around the stem.
- Surface of the copper determine the effectiveness and active life of the device.
- Most IUCD licensed for use over 5-10 years and because of gradual absorption of copper, these IUCD are renewed after 3-5 years.
- Copper salt gives some protection against bacterial infection.

C) Hormone releasing IUCD (Mirena):

- Releasinglevonoregtrel (20ug/24hrs) over at least 5 years.
- Reduce menstrual, blood flow and markedly reduces Blood loss in menorrhagia.
- Protect against pelvic inflammatory disease.
- Cause irregular uterine bleeding for first 6 months following insertion.

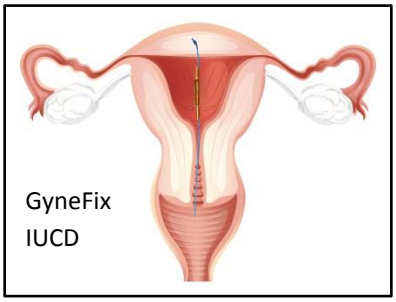
Mechanism of Action:

- All IUCD cause a foreign body reaction in the endometrium with increased prostaglandin production and Leucocyte infeltration. This reaction enhanced by copper which effect endometrial enzymes and oestrogen uptake and also inhibit sperm transport.
- Alteration of uterine and tubal fluid impairs the viability of the gametes.
- The progesterone IUCD (LNG.IUS) cause endometrial suppression and change in the cervical mucus and utro tubal fluid impair sperm migration.

Complications:

- Dysmenorrhoea and Menorrhagia:
  - Antifibrilolytic agent tranexamic acid.
  - Antiprostaglandin agents.
  - Non-steroidal anti-inflammatory drugs.
- Infection – Actinomycosis associated with granulomatous pelvic abscesses.
- Pregnancy rate 1-1.5% most likely in the first 2 years. Copper bearing has lower rate 0.5% and levonoregtrel 0.1%.
- Risk of ectopic pregnancy is greater with IUCD especially progesterone releasing IUCD.
- Expulsion of the device 5-10% in just 6 months.Usually during menstruation.
- Translocation – the IUCD passes through uterine wall into the peritoneal cavity or blood ligament usually a consequence of unrecognized perforation at insertion – laparoscopy should be performed.

Contraindications:



- Pelvic inflammatory disease.
- Menorrhagia.
- History of previous ectopic pregnancy.
- Severe dysmenorrhea.

Choices of Devices:

- Copper T380 is the first choice as it has the lowest failure rate and longest life span.
- Women with:
  - Small uterus
  - Experienced pain
  - Spontaneous expulsion

  GyneFix IUCD
- Women with Menorrhagia are givenLevonorgestrel – releasing (LNG – IUCD)

3- Barrier Method

- Prevent pregnancy by blocking the eggs and sperm from meeting.
- Have higher failure rate than hormonal methods due to design and human errors.

Most important advantage: protection against STD

Barrier Methods:

- **Male:**Condom
- **Female:** Condom (Femidon) –pessaries(diaphragm, cervical cap)in combination with spermicides.

A) Condoms:

- Most common and effective barrier when used properly.
- Thin rubber sheath fits on the penis, it interfere 3-23% with sensation and it is liable to come off as the penis withdraws after the act.
- Widely accessible.
- Inexpensive.
- Reversible.
- Provide protection against STDs including HIV and premalignant disease of the cervix.
- Contraindication to the condom use is latex allergy in either partner.
- Failure rate 3-23%

B) Occlusive pessaries:

- Diaphragm, cervical cap inserted into the vagina, prior to intercourse to occlude the cervix and should be used with spermicide to provide maximum protection and remains6 hours after intercourse.
- Initially need to be fitted by trained person.
- Needs high degree of motivation for successful use (Efficacy 4-20%).

C) Female Condom:

- Polyurethane sheath inserted to and lines the vagina.
- Wildly available.
- Failure rate 5-21%.

D) Vaginal Sponges:

- Made of polyurethane foam and one inserted with spermicide into the vagina and cover the cervix.
- Provide contraception by-acting as Barrier
  - Absorbing the semen.
  - Carrier for spermicide.
  - Higher failure rate.
  - Advantage – protection against STD.

4- Natural Methods

A) Calendar Method (Safe period):

- Relies upon the fact that there are certain days during the menstrual cycle when conception can occur following ovulation, the ovum is viable within reproductive tract for a maximum of 24 hrs.
- The life spam of sperm is longer 3 days.
- During a 28-day menstrual cycle, ovulation occurs around day 14. This means that coitus must be avoided from 8th to 17th day.
- Failure rate is high and so many couples find it difficult to adhere to this method.

B) Ovulation method (The billing's method):

Ovulation prediction can be enhanced by several complementary methods including

Measuring basal body temperature (BBT):

Progesteronecauses rise in temperatureby 0.2-0.4°C following ovulation until the onset of menstruation.

Observing cervical mucus:

- Several days before ovulation mucus appearance of raw egg white, clear, slippery and stretchy (spinnbarkeit). Effect of estrogen
- The final day of fertile mucus is considered to be the day when ovulation is most likely to occur and abstinence must be maintained from first day of fertile mucus until 3 days after the peak day.
- The end of the fertile period is characterized by appearance of (infertile mucus) which is scanty and viscous.(Thick mucus: infertile mucus. Sperm can't penetrate thick mucus)

Failure rate of calendar method and ovulation method is is 2.8 %.

C) Personal fertility monitors:

- Small devices able to detect urine concentration of estrogen and LH indicate start and end of fertile period.
- Failure rate is 6.2%.

Disadvantage of natural methods:No protection against STDs.

Emergency Methods

1) Hormonal methods:

Yuzpe Regime (PC4):

- Ethinylostradiol (100µg) levonorgesterel (500µg). Trade name: Eugynon/ovran.
- First dose is taken with 72 hours of intercourse and second dose taken 12 hours after the first.
- It inhibits or delay ovulation, altering endometrial receptivity.

Progestegen only form of emergency contraception.

