

Labour and delivery for post basic students

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Normal labor and delivery

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NORMAL LABOUR AND DELIVERY

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Definition of terms

Labor :a coordinated effective sequence of involuntary uterine contractions that result in effacement and dilatation of the cervix and a voluntary bearing down effort leading to the expulsion per vaginum of products of conception after 28 weeks of gestation

Delivery : the mode of actual expulsion of the fetus and the placenta

Parturition: the birth process

Parturient: a woman in labor

- Normal labor: Criteria for normal labor (eutocia)
 - At term
 - Spontaneous onset
 - Vertex presentation
 - With out due prolongation
 - Natural termination with minimal aid
 - With out any complication to the mother and/or baby

True Labour Pain	False Labour Pain
Regular.	Irregular.
Increase progressively in frequency, duration and intensity.	Do not.
Pain is felt in the abdomen and radiating to the back.	Pain is felt mainly in the abdomen.
Progressive dilatation and effacement of the cervix.	No effect on the cervix.
Membranes are bulging during contractions.	No bulging of the membranes.
Not relieved by antispasmodics or sedatives.	Can be relieved by antispasmodics and sedatives.

WHAT INITIATE LABOUR “ONSET OF LABOUR”



Mechanism of initiation

- Two school of thoughts or theorems
 - 1. Functional loss of pregnancy maintenance factors
 - 2. Synthesis of factors which induce parturition

Mechanisms of initiation of labor

Pro-pregnancy

- ✓ Progesterone
- ✓ Prostacyclin (PGI-2)
- ✓ Relaxin
- ✓ Parathyroid hormone-related peptide
- ✓ Nitric oxide
- ✓ Calcitonin gene-related peptide
- ✓ Adrenomedullin
- ✓ Vasoactive intestinal peptide

Pro-labor factors

- Estrogen
- Oxytocin
- Prostaglandins
- Prostaglandin dehydrogenase
- Inflammatory mediators

Theories

- **Hormonal factors**

- 1) *Estrogen theory*

- 2) *Progesterone withdrawal theory*

- 3) *Prostaglandins theory*

- 4) *Oxytocin theory*

- 5) *Fetal cortisol theory*

- **Mechanical factors**

- 1) *Uterine distension theory*

Parturition cascade

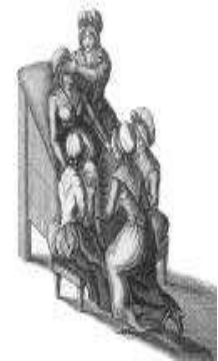
Estrogen theory

- Progesterone inhibits contractibility
- Estrogen increases contractibility
- At 7th month, estrogen still increasing but progesterone drops off slightly
- High Estrogen: Progesterone ratio excites uterus

During pregnancy, most of the estrogens are present in a binding form

During the last trimester, more free estrogen appears increasing

- ✓ the excitability of the myometrium and
- ✓ prostaglandins synthesis



Progesterone withdrawal theory

- **Classical Progesterone Withdrawal Does Not Cause Human Parturition**
 - in humans the inhibition of progesterone action is important for activation phase of parturition
 - Administeration of RU 486, or mifepristone
 - during the latter phase of the ovarian cycle, → induces premature menstruation
 - during early stages of pregnancy, → induces abortion
 - later in pregnancy, → ripening of the cervix and increasing myometrium sensitivity to uterotonins
 - inhibiting the enzyme 3-hydroxysteroid dehydrogenase
Induces labor

– But in primates plasma progesterone levels do

- **Functional Progesterone Withdrawal in Human Parturition**

- Could be mediated in the uterus through several mechanisms, including:

- Changes in the relative expression of the progesterone receptor or of its two **isoforms** (PR-A and PR-B).

- PR-B isoform is more transcriptionally active, and inhibit PR-A activity

- there is a shift in relative ratio of PR-A to PR-B within the myometrium late in gestation

- the ratio is similarly modified in decidua and chorion

- **Posttranslational modifications** of the progesterone receptor causing decreased activity.

- Alterations in progesterone receptor activity through changes in the expression of **co-activators or co-repressors** that directly influence receptor function

Prostaglandins theory:

- Prostaglandins E2 and F2 α are powerful stimulators of uterine muscle activity
- PGF2 α was found to be increased in maternal and fetal blood as well as the amniotic fluid late in pregnancy and during labor

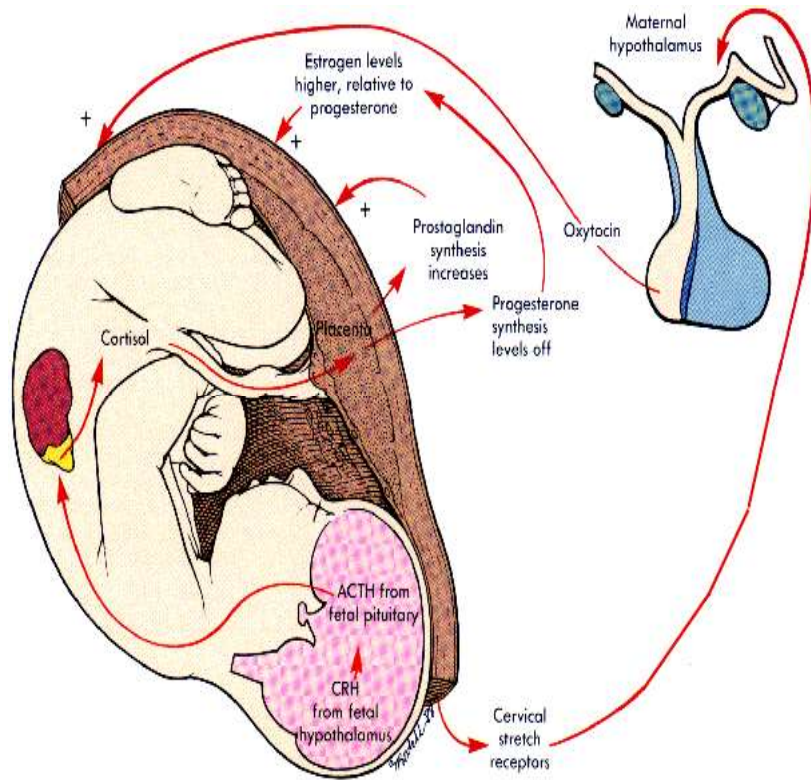
Oxytocin theory:

- Although oxytocin is a powerful stimulator of uterine contraction, its natural role in onset of labour is doubtful
- The secretion of oxytocinase enzyme from the placenta is decreased near term due to placental ischaemia leading to predominance of oxytocin's action

Fetal cortisol theory:

- Increased cortisol production from the fetal adrenal gland before labor may influence its onset by increasing estrogen production from the placenta

Fetal cortisol theory



- Fetal Hypothalamus secretes Corticotropin Releasing Hormone near term which stimulates the
- Fetal Anterior Pituitary to secrete adrenocorticotropin hormone (ACTH)
- ACTH stimulates fetal adrenal cortex to produce cortisol
- Cortisol stimulates secretion of estrogen from placenta, inhibition of P synthesis -> uterine contractions -> stimulates oxytocin -> hyp
- Fetuses with adrenal **hypoplasia** are often post-date and labor is slow to start

Mechanical factors

Uterine distension theory:

- Like any hollow organ in the body, when the uterus is distended to a certain limit, it starts to contract to evacuate its contents. **This explains the preterm labour in case of multiple pregnancy and polyhydramnios.**

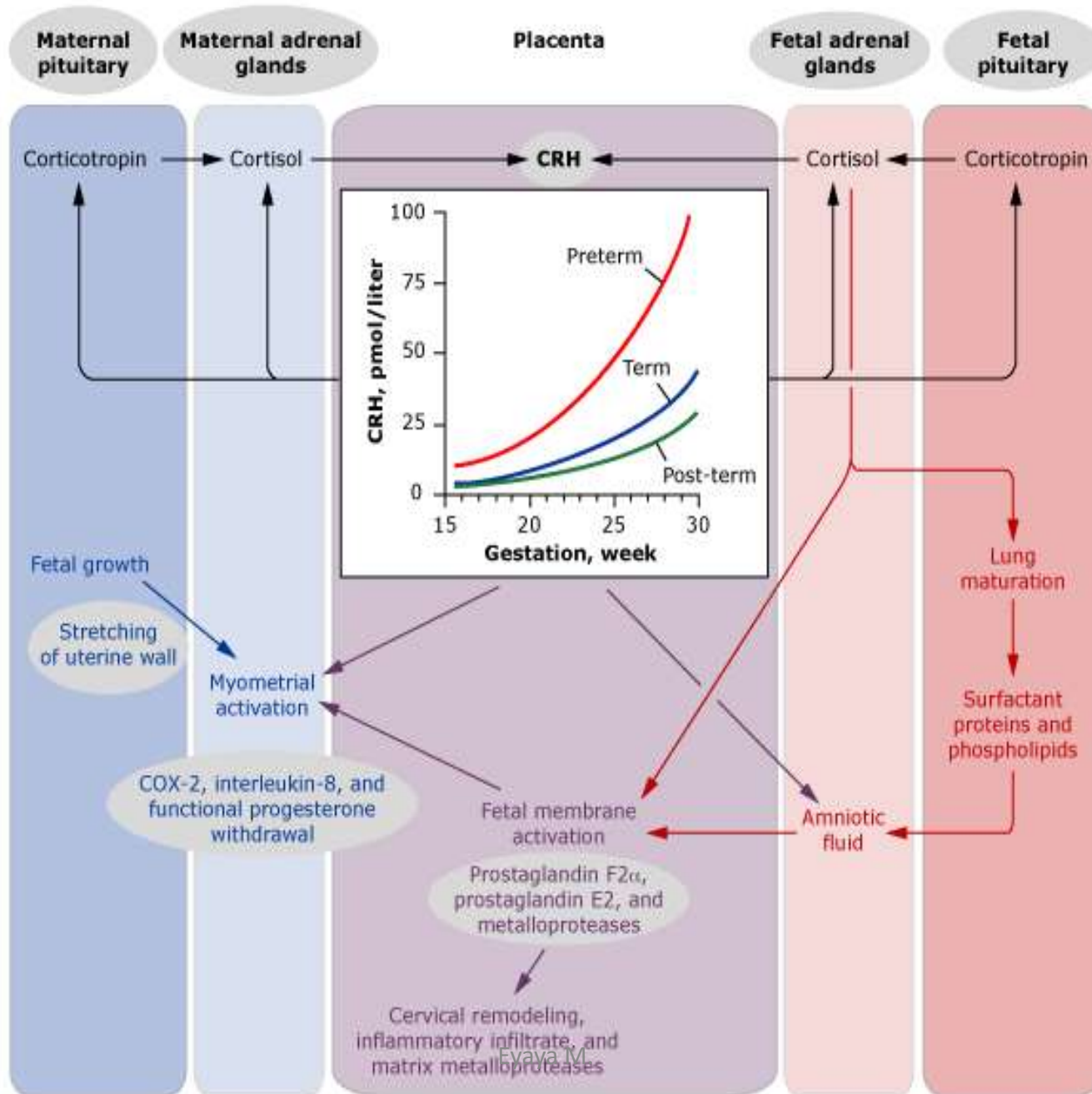
Mechanotransduction

- Stretch of the lower uterine segment by the presenting part near term
 - Stretch increased expression of **the gap junction protein, connexin 43, as well as oxytocin receptors**
 - stretch plays an integrated role with fetal/maternal

Fail-Safe Systems for Uterine Activation

- During the Hippocratic period, the fetus was thought to be positioned head down at term so it could kick its legs up against the fundus of the uterus, thereby propelling itself through the birth canal
- a "**parturition cascade**" removes the mechanisms maintaining uterine quiescence and recruits factors promoting uterine activity
- labor results from a **down-regulation of pathways** that favor uterine quiescence

Parturition Cascade



A far from comprehensive list of substances and categories of substances which are known to participate in the birth process

- *Actin Lipocortin*
- Adenylate cyclase Lipopolysaccharide
- Adhesion molecules Lipoxygenase
- (ICAM, VCAM, etc.) Magnesium
- Adrenaline Matrix metalloproteinases
- Bradykinin *Monocyte chemotactic protein-1*
- *Calcium Myosin*
- Calmodulin Myosin light chain kinase
- Chemokines Neutrophil elastase
- Chondroitin sulphate
- *Dehydroepiandrosterone sulphate*
- *Prostaglandin dehydrogenase*
- Dermatan sulphate *Prostaglandin E2*
- Endothelins *Prostaglandin F2 α*
- Glycosaminoglycans Proteoglycans
- G proteins Relaxin
- Gravidin Sodium
- Inositol trisphosphate Substance
- Nitric oxide
- *Collagen Noradrenaline*
- Collagenases Oestrogens
- *Connexin 43 Oxytocin*
- Corticotrophin (ACTH) Oxytocinase
- Corticotrophin-releasing factor Phosphatases
- *Cortisol Phosphodiesterase*
- *cAMP Phospholipases*
- *cGMP Platelet-activating factor*
- Cyclo-oxygenase-1 Potassium
- *Cyclo-oxygenase-2 Progesterone*
- Cytokines Prostacyclin
- *IL-8 Sulphatase*
- Leukotrienes Surfactant
- Vasopressin
- Substance P

Anatomical and Physiological Considerations of the Myometrium

- unique characteristics of smooth muscle
 - **greater degree of shortening** with contractions is than that in striated muscle cells
 - forces can be exerted in any direction
 - smooth muscle fibers are organized in long, random bundles throughout the cells → greater shortening and force-generating capacity
 - the advantage that **multidirectional force** generation in the uterusâ€™fundus versus lower uterine segmentâ€™permits versatility in expulsive force directionality that can be brought to bear

Regulation of Myometrial Contraction and Relaxation

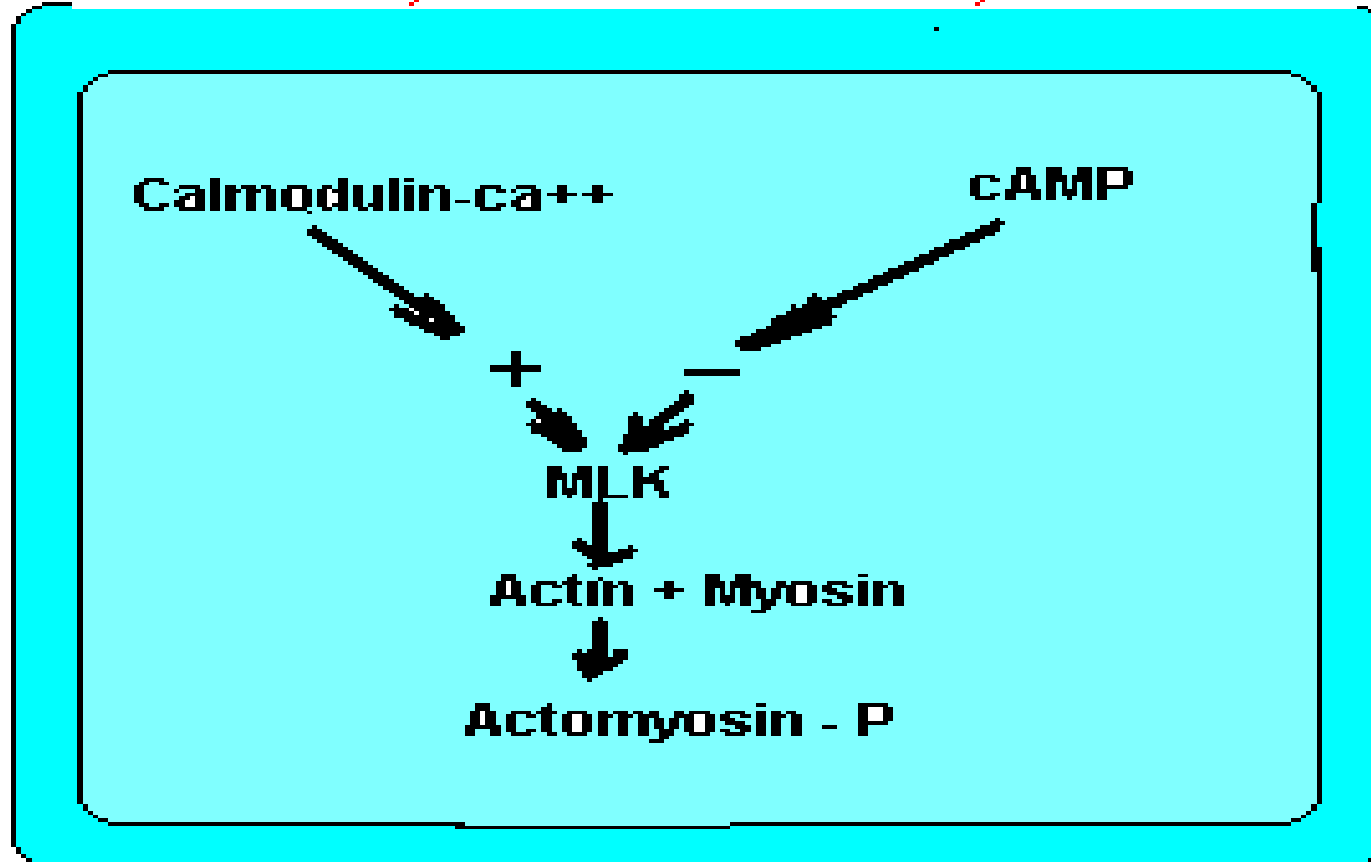
- acute and chronic mechanisms
 - Acutely, the interaction of **myosin and actin**
 - agents that cause an increase in the intracellular cytosolic concentration of calcium ($[Ca^{2+}]_i$) promote contraction
 - cAMP and cGMP act to cause a decrease in $[Ca^{2+}]$
 - chronic **action of hormones** on the contractile status of the cell
 - *Regulation of myometrial response to a hormone can be associated with changes in the course of pregnancy* include
 - channels associated with smooth muscle excitation
- Myometrial Gap Junctions
 - Are transmembrane channels
 - Consist of two protein "hemi-channels," termed **connexons**
 - optimal numbers (area) of functional permeable gap junctions between myometrial cells
 - establishment of electrical synchrony in the myometrium
- Cell Surface Receptors
 - three major classes of cell surface receptors
 - the G-protein-linked,
 - ion channel-linked, and

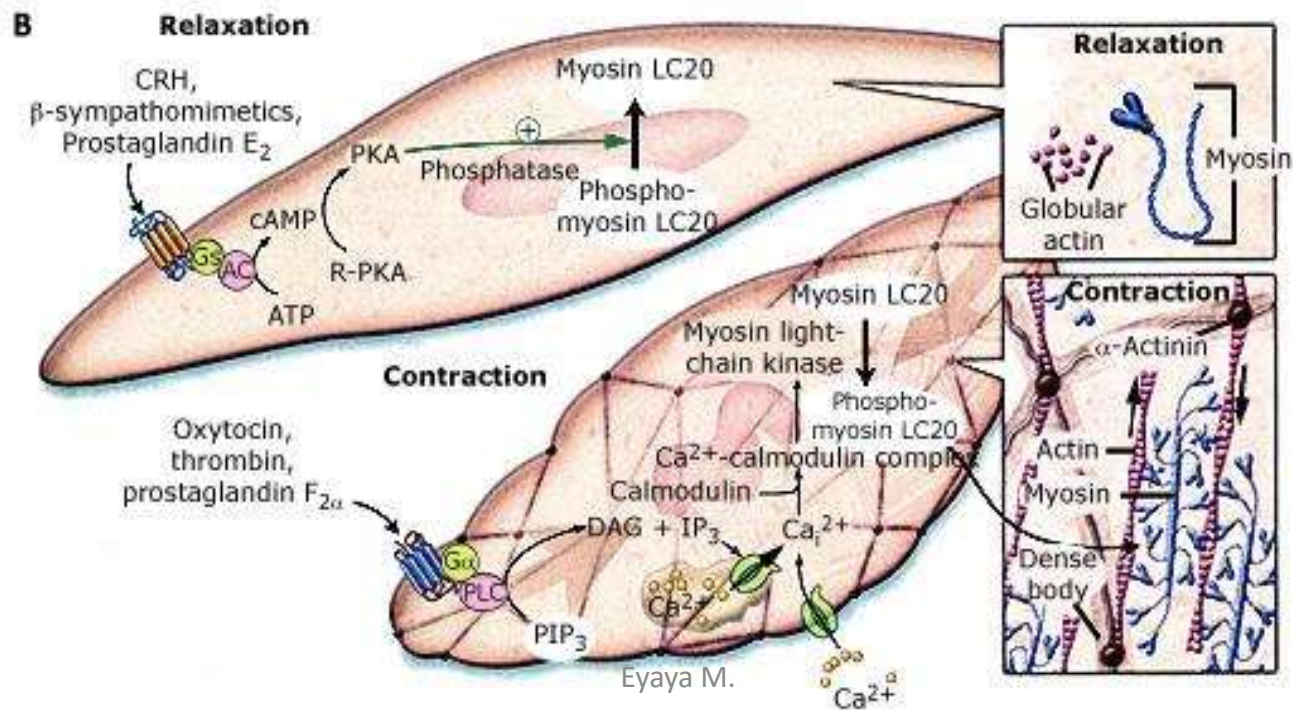
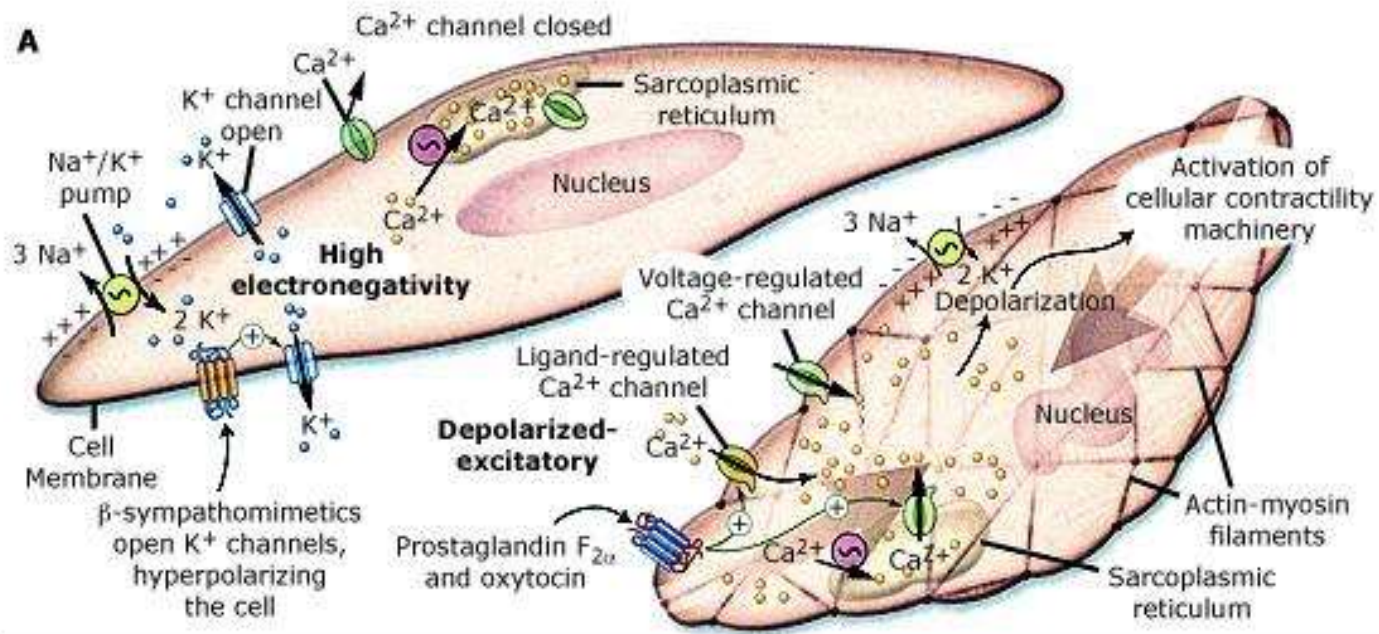
BIOCHEMICAL BASIS OF CONTRACTION

- Muscle contraction is brought about by the sliding of *actin and myosin* filaments fueled by *ATP* and *calcium*
- Unlike the heart, in which the bundle of His is present, no anatomic structures for synchronization of contractions have been found in the uterus

Physiology of uterine contractility

Control of Myometrial Contractility

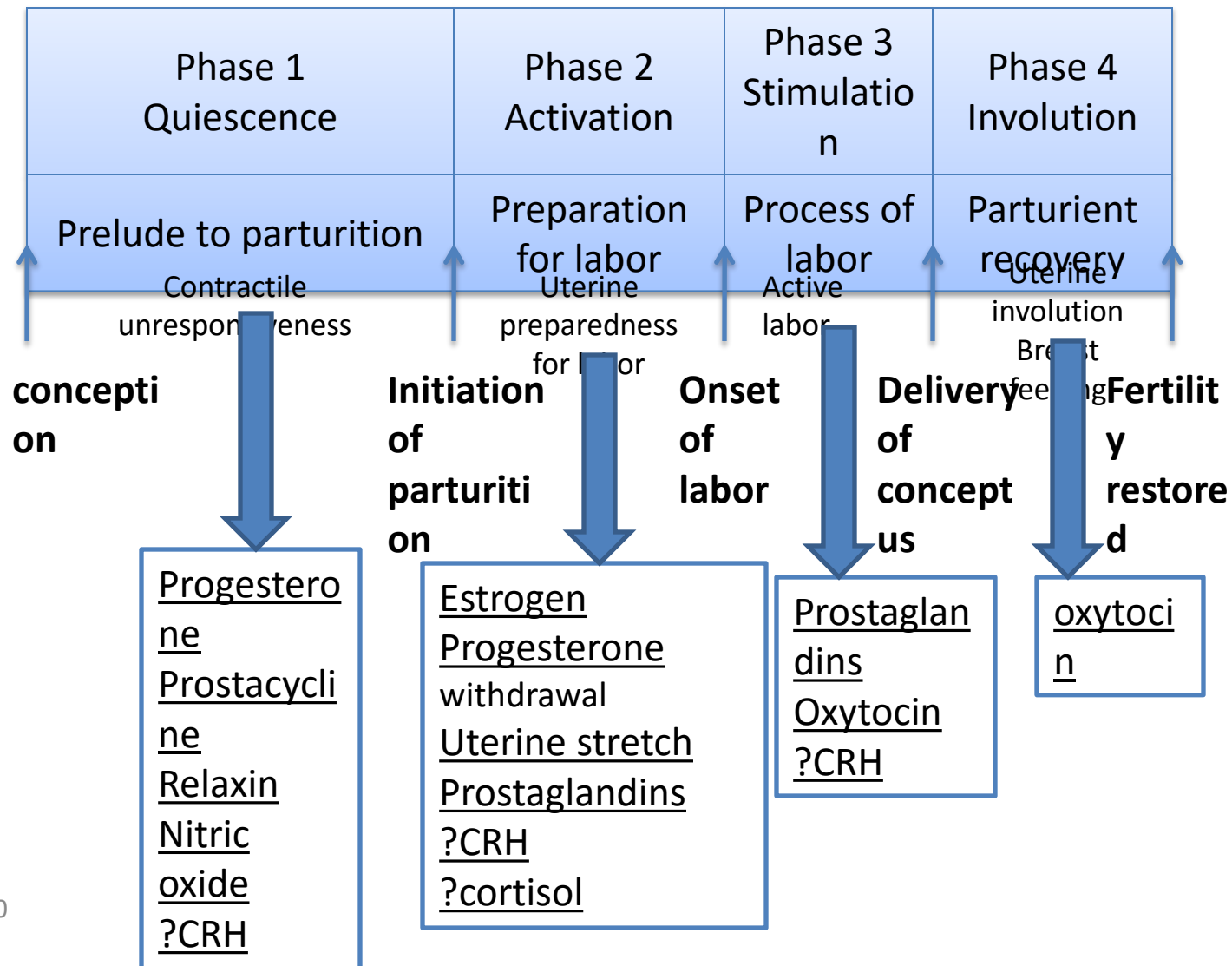




Phases of parturition

- Labor : uterine contractions that effect dilatation of cervix and force fetus through birth canal
- Parturition: bringing forth of young , encompass all physiological processes involved in birthing
 - Phase 1 :Prelude to Parturition
 - Phase 2: Preparation for Labor
 - Phase 3 : Process of Labor
 - Phase 4 : Parturition Recovery

Phases of parturition



Phase 0 of parturition : Uterine Quiescence

- Uterine smooth muscle tranquility with maintenance of cervical structural integrity
- Unresponsive to natural stimuli, **contractile paralysis**
- Myometrium : quiescent state
- 95% of duration of pregnancy spend in this phase
- Begins from the time of implantation
- Cx : firm unyielding
- Successful anatomical structural integrity :essential for successful parturition
- Some myometrial contraction occur toward the end of pregnancy that do not cause Cx dilation → Braxton – Hicks contraction or false labor:

unpredictability in occurrence

low intensity

brief in duration

discomfort – confined to low
abdomen & groin

- Cervix

- Softening : an increase in tissue compliance
 - Hegar sign: softening of the lower segment and the cervix a between 4-6wks of conception
- Structural changes in the extracellular matrix i.e. collagen
 - Increase vascularity
 - Stromal hypertrophy
 - Glandular hypertrophy and hyperplasia
- Reduction in the number and type of collagen crosslinking due to
 - Reduced expression of matricellular proteins

Phase 2 of parturition Myometrial Activation

- (Preparation for Labor, *uterine awakening* or *activation*)
 - Myometrial changes
 - Cervical changes
- Characterized by
 - the development of uterotonin sensitivity,
 - improved intercellular communicability via gap junctions, and
 - alterations in the capacity of myometrial cells to regulate the concentration of cytoplasmic Ca^{2+}
- uterus becomes activated in response to uterotropins, such as estrogen.

4/26/2020 — increased expression of a series of contraction-associated proteins (CAPs) (including myometrial

Premonitory Signs of Labor

1. *Lightening*
2. *shelving*
3. *Braxton Hick's Contractions*
4. *Sudden burst of maternal energy/activity.*
5. *Softening "ripening" of the cervix*
6. *Rupture in the membranes
"BOW"*

Phase 2 of parturition : Cervical change

- Initiation of parturition : Cx soften, yield, more readily dilatable
- Fundus transformed to produce effective contraction that drive fetus through Cx & birth canal
- Failure of coordinated interaction
→ unfavorable preg outcome
- Change of state of bundles of collagen fiber

Collagen breakdown ↑ & rearrangement of collagen fiber bundles (No & size ↓)

Changes in relative amount of glycosaminoglycans (hyaluronic acid, ↑ capacity of Cx to retain water)

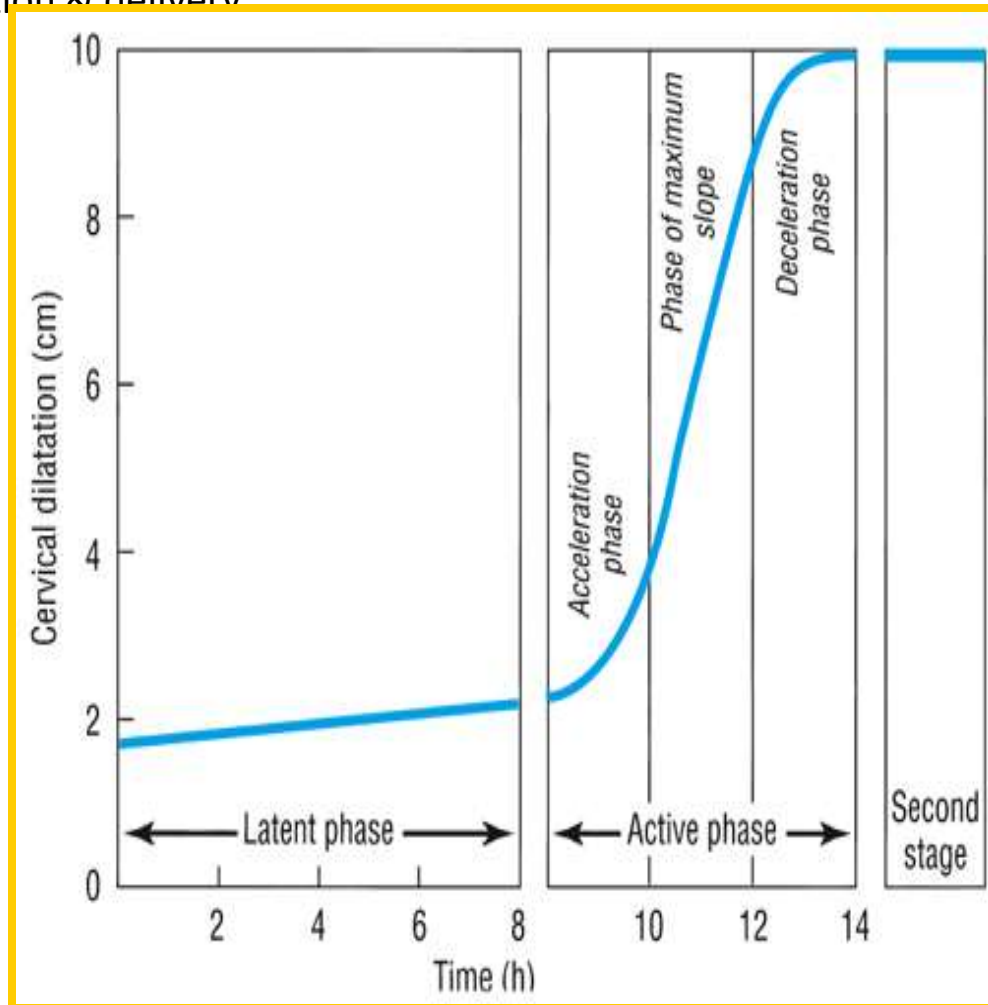
- Dermatan sulfate ↓ (need for collagen fiber cross linking)
- Production of cytokine → degrade collagen → Cx thinning, softening relaxation → Cx initiate dilatation

Myometrial change

- Increase Ut irritability & responsiveness to uterotonins
 - Alterations in expression of key enzyme. CAP(contraction-associated proteins)-control myometrium contractility
 - Myometrial oxytocin R ↑
 - Myometrial cell gap junction protein (ex connexin -43)
- Formation of lower segment

Phase 2 of parturition :process of labor

- Active labor : Ut contrations bring about progressive cervical dilatation & delivery



Three Stages of Labor

1.First stage of labor

Stage of cervical effacement & dilatation

2.Second stage of labor

stage of expulsion of fetus

3.Third stage of labor

stage of separation & expulsion of placenta

1st stage of Labor :

Clinical Onset of Labor

- Show (bloody show)
 - sign of initiation of labor
 - spontaneous discharge of small amount of blood-tinged mucus from vagina – this occurs following effacement of the cervix
 - labor already in progress or ensue during next several hours to days

Uterine Contractions Characteristic of Labor

- Contraction of Ut smooth m during labor : painful
- Involuntary, independent of extra uterine control
 - The intensity, duration and frequency of uterine contraction is unaffected by epidural analgesia or in patients with paraplegia

Cause of pain (not known definitely)

- ① hypoxia of contracted myometrium
- ② compression of nerve ganglia in Cx & lower uterus by interlocking muscle bundles
- ③ stretching of Cx during dilatation
- ④ stretching of peritoneum overlying fundus

Ferguson reflex: mechanical stretching of Cx enhances uterine activity → exact mechanism : not clear

- Some suggest an increase in oxytocin receptors but not proven
- Manipulation of Cx and stripping fetal membranes is associated with increase in PGF_{2α} metabolite (PGFM) in blood → contraction↑

Interval between contractions

: 10 minutes at onset of 1st stage

→ diminishes gradually

➔ 1 minute or less in 2nd stage

- Periods of relaxation between contractions
 - essential to welfare of fetus
 - unremitting contraction of uterus
 - ➔ compromises utero-placental blood flow ➔ fetal hypoxia
- Duration of contraction : in active phase
 - ✓ Duration 30-90 seconds (average 60 seconds)
 - ✓ Amniotic fluid pressure generated by spontaneous uterine contractions averages 40 mmHg but can range 20-60 mmHg

★ *Formation of distinct lower & upper*

2 distinct parts (anatomically & phys

1) Upper segment

- ① actively contracting
- ② becomes thicker and shorter
- ③ quite firm or hard

2) Lower segment

- ① relatively passive
- ② develops into a much thinly walled passage for the fetus
- ③ much less firm



1)Upper segment

- Contract, retract, expel fetus
- Myometrium of upper segment not relax to original length after contraction → fixed at shorter
 - Myometrial tension remains constant
- Upper part of Ut cavity smaller with each successive contraction
- Successive shortening of muscular fibers with contractions
- → **Upper Active Ut segment : progressively thickened throughout 1st & 2nd stage of labor**

2) Lower segment

- ✓ Fibers become stretched with each contraction of upper segment
- ✓ Lower segment not returned to previous
- ✓ length but remain fixed at longer length
- ✓ Tension remain same as before
- ✓ Successive lengthening of fibers in lower segment, as labor progress
- ✓ thinning normally to only a few mm in thinnest part

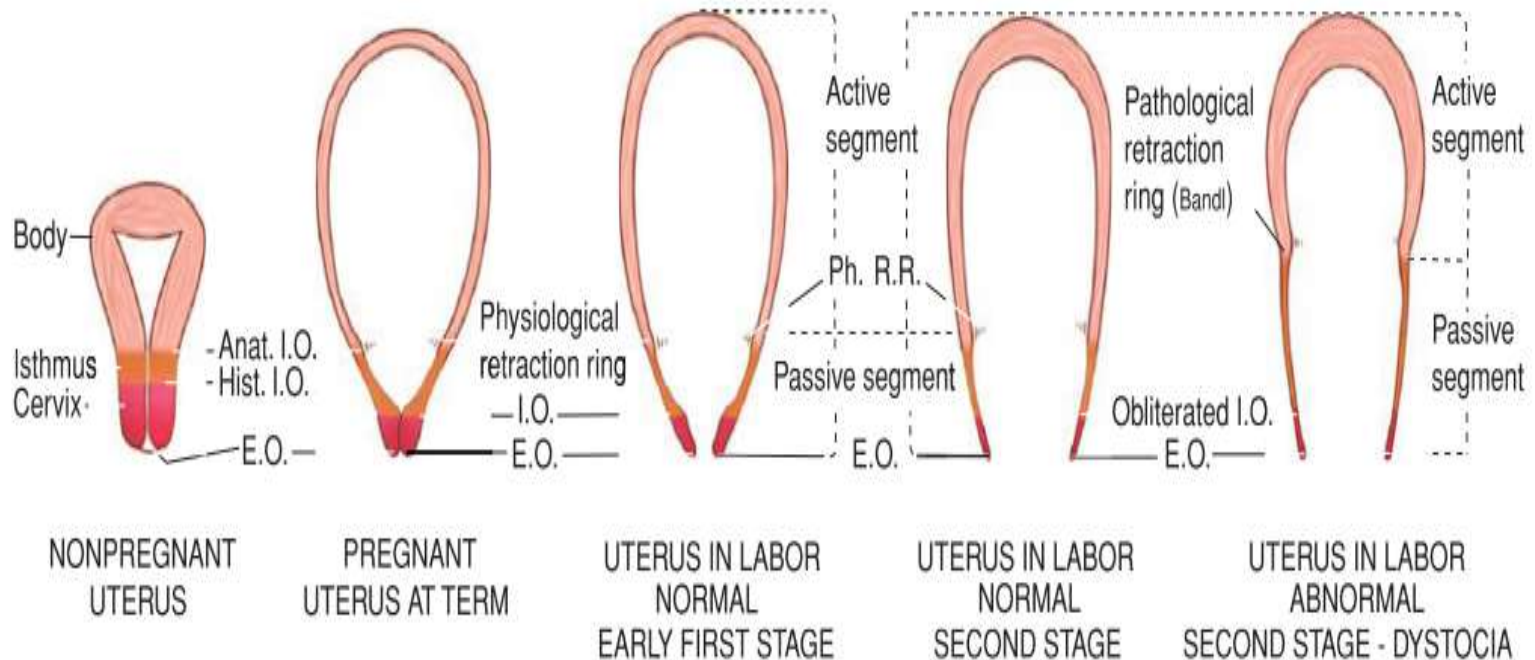
- ***Physiologic retraction ring***

- As result of thinning of lower uterine segment and concomitant thickening of the upper, boundary between the two is marked by ridge on inner uterine surface

- ***Pathologic retraction ring (Bandl ring)***

- When thinning of lower uterine segment is extreme, as in obstructed labor, ring is very prominent

Sequence of development of segment & ring in ut in preg women at term & in labor



★ *Change in Uterine Shape*

Each contraction produces elongation of uterus with decrease in horizontal diameter

→ important effect on labor process

① decrease in horizontal diameter

→ straightening of fetal vertebral column

➔ press upper pole of fetus firmly against fundus while the lower pole drawn downwards

② lengthening of uterus

→ longitudinal fibers are drawn taut → lower segment &

cervix are pulled upward over lower pole of fetus

→ important factor in cervical dilatation

★ **Ancillary Forces in Labor**

: After Cx is dilated fully, most important force in expulsion of fetus → increased maternal intra-abdominal pressure

"Pushing"

- ❖ increased intra-abdominal pressure by contraction of abdominal m, simultaneously with forced respiratory efforts with glottis closed
- ❖ important force in expulsion of fetus
- ❖ similar to defecation

1. Changes Induced in the Cervix with Labor

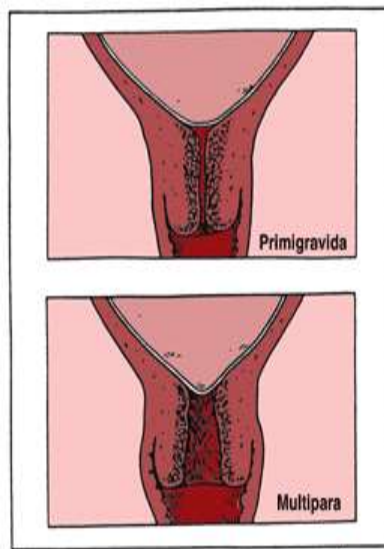
- Effective force of 1st stage of labor is uterine contraction
- As result of action of these forces, two fundamental changes take place in the already ripened cervix
"effacement & dilatation"
- Cx completely (fully) dilated : 10 cm

2. Cervical Effacement

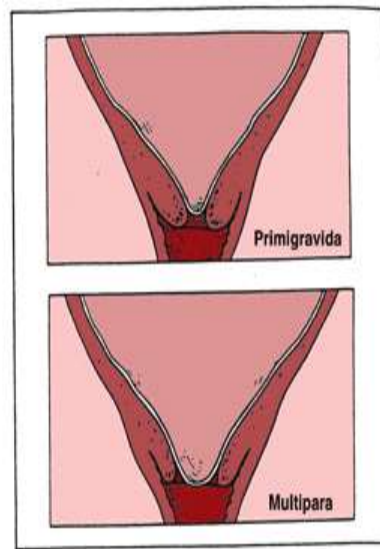
- obliteration or taking up of cervix
- shortening of the cervical canal (2cm → mere circular orifice with almost paper thin edge)
- muscular fibers at about level of internal os are pulled upward or “taken up” lower uterine segment
- external os remains temporarily unchanged

3. Cervical Dilatation

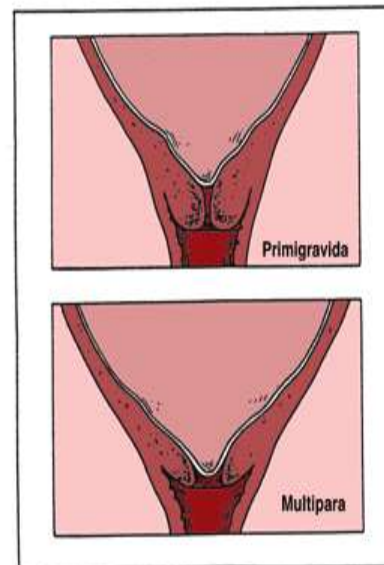
- During contraction centrifugal pull is exerted on Cx leading to distention, process of Cx Dilatation
- As uterine contraction cause pressure on the membranes → hydrostatic action of amnionic sac (pressure of presenting part) in turn dilates the cervical canal



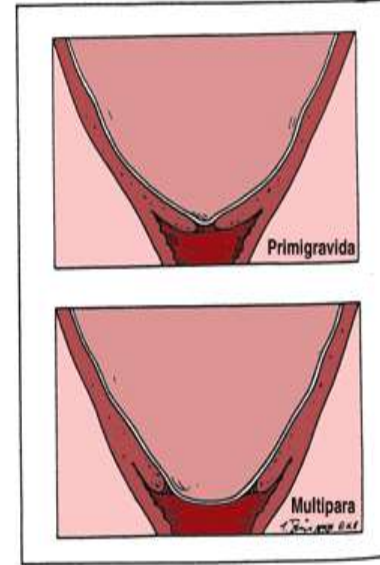
Cervix near the end of pregnancy, but before labor.



Further effacement of cervix

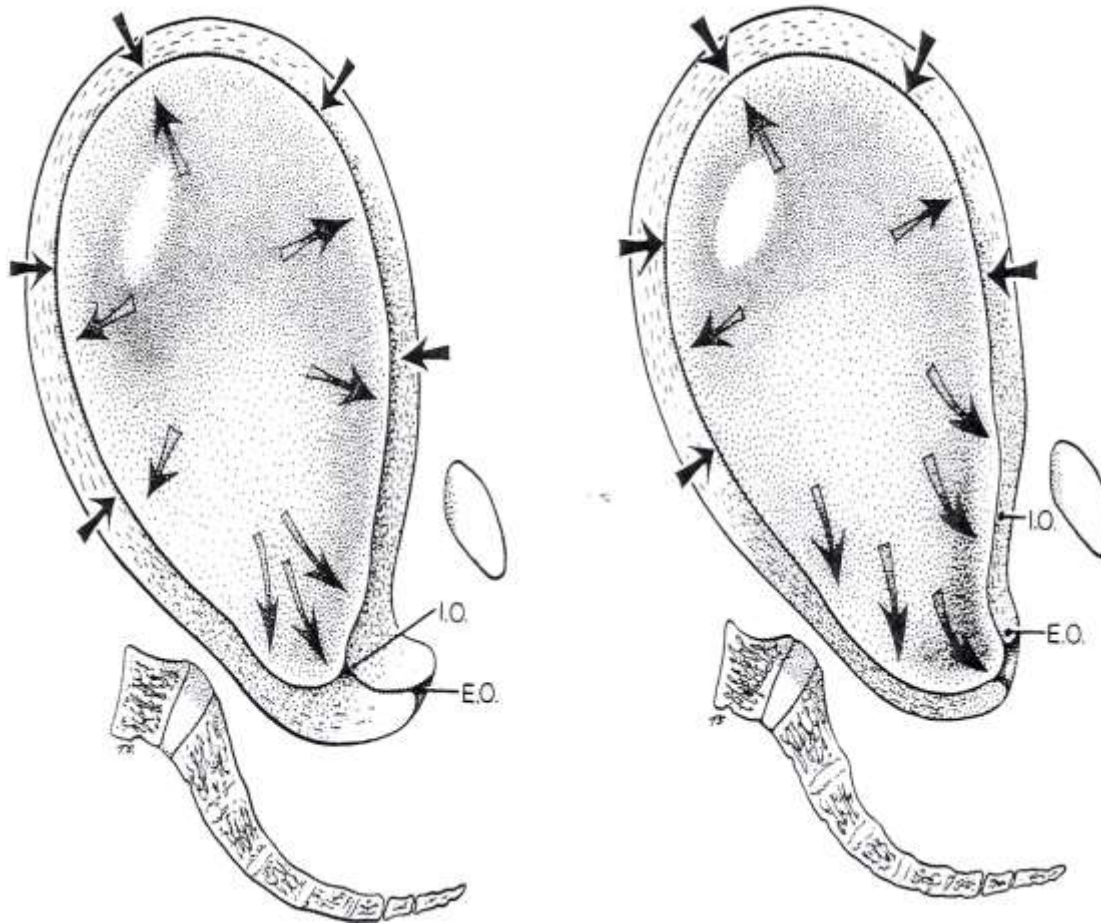


Beginning effacement of cervix. Note dilatation of internal os and funnel-shaped cervical canal.



Cervical canal obliterated, i.e., the cervix is completely effaced.

Cervical change induced during 1st stage of labor



Cervical change induced during 1st stage of labor

2 phases of cervical dilatation

1) Latent phase

: more variable

: subject to sensitive changes by extraneous factors & by sedation (prolongation) & myometrial stimulation (shortening)

2) Active phase

① Acceleration phase

usually predictive of outcome

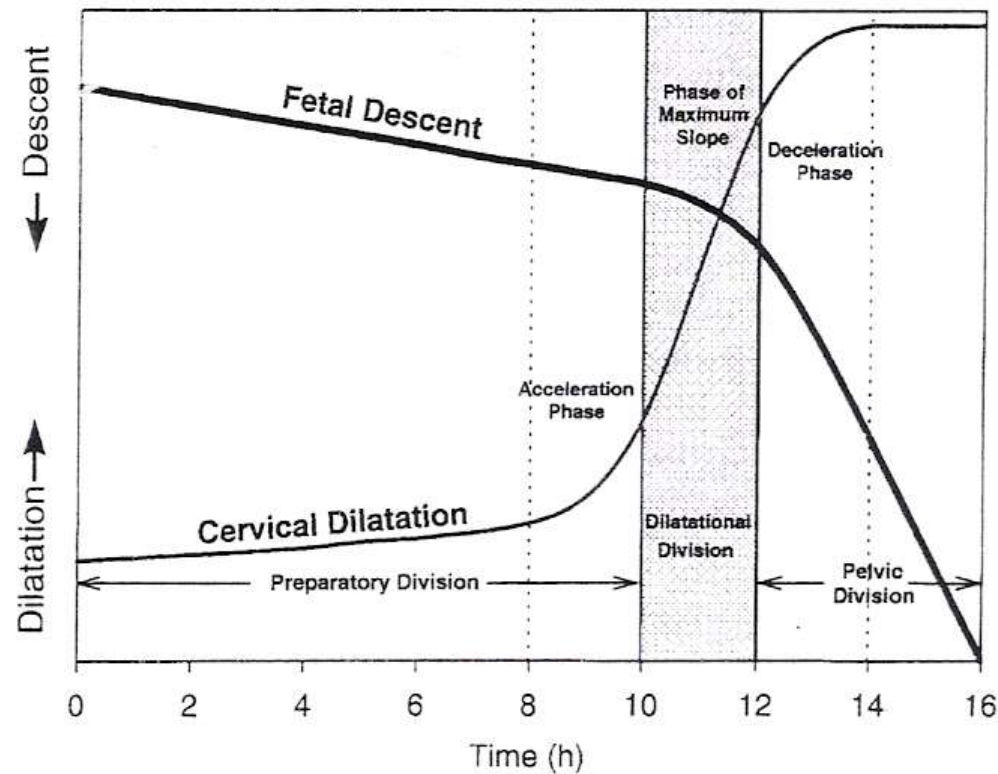
② Phase of maximum slope

③ Deceleration phase

2nd stage of labor : fetal descent

- In many nulliparas
 - ① engagement accomplished before labor begins
 - ② further descent not occur until late in labor
 - ③ increased rates of descent are ordinarily observed during the phase of maximum slope

2nd stage of labor : fetal descent



2nd stage of labor : fetal descent

: Labor course divided functionally on basis of expected evolution of dilatation & descent curves into 3 divisions

- ① Preparatory division**
 - latent & acceleration phases**
- ② Dilatational division**
 - phase of maximum slope of cervical dilatation**
 - most rapid rate of dilatation occur**
- ③ Pelvic division**
 - deceleration phase & second stage while concurrent with phase of maximum slope of fetal descent**

Change in pelvic floor during labor

- Most important structure : levator ani & fascia
- During pregnancy: hypertrophy, forming thick band that extend backward from pubis & encircles vagina about 2cm above plane of hymen
- On contraction, levator ani draw both Rectum & vagina forward & upward in direction of symphysis pubis → acts to close vagina

3rd stage of labor : delivery of PL & membrane

★ Placental Separation

- begins immediately after delivery of fetus, involve separation & expulsion of placenta
- Diminution in Ut size → PL implantation site area ↓ → PL accommodate to reduced area → thickness because of limited PL elasticity → forced to buckle
- Resulting tension → weakest layer of decidua (D. spongiosa) cleavage take place at that site
- As separation proceed → hematoma forms between separating PL & remaining Decidua ← result of separation

Diminution in size of Placental site after birth of infant

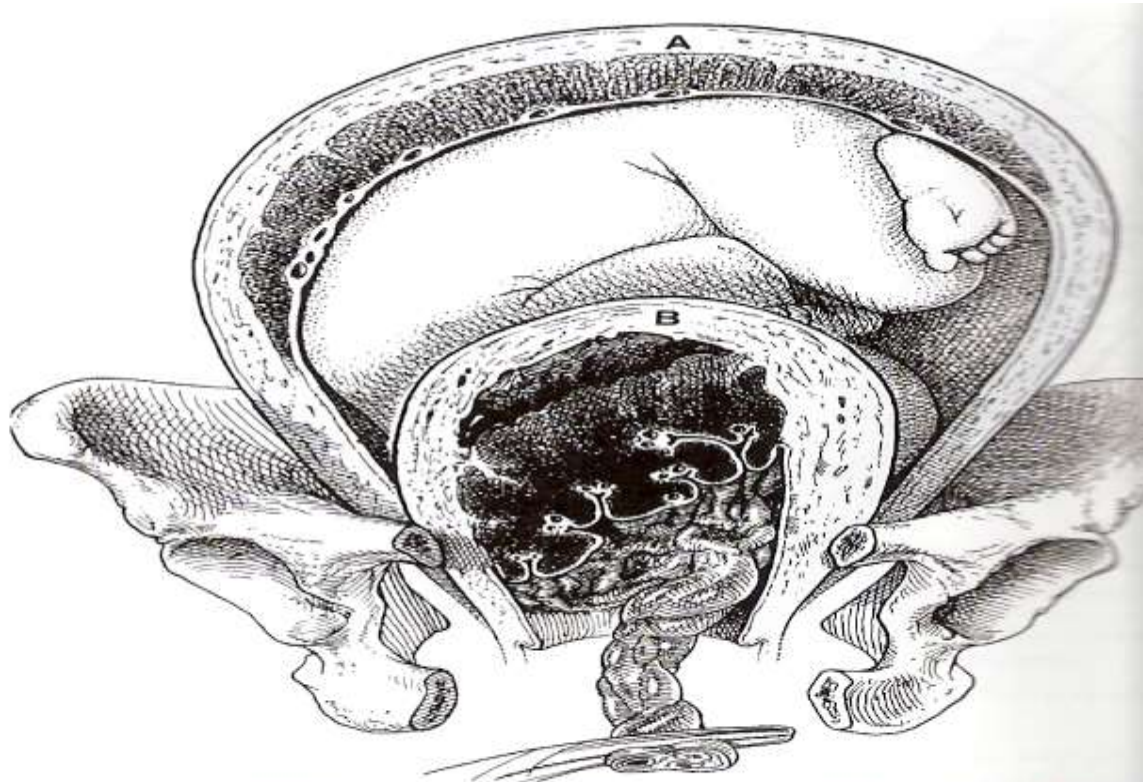


FIGURE 6-13. Diminution in size of the placental site after of the infant. **A.** Spatial relations before birth. **B.** Placental sp relations after birth.