- Levonorgestrel (0.75 mg)
- Given twice within 72 hours of intercourse.
- It also alters cervical mucus, impairing sperm transport and preventsfertilization, whichexplains the greater efficacy 99% compared toYuze regime 77% in prevention of expected pregnancy. If commenced with 24 hours of intercourse.
- Side effects:
    - Nausea and vomiting.
    - Theoretical risk to pregnancy.
    - If pregnancy occurs: increased risk of ectopic pregnancy.

2) Copper IUCD:

- Very effective if used 5 days after coitus or ovulation due to spermicidal and Blastocidal actin of copper.
- Has the lowest failure rate (<1%).
- Age, nulliparity and menorrhagia are NOT contraindications.

Type 2 - Irreversible Methods

A permanent, irreversible method, performed on a man or a women.

1) Female – Tubal ligation:

- Laparoscopic sterilization (mini laparotomy): ring, clips, diathermy, laser
- Pre – counseling include:
    - Irreversible and permanent nature of the procedure.
    - Failure rate 1:200
    - Risk of laparoscopy and chance of requiring laparotomy.

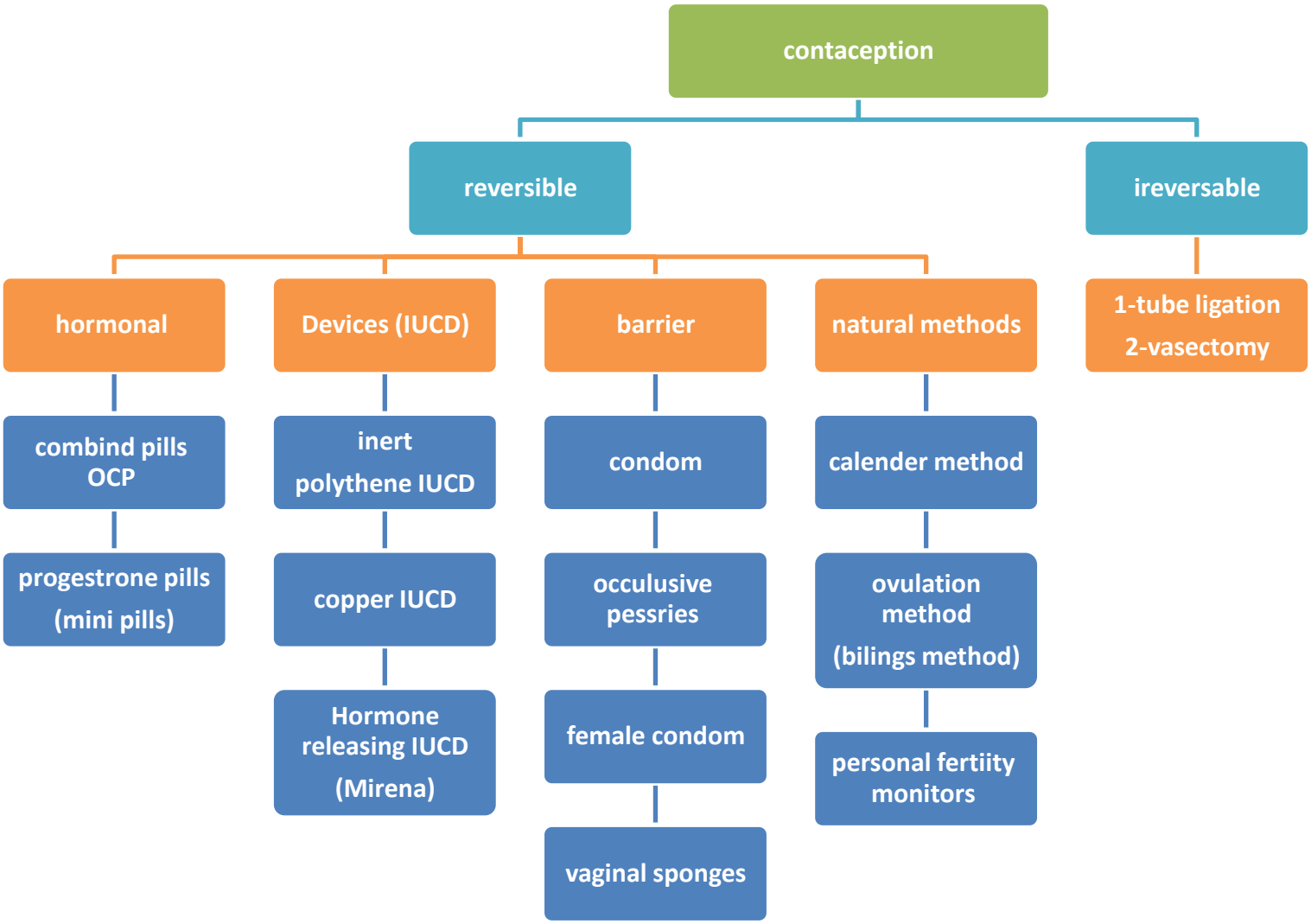
2) Male – Vasectomy:

- Vas deferentia can be divided by removal of a piece of each vas under local anaesthesia.
- Advised to use effective contraception until there are two consecutive semen analyses showing azooospermia.
- Failure rate is 1:2000 and it can occur up to 10 years as a result of late recanalization.
- Minor complication can occur in 5% of patient:
    - Vaso vagal reaction.
    - Hematoma.
    - Mild infection.
    - Sperm auto antibodies – difficulty in reversing the operation.

Summary 1

Category	Mechanism	Advantages	Disadvantages
Combined Oral Contraceptive Pills	-Stops ovulation -Cervical mucus modification -Thins uterine lining - Direct effect on fallopian tubes	Less incidence of: -Dysmenorroea and menorrhagia -Carcinoma of the endometrium and ovary -Benign breast disease -Pelvic inflammatory disease -Ovarian cyst -Acne Protection against rheumatoid arthritis, thyroid disease and duodenal ulceration.	-No protection from STDs -Increase risks of thromboembolism -Weight gain -Effect on insulin secretion -Affects ratio of HDL/LDL -Breast Cancer -Cervical cancer
Progestrone Only Contraceptive	-Stops ovulation -Cervical mucus modification -Endometrial modifications	-Minimal impact on lipid profile and hypertension -Can be used in cardiovascular disease -Used by lactating mother	-Menstrual disturbance -Ammenorrhoea with injection -Functional ovarian cyst -Breast tenderness -Lose of libido -Acne -Headaches
Depot Medroxyprogesterone Acetate (DMPA)	Given once every 3 months	Protection against: -Endometrial cancer -Acute PID -Vaginal candidiasis -Ovarian cancer, endometriosis and fibroids	
IUCD	-A foreign body reaction in the endometrium -Alteration of uterine and tubal fluid -The progesterone IUCD (LNG.IUS) cause endometrial suppression and change in the cervical mucus		-Dysmenorrhoea and Menorrhagia -Infections -Risk of ectopic pregnancy is greater with IUCD especially progesterone releasing IUCD. Contraindications: Pelvic inflammatory disease. -Menorrhagia. -History of previous ectopic pregnancy. -Severe dysmenorrhea.

Summary 2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Devices (IUCD)
barrier
natural methods
1-tube ligation
2-vasectomy
combind pills
OCP
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polythene IUCD
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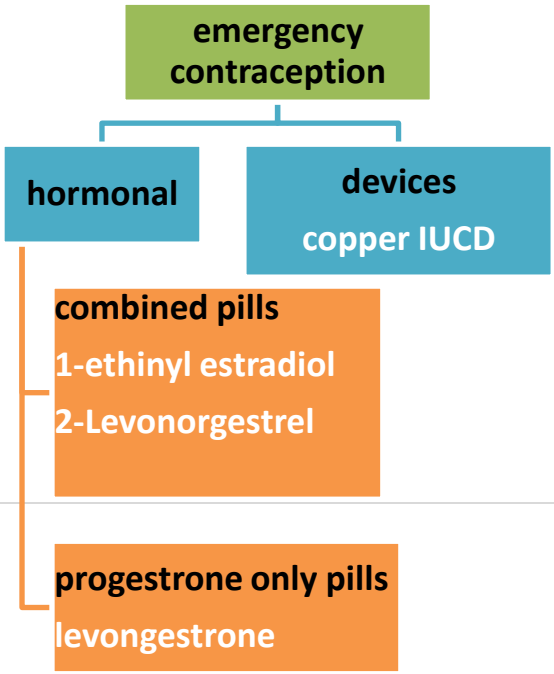

emergency
contraception
hormonal
devices
copper IUCD
combined pills
1-ethinyl estradiol
2-Levonorgestrel
progestrone only pills
levongestrone

Hormones :

	Advantages	Disadvantages
Progesterone pills	Dysmenorrhea DUB PID Overian CA , endomatruim CA, Fibroid	Amenorrhea Menorrhagia Overian cyst Acne , brest tenderness Libido
Combined pills	Overian and endomatruim CA Overian cyst Thyroid ,rheumatoid arthritis Duodenal ulcer	Weight gain Lipid profile Insulin secretion Cardiac diseases MI DVT and thromosis Cervical and breast CA

Women who are (breast feed, HTN or smoker) >>>>> they should only use progesterone only pills (mini pills)

Devices :

Complications:

1-infection (pelvic Actinomycosis abscess)

2-malpostion

3-pregnancy

4-ectopic pregnancy

5-expulsion

6- dysmenorrhea , menorrhagia

Patient Safety.

First, Do No Harm: First basic in medicine.

" Medicine used to be simple, ineffective & relatively safe. Now it is complex, effective & potentially dangerous"

Sir. Cyril Chantler, University College London

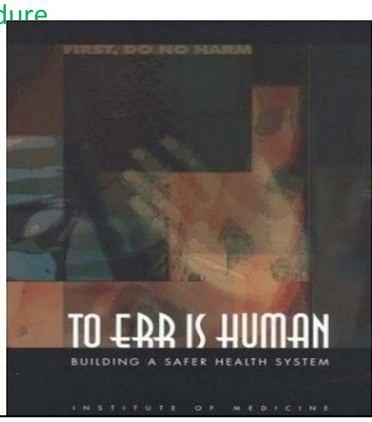
For example Medicine now have organ transplantation which is new and complex procedure

Scope of Problem & History of Patient Safety:

- 1999: IOM

To Err is Human: Building a Safer Health Care System

- 44,000 - 98,000 Americans die each year from medical errors



Published November 1999

Medical Error Theory:

- Four factors contributing to medical errors:

1- Human fallibility . nobody is immune from making mistakes.

2- Complexity . we are dealing with very complex system now.

3- System deficiencies. there is no system is perfect

4- Vulnerability of defensive barriers . there is always gap.

1- Human fallibility

- **"** To err is human": mistakes are part of the human condition.

- **System changes to make it harder to do the wrong & easy to dothe right thing.**
  Like if you had child and there is something harmful to him what will you do ? you will put the harmful thing in far hidden place that is not reachable for the child.

A- Forcing functions.

B- Reminders @ the point of care.

A-Forcing functions:

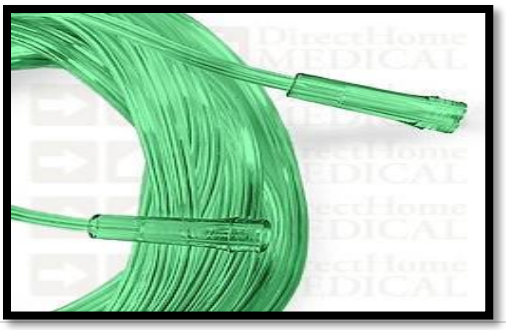
- physical or process constraints that make errors difficult if not impossible.