3rd stage of Labor : Delivery of Placenta & membrane

★ Separation of Amniochorion

:great decrease in surface area of uterine cavity

→ fetal membranes (amniochorion) & parietal decidua to thrown into innumerable folds

→ increase thickness of layer 1mm → 3~4 mm

: membranes usually remain in situ until placental separation is nearly completed

3rd stage of Labor : Delivery of Placenta & membrane

★ Placental Extrusion

- some case abdominal pr↑ PL be expelled
- women in recumbent position frequently cannot expel placenta spontaneously
 - artificial means generally required
 - compress & elevate fundus while exerting minimal traction on umbilical cord

3rd stage of Labor : Delivery of Placenta & membrane

★ Mechanisms of Placental Extrusion

(1) Schultze mechanism

- PL separation occurs 1st at central areas
 - retroplacental hematoma
 - push placenta toward uterine cavity

(2) Duncan mechanism

- ① placental separation occurs first at periphery
- ② blood collects between membranes & uterine wall → escapes from vagina
 - ➔ Maternal surface first to appear at vulva

Phase 3 of parturition : process of labor

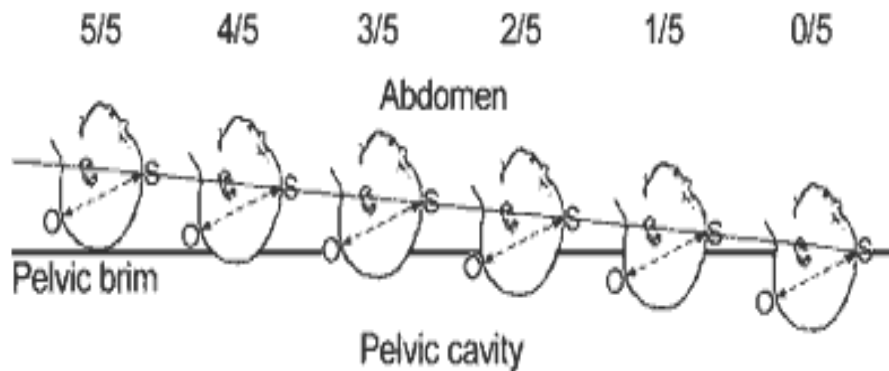
- Immediately after delivery & for 1hr or so thereafter, myometrium in state of rigid & persistent contraction & retraction
 - ➔ effect compression of large Ut vessels
 - ➔ Severe PPH prevented
- Involution of Ut & reinstitution of ovulation
- Complete Ut involution : 4~6 wks
- Infertility persist as long as breast feeding is continued (lactation ➔ anovulation & amenorrhea)

Mechanisms of labor

- Also called as **cardinal movements**
- Refers to the changes in position of fetal head during its passage through the birth canal
- Because of asymmetry in fetal head and maternal pelvis, such rotations are needed for negotiation
- Seven cardinal movements:
 - ✓ Engagement
 - ✓ Descent
 - ✓ Flexion
 - ✓ Internal rotation
 - ✓ Extension
 - ✓ External rotation
 - ✓ Expulsion
- Every Decent Family in Europe Eats Egg

Engagement

- Head normally enters pelvis in the transverse or oblique diameter of the inlet
- Engagement occurred when the widest part of the presenting part has passed successfully through the inlet.
- More than two-fifth palpable abdominally, the head



5/5	4/5	3/5	2/5	1/5	0/5
Completely above	Sinciput high, Occiput easily felt	Sinciput easily felt, Occiput felt	Sinciput felt, Occiput just felt	Sinciput felt, Occiput not felt	None of head palpable



A. Head is mobile above the symphysis pubis = 5/5



B. Head accommodates full width of five fingers above the symphysis pubis

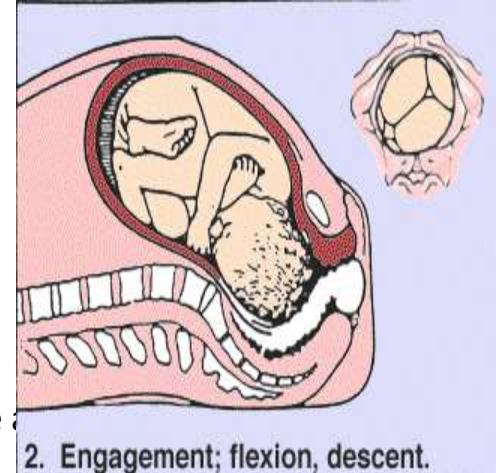


C. Head is 2/5 above symphysis pubis



D. Head accommodates two fingers above the symphysis pubis

Descent

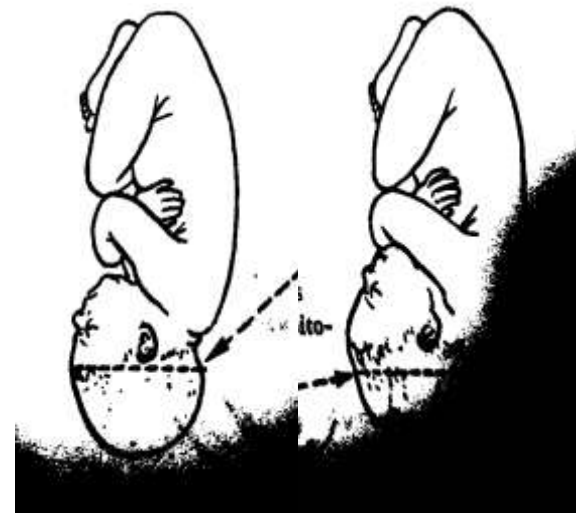
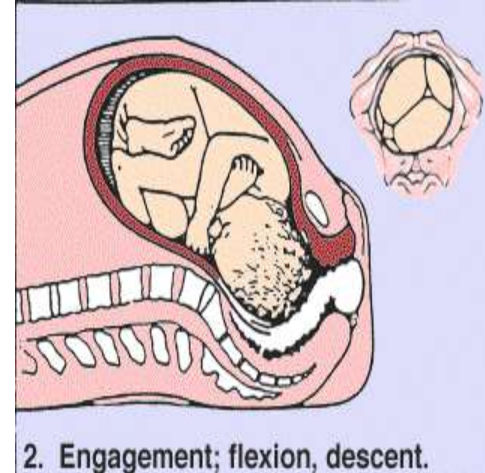


It is continuous throughout labour particularly during the second stage of labour

- ❖ Uterine contractions and retractions
- ❖ Pressure of amniotic fluid
- ❖ The auxiliary forces which is bearing down brought by contraction of the diaphragm and abdominal muscles
- ❖ The unfolding of the foetus i.e. straightening of its body due to contractions of the circular muscles of the uterus

Flexion

- ▶ At the beginning of labour, head of fetus is possible for some degree of flexion.
- ▶ Presenting diameter (11.5cm)
 - a. As the **atlanto-occipital joint** is nearer to the occiput than the sinciput, increased flexion of the head occurs when it meets the pelvic floor according to the lever theory.
 - b. Increased flexion results in:
 - > The suboccipito-bregmatic diameter (9.5 cm) passes through the birth canal instead of the suboccipito-frontal diameter (10 cm).
 - > The part of the foetal head applied to the maternal passages is like a ball with equal longitudinal and transverse diameters as the suboccipito-bregmatic = biparietal = 9.5 cm. The circumference of this ball is 30 cm.
 - > The occiput will meet the pelvic floor.

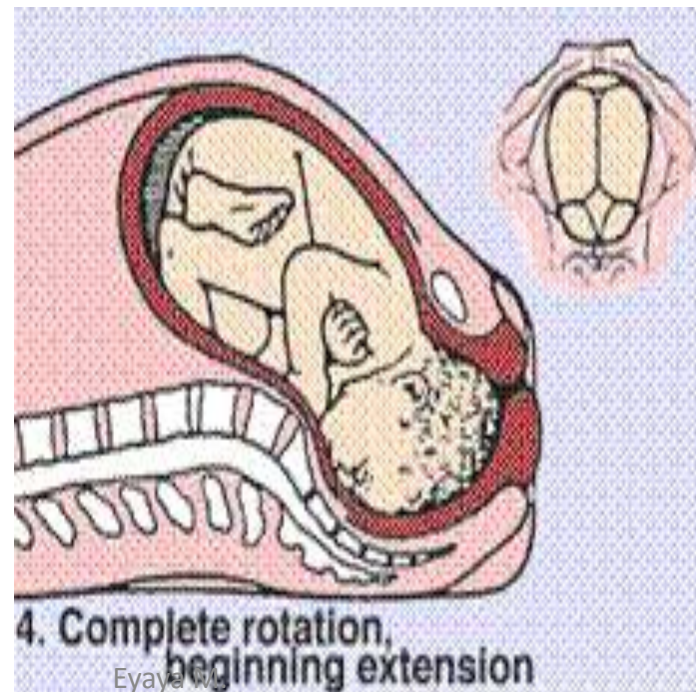
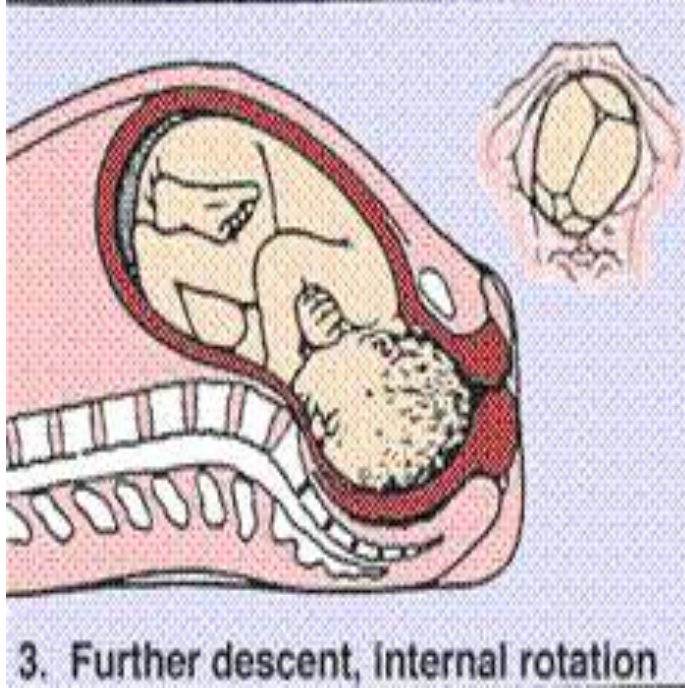


Internal rotation

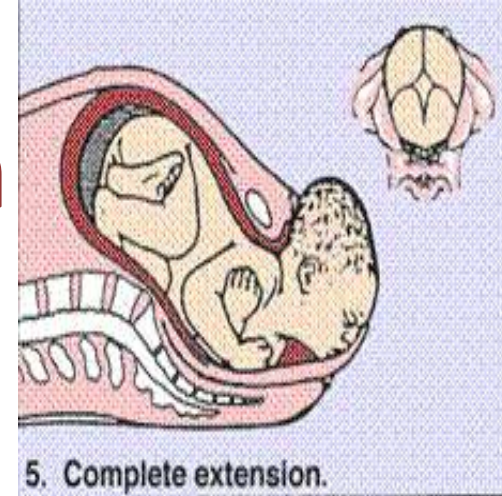
- ▶ Important factor: *Resistance of pelvic floor*
Occiput if head is well flexed → occiput will be leading point → encouraged to rotate anteriorly → sagittal suture now lies in AP diameter
- ▶ Rotates from LOT(Left occipitotransverse (90^0) /LOA-Left occipitoanterior (45^0) position to lie under the subpubic arch.
- ▶ Head now in occipito-anterior (OA) position
- ▶ Shoulders is in left oblique of the brim
- ▶ The internal rotation cause a slight twist in the neck of the fetus (the head is no longer in direct alignment with the shoulder).

The rule is that the part of foetus meets the pelvic floor first will rotate anteriorly. So that its movement is in the direction of levator ani muscles (the main muscle of the pelvic floor) i.e. downwards, forwards and inwards.

In normal labour, the occiput which meets the pelvic floor first rotates anteriorly $1/8$ circle



Extension



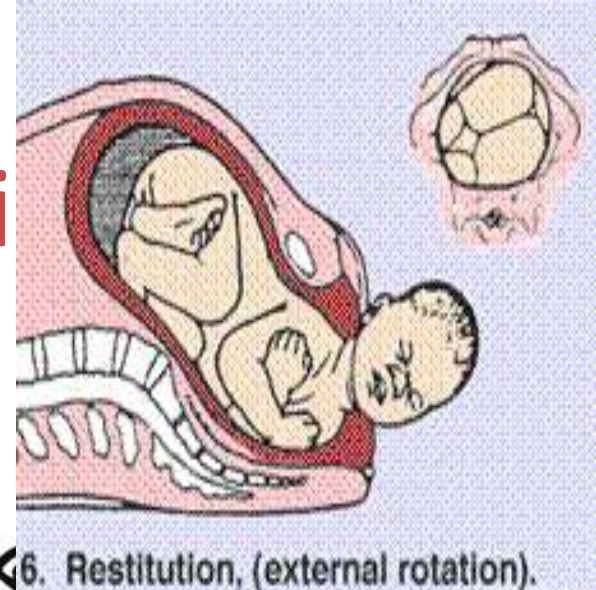
- Occiput is below symphysis pubis.

The head is acted upon by 2 forces:

- > the uterine contractions acting downwards and forwards.
- > the pelvic floor resistance acting upwards and forwards so the net result is forward direction i.e. extension of the head
- The well flexed head now extends and the occiput escapes from underneath the symphysis pubis and distends the vulva.
- **Crowning**
 - That stage of childbirth when the fetal head has negotiated the pelvic outlet and the largest diameter of the head is encircled by the vulvar ring•
- Occiput is delivered followed by bregma, brow and face.

Restituti

After delivery, the head rotates
1/8 of a circle in the opposite
direction of internal rotation
to undo the twist produced by
it

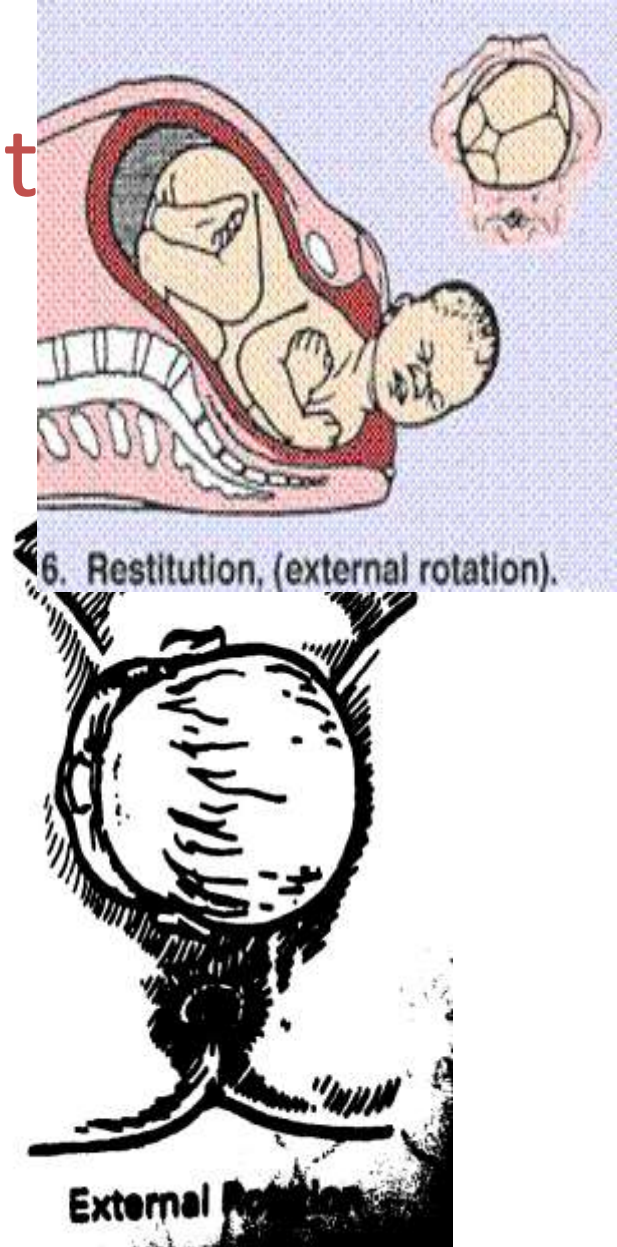


External rotation

The shoulders enter the pelvis in the opposite oblique diameter to that previously passed by the head. When the anterior shoulder meets the pelvic floor it rotates anteriorly $1/8$ of a circle. This movement is transmitted to the head so it rotates $1/8$ of circle in the same direction c restitution

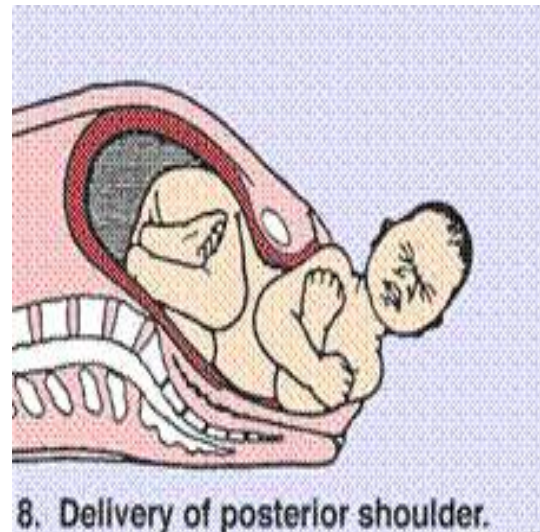
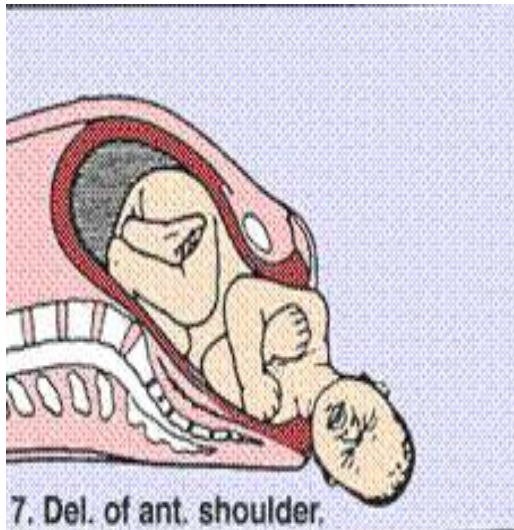
In order to be delivered, the shoulders have to rotate into the direct AP plane(the widest diameter)

- ▶ External rotation cause rotation of the head 45° towards mother left thigh in the same direction as



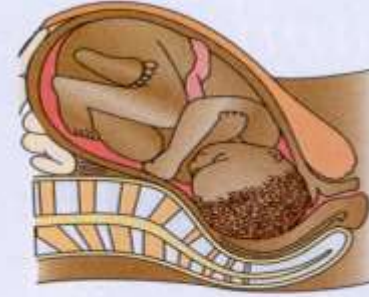
Lateral flexion (Expulsion)

- Shoulders will be in the anterior-posterior position
- Anterior shoulder is under symphysis pubis, delivers first and subsequently posterior shoulder.
- Aided by lateral movement:
The rest of the body is born by lateral flexion with arms folded on the chest and hands under the chin.



- Engagement, descent, flexion
- Internal rotation
- Complete rotation with beginning of extension
- Complete

Engagement,
Descent,
Flexion



Internal Rotation



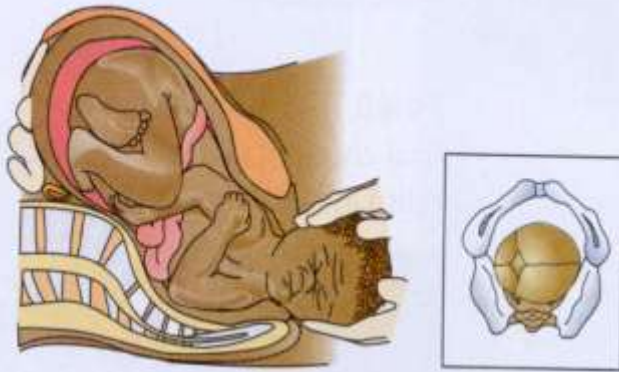
Extension Beginning (rotation complete)



Extension Complete



External Rotation (Restitution)

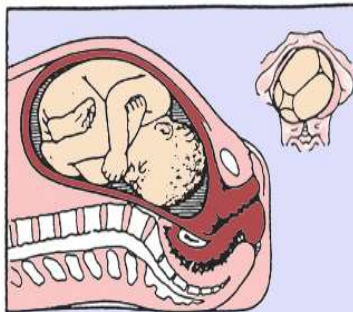


External Rotation (Shoulder rotation)

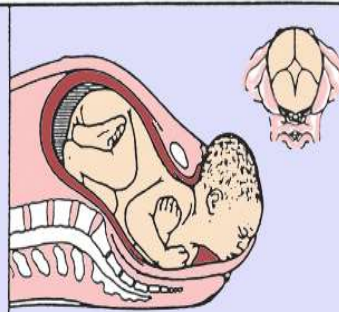


Expulsion

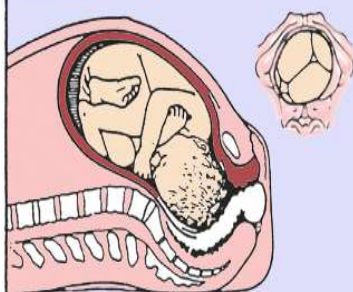
- **External Rotation (Restitution)**
- **External rotation with delivery of Anterior shoulder**
- **Expulsion with delivery of Posterior**



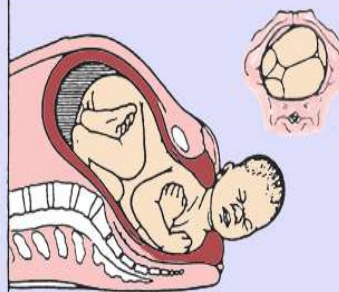
1. Head floating, before engagement



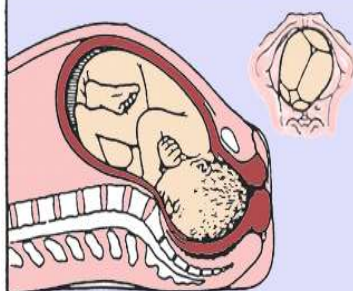
5. Complete extension.



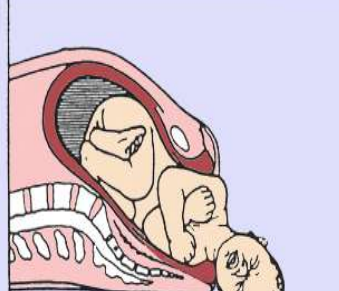
2. Engagement; flexion, descent.



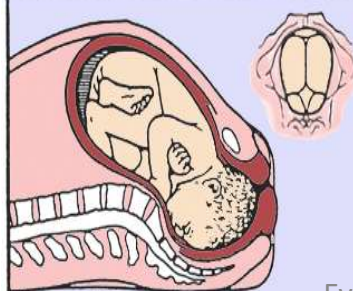
6. Restitution, (external rotation).



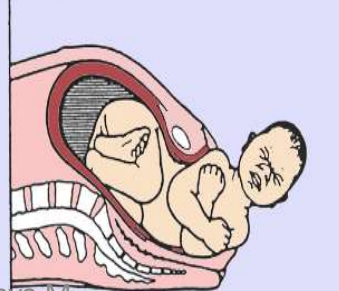
3. Further descent, internal rotation



7. Del. of ant. shoulder.



4. Complete rotation,
beginning extension



8. Delivery of posterior shoulder.

Eyaya VI.