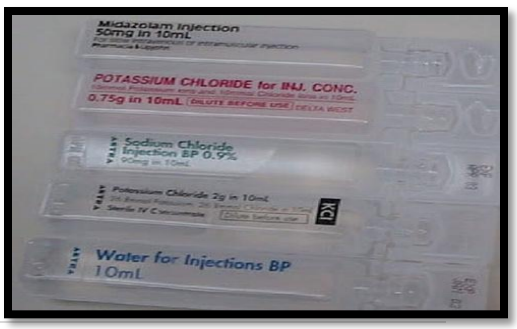
Example: in the past , the couplings connecting the various gases to the anesthesia machine were universal. The oxygen could be connected to the nitrous oxide port and vice versa

KCL:……

the company made changes to the end of tubes like the oxygen tube will not fit in the nitrous oxide tube so here the anesthetic will not by mistake do errors . this is what we mean by making the system harder .

Also don't put similar tubes beside each other.





B-Reminders at the point of care:

- keeping a checklist to help ensure the steps are performed in the proper sequence.

Thermachoice Endometrial Ablation System (Gynecare):

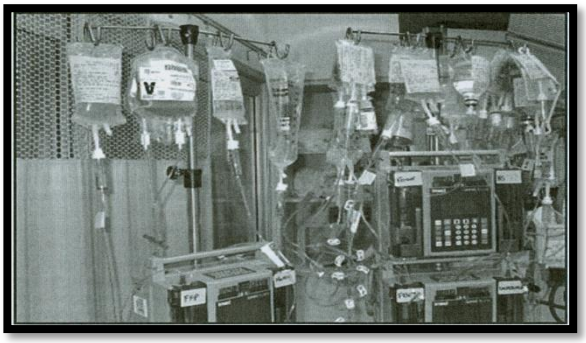
- checklist attached to machine that lists the sequence for the nurse to properly attach the connections.
- machine itself prompts the physician on the order of the steps and monitors the completion of one step before proceeding to the next.

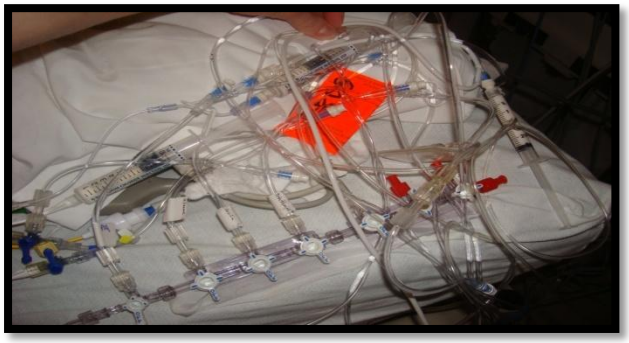
All new machines now have checklist to make it safer .

2- Complexity

Modern health care is the **most complex** activity ever undertaken by human beings.

- **Inpatient medication system**





It like looking for the needle in the haystack

Table 1
Inpatient medication system

Prescribe ⟶	Transcribe ⟶	Dispensing ⟶	Administer ⟶	Monitor
Clinical decision	Receive order	Data entry	Receive from pharmacy	Assess therapy effect
Choose drug	Verify correct	Prepare, mix, compound	Prepare to administer	Assess side effects
Determine dose	Check allergy	Check Accuracy	Verify order and allergy	Review labs
Med record document		Check allergy	Administer drug	Treat side effects
Order		Dispense to unit	Document in MAR	Document

Abbreviation: MAR, medication administration record.

Adapted from Aspden P, Wolcott J, Bootman, JL, et al. Preventing medication errors. Washington, DC: The National Academies Press; 2006. p. 60; with permission.

Before giving any medication you should check if the patient have any allergy or if she is pregnant or lactating .

- it shows the major steps in this process
  - Each of these major steps has several components , all potential sources for error
  - This system is complex and disjointed
  - Strategy to improve medication safety would include simplifying and standardizing the process by using tools e.g., electronic prescribing

The pharmacist before giving medication to patient he/she should ask if there is any allergy or double check with the doctor if this is the right medication for this patient.
So the pharmacist considered the second defense line.

3- System deficiencies& defensive Barriers: MSQ

- 2 major components: Sharp & Blunt Ends

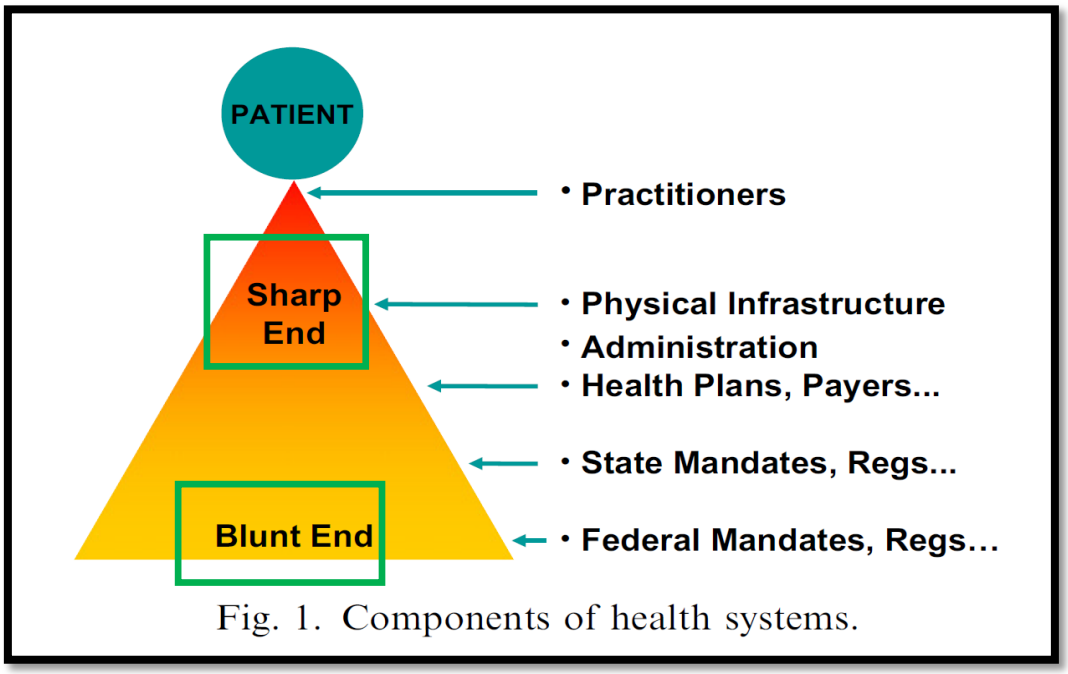


Fig. 1. Components of health systems.

Practitioners such as physicians ,dietitians ,technicians ,nurses….etc people who have direct relationship with patients they are in the sharp end.

The blunt end are those who have indirect relationship with patient s such as Insurance companies if the procedure is so expensive for them to cover which will lead to medical errors for the patient because the patient will undergo less expensive procedure but will not be the ideal procedure for this patient .
Also the tests for women who want to do IVF some of medical system say there is no need for screening for infectious disease this is indirect relationship because the people who put this rules have nothing to do with patients.
Here the press and media always blame people on the sharp end for medical errors .

1- Active Errors (usually practitioners errors):

Example :if a patient want to do right knee surgery but ended up with left knee surgery who will they blame ? maybe the patient came with incomplete documentation ,nurse wrote the wrong side ,surgeon didn't sleep well. these all happen on the sharp end .

-@ **the <u>sharp</u> end** of care.

- **<u>Immediate</u>** effects.

such as: collapse or giving the patient antihypertensive 100 mg instead of 20 mg here the patient will undergo into hypertensive state .

- Generally **unpredictable & unpreventable .**

like if the nurse instead of giving the patient calcium chloride she give sodium chloride.

- Example: inadvertent bladder injury during a hysterectomy for endometriosis with multiple adhesions.

- There is no *" system"* that would prevent this injury .

2- Latent Error(usually system error) : " An Accident Waiting To Happen " unless you take some steps to avoid you will do that mistake.

System deficiencies **<u>hidden</u> in the blunt end** of care.

- *Holes in Swiss cheese*

- We work around these risks until the wrong set of circumstances occur → Patient injury

- **Examples:** understaffing(one nurse responsible for 3 rooms), engineering defects(the monitor is far away from the surgeon in the procedure room) .

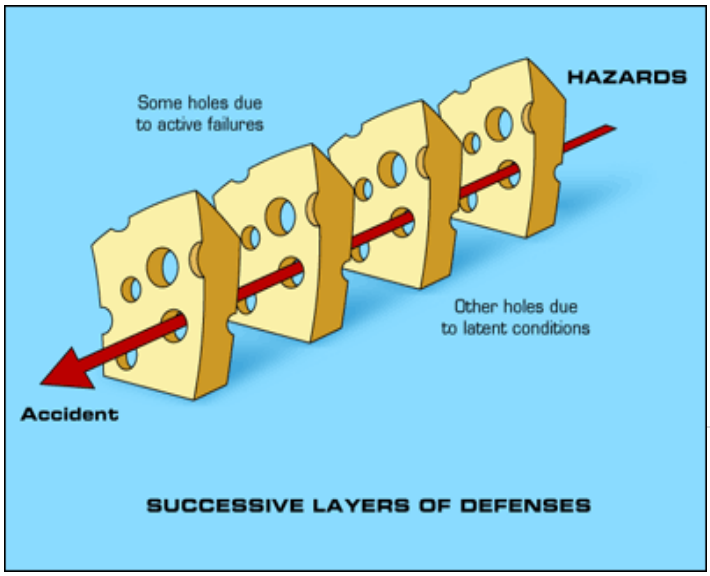
Medical Errors & Swiss Cheese

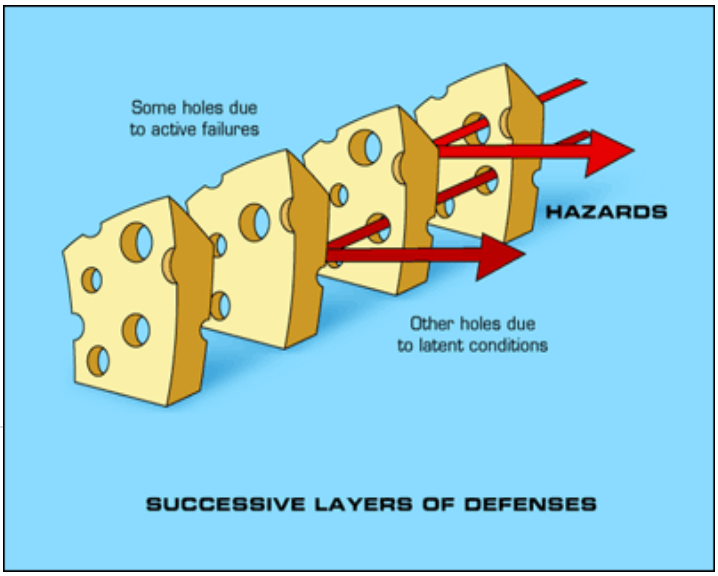
Defensive Barriers: Swiss cheese Model:

Example; if the nurse forget to check allergy the pharmacist will check the allergy.

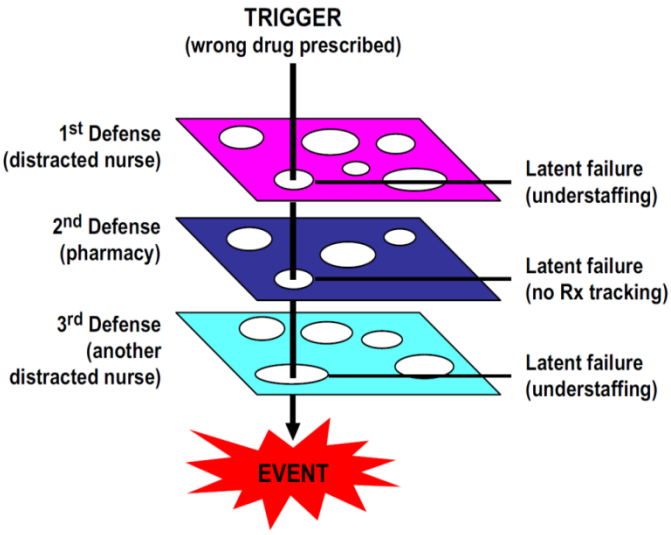
Also in the surgical equipments ,if the surgeon forget there is alarm or notes in the system says that the system is not ready to be used.