FACTORS THAT INFLUENCE PROGRESS OF LABOUR



Power

3 "P"



Passenger



Passage

POWER ► Contractions + Maternal

Uterine contractions: pushing

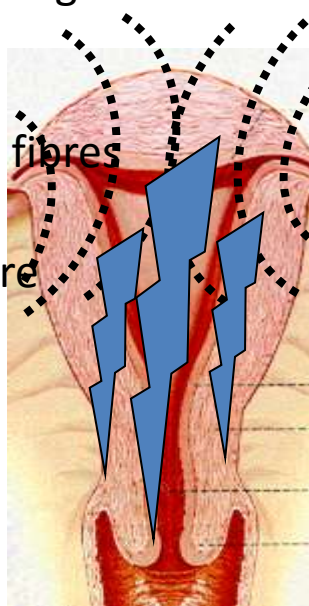
1. Initiate by pacemakers ~ uterotubal junction
2. Contraction waves meet at the fundus
3. Contraction waves progress downward

Additional force

“maternal pushing”

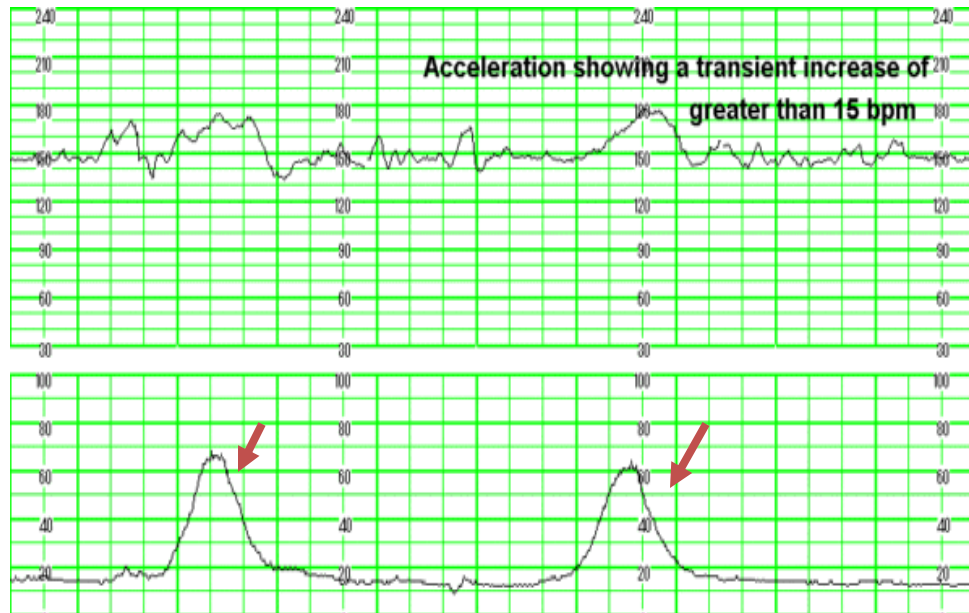
- ☐ Shortening of muscle fibres
- ☐ Retractions
- ☐ ↑ intra uterine pressure

↑ Intra abdominal pressure



EXPULSION OF THE FETUS

UTERINE CONTRACTION



Uterine contractions

ADEQUATE CONTRACTION

1. Frequency ~ one in every 2 – 3 min with at least 1 minute interval
2. Intensity ~ strong (> 50 mmHg)
3. Duration ~ 45 – 60 sec

There are 3 phases of uterine contractions:

Increment/Crescendo –intensity of the contraction increases

Apex/Acme –the height or peak of the contraction

Decrement/Decrescendo –intensity of the contraction decreases

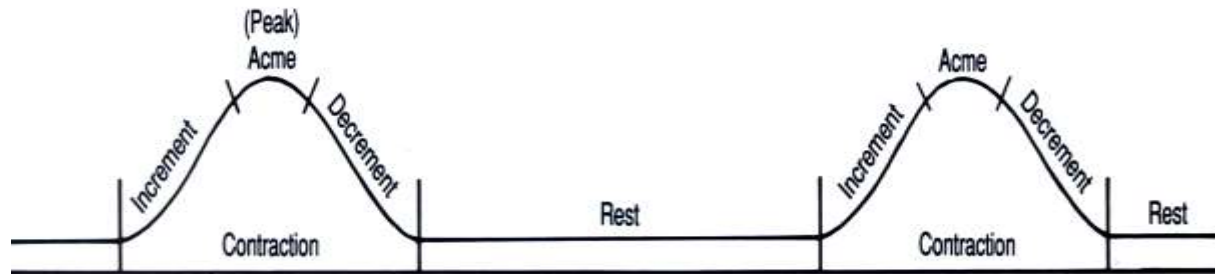


Figure 1.4. The segments of a contraction.

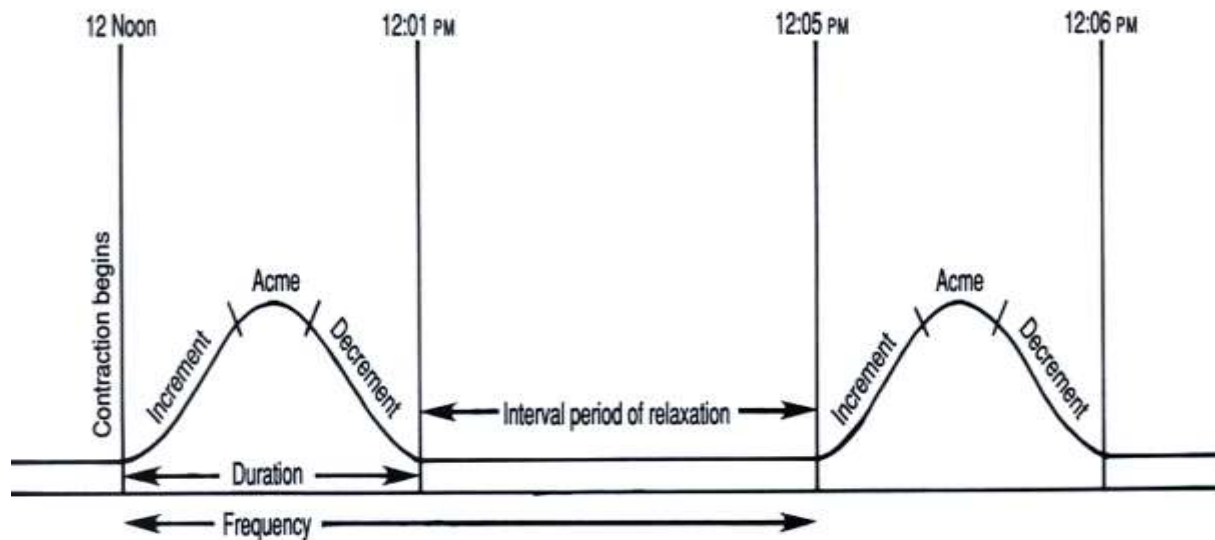


Figure 1.5. Frequency and duration of contractions.

Methods of uterine activity assessment

- Simple observation,
- Manual palpation,
- External tocodynamometry:
 - Contractions Abdominal shape change Graphic uterine activity
 - Correlates FHR with uterine activity BUT NOT contraction intensity or basal in uterine tone.
- Internal tocodynamometry via internal uterine pressure catheter
 - Most precise method
 - Performed with indication
 - Risks: uterine perforation, placental disruption, intrauterine infection (HIV)

Contraction measurement

- *Montevideo unit: most common objective measure*
 - Measures average frequency and amplitude above basal tone
 - Average strength of contractions in mmHg multiplied by number of contractions per 10 minutes
 - Adequate labor in the active phase of labor: 200 to 250 MU
- Abnormal uterine activity
 - Tachysystole: more than 5 contractions in 10 minutes for at least 20 minutes
 - Hyperstimulation: tachysystole accompanied by abnormal FHR

The Fetus (Passenger):

Fetal variables influence course of labor & delivery

- Fetal *size*: abdominal palpation or ultrasound
 - Macrosomia: actual birth weight greater than 4,000 g
 - Increased likelihood of failed trial of labor
- *Lie* : longitudinal axis of the fetus relative to the longitudinal axis of the uterus
 - Longitudinal, transverse, or oblique
- *Malpresentation*: any presentation other than vertex
 - 5% of all term labors

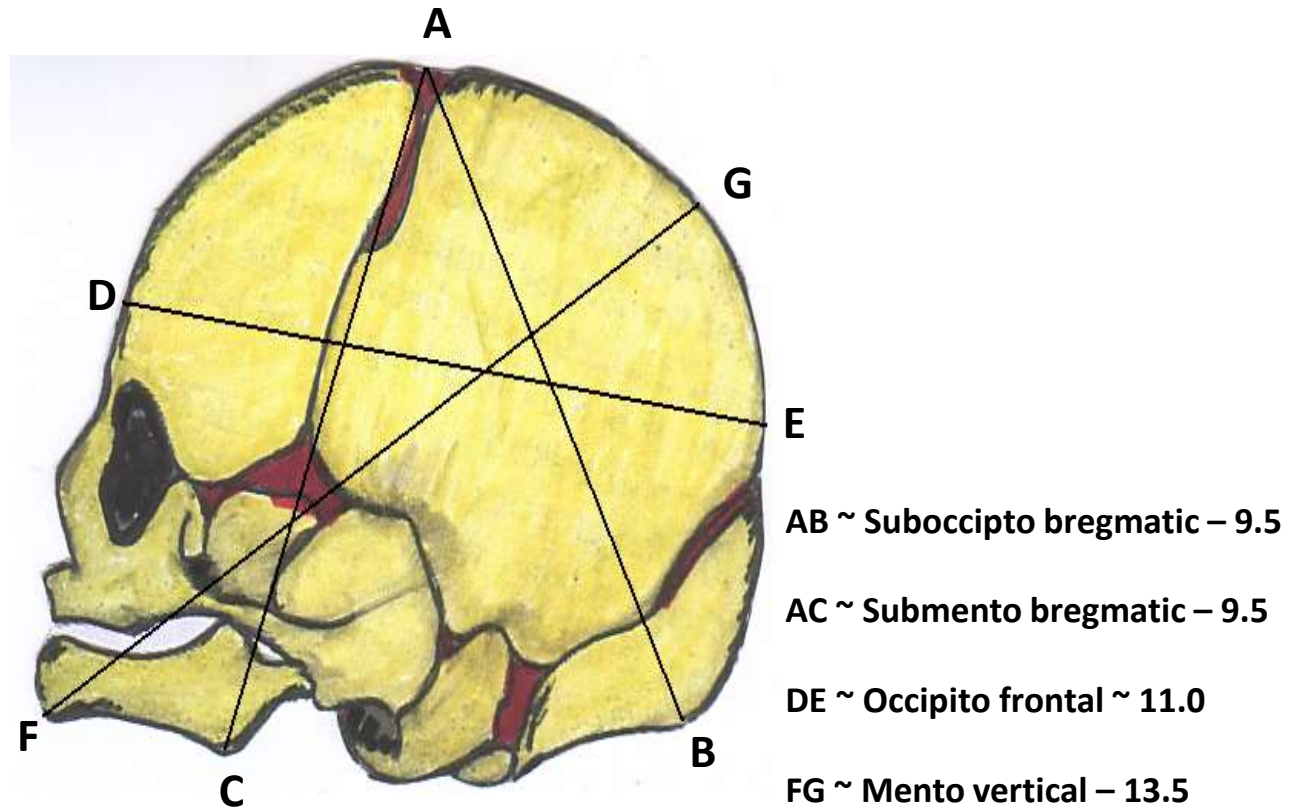
The Fetus (Passenger):

Fetal variables influence course of labor & delivery

- *Attitude*: position of head with fetal spine
 - Flexion facilitates *engagement*
 - Chin optimally flexed onto the chest: suboccipitobregmatic diameter (9.5 cm)
 - Deflexed (extended) head: brow and face
- *Position*: relationship of the fetal presenting part to the maternal pelvis
 - *Malposition* refers to any position in labor that is not ROA, OA, or LOA
- *Station*: measure of descent of the bony presenting part of the fetus through the birth canal
 - Classification (-5 to +5) based on a quantitative measure in centimeters of the distance of

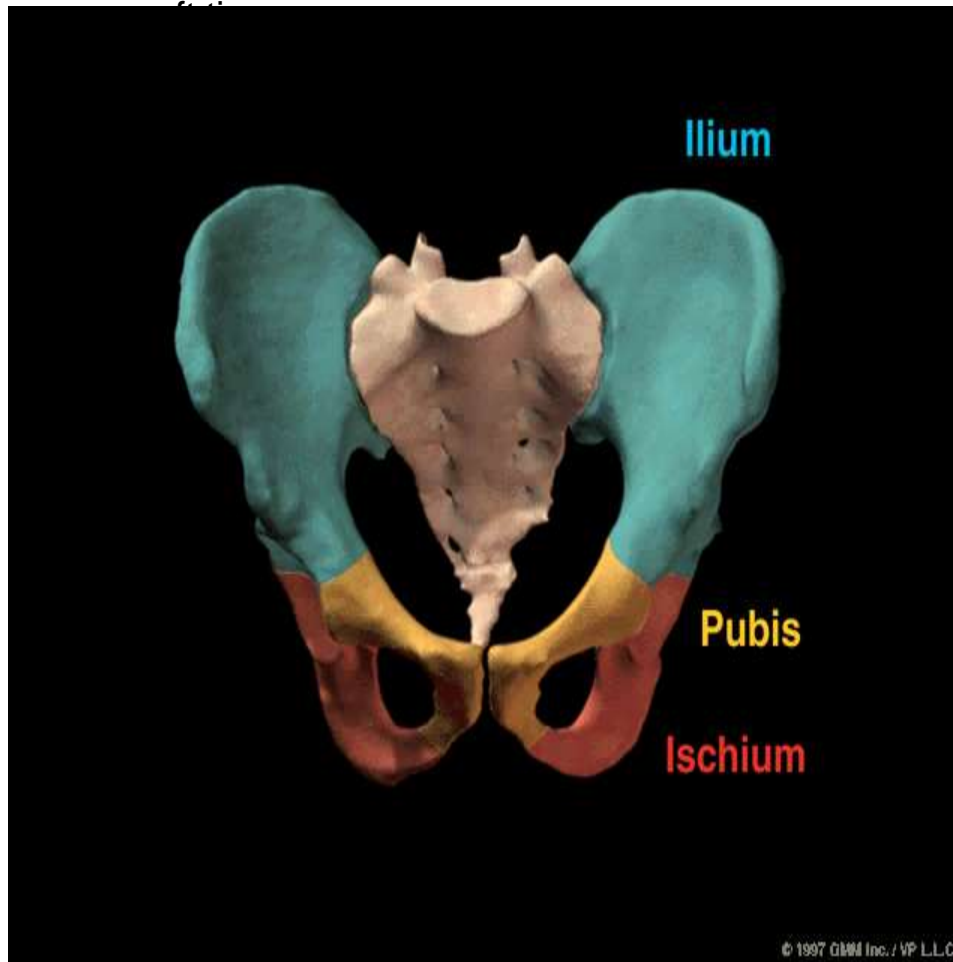
THE FETAL SKULL

Diameters of the fetal skull – anterior posterior diameters



The Maternal Pelvis (Passage)

- Consists:
 - bony pelvis (composed of the sacrum, ilium, ischium, and pubis)

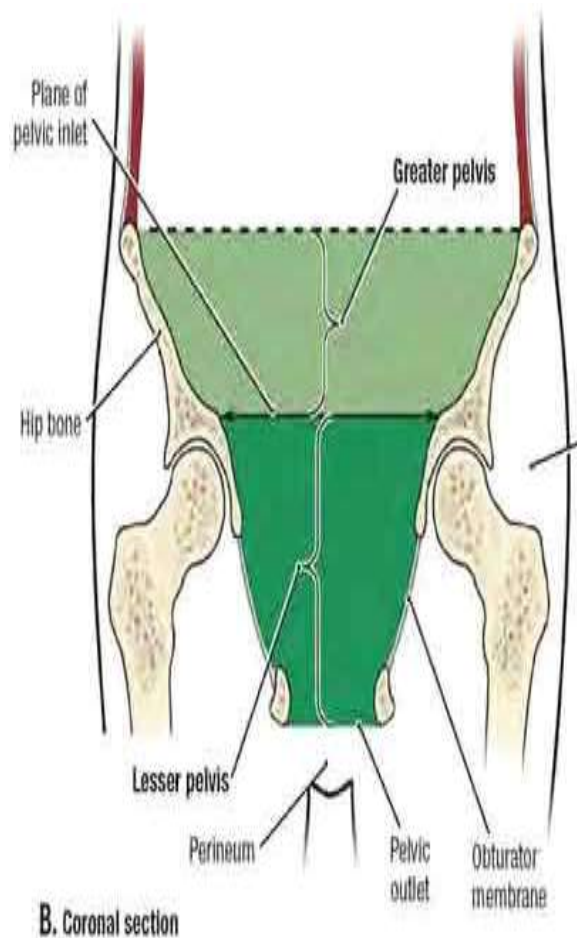


The female pelvis provides the basic framework of the birth canal.

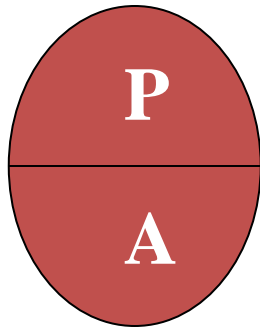
The obstetric pelvis is divided into *false(greater)* and *true(lesser)* pelvis by the *pelvic brim* or inlet

The true pelvis is important, for it is through this confined space that the fetus must pass on its journey through the birth canal.

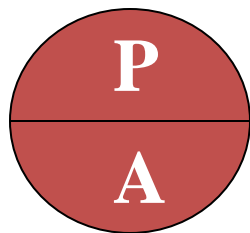
The true pelvis is composed of *inlet*, *cavity* and *outlet*



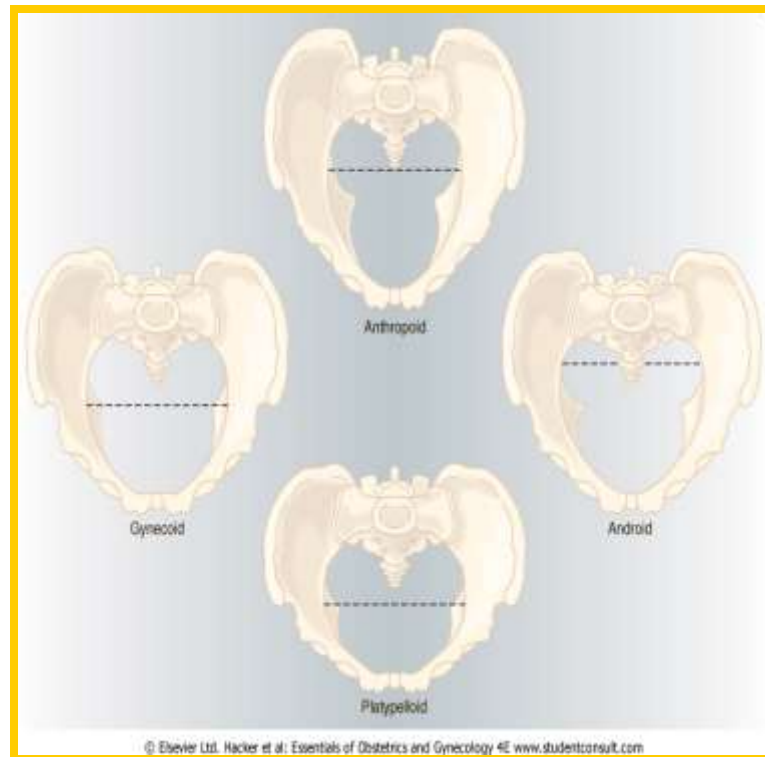
Caldwell-Moloy Classification



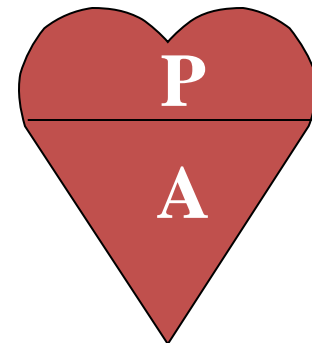
Gyneco
id



Platypello
id



**Anthrop
oid**



**Androi
d**



1. The brim is slightly oval

The ideal normal female gynaecoid pelvis:

2. The sacral promontory is not prominent.
3. The transverse diameter is slightly longer than the anteroposterior.
4. The sidewalls are parallel and straight.
5. The ischial spines are not prominent.
6. The sacrosciatic notches are wide.
7. The sacrum has a good curve.
8. The pubic arch angle are wide, i.e. more than 90°
9. Inter-tuberos diameter is wide

Favorable pelvic shape for vaginal delivery:

Favorable: gynecoid, anthropoid

Less favorable: android, platypelloid

Many pelvis of women fall into intermediate categories

TABLE 12-1**AVERAGE AND CRITICAL LIMIT VALUES
FOR PELVIC MEASUREMENTS BY
X-RAY PELVIMETRY**

DIAMETER	AVERAGE VALUE	CRITICAL LIMIT*
Pelvic Inlet		
Anteroposterior (cm)	12.5	10.0
Transverse (cm)	13.0	12.0
Sum (cm)	25.5	22.0
Area (cm ²)	145.0	123.0
Pelvic Midcavity		
Anteroposterior (cm)	11.5	10.0
Transverse (cm)	10.5	9.5
Sum (cm)	22.0	19.5
Area (cm ²)	125.0	106.0

Modified from O'Brien WF, Cefalo RC. Labor and delivery. In: Gabbe SG, Niebyl JR, Simpson JL, eds. *Obstetrics: Normal and Problem Pregnancies*, ed 3. New York: Churchill Livingstone; 1996;377.

*The critical limit values cited imply a high likelihood of cephalopelvic disproportion.

Management of normal labor and delivery



AIMS IN THE MANAGEMENT OF LABOUR

The AIMS include:

- **To achieve delivery of a normal healthy child**
- **To anticipate, recognize and treat potential abnormal conditions before significant hazard develops for the mother or the fetus.**

PRINCIPLES IN THE MANAGEMENT OF LABOUR



The principles include:

- **Diagnosis of labour**
- **Monitoring the progress of labour**
- **Ensuring maternal well-being**
- **Ensuring fetal well-being.**



MANAGEMENT FIRST STAGE OF LABOUR



MANAGEMENT OF THE FIRST STAGE OF LABOUR

- **On admission:**

When the woman presents at hospital, the woman's antenatal record is reviewed to discover whether there have been any abnormalities during her pregnancy. When there are no records of antenatal care a complete history must be taken.

- **General examination of the mother**

- a) General conditions – evaluate the mother general health condition. Look for pallor, edema, abdominal scar (LSCS) and maternal height.
- b) Vital signs – Blood pressure, pulse, respiration and temperature are taken and recorded
- c) Heart and lungs
- d) Urine analysis – for protein, sugar and ketones



MANAGEMENT OF THE FIRST STAGE OF LABOUR²

- **Abdominal examination:**

- a) A detailed abdominal examination should be carried out and recorded.
- b) Determine the presentation and position of the fetus and also the engagement
- c) Auscultate the fetal heart
- d) Evaluate the uterine contraction

- **Vaginal examination – the purpose is to**

- a) To make a positive diagnosis of labour
- b) To make a positive identification of presentation
- c) To determine whether the fetal head is engaged in case of doubt
- d) To ascertain whether the fore waters have ruptured or to rupture them artificially
- e) To exclude cord prolapse after rupture of the fore waters
- f) To confirm the degree of cervical dilatation and position of the presenting part
- g) To assess progress of labour.
- h) To assess the adequacy of the pelvis.



- **Bowel preparation:**

If there has been no bowel action for 24 hours or the rectum feels loaded on vaginal examination an enema is given.

- **Bladder care**

- A full bladder may initially prevent the fetal head from entering the pelvic brim and later impede descent of the fetal head. It will also inhibit effective uterine action.
- The woman should be encouraged to empty her bladder every 1½ - 2 hours during labour.
- The quantity of urine passed should be measured and recorded and a specimen obtained for testing.

- **Nutrition in early labour**

When labour is established no oral feeding is allowed, but sips of water.

> 15 ml magnesium trisilicate is given every 2 hours as an oral antacid to guard against bronchospasm occurs if the acid vomitus is inhaled during general anaesthesia "Mendelson's syndrome". Antacid injections may be used instead.

>If labour is delayed more than 8 hours, IV drip of glucose 5% or saline-glucose solution is given



MANAGEMENT OF THE FIRST STAGE OF LABOUR⁴

- **Position of labouring mother:**

As long as the patient is healthy, the presentation normal, the presenting part engaged, and the fetus in good condition, the patient may walk about or may be in bed, as she wishes

- **Monitoring the progress of labour**

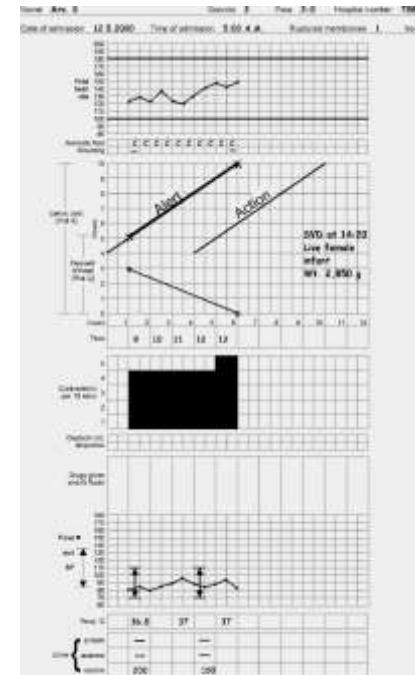
Once labour has become established, all events during labour should be recorded on a partogram.

- a) The well-being of the fetus
- b) The well-being of the mother
- c) The progress of the labour

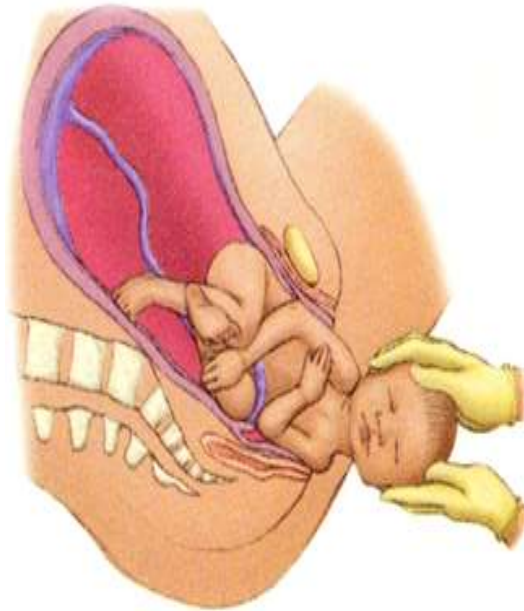
- **Pain relief**

When the pains are severe an analgesic preparation may be given.

- a) Opiate drugs – e.g. Pethidine given intramuscularly every 4 hour
- b) Inhalational analgesia – e.g. Entonox
- c) Epidural analgesia



RECORDING THE PROGRESS OF LABOUR



MANAGEMENT SECOND STAGE OF LABOUR

Second Stage of Labour

- a. The patient feels the desire to defecate.
- b. The contractions become more prolonged and painful.
- c. Reflex desire to bear down during the contractions.
- d. The expulsive effort is accompanied by sustained expiratory grunt.
- e. Rupture of membranes, although this is not specific as it may occur earlier even before start of labour " prelabour rupture of membranes" or later even to the degree that the foetus is delivered in an intact sac.
- f. Full dilatation of the cervix (10 cm) in between uterine contractions is the most imminent sign

Second Stage of Labour

Delivery room:

- a. The patient is transferred on a wheel or trolley to the delivery room.
- b. Put her in the lithotomy position.
- c. The lower abdomen, upper parts of the thighs, vulva and perineum are swabbed with antiseptic lotion.
- d. Sterile legs and towels are applied.

Bearing down

- Ask the patient to bear down during contractions and relax in between

Second Stage of Labour

Delivery of the head

- The main aim during delivery of the head is to prevent perineal lacerations through the following instructions
- Support of the perineum
- When the labia start to separate by the head, a sterile pad is placed over the perineum and press on it with the right hand during uterine contractions. This is continued until crowning occurs to maintain flexion of the head

Second Stage of Labour

Crowning

- a. is the permanent distension of the vulval ring by the foetal head like a crown on the head. The head does not recede back in between uterine contractions.
- b. This means that the biparietal diameter is just passed the vulval ring and the occipital prominence escapes under the symphysis pubis.
- c. After crowning, allow slow extension of the head so the vulva is distended by the suboccipito frontal diameter 10 cm
- d. If the head is allowed to extend before crowning the vulva will be distended by the occipito-frontal 11.5 cm increasing the incidence of perineal lacerations
- e. Ritgen manoeuvre: upward pressure on the perineum by the right hand and downward pressure on the occiput by the left hand to control the extension of the head

Episiotomy: It is done at crowning when the perineum is stretched to the degree that it is about to tear

Swab and aspirate: the mouth and nose once the head is delivered before respiration is initiated and the liquor, meconium or blood is inhaled.