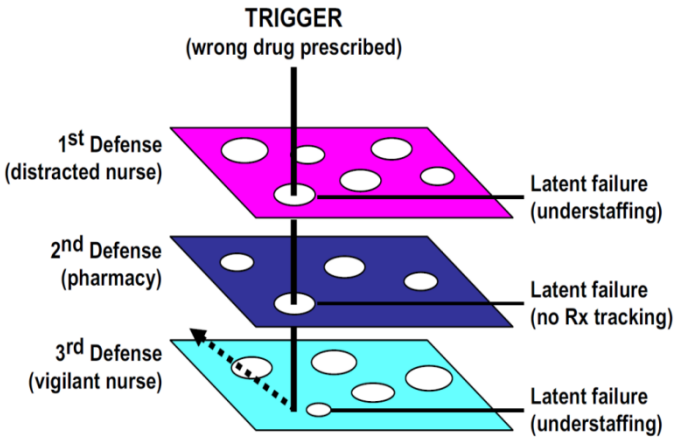
These will prevent errors from happening so remember if Swiss chess in straight line once we do something the error will not happen .





Trajectory of Error & Defensive Barriers



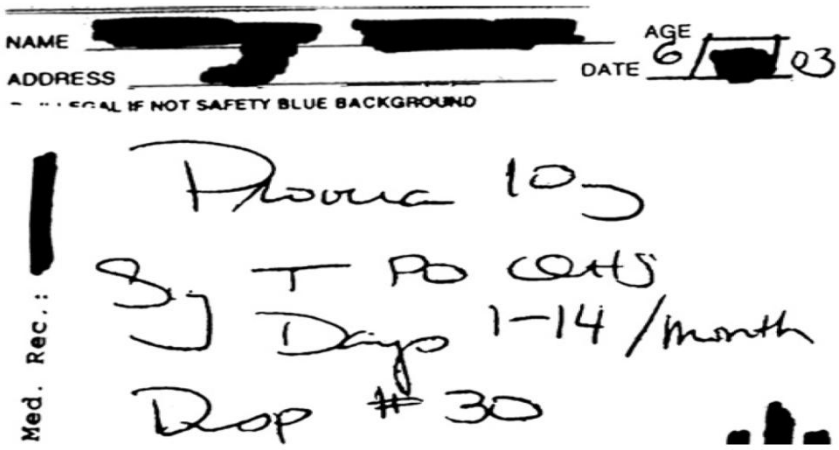


Defensive Barriers

- No defensive barrier is perfect.
- Each has inherent vulnerabilities (holes) that, under the wrong circumstances, can be pierced by the trajectory of error.
- Complex medical processes often have multiple layers of such barriers.
- When the potential defects in each of these barriers <u>align</u> in just the wrong way, errors will not be deflected and patient injury/death will results.
- Preventing harm: By interposing another piece of " Swiss cheese" between the hazard and the potential injury.

Practical solutions to improve safety in OB & GYN:

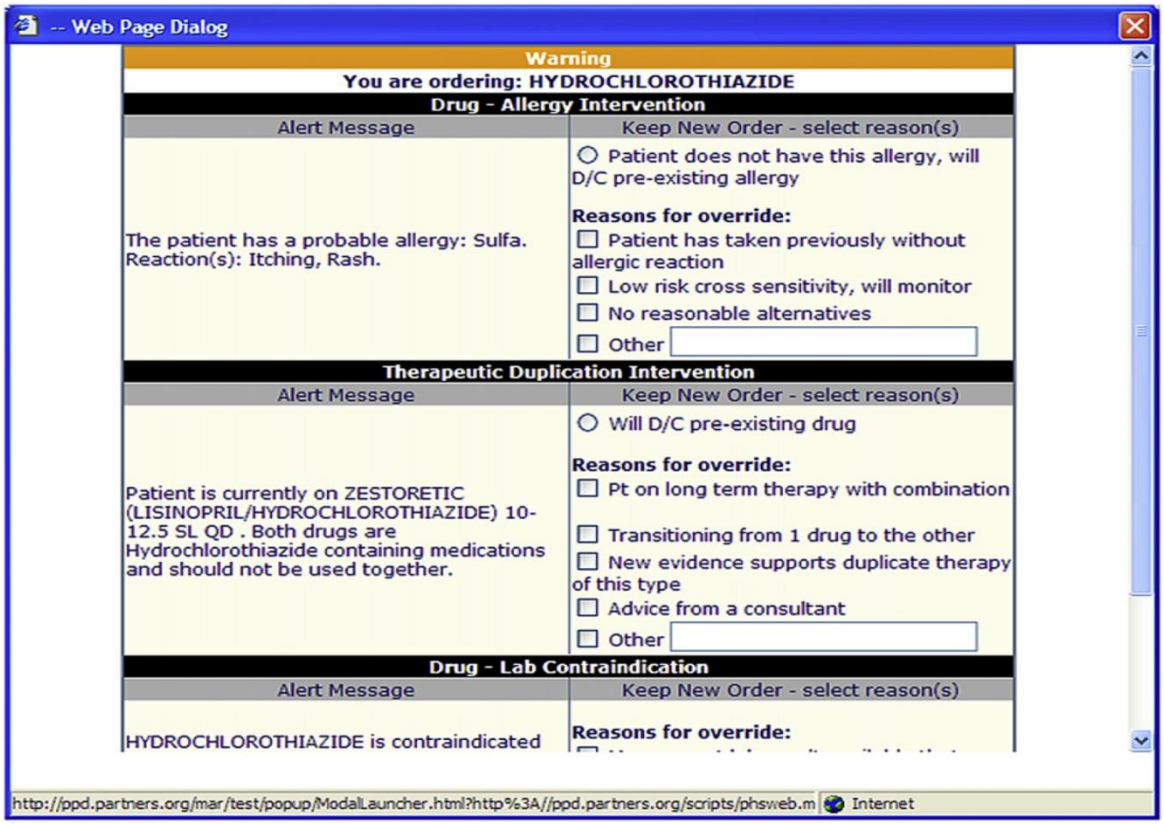
- **Medication errors** account for the **<u>largest</u>** # of errors in health care



The patient was given Prozac (instead of the intended Provera (medroxyprogesterone)).

The solution for these medication errors is **electronic system.**

Medication Error: Advance Decision Support Alert

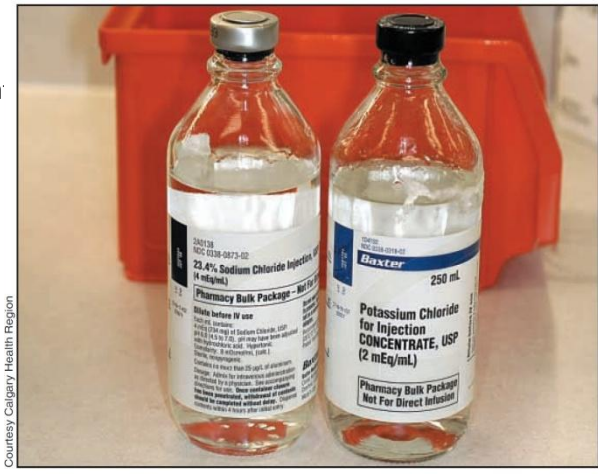


March 2004 : Unexplained hyperkalemiain in an elderly patient undergoing continuous renal replacement therapy in the ICU a led to an analysis of the dialysate solution.

- The solution was found to contain 6 mmol/L sodium (should have been 110 mmol/L) and 60 mmol/L potassium (should have been zero).

the nurse instead of giving the patient with(renal dialysis) sodium chloride she give potassium chloride the patient went into arrhythmia and she was about to die but they revived her ,when they did blood tests they discovered that the potassium is 100 times the normal range .

to prevent this from happening don't put these bottles side by side or put them in the pharmacy

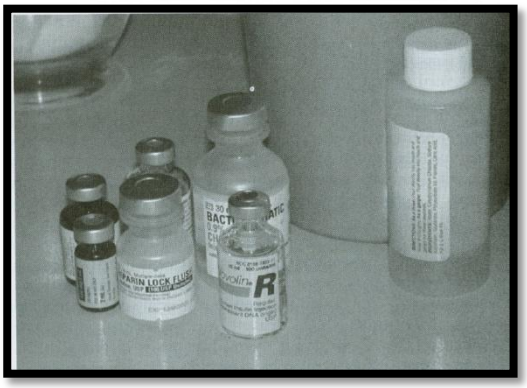


Sodium chloride and potassium chloride bottles: a dangerous similarity

Indiana Hospital: September 2006

Similar vials of heparin involved in fatal dispensing error in neonatal setting (the doses for adults and infants were similarly packaged)3 preterm infants died as a result of lethal overdoses of IV heparin.

Heparin and insulin vials on a bedside tray



To prevent errors here each nurse should bring her Own medication with her , if morning nurse give heparin she should take it back and if patient need insulin at night the night nurse should give night dose and take it back so, we don't collect the medication by the bed side . here we will prevent the hole in Swiss chess.

Medication Safety & Errors:

- Clear handwriting
- Distinguishing between look-alike and sound-alike drugs. such as: gentamicin , erythromycin
- Avoid using abbreviations/ non-standard abbrev. Such as :o/d instead of once a day.
- Electronic system for generating & transmitting Rxs .
- All prescriptions should include detailed instructions to pt for using the medications.
- Comprehensive recommendations/guidelines published by ACOG, ACS & Joint Commission.

JCAHO's "do not use" list

To comply with Goal 2, hospitals are required develop a list of abbreviations, acronyms, and symbols that must not be used in orders or other medication-related documentation that are handwritten, are entered into a computer, or appear on pre-printed forms. JCAHO has created its own "do not use" list that facilities can emulate.

Do not use	Potential problem	Use instead
U (unit)	Mistaken for "0" (zero), the number "4", or "cc"	Write "unit."
IU (international unit)	Mistaken for IV or the number 10	Write "International Unit."
Q.D., QD, q.d., qd (daily) and Q.O.D., QOD, q.o.d., qod (every other day)	Mistaken for each other. Period after the Q mistaken for "I" and the "O" mistaken for "I"	Write "daily" or "every other day."
Trailing zero (X.0 mg) Lack of leading zero (.X mg)	Decimal point may be missed.	Write "X mg" or "0.X mg." (Trailing zero may be used only when required to demonstrate the level of precision of the value being reported, such as for lab results, imaging studies that report the size of lesions, or catheter/tube sizes.)
MS	Can mean morphine sulfate or magnesium sulfate	Write "morphine sulfate" or "magnesium sulfate."
MSO_4 and $MgSO_4$	Mistaken for each other	Write "morphine sulfate" or "magnesium sulfate."

In addition, JCAHO is considering the following items for inclusion on its do not use list: All abbreviations for drug names; the symbols "<" (less than), ">" (greater than), and "@" (at); the abbreviations "cc" and "μg"; and apothecary units. While these items are not currently prohibited, eliminating them now will make it easier to meet this requirement if JCAHO does add them to the list in coming years.

Source: Joint Commission on Accreditation of Healthcare Organizations. "The official Do Not Use list." 2006. www.jointcommission.org/PatientSafety/DoNotUseList2006 (11 Sept. 2006).

Patient Role in her safety: patient have a rule in preventing errors.