Coils of the umbilical cord: if present around the neck are slipped over the head but if tight or multiple they are cut between 2 clamps

Delivery of the shoulders

- Gentle downward traction is applied to the head till the anterior shoulder slips under the symphysis pubis. The head is lifted upwards to deliver the posterior shoulder first then downwards to deliver the anterior shoulder

Delivery of the remainder of the body

- Usually slips without difficulty otherwise gentle traction is applied to complete delivery.

Clamping the cord

- This may be enhanced by milking the cord towards the baby, to add about 100 ml of blood to its circulation.
- The cord is divided between 2 clamps to avoid bleeding from a possible 2nd uniovular twin. Eyaya M.

Third Stage of Labour

Delivery of the placenta

- **Conservative method:**

- a. Put the ulnar border of the left hand just above the fundus at the level of the umbilicus to detect any bleeding inside the uterus known by rising level of the atonic uterus.
- b. Wait for signs of placental separation and descent but do not massage the uterus
- c. As soon as they are detected massage the uterus to induce its contraction, ask the patient to bear down and push the uterus downwards to deliver the placenta.
- d. Hold the placenta between the two hands and roll it to make the membranes like a rope in order not to miss a part of it.
- e. Give ergometrine 0.5 mg or oxytocin 10 units IM after delivery of the placenta to help uterine contraction and minimise blood loss. These may be given before delivery of the placenta.

Third Stage of Labour

Signs of placental separation and descent:

- a. The body of the uterus becomes smaller, harder and globular.
- b. The fundal level rises as the upper segment overrides the lower uterine segment which is now distended with the placenta.
- c. Suprapubic bulge due to presence of the placenta in the lower uterine segment.
- d. Elongation of the cord particularly on pressing on the uterine fundus and it does not recede back into the vagina on relieving the pressure.
- e. Gush of blood from the vagina.

Third Stage of Labour

The active method (Brandt-Andrews method):

- a. With delivery of the anterior shoulder, 0.5 mg ergometrine or syntometrine (0.5 mg ergometrine + 5 units oxytocin) is given IM.
- b. When the uterus contracts, put the left hand suprapubic and push the uterus upwards while gentle downward and backward traction is applied on the cord by the right hand when the placenta is delivered it is rolled as in the conservative method.
- c. Advantage: reduction of the blood loss.
- d. Disadvantages:
 - > Constriction ring may occur with retention of the placenta.
 - > Avulsion of the cord if undue pressure is applied.
 - > Inversion of the uterus if fundus is pressed while the uterus is lax.

Third Stage of Labour

- **Routine examinations**

- Examination of the placenta and membranes

- >by exploring it on a plain surface to be sure that it is complete. If there is missed part, exploration of the uterus is done under general anaesthesia.

- >Explore the genital tract:

- >For any lacerations that should be immediately repaired

Fourth Stage of Labour

- a. Observation for the patient particularly atony of the uterus and vaginal bleeding
- b. Care of The Newborn:
- c. Clearance of the air passages
 - >The newborn is placed in supine position with the head lower down. A metal, rubber or better disposable plastic catheter is used to aspirate the mucus from the pharynx and mouth
 - >Crying of the baby is usually occurs within seconds, if delayed slapping its soles, flexion and extension of the legs and rubbing the back usually stimulate breathing

Fourth Stage of Labour

d.The umbilical cord

- A disposable plastic umbilical clamp is applied about 5 cm from the umbilicus to avoid the possibility of tying an umbilical hernia then cut about 1.5 cm distal to the clamp. Inspect for bleeding and paint it with alcohol.
- If the plastic umbilical clamp is not available, 2 ligatures of silk are applied instead of it.
- The umbilical stump is painted daily with an antiseptic till its fall within 10 days

e.Congenital anomalies

- The newborn is examined for injuries or congenital anomalies as imperforate anus, hypospadias (not to be circumcised as the cut skin will be used in the repair later on), cyanotic heart diseases.... etc.

f. Weight the newborn and record it

g. Dressing

Dressing as well as all previous procedures should be done in a warm place better under radiant warmer to prevent heat loss which occurs rapidly after delivery increasing the metabolism and acidosis.

Fourth Stage of Labour

- Care of the eyes:

An antibiotic eye drops as chloramphenicol are instilled into the eyes as a prophylaxis against ophthalmia neonatorum.

- Identification:

of the baby by a plastic bracelet on which its mother's name is written.

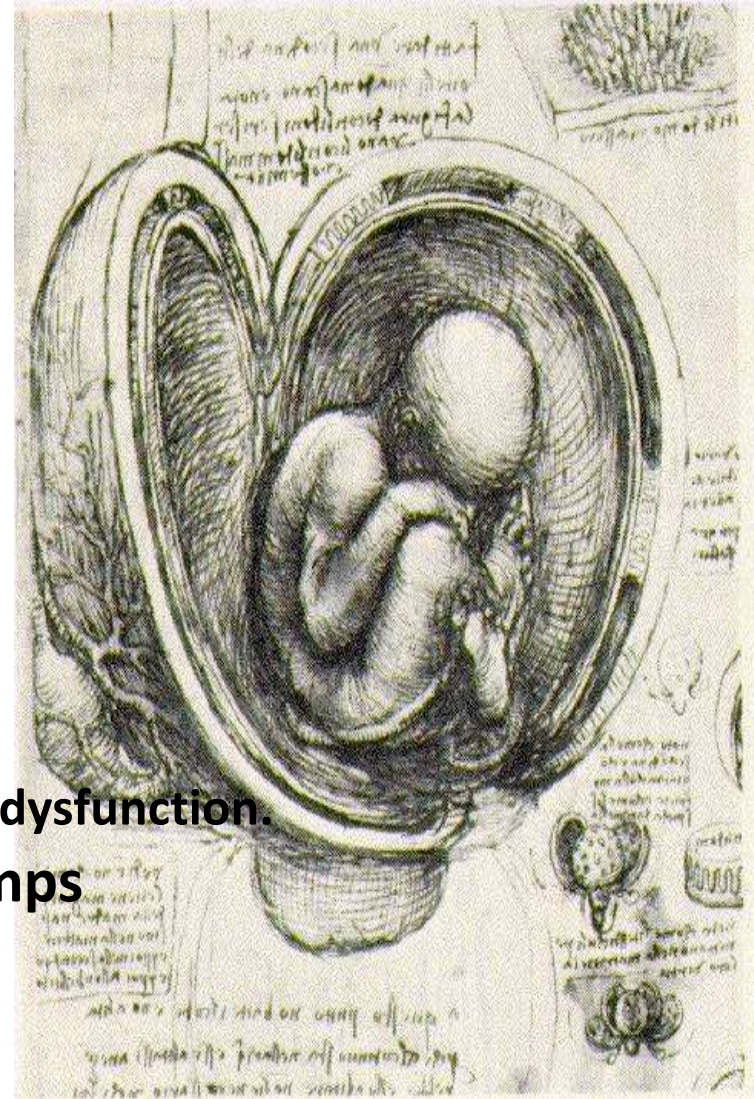
A celestial scene with a large, glowing planet in the center, partially obscured by the text. The planet has a bright, orange-yellow ring of light around its equator. A small, crescent moon is visible in the upper left quadrant of the image. The background is a dark, starry space.

**Thank
you**

Minor Disorders of Pregnancy

Topics to be covered..

1. Nausea and Vomiting
2. Gastric Reflux (Heartburn)
3. Constipation
4. Respiratory Distress
5. Fatigue and Insomnia
6. Pruritus
7. Edema and varicose veins
8. Hemorrhoids
9. Vaginal discharge
10. Skin Changes
11. Pelvic Pain, Backache and Symphysis pubis dysfunction.
12. Peripheral paraesthesia and Leg cramps



Nausea and Vomiting

- Nausea and vomiting of pregnancy (**NVP**) **is the most common medical condition in pregnancy**
- Occurs in approximately 50% of pregnancies
- Most marked at gestational weeks 2–12
- It is usually most severe in the morning (Morning Sickness) but can occur at any time and may be precipitated by cooking odors and strong sharp smells
- The pathogenesis of NVP is poorly understood and the etiology is likely to be **multifactorial**

Nausea and Vomiting

- The nausea probably results from rapidly rising serum levels of **human chorionic gonadotropin- hCG**
- During the first trimester, serum hCG levels may be as high as 100,000 mIU/mL
- Emotional tension may play a role in the severity of nausea and vomiting
- Extreme nausea and vomiting may be a sign of **multiple gestation or molar pregnancy** and SHOULD be distinguished from **idiopathic NVP**

Treatment of NV

- For uncomplicated nausea consists of light dry foods, small frequent meals, and emotional support
- Some improvement can be seen with the addition of high-dose B6 therapy and the preconceptional use of prenatal vitamins
- Alternative therapies, such as ginger supplementation, acupuncture, and acupressure, may be beneficial
- Antinauseant drugs ,Promethazine, prochlorperazine and Metoclopramide are used only as a final measure

Nausea and Vomiting

- Protracted vomiting associated with dehydration and ketonuria (**hyperemesis gravidarum HG**) is defined as persistent vomiting that leads to weight loss greater than **5%** of pre-pregnancy weight, with associated electrolyte imbalance and ketonuria

Gastric Reflux (Heartburn)

- Gastric reflux commonly occurs as a result of
 - Delayed gastric emptying
 - Decreased intestinal motility, and
 - Decreased lower esophageal sphincter tone
- Information on **lifestyle modification** includes awareness of posture, maintaining upright positions (especially after meals), sleeping in a propped up position and **dietary modifications** (e.g. small frequent meals, eat slowly, reduction of high-fat foods and caffeine)
- Antacid Preparations containing aluminum hydroxide are favored

Constipation

- **Constipation during Pregnancy is due to :**
 - Reduced motility of large intestine (progesterone effect)
 - Increased water reabsorption from large intestine (aldosterone effect)
 - Pressure on the pelvic colon by the pregnant uterus
 - Sedentary life during pregnancy
- Advice includes drinking plenty of fluids, high fiber foods and get plenty of exercise
- When fiber supplementation is not effective, stimulant laxatives have been shown to be more effective
- **No evidence currently exists for the effectiveness or safety of osmotic laxatives (e.g. lactulose) or faecal softeners in pregnancy**

Respiratory distress

- The enlarged uterus displaces the diaphragm up to ± 4 cm

This result in :

- 1.The diaphragmatic mobility is reduced and respiration becomes mainly thoracic
- 2.Widen the subcostal angle and increases the transverse diameter of the chest

- **Over breathing (deep respiration)** occurs due to the effect of excess progesterone
- **Shortness of breath** (the need to breath becomes a conscious one) and **dyspnea** are common complaint of the pregnant women which may be due to unfamiliarity with low **CO₂** tension in the alveolar capillaries

Fatigue and insomnia

- Fatigue is very common in early pregnancy and reaches a peak at the **end of the first trimester**
- Rest, lifestyle adjustment and reassurance are usually all that is required
- Fatigue also occurs in late pregnancy, **when anemia should be excluded**
- Insomnia is also very common and due to a combination of anxiety, hormonal changes and physical discomfort
- Mild physical exercise before sleep may help but drug **treatment** should be avoided

Pruritus

- Local causes are usually due to infections
- Generalized itching is common in the **third trimester** and disappears after delivery
- Treatment is with simple emollients but...

Cholestasis of pregnancy needs to be excluded by checking liver function tests (raised AST/ALT; alkaline phosphatase)

Edema and varicose veins

Edema and varicose veins in the lower limbs & vulva are due to:

- I - ↑ Venous pressure
- ii - Relaxation of the smooth muscles in the wall of the veins by progesterone
- iii - ↓ Osmotic pressure in blood
- iv - ↑ Capillary permeability (due to progesterone and aldosterone)
- v - ↑ Interstitial pressure (Na retention)

Varicose Veins

treatments

1. Avoid long periods of standing and encourage active exercise
2. Avoid constricting clothes
3. Keep the legs elevated while sitting and during sleep

4. Use of elastic stockings:

These should be removed at night and applied with leg elevated before getting out of bed in the morning (empty veins)

5. Stretch panties may be necessary for vulval varicosities

Hemorrhoids :

- They occur due to:
 - Mechanical pressure on the pelvic veins
 - Laxity of the walls of the veins by progesterone
 - Constipation

Treatment

- includes diet modification, topical soothing preparations and surgery
- However, surgery is rarely considered an appropriate intervention for the pregnant woman since hemorrhoids may resolve after delivery

Vaginal discharge

- Women usually produce more vaginal discharge during pregnancy
- If the discharge has a strong or unpleasant odor, is associated with itch or soreness or associated with dysuria, then infection needs to be excluded
- A topical **imidazole** is an effective treatment for thrush **which is common during pregnancy** but the effectiveness and safety of oral treatments for thrush in pregnancy is uncertain and these should be avoided

Skin Changes

- Spider telangiectasis & palmar erythema :
Due to increased estrogen or cutaneous vasodilatation.
- **Hyperpigmentation:**
Due to increased **estrogen or melanocyte** stimulating hormone or ACTH

Chloasma gravidarum :

(mask of pregnancy)) a butterfly pigmentation on the cheeks and nose

It usually disappears few months after labour



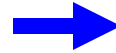
Il Linea Nigra



III Stria gravidarum

starts pink (stria rubra)
then becomes pale to become
white (stria albicans) after
delivery, which persists

(Primigravida has stria rubra only
,while multigravida has both S.R
and S.A).



- **Pelvic pain** As the uterus grows, pulling and stretching of pelvic structures causes ligament pain which usually resolves by 22 weeks
- **Backache:**
- it often first develops during the **5th to 7th** months of pregnancy
 - Encourage light exercise and simple analgesia, and consider physiotherapy referral
 - Exercising in water, massage therapy

Hyperemesis gravidarum

Definition

- Extreme form of nausea & vomiting in the 1st TM of pregnancy
- The adverse effects of severe vomiting are:
 - DHN,
 - metabolic acidosis(from starvation),or
 - alkalosis(from loss of HCl),
 - electrolyte imbalance(hypokalemia) &
 - Wt loss

Incidence

- Decreasing due to:
 - Better application of FP w/c reduces unplanned Px.
 - Early visit to ANC
 - Potent antihistamic, antiemetic drugs

Etiology

- Obscure but 2 facts are 2 known facts:
 - It is mostly limited to 1st TM (peak in 9th & 10th wk)
 - It is more common in 1st Px, with a tendency to recur again in subsequent Pxs
 - Family Hx-genetic
 - More common in molar Px & multiple Px

Theories

1.Hormonal:

- Excess hCG or higher biologic activity of hCG
- This is proved by the frequency of vomiting at the peak level of hCG & also the increased association with molar Px or multiple Px when z hCG titer is very raised.
- High level of estrogen
- Excessive progesterone
- Other hormones

Con't...

2.Psychogenic

3.dietetic def:- low CHO,def of vitB6,vit B1 & proteins

4.Allergy or immunologic basis

5.Decreased gastric motility;-causes nausea

Metabolic, biochemical,& circulatory changes

- **Metabolic**-glycogen depletion due to poor intake
- **Biochemical**-loss of H₂O & salt in vomitus cause fall in plasma Na,K,Cl
- Urinary Cl fall significantly
- Hepatic dysfunction results in acidosis & ketosis with rise in blood urea & uric acid;hypoglycemia;hyponatremia
- **Circulatory**:hemoconcentration leads to rise in Hgb % tage,RBC count & Hct value
- Reduction in ECF occurs

Risk factors

- Younger age
- Low pregnancy body mass
- Female fetus
- Hx of motion sickness & migraine
- Smoking & obesity are associated with decreased risk of hyperemesis

Clinical course

- The onset is insidious
- *early:*
 - Vomiting occurs throughout the day
 - Normal day to day activities are curtailed
 - There is no evidence of DHN or starvation

Con't...

- **Late(moderate to severe):**

➤ Sxs:

- vomiting increases with retching
- Urine quantity is diminished even to the level of oliguria
- Epigastric pain, constipation may occur
- Complication may appear if not treated

Con't...

- Signs

- Features of DHN & ketoacidosis:

- Dry coated tongue
- sunken eyes
- acetone smell in breath
- Tachycardia
- Hypotension
- rise in Temp may be noted

- jaundice is a late feature

Investigations

- ***U/A:***
 - small, dark color, high specific gravity, acetone, diminished or even absent Cl
- ***Biochemical & circular change:***
- ***Ophthalmologic exam***
 - Retinal hemorrhage & detachment is **unfavorable signs**
- ***ECG***
 - Abnormal serum potassium level
- ***Transient Lab. abnormalities occur:***
 - Suppressed TSH or elevated free thyroxin

Diagnosis

- The Px is to be confirmed first
- ***U/S:***
 - to confirm Px
 - To exclude other obstetrics(hydatid mole, multiple Px, gynecologic, surgical or medical causes of vomiting)

Rx of HEG

Mx principles

- ✓ To control vomiting
- ✓ To correct fluid & electrolytes
- ✓ To correct metabolic disturbance (acidosis & alkalosis)
- ✓ To prevent serious complications

☐ *If it is primarily symptomatic*

- Frequent, small, dry meals that favor protein over carbohydrates & liquids over solids

Con't...

- **Fluid**

- Withheld oral fluids for at least 24hr
- Total amount of fluid/24 hr approximates 3L of w/c ½ is 5% dextrose & ½ is R/L solution
- Extra amount of 5% dextrose equal to the amount of vomitus & urine in 24 hr, is to be added

- **Drugs**

- antiemetic drugs:promethazine 25mg or prochlorperazin 5mg ,metoclopramide
- Hydrocortisone 100mg Iv in the drip in case of hypotension

HEG Chart should be used daily to know the progress

—includes:

- V/S twice daily
- Intake-out put
- Urine for acetone, protein, bile,
- Blood biochemistry
- ECG

—Termination of pregnancy

- *Only in case of intractable HEG*

Amniotic fluid disorders

Definition:-

-Liquid that surrounds the fetus after the first few weeks of conception

Cont'd

• Source

- - Derived from - the fetus (almost entirely)
- maternal serum

• The relative contribution from each source changes across gestation

1. Early pregnancy - derived from **3 sources**:

- The fetal surface of the placenta
- Transport from the maternal compartment across the amnion (transmembranous)
- Secretions from the surface of the body of the embryo

2. late gestation (16 weeks)

- Fetal urine begins to enter the amniotic sac & the fetus begins to swallow AF

Regulation of Amniotic Fluid

Volume Production

- Fetal urination
- Lung liquid secretion

Absorption

- Fetal swallowing
- Intramembranous (placenta cord) absorption
- transmembranous (amniotic membranes)

➤ The human fetal urine production rate can be seen to be approximately **1,000 to 1,200 ml/day** at term



➤ Fetal swallowing rate was seen to be **72 to 262 ml/kg/day**

Functions of amniotic fluid

- ✓ Helps to protect the fetus from trauma
- ✓ Cushions the umbilical cord from compression
- ✓ Has antibacterial properties
- ✓ Serves as a reservoir of fluid & nutrients for the fetus
- ✓ Provides fluid, space, & growth factors to permit normal development of the fetal lungs, MSS & GI systems

Assessment of Amniotic

1. Direct fluid volume

- most accurate

2. Dye dilution test

- the second most accurate method
- done by injecting known concentration of dye into amniotic cavity
- followed by withdrawal of amniotic fluid to see the concentration of the dye and calculate the amniotic fluid volume
- cumbersome, invasive, and require specialized technical skills and laboratory

Cont'd

3. Ultrasound estimation methods

a. Single deepest pocket technique

- **Interpretation**

- 0 to 2 cm - Oligohydramnios
- 2.1 to 8 cm – Normal
- > 8 cm – Polyhydramnios

Cont'd.

b. Amniotic fluid index

- **Interpretation**

- **Oligohydramnios — 0 to <5 cm**
- **Normal — 5 to 25 cm**
- **Polyhydramnios — > 25 cm**

Cont'd.

c. Subjective assessment AF

- **Visual interpretation and by palpation without sonographic measurements**
- **Reports the amniotic fluid volume as**
 - **oligohydramnios**
 - **normal**
 - **polyhydramnios**

Amniotic fluid abnormalities

- Could be low (oligohydramnios) or high (polyhydramnios)
- Both low (oligohydramnios) and high (polyhydramnios), are associated with a multiple pregnancy-related problems and adverse perinatal outcomes
- Fetal congenital anomalies (eg, GI or UT obstruction), chromosomal abnormalities associated with abnormalities of AFV

1.Oligohydramnios

Causes

- ▶ Maternal Medical or obstetrical conditions associated with uteroplacental insufficiency
- ▶ Medications
- ▶ Twin to twin transfusion
- ▶ Fetal Chromosomal abnormalities
- ▶ Congenital abnormalities, especially those associated with impaired urine production
- ▶ IUGR
- ▶ Post term pregnancy
- ▶ PROM
- ▶ Idiopathic

Evaluation of pregnancies with oligohydramnios

- ➡ A maternal history and targeted physical examination is performed to look for maternal conditions
- ➡ Comprehensive sonographic evaluation
- ➡ If PROM is suspected the diagnosis may be confirmed using sterile speculum examination
- ➡ If there are fetal anomalies, amniocentesis may reveal an abnormal

Treatment

- ✓ No effective long-term treatment
- ✓ The prognosis and management depends on **cause and gestational age** at the time of diagnosis
- ✓ **Adverse outcomes are related to**
 - umbilical cord compression
 - uteroplacental insufficiency, and
 - meconium aspiration
- ✓ Delivery is indicated in women with oligohydramnios attributable to a specific condition (eg, preeclampsia, PROM, IUGR, congenital anomaly, post term pregnancy)
- ✓ For women with idiopathic oligohydramnios, delivery is suggested at **37 to 38** completed weeks

2. Polyhydramnios

Definition

➤ Excessive accumulation of amniotic fluid

Etiology

- ❏ Fetal malformations
- ❏ Chromosomal abnormalities
- ❏ Maternal diabetes mellitus
- ❏ Multiple gestation
- ❏ Fetal anemia
- ❏ Congenital infection
- ❏ Idiopathic

✗ The most common structural defects associated with polyhydramnios are those that interfere with fetal **swallowing and/or absorption of fluid**, such as gastrointestinal obstruction due to duodenal, esophageal, or intestinal atresia

✗ Decreased swallowing may also be due to anencephaly

Clinical manifestation and diagnosis

- ✚ the uterus will be large for gestational age
- ✚ the fetus could not be easily palpated by Leopold's maneuvers
- ✚ typically diagnosed by ultrasound examination and may be described qualitatively or quantitatively by various methods
 - Using single deepest pocket it is defined when **SDP >8cm**
 - **mild**
 - **moderate**

Evaluation of pregnancies with Polyhydramnios

- Comprehensive sonographic evaluation should be performed to determine whether **fetal anomalies or fetal hydrops** is present
- Laboratory evaluation may include as clinically indicated
 - glucose challenge test to screen for gestational diabetes
 - maternal serology to determine exposure to infectious agents
- Amniocentesis can be undertaken for karyotype analysis

Outcome

- Increased risk of several obstetrical complications related to uterine over distention
 - Maternal respiratory compromise
 - Preterm labor
 - PROM
 - Umbilical cord prolapse
 - Postpartum uterine atony
 - Fetal macrosomia (due to the association with maternal diabetes)

▪ The **earlier in gestation** it occurs and the greater the amount of fluid, **the higher the morbidity and mortality**

Treatment

- Amniotic fluid volume reduction
- Maternal administration of prostaglandin synthetase inhibitors(indometacin)

**** Indomethacin is discontinued after 32 to 34 weeks**

HYPERTENSIVE DISORDERS OF PREGNANCY



Hypertensive Disorders of Pregnancy (HDP)

- The most common medical complications of pregnancy
- HDP affect **5% to 10%** of all pregnancies
- The spectrum of disease ranges from mildly ↑ BP with minimal clinical significance to severe hypertension & multiorgan dysfunction

4 types of HDP

1. Gestational hypertension

Classification of HDP with Definitions

1. Gestational hypertension

:Hypertension developing after 20 wks gestation without proteinuria or other signs of preeclampsia & resolves by 12 wks postpartum

2. Chronic hypertension :

Hypertension diagnosed prior to pregnancy, prior to 20 weeks gestation, or persists > 12 weeks postpartum

Classification of HDP with Definitions

3. Preeclampsia or eclampsia:

Hypertension typically developing after **20 weeks** gestation with proteinuria

4. eclampsia is the occurrence of seizure activity without other identifiable causes.

5. Preeclampsia superimposed :The development of preeclampsia or eclampsia in a woman with **pre-existing or chronic hypertension**

HDP---

- About **30% & 70%** of HDP are due to chronic & gestational hypertension respectively
- Common causes of adverse maternal & perinatal outcome
- Severe preeclampsia or eclampsia have highest Morbidity & mortality
- The most dangerous type of **HDP** is superimposed PE/E (on chronic

Gestational Hypertension

- a) Systolic BP ≥ 140 or diastolic BP ≥ 90 mm Hg for 1st time after mid-pregnancy (20 weeks)
- b) No proteinuria
- c) BP returns to normal < 12 weeks postpartum
- d) Final diagnosis made only postpartum

❖ It is the most frequent cause of hypertension during pregnancy

❖ Gestational hypertension occurs

Gestational Hypertension---

- In almost 50%, it can subsequently develop PE
- Most commonly when it develops early ,before 30 wks of gestation
- *Transient hypertension* → if no evidence for preeclampsia & the BP returns to normal by 12 wks postpartum

Superimposed Preeclampsia upon chronic hypertension

Rate of superimposed preeclampsia
ranging from 10% to 25%

- 1) New-onset proteinuria ≥ 300 mg/24 hours in hypertensive women but no proteinuria before 20 weeks' gestation
- 2) Women with hypertension & proteinuria before 20 wks develops
 - ✓ a sudden \uparrow in proteinuria or BP to the severe range (SBP \geq 160 mmHg or DBP \geq 110 mmHg) or
 - ✓ thrombocytopenia ($<100,000/\text{mm}^3$)

Chronic Hypertension

- ✓ About 3-5% of pregnancies
- ✓ It can be primary (essential hypertension) or
- ✓ secondary to a variety of medical disorders
- ❖ Systolic BP ≥ 140 mmHg, diastolic BP ≥ 90 mmHg, or both
 - a) before pregnancy (with /out anti-hypertensives) or
 - b) diagnosed before 20 wks' gestation not attributable to GTD or

Causes of Chronic Hypertension

- ✓ Idiopathic
- ✓ Essential hypertension
- ✓ Vascular disorders
- ✓ Endocrine disorders
- ✓ Renal disorders etc

Preeclampsia

- ❖ A form of hypertension that is unique to human pregnancy
- ❖ A syndrome characterized by the new onset of hypertension & proteinuria after **20 wks** of gestation in a previously normotensive woman
- ❖ A *pregnancy-specific syndrome that can affect virtually every organ system*

Minimum criteria:

- BP \geq 140/90 mm Hg after 20 wks' gestation

Preeclampsia----

- a) in 3 to 14% of all pregnancies worldwide
- b) about 5 to 7% of pregnancies in the United States
- Mild Preeclampsia occurs in 75%, & severe in 25% of cases in the United States
- 10% of preeclampsia occurs in pregnancies <34 wks of gestation
- **Bimodal frequency distribution**
 - a) 1st peak in young, nulliparous &
 - b) 2nd peak in multiparous women >35 years of age
- Preeclampsia occurs more frequently in

Increased certainty of preeclampsia

- BP $\geq 160/110$ mm Hg
- Proteinuria ≥ 2.0 g/24 hours or $\geq 2+$ dipstick
- Serum creatinine >1.2 mg/dL unless previously \uparrow
- Platelets $< 100,000/L$
- Microangiopathic hemolysis— \uparrow LDH
- \uparrow serum transaminase levels—ALT or AST
- Persistent headache or other

Proteinuria

A 24-hour urinary protein excretion ≥ 300 mg is the gold standard for the diagnosis of proteinuria

Urine concentrations & dipstick readings vary widely during the day \rightarrow 1+ to 2+ value from concentrated urine specimens but 24hr excretion < 300 mg/day

The stages of PIH

- **Women with PIH may progress from mild disease to a more serious condition**
 - 1) Hypertension with proteinuria or edema
 - 2) Mild preeclampsia
 - 3) Severe preeclampsia
 - 4) Eclampsia

In **PIH** there may be no symptoms & the only sign may be **hypertension**

Proteinuria is a late sign of

Indicators of Severity of Gestational Hypertensive Disorders

Abnormality	Nonsevere ^b	Severe
Diastolic BP	< 110 mm Hg	≥ 110 mm Hg
Systolic BP	< 160 mm Hg	≥ 160 mm Hg
Proteinuria ^c	None to positive	None to positive
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion (eclampsia)	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia (< 100,000/ μ L)	Absent	Present
Serum transaminase elevation	Minimal	Marked
Fetal-growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

Risk factors for preeclampsia

1. Nulliparity
2. Age >40 years or <18 years (age >40 is independent factor for PE)
3. Hydatidiform mole
4. **Multifetal gestation**
5. DM (pre-gestational & gestational)
6. **Family history of preeclampsia**
7. **Obesity**
8. Previous history of preeclampsia
9. **Previous unexplained Bad Obstetric History (IUGR, P. abruption, IUFD)**

Risk factors...

- 11. Chronic renal disease
- 12. Male partner whose mother or previous partner had preeclampsia
- 13. Hydrops fetalis
- 14. Thyroid disease
- 15. Woman herself was SGA**
- 16. Prolonged inter-pregnancy interval
- 17. Connective tissue disease**
- 18. Inherited thrombophilia**

Protective factors of PE

1. **Smoking** has consistently been associated with a ↓ *risk* of hypertension during pregnancy why???
2. Placenta previa has also been reported to ↓ the risk of hypertensive disorders in pregnancy

Etiopathogenesis

- **Hypertensive disorders of pregnancy are more likely to develop in women who:**
 - a) Are exposed to chorionic villi for the 1st time
 - b) Are exposed to a superabundance of chorionic villi, as with twins or hydatidiform mole
 - c) Have pre-existing renal or cardiovascular disease
 - d) Are genetically predisposed to

Etiopathogenesis

- Preeclampsia is not simply "one disease,"
- It involves culmination of maternal, placental, & fetal factors

➤ **Cascade of abnormal host events that leads to the preeclampsia syndrome involves**

1. vascular endothelial damage
2. subsequent vasospasm
3. transudation of plasma,&
4. ischemia & thrombotic sequelae

Etiology of Preeclampsia

- The exact etiology is **unknown**
- Currently accepted theories (most of them are not proven)
 - Abnormal cytotrophoblast trophoblastic invasion of uterine vessels
 - Abnormal or ↑ immune response (Immunological maladaptive tolerance b/n maternal, paternal placental, & fetal tissues)
 - Maternal maladaptation to cardiovascular or inflammatory

Prevention

Some Methods to Prevent
Preeclampsia That Have Been
Evaluated in Randomized Trials

1. **Dietary manipulation**—low-salt diet, calcium supplementation, fish oil supplementation
2. **Cardiovascular drugs**—diuretics, antihypertensive drugs
3. **Antioxidants**—ascorbic acid (vitamin C), α -tocopherol (vitamin E)
4. **Antithrombotic drugs**—low-dose aspirin, aspirin/dipyridamole

- High-protein and low-salt diet
- Nutritional supplementation (protein)
- Calcium
- Magnesium
- Zinc
- Fish and evening primrose oil
- Antihypertensive drugs, including diuretics
- Antithrombotic agents
- Low-dose aspirin
- Dipyridamole
- Heparin
- Vitamins E and C
- Sildenafil

Fetal Assessment in Chronic Hypertension

- Early U/S (anomalies, gestational age Doppler velocimetry U/S fetal growth monitoring)
- Antepartum fetal monitoring started at 32–34 wks

Management of Mild chronic HTN

At each visit

- signs & symptoms of preeclampsia (headache, abdominal pain, blurred vision, rapid weight gain, or marked swelling of the hands &/or face)
- BP, urine protein, & fundal height
- Starting antepartum fetal monitoring at 32–34 wks
- Delivery at 39–40 wks

Mx. of Severe Chronic Hypertension

- *Indications of antihypertensive medication*
 - a) sustained BP **180/110 mm Hg** or
 - b) evidence of renal disease
 - c) at higher risk for serious complications(heart attack, stroke, or progression of renal disease)
- **Frequent prenatal visits;** at each visit, assess fetal growth, BP, & proteinuria & evidence of superimposed preeclampsia

Prognosis of chronic hypertension

- usually good with mild chronic hypertension with no other serious medical conditions
- **Poor prognosis**
- severe hypertension early in pregnancy
- evidence of end-organ compromise(renal insufficiency &/or cardiovascular disease)
- **The most common complications**

Gestational Hypertension

- progression to either severe hypertension, preeclampsia, or eclampsia esp. with a lower gestational age

Maternal evaluations require

- weekly prenatal visits
- education about reporting preeclamptic symptoms,
- evaluation of complete blood count, platelet count, and liver enzymes

Fetal evaluation includes

Management of PE

The basic management objectives for any pregnancy complicated by PE are:

- 1) Termination of pregnancy with the least possible trauma to mother and fetus
 - 2) Birth of an infant who subsequently thrives
 - 3) Complete restoration of health to the mother
- Induction of labour for PE,

Management

- Immediate delivery (mother vs. fetus interest)
- expectant management for extreme prematurity
corticosteroids to accelerate fetal maturation
- Several factors affecting decision for delivery
 - a) disease severity
 - b) fetal maturity
 - c) maternal and fetal condition,&

Preeclampsia with out severity features

- Many cases are sufficiently mild & near enough to term that they can be managed conservatively until
 - A. labour commences spontaneously or
 - B. until the cervix becomes favourable for labour induction

Preeclampsia with out severity features

Goals of such management include

- early identification of worsening preeclampsia and a plan for timely delivery
- Reduced physical activity throughout much of the day
- High protein& calories diet, sodium & fluid intake should not be limited or forced

Further management depends on:

(1) severity of preeclampsia

Mild Preeclampsia----

- Delivery at ≥ 40 weeks irrespective of cervix status
- Induction at 37–40 wks if favourable cervix status
- If cervix is unripen b/n 37 & 40 wks
 - a) cervical ripening agents
 - b) Expectant management with
 - bed rest
 - antepartum fetal surveillance, &
 - close monitoring of maternal condition, including:-
 - ✓ BP measurement every 4–6 hours &
 - ✓ daily assessment of patellar reflexes

Severe Preeclampsia

- hospitalization
- Delivery is indicated if
 - a) the gestational age is ≥ 34 wks
 - b) fetal pulmonary is confirmed, or
 - c) evidence of deteriorating maternal or fetal status
- Acute BP control with hydralazine, labetalol, or nifedipine
- The goal of antihypertensive therapy is **SBP < 160 mm Hg & DBP < 110 mm Hg**

Contraindication to Expectant management

1. uncontrollable hypertension
2. eclampsia,
3. DIC
4. HELLP syndrome,
5. pulmonary edema
6. Oliguria < 500 mL/24 hr or serum creatinine ≥ 1.5 mg/dL
7. Persistent platelet counts $< 100,000/L$

Complications of Severe Preeclampsia

Maternal

1. Eclampsia
2. HELLP syndrome /Disseminated coagulopathy
3. Pulmonary edema/aspiration
4. Acute renal failure
5. Abruptio placentae
6. Liver failure or hemorrhage
7. Stroke (rare)
8. Death (rare)
9. Long-term cardiovascular morbidity

Fetal

1. Preterm delivery
2. Fetal growth restriction
3. Hypoxia & neurologic injury
4. Perinatal death

Management of HDP

The steps of management include:

1. General measures - supporting the specific treatments
2. Prevent convulsion with magnesium sulphate or diazepam/valium
3. Control hypertension
4. Delivery as soon as possible

General Measures

- Admit the patient urgently
- Manage in left lateral position
- Set up IV line (using canulla) & infuse fluids to replace estimated loss (from bleeding, vomiting diarrhea, sweating) (500ml in 1st 1/2 hr)
- then ongoing loss + urine output + insensible loss (700 ml/24 hrs (PO & IV)
- Place an indwelling catheter to

General Measures----

- Maintain a strict fluid balance chart
- Prepare equipment for convulsion management, at bedside (mouthpiece, airway, suction equipment, mask & bag, oxygen)
- Never leave the patient alone (if convulsion occurs, aspiration may cause death)
- Observe vital signs, FHB & reflexes hourly

General Measures---

- Auscultate the lung bases for crepitation
- The immediate treatment should include managing symptoms
- Anti emetic
- Anti pain
- Stress Reduction

3. Anticonvulsant therapy (seizure prophylaxis)

Seizure prophylaxis should be instituted

1. In all preeclampsics during labour & continued for 24 hrs after delivery
 2. In all severe preeclampsics during admission & continued during period of evaluation & observation
- This is achieved by proper sedation & cutting off all peripheral stimuli

Delivery for Severe preeclampsia

Termination of pregnancy is the only cure for preeclampsia.

The primary objectives are to

- Anticonvulsant to prevent convulsions
- antihypertensive therapy to prevent intracranial hemorrhage & serious damage to other vital organs
- deliver a healthy infant.

WHO Should Be Given Magnesium Sulphate ?

Seizure prophylaxis should be instituted

- In all preeclampsics during labour & continued for 24 hrs after delivery
- In all severe preeclampsics during admission & continued during period of evaluation & observation

WHO Should Be Given

Magnesium Sulfate

- woman with **new-onset proteinuric** hypertension, at least one of the ***following criteria is required--***

- Systolic BP ≥ 160 or diastolic BP ≥ 110 mm Hg
- Proteinuria $\geq 2+$ as measured by dipstick in a catheterized urine specimen
- Serum creatinine > 1.2 mg/dL
- Platelet count $< 100,000/L$
- Aspartate transaminase (AST) $\uparrow 2X$ above upper limit of normal range

Magnesium Sulphate to Control Convulsions

1. Intermittent Intramuscular Injections

- Give 4 g of magnesium sulfate (MgSO_4) as a 20% solution IV at a rate not to exceed 1 g/min
- Follow promptly with 10 g of 50% magnesium sulfate solution, one-half (5 g) injected deeply in the upper outer quadrant of both buttocks through a 3-inch-long 20-gauge needle

Intermittent IM Injections---

- (Addition of 1.0 mL of 2% lidocaine minimizes discomfort.) If convulsions persist after 15 min, give up to 2 g more intravenously as a 20% solution at a rate not to exceed 1 g/min.
- If the woman is large, up to 4 g may be given slowly
- Every 4 hr thereafter give 5 g of a 50% solution of magnesium sulfate injected deeply in the upper outer

Before repeat administration, ensure that:

- Respiratory rate is at least 16 per minute
- Patellar reflexes are present
- Urinary output is at least 100 ml over 4 hours.

Withhold or delay drug if:

- Respiratory rate falls below 16 per minute.
- Patellar reflexes are absent
- Urinary output falls below 30 mL per hour over preceding 4 hours

Diazepam

- Diazepam is an effective alternative, but it increases the risk of respiratory depression & newborn asphyxia

Diazepam schedule for severe Pre-eclampsia & eclampsia

I) Intravenous administration

i) Loading dose

- Diazepam 10 mg IV slowly over 2 minutes

- If convulsion recur, repeat loading

Diazepam---

ii) Maintenance dose

- Diazepam 40 mg in 500 ml IV fluids (N/S or Ringer's lactate L)
- Diazepam may be given rectally when IV access is not possible.
- Peak levels are reached within 10-20 minutes
- Drops titrated to keep the woman sedated but arousable

Eclampsia

- Eclampsia is most common in the **last trimester** & ↑ more frequent as term approaches
- An ↑ shift in the incidence of eclampsia toward the postpartum period related to
 1. improved access to prenatal care
 2. earlier detection of preeclampsia, &
 3. prophylactic use of magnesium sulphate

Diagnosis of eclampsia

- Hypertension is assumed as the **hallmark** for the diagnosis of eclampsia.
- severe hypertension, severe proteinuria, & generalized edema
- absent or minimal hypertension, no proteinuria, & edema in 20% to 25% of cases
- Severe hypertension (≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic) in 20 to 54% or mild (SBP:140-160

DIFFERENTIAL DIAGNOSIS of Eclampsia

1. Stroke (haemorrhage, arterial or venous thrombosis)
2. Hypertensive disease
3. Infection (meningitis, encephalitis)
4. Cerebral malaria
5. Space-occupying lesions of the CNS (brain tumor etc
6. Metabolic disorders (hypoglycemia, uremia, **hyponatremia**)
7. ruptured cerebral aneurysm

Management of

Eclampsia

1. General measures

2. Control of convulsions (to stop ongoing convulsion & prevent repeated convulsion)
3. Intermittent administration of an antihypertensive medication to lower BP whenever it is considered dangerously high & stabilization of the condition of the mother & fetus
4. Delivery of the fetus to achieve a "cure." & intra partum/post

General Measures in the Mx of Eclampsia

1. ABCD rule of resuscitation
 - Set up IV line & maintain intravascular volume & replace ongoing losses; avoid overload (if not done already)
 - make the airway is clear & prevent injury & aspiration of gastric contents from the mouth & throat
2. Position the patient on her side (left lateral) & in Trendelenburg (head down) position to reduce risk of

General Measures ---

5. Avoid tongue bite by placing an airway or padded tongue blade between the teeth & protect the Woman from injury but do not actively restrain
6. Place an indwelling catheter to monitor urine output & urine test for protein (if not done already)
 - Observe vital signs, FHB & reflexes frequently & auscultate the lung bases hourly for

Fluid Therapy

- Conservative fluid administration(↓ plasma volume)
- Except unusual fluid loss from vomiting, & diarrhea, or more likely, excessive blood loss with delivery
- infusion of large fluid volumes appreciably ↑the risk of pulmonary & cerebral edema
- To avoid pulmonary edema, total IV fluids should be 60-125

Intrapartum Management

- labor may begin spontaneously shortly after convulsions ensue & may progress rapidly.
- If the convulsion occurs during labor
 1. contractions may ↑ in frequency & intensity, &
 2. the duration of labor may be shortened.
- Maternal hypoxemia & lactic acidemia by convulsions → fetal

Intrapartum Management

- if the bradycardia &/or recurrent late decelerations persist beyond 10–15 minutes despite all resuscitative efforts
 - a) ? abruptio placentae or
 - b) persistent nonreassuring fetal status
- Uterine contractions can ↑ in frequency & tone.
- No rush for emergency C/S especially if the maternal

Is Eclampsia Preventable?

- At present, there is no effective preventive therapy for preeclampsia.
- Prevention of eclampsia can be
 1. primary by preventing the development of preeclampsia or
 2. secondary by using pharmacologic agents that prevent convulsions in women with established preeclampsia.
 3. tertiary by preventing

HELLP Syndrome

All of the following Criteria are required for diagnosis

1.Hemolysis

- a) Abnormal peripheral blood smear**
- b) \uparrow bilirubin (>1.2 mg/dl)**
- c) \uparrow lactic dehydrogenase(LDH >600 IU/L)**

2.Elevated liver enzymes($\uparrow \geq 2x$)

- a) \uparrow ALT/AST(≥ 72 IU/L)**
- b) \uparrow LDH >600 IU/L**

Management of HELLP Syndrome

- Immediate delivery is indicated if the
 - a) $G/A \geq 34$ wks or
 - b) $G/A < 34$ wks with
 1. multiorgan dysfunction
 2. DIC
 3. liver infarction
 4. hemorrhage
 5. renal failure
 6. suspected abruptio placentae or

Conservative management of HELLP Syndrome

- Bed rest
- Antihypertensive agents
- Chronic parenteral magnesium sulfate
- Antithrombotic agents (low-dose aspirin, dipyridamole)
- Plasma volume expanders (crystalloids, albumin, FFP)
- Steroids (dexamethasone, or betamethasone)

Long-Term Sequelae

- any hypertension during pregnancy is a marker for ↑ rates of
 1. Chronic hypertension
 2. ischemic heart disease
 3. Stroke
 4. venous thromboembolism
 5. all-cause mortality
 6. chronic renal disease

Antepartum hemorrhage

OUTLINE OF PRESENTATION

- Definition
- Etiologies
- Abruptio Placenta
- Placenta Previa
- Vasa Previa

Defn.

- *Antepartum hemorrhage (APH) refers to any bleeding from the genital tract following fetal viability (20 or 28 weeks) and before the delivery of the fetus*
- *APH covers the antepartum period after fetal viability and any bleeding during the first and second stages of labor*
- *Covers*
 - 2-3% of all pregnancies
 - $\geq 50\%$ the cause is unknown

Etiologies of antepartum hemorrhage

- **Placental causes –**
 - **Abruptio placentae**
 - **Placenta previa**
 - **Vasa previa**
 - **Marginal sinus rupture**
 - **Circumvallate placenta**
- **Non-placental causes**
 - **Local causes – cervicitis, cervical tumors, vaginal tumors or trauma**
 - **Uterine rupture – antepartum or intrapartum**
 - **DIC and other bleeding diathesis**
 - **“Heavy Show”**
- **Idiopathic /undetermined**

ABRUPTIO PLACENTAE

INTRODUCTION

- Placental abruption is defined as **decidual hemorrhage** leading to the premature separation of the placenta prior to delivery of the fetus
- Although there are **no standard diagnostic criteria** for placental abruption
- the **clinical hallmarks** of the condition are
 - vaginal bleeding and pain

INTRODUCTION

- The immediate cause of the placental separation is often the rupture of defective maternal vessels in the decidua basalis, where it interfaces with the anchoring villi in the placenta
 - Rarely, the bleeding can originate from the fetal-placental vessels
- The accumulating blood splits the decidua, separating a thin layer of decidua with its placental

INCIDENCE

- Placental abruption complicates about **1 in 100 births**
- The incidence **peaks at 24 to 26 weeks** of gestation , although gestational age-specific incidence rates vary considerably according to whether the abruption is due to an **acute or chronic etiology**

PATHOGENESIS

- Both **acute and chronic processes** lead to the development of placental abruption
- Separation of the placenta from the uterine wall can be the result of
 - **mechanical force** (eg, blunt trauma to the abdomen or rapid uterine decompression)
 - related to **abnormalities of the uteroplacental vessels** (thrombosis, decidual vasculopathy, inflammation).

CLINICAL MANIFESTATION

- The clinical manifestations of placental abruption are **dependent on a variety of factors**, including whether the hemorrhage is
 - (1) acute or chronic
 - (2) concealed or clinically evident, and
 - (3) mild or severe

ACUTE ABRUPTION

- An acute, clinical abruption classically presents with
 - painful vaginal bleeding
 - abdominal and/or back pain
 - uterine contractions.
- The contractions are usually **high frequency and low amplitude**, but a mild to moderate contraction pattern is also possible.
- The uterus may be **rigid and tender**.
- With greater degrees of placental separation, **fetal heart rate abnormalities or fetal death** may occur
- Unfortunately, the **amount of vaginal bleeding**

ACUTE ABRUPTION

- Occasionally, a woman with placental abruption will present with **only preterm labor, and no vaginal bleeding**
 - "Concealed" hemorrhage occurs in as many as **10 to 20 percent** of abruptions
 - all or most of the blood is trapped between the fetal membranes and decidua, rather than escaping through the cervix and vagina
 - Therefore, even small amounts of vaginal bleeding in the setting of abdominal pain and uterine contractions should prompt **close maternal and**

ACUTE ABRUPTION

- In the presence of a **severe abruption (≥ 50 percent placental separation)**
 - both **fetal and maternal compromise** may occur.....
 - **Acute DIC..**
 - **2o to** blood is exposed to large amounts of tissue factor over a brief period of time, with massive generation of **thrombin**, resulting in the acute triggering of coagulation.

ACUTE ABRUPTION

- Acute DIC
 - The clinical consequence is a
 - profound systemic bleeding
 - Common manifestations of acute DIC, in addition to bleeding and shock, include
 - dysfunction of the kidney, liver, lungs, and central nervous system
 - thromboembolism.

CHRONIC ABRUPTION

- placental abruption can be a **chronic process** and a manifestation of **ischemic placental disease**
- Pathologically, the placental separation in chronic abruption
 - typically occurs **at the periphery**
 - caused by **low pressure venous hemorrhage**, often associated with **inflammatory necrosis**
- Affected patients experience relatively **light, chronic, intermittent bleeding**
- exhibit clinical manifestations that **develop over time**, such as
 - oligohydramnios

CHRONIC ABRUPTION

- By comparison, acute abruption is typically caused by
 - high pressure arterial hemorrhage in more central areas of the placenta.
 - Fetal heart rate abnormalities and DIC occur when there is significant acute placental separation

DIAGNOSIS

- The diagnosis of abruptio placentae is **primarily clinical**
 - but **radiologic, laboratory, and postpartum pathologic findings** can be used to support the clinical diagnosis.
- The diagnosis should be considered in pregnant women who present with one or more of the following
 - vaginal bleeding, abdominal pain, preterm labor, or trauma

DIAGNOSIS

- A **retroplacental clot** is the **classic ultrasound description** of placental abruption
 - however, its identification is dependent on the
 - extent of the hemorrhage
 - the chronicity of the bleeding
 - whether there has been escape of blood through the cervix

RETROMEMBRANEOUS BLEED



DIAGNOSIS

- Laboratory testing is **not useful** in making the diagnosis of abruptio placentae
 - but coagulopathy
 - Disseminated intravascular coagulation (DIC) occurs in **10 to 20 percent** of cases of **severe abruption** with death of the fetus

RISK FACTORS

- the etiology of placental abruption is **heterogeneous and speculative.**
- Risk factors for placental abruption can be classified as
 - (1) those associated with an acute etiology
 - (2) medical and obstetrical risk factors
 - (3) maternal sociodemographic and behavioral risk factors.

ACUTE EVENTS

- **Maternal trauma** during pregnancy
 - may cause external **compression-decompression**
 - In motor vehicle accidents, an additional factor is **rapid acceleration-deceleration**
 - This cascade of events may ultimately culminate in placental abruption.
 - tend to be **severe and generally present within 24 hours of the precipitating event**

ACUTE EVENTS

- Rapid uterine decompression
 - such as after rupture of membranes in the setting of **polyhydramnios or after delivery of a first twin**, can also trigger placental abruption
- placental implantation over a uterine anomaly or leiomyoma.
 - **mechanically and biologically unstable sites**
 - subject to both **torsion and inadequate decidualization**.