- Speak up if you have questions or concerns
- Pay attention to the care you're receiving
- Educate yourself about your diagnosis , tests you are undergoing and your treatment plan
- Know what medications you take and why you take them *(medication errors are the most common healthcare errors)*
- Participate in all decisions about your treatment

Examples:

There was a patient allergic to penicillin her doctor was in holiday and there was a new doctor in the hospital and he is new to the system he prescribed for her **Augmentin** (penicillin) she knew how to read English and she knew that she is allergic to penicillin and yet she take it.

Another patient prescribed for her **provera** taking on the 3 day of period when she went to the pharmacy the y give her **Prozac** taking before breakfast every day but she was smart enough to know the mistake and said that her doctor says that she should take it on 3 day of period here the pharmacist knew that there was a mistake .

Let our Residents Rest!

- 2003: work-hour limitations promulgated by the ACGME
- 2010: new standards

(ACGME) : The Accreditation Council for Graduate Medical Education

- **US National Traffic Safety Administration**

sleepy drivers are responsible for at least 100,000
automobile accidents, 40,000 injuries and 1500
deaths annually

- Sleep deprivation increases errors in performing <u>even</u> simple familiar tasks
  - needle sticks
  - puncture wounds
  - lacerations
  - medical errors
  - motor vehicle

Sleep deprivation affects human cognitive and physical function

- It has long been recognized that fatigue can affect human cognitive and physical function
- There is increasing awareness within the patient safety movement that fatigue, even partial sleep deprivation, impairs performance

Surgical Environment:

- In **Obstetrics & Gynecology** , the risks of surgical error may have increased because :

- ↑C.S
- ↑MIS
- Robot-assisted laparoscopy.
- Pressure for shorter lengths of stay post op.
- More outpatient procedures .

Retained Foreign Objects:

- Sponges, surgical instruments
- Indefensible!
- "Correct sponge count" does not exonerate the surgeon

If they forget objects inside the patient during a procedure and he raises a lawsuit against the nurse or surgeon the patient always win the lawsuit there is no excuse for them to forget these objects because they have a check list for every objects and they should double check .

So before finishing the procedure wait for the nurse to tell that the count is correct and they repeat it twice.

Table 1. Characteristics of 54 Cases of a Retained Foreign Body after Surgery.

Characteristic	No. of Cases (%)
Type of foreign body retained	
Sponge	37 (69)
>1 Sponge	4 (7)
Clamp	4 (7)
Other (e.g., retractor or electrode)	13 (24)
Cavity in which foreign body was left	
Abdomen or pelvis	29 (54)
Vagina	12 (22)
Thorax	4 (7)
Other	9 (17)
Outcomes	
Death	1 (2)
Readmission to hospital or prolonged hospital stay	32 (59)
Sepsis or infection	23 (43)
Reoperation	37 (69)
Fistula or small-bowel obstruction	8 (15)
Visceral perforation	4 (7)

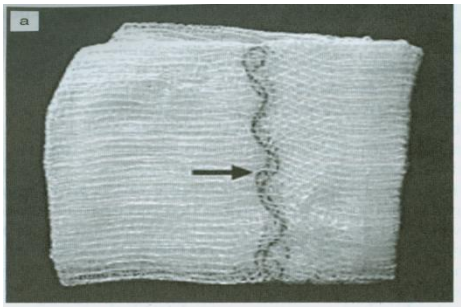
Table 3. Risk Factors for Retention of a Foreign Body after Surgery.*

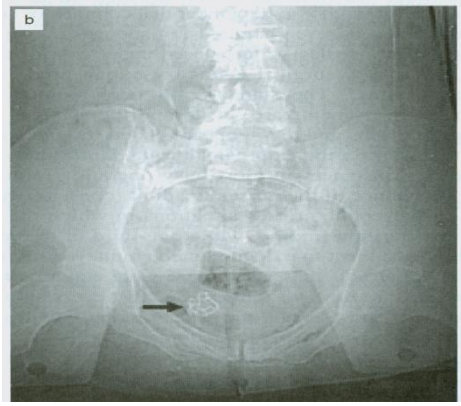
Characteristic	Risk Ratio (95% CI)	P Value
Operation performed on an emergency basis	8.8 (2.4–31.9)	<0.001
Unexpected change in operation	4.1 (1.4–12.4)	0.01
>1 Surgical team involved	3.4 (0.8–14.1)	0.10
Change in nursing staff during procedure	1.9 (0.7–5.4)	0.24
Body-mass index (per 1-unit increment)	1.1 (1.0–1.2)	0.01
Estimated volume of blood lost (per 100-ml increment)	1.0 (1.0–1.0)	0.19
Counts of sponges and instruments performed	0.6 (0.03–13.9)	0.76
Female sex	0.4 (0.1–1.3)	0.13

Sponge is the most common forgetful object , and most common place they forget it in Are abdomen and pelvis .

A- surgical sponge with an embedded radiopaque thread on X-ray
if the forget the sponge they can detect the thread on x-ray .

Surgical Environment





2- Surgical Fire

rare

- We in O & G have all the 3 elements necessary to start / support fires:

1- oxidizers: supplies of oxygen gas

2- ignition sources: electrocautary, fiberoptic light cables, lasers

3- flammable fuels: surgical drapes, **alcohol-based** prepping agents, anesthetic gases

Medication errors

- Prophylactic ABX: demonstrated effectiveness in reducing surgical morbidity.
- Failure to use them when appropriate is a medication error
  - inappropriate choice of agent
  - ineffective start of administration
  - incorrect duration of exposure

4- Venous thromboembolism

- Failure to use accepted surgical thromboprophylaxis is another class of surgical error in patient safety
- Without effective thromboprophylaxis, major gynecologic surgery is associated with a prevalence of DVT 15 - 40%
- ACOG recommends:
  - Low
  - Medium
  - High
  - Highest

Transition & Handoff Errors

- **“** Care transition ” , “ Hand over ”or “ shift change”
- these are the most dangerous times for example: on weekends there is only the on call doctor and they don’t know about the patient who came on Sunday or Monday this will lead to medical errors .
- Risky time:

1- Provider handoff

2- Patient handoff

-Involves breakage of the continuity of care

-breakdowns and inconsistencies in the handoff process contribute to medical errors