MEDICAL AND OBSTETRIC RISKS

- include
 - hypertensive disorders
 - premature rupture of membranes
 - chorioamnionitis
 - ischemic placental disease in the current or previous pregnancy
 - inherited thrombophilia

SOCIODEMOGRAPHIC OR BEHAVIORAL FACTORS

- Maternal age
- Parity
- Smoking
- Male infant sex

COMPLICATIONS

- Maternal
 - Hypovolemia related to blood loss
 - Need for blood transfusion
 - Disseminated intravascular coagulopathy
 - Renal failure
 - Respiratory Distress Syndrome
 - Multisystem organ failure
 - Death

COMPLICATIONS

- Fetal
 - Growth restriction (with chronic abruption)
 - Fetal hypoxemia or asphyxia
 - Preterm birth
 - Perinatal mortality
- The risks to the mother are primarily related to the **severity of the abruption**, while the risks to the fetus are related to **both the severity of the abruption and to the**

MANAGEMENT

- Initial management
- Blood and blood product replacement
- Subsequent management

INITIAL MANAGEMENT

- Patients suspected to have a placental abruption should undergo a **rapid initial evaluation**.
- Subsequent management is determined on a **case-by-case basis**
 - will depend upon the
 - severity of the abruption
 - gestational age
 - maternal status
 - fetal status

INITIAL MANAGEMENT

- secure **intravenous access** In severe cases, a **Foley catheter**
- monitor maternal urine output hourly
- The urine output should be maintained **at above 30 ml/hour.**
- A complete blood count, blood type and Rh, and coagulation studies are obtained.
- Maintain the hematocrit above 30 percent

LIVE FETUS AT OR NEAR TERM

- The fetus should be **delivered expeditiously** by the quickest, safest method if it is alive, the pregnancy is **at least 34 weeks** of gestation, and abruption is suspected
 - **Vaginal delivery** is reasonable if the maternal status is stable and the fetal heart tracing is reassuring.
 - **Prompt cesarean delivery** is indicated if significant maternal or fetal compromise occurs and vaginal

LIVE FETUS AT OR NEAR TERM

- Cesarean delivery is indicated if
 - the fetal heart tracing is **non reassuring**
 - there is ongoing major blood loss or other serious maternal complications
 - vaginal delivery is contraindicated.
- It is reasonable **to consider delivery by 37 to 38 weeks** because of the increased risk of stillbirth

Placenta previa

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- Although there are **no standard diagnostic criteria** for placental abruption
- the **clinical hallmarks** of the condition are
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 - uterine hypertonicity, tetanic uterine contractions
 - non reassuring fetal heart rate pattern

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 - related to **abnormalities of the uteroplacental vessels** (thrombosis, decidual vasculopathy, inflammation).
- Decidual hemorrhage and formation of a retroplacental clot cause a **cascade of events** that result in reduced maternal-fetal oxygen and

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Placenta previa

DEFINITION

- Placenta previa refers to the presence of placental tissue **overlying or proximate** to the internal cervical os
 - Bleeding, which ranges from **spotting to hemorrhagic**, is the main complication
- Four placental configurations have been defined:
 - Complete placenta previa (PP TOTALIS)
 - The placenta **completely covers** the internal os
 - **Twenty to 30 percent** of placenta previas are central

DEFINITION

- Types of PP
 - Partial placenta previa
 - The placental edge partially covers the internal cervical os, which must be partly dilated for this to occur
 - Marginal placenta previa
 - The placenta is adjacent to the internal os, but does not cover it

DEFINITION

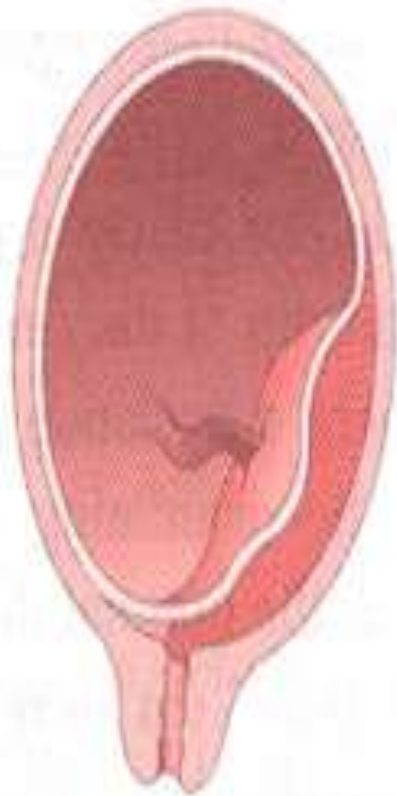
- Types of PP

- **Low-lying placenta**

- This term is used in several ways:
 - (1) to describe an apparent placenta previa in the **second trimester**,
 - (2) to describe a placenta that lies in the lower uterine segment, but the exact relationship of the placenta to the os has not been determined, or
 - (3) to describe a placental edge that lies **within 2 to 3 cm of the internal os**
 - Low lying placentas are also associated with an increased risk of bleeding, and possibly other adverse perinatal outcomes, although less than with true placenta previas



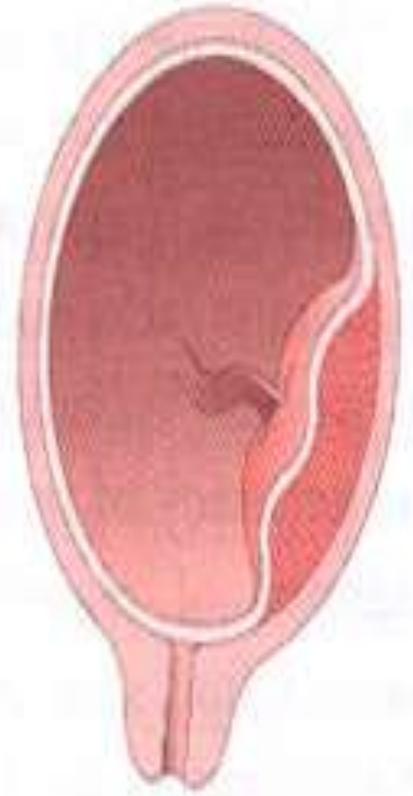
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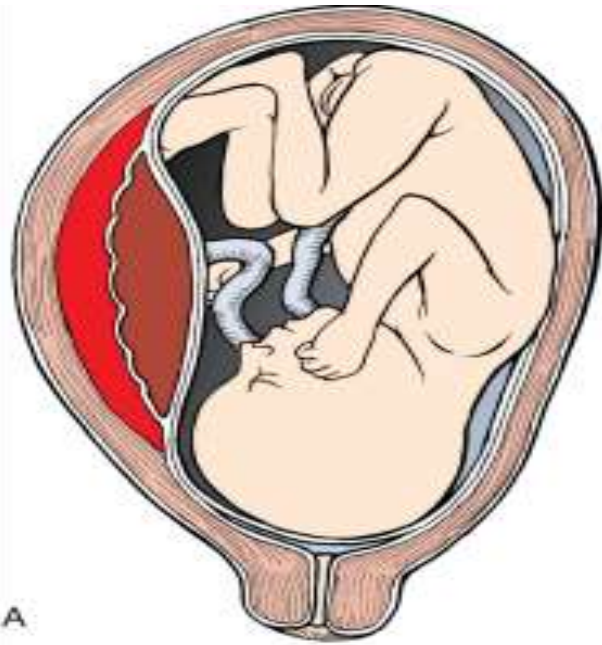
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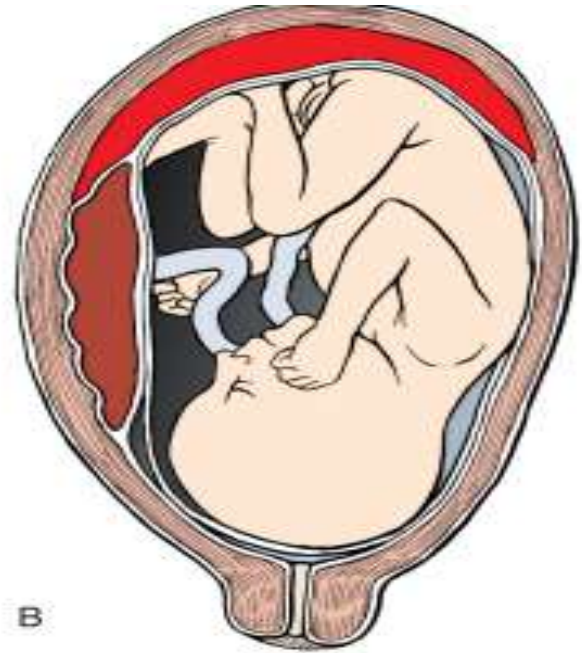
Marginal



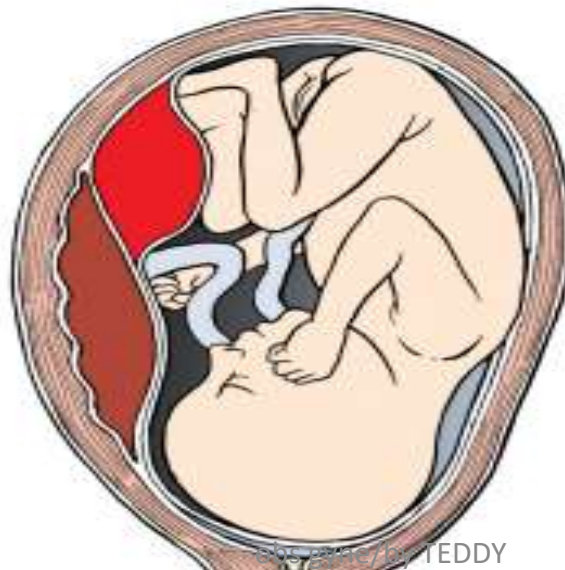
Low lying



A



B



INCIDENCE

- Placenta previa complicates approximately
 - 4 per 1000 pregnancies that are over 20 weeks of gestation

RISK FACTORS

- Risk factors can be grouped according to the **pathogenetic mechanism involved**
- (1) **Endometrial scarring** in the upper segment of the uterus may promote
 - either initial trophoblastic nidation into the relatively unscarred lower uterine segment or unidirectional growth toward the unscarred lower uterine segment
 - **prior cesarean deliveries**
 - **Increasing parity**
 - **Increasing maternal age**
 - **prior curettages** for spontaneous or induced

RISK FACTORS

- 2) The need for **increased placental surface area** to compensate for a reduction in uteroplacental oxygen or nutrient delivery
 - include
 - Maternal smoking
 - Residence at higher altitudes
 - Multiple gestation
- 3) **Early gestational age** is a risk factor for placenta previa because **placental migration** away from the cervical os occurs as pregnancy progresses

CLINICAL MANIFESTATIONS

- The characteristic clinical presentation of placenta previa is **painless vaginal bleeding** after 20/28 weeks of gestation
 - this occurs **in 70 to 80 percent** of patients
 - **10 to 20 percent** of women present with **uterine contractions** associated with bleeding
 - **fewer than 10 percent** are **incidentally detected** by ultrasound examination and remain asymptomatic

CLINICAL MANIFESTATIONS

- Bleeding is likely to occur during the **third trimester** because of
 - development of the lower uterine segment
 - the increasing presence of **uterine contractions**, which dilate and efface the cervix
- Changes in the cervix and lower uterine segment apply **shearing forces** at the **inelastic placental attachment site**, resulting in placental detachment and bleeding
- Separation can also be caused by **vaginal**

ASSOCIATED CONDITIONS

1. Placenta accreta
2. Malpresentation
3. Preterm premature rupture of the membranes (PPROM)
4. Intrauterine growth restriction
5. Vasa previa and velamentous umbilical cord
6. Congenital anomalies
7. Amniotic fluid embolism

DIAGNOSIS

- The diagnosis of placenta previa is based upon results of **ultrasound examination**
- Placenta previa should be suspected in any woman beyond **24 weeks** of gestation who presents with painless vaginal bleeding
 - Classically, the **absence of abdominal pain and uterine contractions** has been used to distinguish placenta previa from abruptio placentae
 - some women with placenta previa have painful Ux contraction diagnosis of PP must be determined by **sonographic examination**

DDx

- Third trimester bleeding complicates 3 to 4 percent of pregnancies
- The differential diagnosis includes
 - abruptio placentae (31 percent)
 - placenta previa (22 percent)
 - a variety of other causes (47 percent)
 - decidual or cervical bleeding associated with labor or neoplasms
 - Sometimes a cause cannot be identified

U/S

- TVS
- Transabdominal ultrasonography is used for initial placental localization

NOTES!!!

- Mild degree pp=type I and type II anterior
 - Can be allowed vaginally
- Major degree=type II posterior, III and IV
- Dangerous=type II posterior b/c
 - Overlies sacral promontory and prevent engagement + increase cord prolapse and compression

DIFFERENTIATION OF PP AND AP

- Bleeding
 - PP=bright red,causeless,painless,recurrent
 - AP=dark menstrual like,painful,associated with cause,non recurrent
- Shock
 - PP=proportional to blood loss but not in AP
- Uterine tenderness
 - Abscent in PP and present in AP
- Fetal condition
 - Ok in PP and distress/dead in AP
- Fetal presentation

MANAGEMENT

- The management of pregnancies complicated by placenta previa is best considered in terms of the clinical setting:
 - asymptomatic women
 - women who are actively bleeding
 - women who are stable after one or more episodes of active bleeding

Asymptomatic PP

- General principles of management of asymptomatic placenta previa after 20 weeks of gestation include:
 - Sonographic reassessment to determine placental position
 - Avoidance of coitus and digital cervical examination in the third trimester
 - Counseling to seek immediate medical attention if contractions or vaginal bleeding occur

ACUTE CARE OF SYMPTOMATIC PP

- Establish intravenous access and administer crystalloid
- Blood bank and laboratory monitoring
- Fetal monitoring
- Maternal monitoring
- Tocolysis
- Indications for delivery
- Anesthesia

ACUTE CARE OF SYMPTOMATIC PP

- ❖ An actively bleeding placenta previa is a potential obstetrical emergency
 - ❖ women should be **admitted to the Labor and Delivery Unit** for maternal and fetal monitoring
 - ❖ On the way to the hospital, **anti-shock garments** may restore an adequate blood pressure in women who are hemodynamically unstable from hemorrhage

OPEN IV LINE

- One or two large bore intravenous lines are inserted
 - and crystalloid (Ringers lactate or normal saline) is infused to maintain
 - hemodynamic stability
 - adequate urine output (at least 30 mL/hour)

DELIVERY

- Delivery is indicated if any of the following occur:
 - A non reassuring fetal heart rate tracing unresponsive to maternal oxygen therapy, left-sided positioning, and intravascular volume replacement
 - Life-threatening refractory maternal hemorrhage
 - Significant vaginal bleeding after 34 weeks of gestation
- Cesarean delivery is the delivery route of choice

Conservative Mgt After Acute Bleed

- **Hospitalization versus outpatient management**
- Correction of anemia
- Autologous blood donation
- Antenatal corticosteroids
- Rh(D) immune globulin
- Fetal assessment
- Tocolysis
- Cerclage

CONSERVATIVE Mgt AFTER ACUTE BLEED

- Most women who initially present with symptomatic placenta previa respond to supportive therapy and do not require immediate delivery

Hospitalization Vs Out Patient Mgt

- Symptomatic women are often hospitalized from their initial bleeding episode until delivery
 - Since the frequency and severity of recurrent bleeding episodes are unpredictable, maintaining close proximity to the labor and delivery unit is necessary for emergency cesarean delivery when needed
- Select women with placenta previa may be discharged if bleeding has stopped for a minimum of 48 hours and there are no other pregnancy complications

Hospitalization VS Out Patient Mgt

- candidates for outpatient care should:
 - Be able to return to the hospital quickly
 - Have an adult companion available 24 hours a day who can immediately transport the woman to the hospital if there is light bleeding or call an ambulance for severe bleeding
 - Be reliable and able to maintain bed rest at home
 - Understand the risks entailed by outpatient management

DELIVERY

- **Timing**
- In **stable** women, most clinicians perform amniocentesis **at 36 weeks** of gestation to assess **fetal pulmonary maturity** and then schedule cesarean delivery if the results predict mature lungs
 - The rationale for this approach is **to avoid emergency delivery** necessitated by placental hemorrhage

DELIVERY

- timing
 - If test results are not predictive of maturity at 36 weeks, subsequent management is predicated upon the **patient's condition**
 - Patients who do not have bleeding, contractions, or cervical shortening can continue with expectant management
 - We repeat the amniocentesis at 37 weeks of gestation and deliver if pulmonic indices are mature
 - If the indices are still immature, we deliver at **38 weeks of gestation without further assessment of fetal pulmonary status**

DELIVERY

- Route
 - Complete previa
 - A **cesarean delivery** is always indicated when there is sonographic evidence of a complete placenta previa and a viable fetus.
 - **Vaginal delivery** may be considered in rare circumstances, such as in the presence of a **fetal demise or a previable fetus**, as long as the mother remains **hemodynamically stable**

DELIVERY

- Route
 - Low-lying placenta
 - Studies showed there appears to be a reasonable possibility of vaginal delivery without hemorrhage when the placenta is **greater than 2.0 centimeters** from the internal os.

DELIVERY

- Route
 - Marginal previa
 - We suggest scheduled cesarean delivery for these pregnancies to minimize the risk of emergent delivery and hemorrhage.

OUTCOME

1. Hemorrhage
2. Maternal mortality
3. Neonatal morbidity and mortality
4. Placenta accreta
5. Complication of Prior cesarean delivery

VASA PREVIA AND VELAMENTOUS UMBILICAL CORD

INTRODUCTION

- Vasa previa and velamentous insertion of the umbilical cord
 - refer to abnormalities in the position and structure of the umbilical cord
- Both are associated with increased risks of fetal hemorrhage and cord compression

VELAMENTOUS UMBILICAL CORD

- **Definition**

- A velamentous cord refers to vessels surrounded only by fetal membranes, with no Wharton's jelly
- This typically occurs in the portion of the umbilical cord proximate to its placental insertion
- Membranous vessels can also occur between lobes of a bilobed placenta

- **Prevalence**

- occurs in up to 1 percent of singleton gestations , but is observed in almost 15 percent of fetuses from monochorionic twin gestations

VASA PREVIA

- Definition and clinical significance
 - Vasa previa refers to **vessels that traverse the membranes located in the lower uterine segment** in advance of the fetal presenting part
 - Rupture of these vessels can occur with or without rupture of the membranes and result **in fetal exsanguination**
 - In **monochorionic twin gestations**, the perinatal mortality rate is high for both twins, even if the vasa previa is associated with only one twin, due to the presence of placental vascular anastomoses
 - The vessels are **at risk of compression** from the fetal presenting part since they are not protected by the

VASA PREVIA

- **Diagnosis**

- Diagnosis of vasa previa is based upon sonographic, clinical, and pathological findings
- The diagnosis of vasa previa should be considered in the setting of vaginal bleeding that occurs upon rupture of the membranes

Premature Rupture of Membrane and preterm labour

- Defined as spontaneous rupture of membranes prior to the onset of labor at any stage of gestation
- **Term PROM** is rupture of membranes after 37 completed weeks of gestation
- **Preterm PROM** → PPRM >20weeks but <37 weeks (WHO)
- (Ethiopia) preterm PROM → b/n 28 & 37 weeks
- Rupture of membrane <28weeks → inevitable abortion
- PROM occurs in about 10-15% of all delivery

INTRODUCTION

- The membranous structure is derived from **fetal tissue** and is composed of **two layers**:
 - the amnion (inner layer)
 - the chorion (outer layer).
- The amnion is a **translucent structure** adjacent to the amniotic fluid, which provides necessary nutrients to the amnion cells
- The chorion is a **more opaque membrane** that is attached to the decidua (ie, maternal tissue that lines the uterus during pregnancy).

PPROM

- Premature rupture of membranes (PROM) refers to membrane rupture **before the onset of uterine contractions**
- preterm PROM (PPROM) is the term used when the pregnancy is **less than 37 completed weeks** of gestation.
 - PPRM occurs in **3 percent of pregnancies** and is responsible for approximately **one-third of preterm births**.

ETIOLOGY AND RISK FACTORS

RISK FACTOR

- History of PPRM,
- Genital tract infection,
- Antepartum bleeding, and
- Cigarette smoking have a particularly strong association with PPRM

Others

1. Stress
2. Occupational fatigue
3. Excessive or impaired uterine distention
4. Cervical factors
5. Infection
6. Placental pathology

Previous PPROM

- Studies have consistently shown that a history of PPROM is a **significant risk factor for recurrence**.
 - women with a history of PPROM had a **13.5 percent rate of PPROM** in a subsequent pregnancy compared to **4.1 percent in women with no such history**
 - Women with a history of PPROM are at risk for recurrent PPROM or preterm birth without PROM

- Genital infection
 - Genital tract infection is the **single most common identifiable risk factor** for PPRM.
 - Three lines of epidemiologic evidence strongly support this association:
 - (1) women with PPRM are significantly more likely than women with intact membranes to have **pathogenic microorganisms in the amniotic fluid**

- (2) women with PPRM have a significantly **higher rate of histologic chorioamnionitis** than those who deliver preterm without PPRM
- (3) the frequency of PPRM is significantly higher in women with certain **lower genital tract infections** (eg, bacterial vaginosis) than in uninfected women

- Antepartum bleeding
 - Antepartum bleeding in **more than one trimester increases** the risk of PPROM **three to seven-fold**
- Cigarette smoking
 - The risk of PPROM among smokers is **increased two to four-fold** compared to nonsmokers.

OUTCOME

- Approximately **one-third** of women with PPRROM develop potentially **serious infections**, such as **intraamniotic infection**, **endometritis**, or **septicemia**.
- The **fetus and neonate** are at greater risk of PPRROM-related **morbidity and mortality** than the mother.
 - The majority of pregnancies with PPRROM **deliver preterm and within one week** of membrane rupture.

- PPRM is also associated with increased risks of **abruptio placentae and prolapse of the umbilical cord.**
- **Fetal malpresentation** is common, given the preterm gestational age and the frequent occurrence of reduced amniotic fluid volume.

- **Early, severe, prolonged oligohydramnios** can be associated with **pulmonary hypoplasia, facial deformation, and orthopedic abnormalities**

CLINICAL MANIFESTATION AND DIAGNOSIS

- History
- Diagnostic evaluation
 - Physical examination
 - Nitrazine and fern tests
 - Ultrasonography
 - Instillation of indigo carmine
 - AmniSure

HISTORY

- The classic clinical presentation of PPRROM is a sudden "gush" of clear or pale yellow fluid from the vagina.
- However, many women describe intermittent or constant leaking of small amounts of fluid or just a sensation of wetness within the vagina or on the perineum.
- A clinical history suggestive of PPRROM should be confirmed by visual inspection or laboratory tests to exclude other causes of wetness, such as

PHYSICAL EXAMINATION

- The best method of confirming the diagnosis of PPROM is
 - direct observation of amniotic fluid coming out of the cervical canal or pooling in the vaginal fornix.
- If amniotic fluid is not immediately visible, the woman can be asked **to push on her fundus, Valsalva, or cough** to provoke leakage of amniotic fluid from the cervical os.
- **Digital examination** should be avoided because it may

NITRAZINE AND FERN TEST

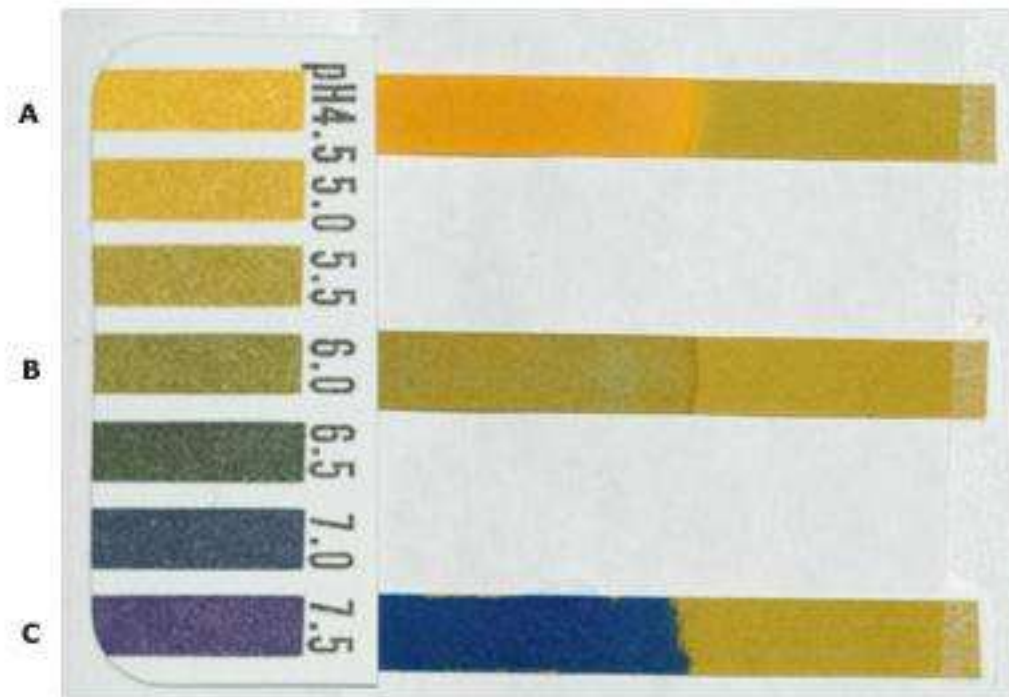
- If the diagnosis is not obvious after visual inspection, the diagnosis can be confirmed by **testing the pH** of the vaginal fluid, which is easily accomplished with nitrazine paper.
 - Amniotic fluid has a pH range of **7.0 to 7.7** compared to the normally acidic vaginal pH of **3.8 to 4.2**
- **False-negative and false-positive** test results occur in up to **5 percent** of cases
 - False negative tests results can occur when leaking is intermittent or the amniotic fluid is **diluted by**

NITRAZINE AND FERN TEST

- A second confirmatory test is the presence of **arborization (ferning)**.
 - Fluid from the **posterior vaginal fornix** is swabbed onto a glass slide and allowed to dry for at least **10 minutes**.
 - Amniotic fluid produces a **delicate ferning pattern**, in contrast to the **thick and wide arborization pattern of dried cervical mucus**.
 - **Well-estrogenized cervical mucus or a fingerprint on the microscope slide may cause a false-positive fern test**

NITRAZINE PAPER TEST

A=normal, B=bacterial
vaginosis, C=ROM(AF)



U/S

- Ultrasound examination may be of value in the diagnosis of PPRM.
- **Fifty to 70 percent** of women with PPRM have low amniotic fluid volume on initial sonography
 - A **mild reduction** of amniotic fluid volume may have many etiologies.
 - On the other hand, the finding of **anhydramnios or severe oligohydramnios**, combined with a characteristic history, is highly suggestive of rupture of membranes, although **renal agenesis**,

MANAGEMENT

- The management of pregnancies complicated by PPRM is based upon consideration of several factors, which are assessed upon presentation:
 - Gestational age
 - Availability of neonatal intensive care
 - Presence or absence of maternal/fetal infection
 - Presence or absence of labor
 - Fetal presentation (Breech and transverse lies are unstable and may increase the risk for cord prolapse)
 - Fetal heart rate (FHR) tracing pattern
 - Likelihood of fetal lung maturity

INITIAL EVALUATION

- Expeditious delivery of women with PPROM is indicated if
 - intrauterine infection
 - abruptio placentae
 - repetitive FHR decelerations
 - a high risk of cord prolapse is present or suspected
 - In each of these conditions, fetal well-being can deteriorate with expectant management, and there are no therapeutic interventions available other than delivery
- We and others posit that pregnancies ≥ 32 weeks of gestation with documented fetal lung maturity achieve better maternal and neonatal outcomes with delivery rather than expectant management, as long as expert neonatal care is available

ANTENATAL GLUCOCORTICOIDS

- Glucocorticoids are given **as late as 34 weeks of gestation** to women with **intact membranes** at risk for preterm delivery
- The use of antenatal glucocorticoids after 32 weeks in women with PPRM is more controversial, as treatment at this gestational age has not consistently resulted in benefit.

ANTIBIOTIC PROPHYLAXIS

- The rationale for antibiotic prophylaxis is that **infection** appears to be **both a cause and consequence** of PPRM and is related to preterm delivery.
- The goal of antibiotic therapy is to
 - reduce the frequency of maternal and fetal infection and
 - delay the onset of preterm labor (ie, prolong latency).

ANTIBIOTIC PROPHYLAXIS

- We administer a **seven-day course** of antibiotic prophylaxis to all women with PPROM who are being managed expectantly.
- Our preference is to give
 - **ampicillin** 2 g intravenously every six hours for **48 hours**, followed by **amoxicillin** (500 mg orally three times daily or 875 mg orally twice daily) for an additional **five days**.

TOCOLYSIS

- The use of tocolysis is **controversial**
 - there are inadequate data on which to make an evidence-based recommendation for or against their use.
- Tocolytics are unlikely to be effective in women with **advanced labor**.
- Many clinicians administer tocolytics for **48 hours to women at less than 32 weeks** of gestation with contractions, but not in advanced labor, in an attempt to delay delivery to allow administration of antenatal

HOSPITALIZATION

- We hospitalize women with PPRM who have a **viable fetus** from the time of diagnosis **until delivery**, with few exceptions.
 - Women are typically kept at modified bedrest and frequently assessed for evidence of **infection or labor**

MATERNAL SURVEILLANCE

- All women with PPRM should be monitored for **signs of infection**
 - however, there is **no consensus as to the best approach**.
 - At a minimum, routine clinical parameters (eg, **maternal temperature, uterine tenderness and contractions, maternal and fetal heart rate**) should be monitored.

— FETAL SURVILIANCE

- Some type of fetal surveillance is generally employed (eg, kick counts, nonstress tests,

TIMING OF DELIVERY

- Gestational age <32 weeks
 - In general, prematurity is the greatest risk to the fetus with PPROM at less than 32 weeks of gestation.
 - Therefore, we manage pregnancies at this gestational age **expectantly** in the absence of complications (eg, infection, abruption, cord prolapse, unstable fetal presentation, nonreassuring fetal assessment).
 - As discussed above, we administer a course of
 - antenatal glucocorticoids

TIMING OF DELIVERY

- Gestational age between 32 and 34 weeks
 - For women admitted with PPRM at 32 to 34 weeks of gestation with fetal lung maturation confirmed via amniocentesis or collection of amniotic fluid from the vaginal pool, the risks associated with expectant management probably exceed those of delivery
 - We believe such women are **best managed by delivery** as long as they are at a center capable of caring for a preterm infant.
 - If fetal lung maturity cannot be confirmed, we **administer**

TIMING OF DELIVERY

- Gestational age greater than 34 weeks
 - Women admitted with PPROM at ≥ 34 weeks of gestation are **delivered**.
 - The risks associated with prematurity are small at this gestational age compared to potential complications that may arise during expectant management

PROM

- There are a variety of options for management of PROM depending upon
 - the gestational age at occurrence
 - the patient's clinical condition.
- The initial evaluation of all pregnancies in which PROM is suspected should include
 - confirmation of membrane rupture
 - confirmation of gestational age
 - assessment of fetal well-being.

- The diagnosis of PROM is based upon a characteristic history (ie, leaking fluid per vagina), physical examination (ie, visualization of fluid flowing from the cervical os), supplemented by laboratory tests in cases of diagnostic uncertainty.
 - The clinical manifestations and diagnosis of PROM are the **same across gestation**

- Gestational age is determined according to the usual parameters.
- Fetal well-being is generally assessed via an external fetal heart rate monitor.

DELIVERY VS EXPECTANT MANAGEMENT

- **Prompt delivery** for women with term or near term PROM.
 - Labor is induced, unless there are contraindications to labor or vaginal delivery, in which case cesarean delivery is performed.
- **Rationale**
 - Most women with term PROM who are followed expectantly will go into spontaneous labor and deliver **within 24, 48, and 72 hours of PROM in 70, 85, and 95 percent of women**, respectively

PTL

Definition

- Preterm labour is the presence of regular uterine contractions associated with dilatation and/or effacement of the cervix prior to 37 weeks gestation.

Diagnosis

- Labour is diagnosed on the basis of regular contractions which are associated with effacement and/or dilatation of the cervix.
- The same diagnostic criteria apply to preterm labour.

Management

Objectives

- The evidence available shows that the aims of treatment of pre-term labour should be to establish effective suppression of labour with tocolytic therapy (unless contraindicated) prior to 34 weeks gestation without undue delay in order to postpone delivery of the fetus for at least 48 hours whilst steroids are given. There is no evidence-base for tocolytic therapy being employed for longer than 48 hours or at a gestation greater than 34 weeks.

Management Details

IF MEMBRANE INTACT

ADMISSION AND INVESTIGATION

- On admission, thorough assessment of the patient should include:

- **History** - particularly relating to rupture of the membranes and antepartum haemorrhage.
- **Gestational age** should be confirmed by the best available information regarding menstrual history and any available previous ultrasound data.
- **Examination** - noting particularly temperature, uterine tone and tenderness, liquor volume and fetal size and presentation.
- **Vaginal examination** - a speculum examination should be performed with full aseptic technique, not touching the cervix with the speculum, and cervical swabs taken for bacteriological

- Urine microbiology - if a mid-stream urine is unsatisfactory a catheter specimen of urine should be obtained for microscopy and culture.
- • Ultrasound - this may assist with assessment of presentation, gestation, fetal weight, amniotic fluid volume and fetal normality.

- **Cardiotocography (CTG) - should be performed to assess fetal wellbeing.**
- **Amniocentesis - this investigation may be appropriate to assess the presence or absence of intra-amniotic sepsis, or (rarely) to assess fetal lung maturity.**

TOCOLYTICS

- Nifedipine:
- Indomethacin
- Salbutamol

MATERNAL FEVER:

- Any maternal temperature of 37.2⁰C or more MUST lead to formal review of the patient and of the treatment plan.

Tocolytics

- Nifedipine:
- Indomethacin
- Salbutamol

IUGR

(Intrauterine Growth Restriction)

Introduction

- Terms used include small for gestational age (**SGA**), **IUGR**, and fetal growth restriction (**FGR**).
- Most often SGA refers to the infant, whereas IUGR to the fetus
- **Definition (IUGR):-**
 - No universal definition
 - Failure to achieve intrauterine growth potential
 - Usually defined as estimated fetal weight (EFW) at or below the **10th percentile (or <2SD)** for gestational age.
- 3 -10% of infants are growth restricted
- 25 -60 % of infants conventionally diagnosed to be SGA are simply **constitutionally small**
- Constitutionally small babies are at less risk of complications than those who are SGA from pathologic process.

Maternal risk factors

- Constitutionally small (Ht & Wt)
- Poor maternal weight gain & nutrition
- Extremes of age.
- Cardio-vascular disease: *preeclampsia, hypertension, cyanotic heart disease, cardiac disease Gr III & IV, diabetic vascular lesions.*
- Maternal anemia
- Anti phospholipid Ab syn.
- Previous Hx of IUGR.
- Substance abuse (tobacco, narcotics, alcohol).
- Social deprivation
- Multiple pregnancy.
- High altitude.
- Drugs like anticoagulants, anticonvulsants.
- Chronic kidney disease
- Chronic infection- *UTI, Malaria, TB, genital infections*

Fetal Risk Factors

- ***Constitutional*** – genetically small, but genetically normal
- ***Fetal infection*** - rubella, cytomegalovirus, herpes simplex, tuberculosis, syphilis, toxoplasmosis, TB, Malaria,
- ***A birth defect*** (cardiovascular, renal, anencephally, limb defect, etc).
- ***Chromosomal abnormalities*** - 21(Down's syndrome), trisomy-18 (Edwards' synd.), 16, 13, xo (turner's syndrome).
- Chondro-dystrophies: a primary disorder of ***bone/cartilage***.
- ***Ectopic*** pregnancy (abdominal).
- ***Multiple fetus***
- ***Fetal sex:*** female are smaller at term by 5% (~150 g) and 2% (1~cm) shorter

Placental Factors

- ***Uteroplacental*** insufficiency resulting from -.
 - Improper / inadequate trophoblastic invasion and placentation in the first trimester.
- ***Foetoplacental*** insufficiency due to-.
 - Vascular anomalies of placenta and cord; (e.g. chorioangioma, twin-twin transfusion syndrome)
 - Decreased placental functioning mass-.
 - Small placenta,
 - Abruptio placenta & placenta previa.
 - Extensive infarcts,
 - Chorionic villitis,
 - Placental malformations (circumvallate placenta, battledore placenta)

Classification

Symmetrical (type -2)

- The baby's head and body are proportionately small.
- May occur when the foetus experiences a problem during early development.

Asymmetrical (type -1)

- Baby's brain is abnormally large when compared to the liver.
- May occur when the foetus experiences a problem during later development

- In a normal infant, the brain weighs about 3X more than the liver.
- In asymmetrical IUGR, the brain can weigh 5-6X more than the liver.

Classification & characteristics of IUGR

Symmetric

- Account for 20%
- From early pregnancy insult
- Constitutional or “normal” small
- Decreased growth potential
- Normal ponderal index
 $[(\text{birthweight (g)} / \text{length}^{[3]}) \times 100]$
- Lower transitional problems risk
- Brain symmetrical to body
- Examples
 - Genetic causes, chromosomal
 - TORCH infections
 - Anomalad Syndromes

Asymmetric

- Account for 80%
- Late pregnancy insult
- Environmental
- Growth arrest
- A low ponderal index
- Higher risk for transitional problems
- Brain sparing
- Examples
 - Chronic hypoxia
 - Preeclampsia (PIH, PET)
 - Chronic hypertension
 - Malnutrition

Short Term Risks of IUGR

- Increased **perinatal morbidity and mortality.**
 - Perinatal mortality = **5-20x** that of AGA
 - Infants at greater risk of dying b/c of neonatal complications
 - *Asphyxia, acidosis, meconium aspiration syndrome, infection, hypoglycemia, hypothermia, sudden infant death syndrome.*
- Intrapartum foetal acidosis may occur in as many as 40 % of IUGR, leading to a high incidence of LSCS.
- Susceptible to infections because of **impaired immunity**
- Hyperviscosity - **polycythemia** syndrome
- **Thrombocytopenia**
- Pulmonary hemorrhage
- Hypocalcemia

Long term Prognosis

- Increased risk for ***death, low blood sugar, low body temp., and abnormal development*** of the nervous system (eg. Cerebral palsy) . The risks increase with increasing severity.
- Infants with asymmetrical IUGR are more likely to catch up in growth after birth.
- If IUGR is related to a disease or a genetic defect, the future of the infant is related to the severity and the nature of that disorder.
- Impaired ***immuno-competence***.
- Predisposed to ***adult adult-onset, degenerative diseases*** like maturity onset diabetes and cardiovascular diseases.

Diagnosis

- Can be difficult to diagnose until the baby is born.
- **Clinical:**
 - Inadequate growth detected by serial measurement of *maternal Wt., abdominal girth and fundal Ht.*
- **Biophysical:**
 - Ultrasound to evaluate the foetal growth.
 - AC,
 - Inadequate foetal growth,
 - Reduced AFI, placental calcification,
 - HC:AC, FL:AC

Prevention

- Because many causes are nonpreventable, few interventions have proved effective for prevention.
- Interventions that have shown benefit include:-
 - Smoking cessation,
 - Antimalarial chemoprophylaxis, and
 - Balanced protein and energy supplementation.
 - Treatment of anaemia,
 - Prevention and treatment of
 - Hypertensive disorders,
 - Foetal compromise
 - Infection.

Treatment

- Once IUGR is suspected , intensive effort should be made to determine if GR is present and if so, its type and etiology.
- Most are constitutional and need only follow-up.
- Early diagnosis and treatment of the underlying problem may reduce the chance of serious outcome.
- The treatment consists of either:-
 - ***Delivery***; or
 - ***Remaining in utero*** and improving blood flow to the uterus.
 - Maternal bed rest
 - Fetal surveillance
- Other forms of treatment that have been studied are nutritional supplementation, zinc supplementation, fish oil, hormones and oxygen therapy.

Treatment; contd...

- **Timing of delivery:**

- Proper timing of delivery is critical.
- In the presence of significant oligohydramnios most fetus will be delivered if G.A has reached >34 wk.
- Often tolerate labor less than AGA and C/S is indicated for intrapartum fetal compromise.
- Uncertainty about the diagnosis of GR should preclude intervention until fetal lung maturity is assured.
- IUGR babies who are at or near term have the best outcome if delivered promptly.

- **Labour and delivery:**

- Close monitoring
- Prevention of hypoglycemia, hypothermia,...

Judge Optimum Time Of Delivery

Immediate delivery

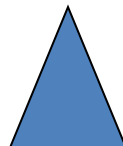


- Risk of prematurity
- Difficult extra uterine existence

Delaying delivery



- Risk of IUFD
- Hostile intra uterine environment



IUFD(intrauterine fetal death)

Definition:

- Only deaths occurring in utero in which the fetus or neonate weighs 500 gm or more (WHO)
- Only deaths occurring in utero in which the fetus or neonate weighs 500 gm or more and/or deaths occurring at 22 weeks of gestation or greater (ACOG)
- Only deaths occurring in utero in which the fetus or neonate weighs 1000 gm or more and/ or deaths occurring at 28 weeks of gestation or greater (Ethiopia)

Introduction

- Not all conceptions result in a live born infant
- Of the clinically recognized pregnancies, 10-15 % are lost.
- Almost 1% of women entering the 2nd half of pregnancy will suffer the loss of their baby
- Almost 80% of still births occur before term and
- More than half occur before 28 weeks.
- Still births are much more common with decreasing gestational age

Magnitude

- World wide 3 million still births occur yearly
- In USA, 6.8/1000 live births

Etiology:

- Fetal- 25-40%
- Placental- 25-35%
- Maternal- 5-10%
- Unexplained-25-35%

Fetal causes

- Chromosomal anomalies
- Non chromosomal birth defects
- Non- immune hydrops
- Infections-viruses, bacteria, protozoa

Placental causes

- Abruption
- Feto-maternal hemorrhage
- Cord accident.
- Placental insufficiency
- Intrapartum asphyxia
- Placenta previa
- Twin-to –twin transfusion
- Chorioamnionitis
- PROM

Maternal causes

- **Anti phospholipid antibodies**
- **Diabetes**
- **Hypertension disorders**
- **Trauma**
- **Abnormal labor**
- **Sepsis**
- **Acidosis**
- **Hypoxia**
- **Uterine rupture**
- **Post term pregnancy**
- **Drugs**
- **Rh disease**

Pathology

- Maceration- blistering & peeling of skin occurs between 12-24 hours after death.
- Initial pathology after death is fetus swells and appear dusky red.
- Maceration is a result of aseptic autolysis affecting different structures

Diagnosis

- Repeated examination is needed to confirm the diagnosis usually in the absence of ultrasound

Signs & symptoms

- Absent fetal movement
- Pregnancy symptoms absent or diminishing
- White milk expression during pregnancy
- Fundal height-same or decreased.
- Smaller uterus than expected

Diagnosis-cont'd

- Abdominal girth same or decreased.
- Gradual retrogression of the fundal height.
- Egg- shell cracking feel of the fetal head (late feature)
- Fetal movements not felt during palpation.
- Absent FHB-pinardes stethoscope

Diagnosis-cont'd

Laboratory:

- An abnormal blood level of HCG.
- Urine pregnancy test could be positive or negative.

X-Ray

- Spalding's sign- the irregular overlapping of the cranial bones on one another and the rolled up appearance of the fetal trunk. Occurs 7 days after fetal death

X-ray- cont'd

- Robert's sign- the appearance of gas bubbles in the thoracic cavity of the fetus within the heart chambers or great vessels. Occurs 12 hours after death
- Kehrer's sign- hyper flexion of the spine.

Ultrasound:

- Absent cardiac activity.
- Absent fetal movement.
- Oligohydramnios and collapsed cranial bones

Complications

1. Psychological upset or stress.
2. Infection.
3. Blood coagulation disorders.
4. During labor-uterine inertia, retained placenta, PPH
5. Maternal death

Work up

- **ABO and Rh grouping**
- **VDRL**
- **Post prandial blood sugar (FBS) level**
- **Thyroid profile**
- **TORCH screening**
- **Lupus anticoagulant and anticardiolipin Abs**
- **Complete blood count**
- **Urine toxicology screen**
- **Indirect coomb's (anti body screen)**
- **Prothrombin time (PT)**
- **Partial thromboplastin time (PTT)**

Work up-cont'd

- **U/A**
- **Platelet count**
- **Fibrinogen level.**
- **Cord or cardiac blood for**
 - **Culture and sensitivity**
 - **Estimation of Ig level**
 - **Coomb's if cord blood is available**
 - **Cytogenetic studies**
- **Post mortem examination**
- **Naked examination of placenta, cord & fetus**

Management

Principles:

- Confirm diagnosis by ultrasound
- Search for cause
- Determine fibrinogen level & PTT

Management could be expectant or interference

- **Expectant:** in 80% of cases spontaneous expulsion occurs in 2 weeks time.
- Follow with coagulation profile like PT, PTT, Fibrinogen & platelet count weekly

Methods of termination

- Oxytocin infusion
- Prostaglandins
- Catheter methods
- C/S or hysterectomy
- Destructive deliveries

NB: bereavement management by managing staff is important.

Prevention

- Total prevention is not possible but include:
 - Preconceptual care
 - Regular antenatal care
 - Screen at risk mothers

Prolonged Pregnancy

Introduction

- **Definition**
 - **Pregnancy that advances beyond 42 completed weeks from the LNMP or 40 weeks' gestation from the time of conception.**
- **Incidence**
 - **Range from 6% to 12% of all pregnancies.**
- **Perinatal mortality is two to three times higher as compared to term gestations.**
 - **20% to 30% of postterm pregnancies develop a dysmaturity syndrome**
- **Faulty recall of the dates of menstruation is common reason of prolonged pregnancy.**

- **Evaluation**

- **To make the diagnosis - confirm the gestational age by**

- Clinical parameters (eg, LMP, quickening, detection of fetal heart tones).
 - Failure to recall & Delayed ovulation are the problems
 - Most pregnancies that are reliably 42 completed weeks beyond the last menses probably are not biologically prolonged.
 - Conversely, a few that are not yet 42 weeks might be postterm.
 - Because there is no method to identify pregnancies that are truly prolonged, all pregnancies judged to be 42 completed weeks should be managed as if abnormally prolonged.

– Early pregnancy tests and ultrasound examinations,

- Ultrasound is most useful when performed before the 20th week of gestation**
- First trimester (accuracy \pm 1wk). Gestational sac, crown rump length.**
- Second trimester (accuracy \pm 10-14 days). Femur length, Biparietal diameter.**
- Third trimester (accuracy \pm 2-3 wks), Biparietal diameter, femur length.**

– The exact time of conception

– Incidence varies from 7.5 % to 2.6 to 1.1 percent

Risk factors

- The majority of postterm pregnancies have no known cause
- Maternal demographic factors
 - Parity (Primigravidity),
 - Prior postterm birth,
 - Socioeconomic class, and
 - Male fetal gender
 - Maternal obesity
 - Age
- **Fetal–placental factors**
 - Anencephaly,
 - Adrenal hypoplasia, and
 - X-linked placental sulfatase deficiency

Complications

- It is a high risk pregnancy associated with fetal and maternal complications
- Increases perinatal mortality
 - All components of perinatal mortality— antepartum, intrapartum, and neonatal deaths— were increased at 42 weeks and beyond.
 - The most significant increases occurred intrapartum.
 - It is due to cord compression secondary to the reduced amniotic fluid volume and viscous meconium which are results of placental dysfunction

- **Postmaturity Syndrome**

- **20%-30% postmature infants present a unique and characteristic appearance.**
- Features include wrinkled, patchy, peeling skin; a long, thin body suggesting wasting; and advanced maturity because the infant is open-eyed, unusually alert, and appears old and worried-looking.
- Skin wrinkling can be particularly prominent on the palms and soles.
- The nails are typically quite long.
- **Most such postmature infants are not growth restricted because their birthweight seldom falls below 4000 g.**

- **Long term sequel**
 - **In the absence of perinatal asphyxia, growth restriction, or meconium aspiration, prolonged pregnancy is associated with normal, long-term neonatal developmental outcome.**

- **Management**

- **General**

- Confirm the gestational age
- Review the prenatal case document
- Do physical examination to
 - Estimate fetal size; ascertain viability and fetal well being.
 - Assess the adequacy of the pelvis, and favorability of the cervix using Bishop score

- **Specific**

- Induce at
 - 42 completed weeks in all cases with favorable cervix.
 - 42 completed weeks by ripening the cervix if unfavorable.

- **Antenatal Surveillance Testing Methods**
 - **Ante partum surveillance generally begins at 41 weeks, because the perinatal morbidity and mortality begin to rise before 42 weeks of amenorrhea.**
 - **Kick count**
 - **Biophysical Profile (BPP)**
 - **Non stress test: done twice weekly: If non reactive, do Oxytocin challenge test (OCT/CST).**
 - **Oxytocin challenge test weekly. Terminate pregnancy if test is positive**

- **INTRAPARTUM**

- Patients are scheduled for induction from outpatient unless otherwise induction on emergency ground or admission for other obstetrical risk factors is indicated
- Continuous electronic fetal monitoring must be employed during the induction of labor if possible otherwise intermittent auscultation every 15 minutes in relations to contraction in the first stage of labour
- The patient should be encouraged to lie on her left side.

- **Cesarean section is indicated for fetal distress.**
- It should not be delayed because of the decreased capacity of the postterm fetus to tolerate asphyxia and the increased risk of meconium aspiration.
- **Second stage of labor:** anticipate the following maternal and fetal complications.
 - **Maternal:** Shoulder dystocia, PPH, Genital (birth canal) trauma,
 - **Fetal:** Fetal distress, Meconium aspiration syndrome.
- **If meconium is present, neonatal asphyxia should be anticipated, and a neonatal resuscitative team should be present at**

Multiple Pregnancy



Definition



- Presence in Utero or birth of more than one fetuses.



- According to their number, they could be categorized into:
 - twins (most common),
 - triplet,
 - quadriplet, ...etc.
- **Incidence:**
 - According to Hellin's law [$1:80^{(n-1)}$, n = no. of gestation]
 - the incidence of twins is 1:80,
 - for triplet $1:(80)^2$, for quadriplet $1:(80)^3$, and so on.
 - This rule applies for spontaneously occurring multiple pregnancies and not for those induced by ovulatory stimulating drugs.

INCIDENCE



- Highest in Nigeria
- Lowest in Japan or east Asia
- Spontaneous ovulation twins 1%
- Clomiphene induced ovulation 10 %
- HMG (gonadotrophins) induced 30%

1. BY ZYGOSITY(number of ovum)



- **Monozygotic**

- Fertilization of a single ovum,
- Similar sex.
- Identical in every way including the HLA genes
- Not genetically determined
- Constant in all races; its prevalence: 1/25. ,incidence is 4-5 per 1000LB

- **Dizygotic**

- Fertilization of 2 separate ova
- Its etiology and prevalence varies, with racial / hereditary difference,
- Its actual prevalence is increasing due to:
 - Early diagnosis by U/S.
 - Induction of ovulation
 - Change of the ages of women experiencing their first pregnancy and delivery (> 35 years age).



2. BY CHORIONICITY(Number of placenta):-

Monochorionic(shared placenta),
Dichorionic(Two placentae) it could be either
Mono or Dizygotic, Placenta fused or separate,
and septum has four layers.

- **3. BY AMNIOCIITY:- Number of amniotic sacs.**

One sac- Monoamnionic(Monozygotic)

Two Sacs- Diamnionic.

Ctd..



- MZ twins (30%) a single fertilized ovum splits into two distinct individual after a variable number of division
- Timing of egg division Determines placentation in twins
- DC DA placentation occurs with division prior to the morula stage (within 3 days postfertilization)
- MC DA placentation occur with division b/n days 4 and 8 post fertilization
- MC MA placentation occurs with division b/n days 8 and 12 post fertilization
- Division at or after day 13 results in conjoined twins

Monozygotic Twins...

Different Scenarios of Cleavage



Scenario 1

Monozygotic twin pregnancy
Bi-chorial and bi-amniotic

**If the separation takes place just after the first cellular division [1st 3 days]
both of the twins will have their own placenta and an amniotic sac each.**



Scenario 2
Monozygotic twin pregnancy
Mono-chorial and bi-amniotic.

**Separation can also take place a little later in the development [4-8 days]
of the embryonic cells but before the blastocyte has defined the roles of each cell.**

Twins will be in the same placenta, but they will have 2 amniotic sacs.



Scenario 3
Monozygotic twin pregnancy
Mono-chorial & mono-amniotic

**Separation takes place at the stage when the amniotic bag is already being formed
[day 8-14]**

Twins will be in the same placenta, and in the same amniotic sac.

Conjoined Twins

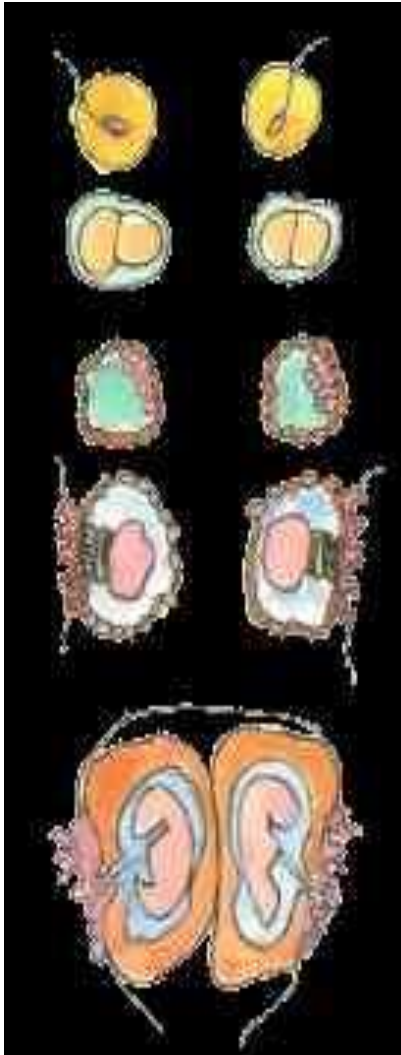


- If the division occurred just after embryonic disc formation, incomplete or conjoined twins will occur.

They may be joined

- anteriorly [thoracopagus-commonest],
- posteriorly [pygopagus]
- cephalad [craniopagus] or
- caudal [ischiopagus].





**Dizygotic twin pregnancy
Bi-chorial and bi-amniotic.**

**Dizygotic twins, are descended from a double ovulation and a double fertilization.
The 2 eggs are completely independent.
This configuration represents two thirds of all twin pregnancies.**



Etiology



1. Elevation of FSH level
2. Ovarian stimulation- clomiphene citrate increased twin rate by 5-50 %.
3. In vitro fertilization-
 - ✓ based on the no of embryo transferred.
 - ✓ the in vitro environmental exposure increases the chance of division & resulting monozygotic twins

Risk factors



1. Race & geographic area
2. Hereditary: tendency to release multiple ova is inherited.
3. Maternal age & parity

Age- rate of natural twinning is 0 at **puberty**
& peaks around age of **37**

Parity- 1st pregnancy – 1.3%

4th pregnancy-2.7%

Risk factor cotd...



4.Nutritional factors-

Maternal BMI $>30 \text{ k.g/m}^2$ & height $>164\text{cm}$ is associated with high twining rate(25-30%greater)

Fetal / Neonatal Complications



- Increased abortion rate:
- Increased intra-uterine fetal death (IUFD) (Unexplained in 1/3rd of the cases, associated structural & placental disorders in remaining)
 - More in MZ > DZ.(**3 to 4 fold increase**)
 - Vanishing twin syndrome: (incidence rate 21%)
 - Early death = Fetus compressus (papyraceous fetus).
 - Later death = macerated fetus.
 - Death during delivery:
 - second fetus: [cord prolapse, due to excess sedation, premature separation of placenta, constriction ring ,dystocia, operative manipulation, hypoxia].

Single Fetal Demise...??



- First trimester Fetal loss of a twin
 - does not appear to impair the development of the surviving twin.
- Midgestation fetal death occurring after (17 weeks' gestation)
 - Increase the risk of IUGR, preterm labor, preeclampsia and perinatal mortality (17-50% in MC and if TTT)
 - Antenatal necrosis(Ischemic) of the cerebral white matter has been associated with the presence of intrauterine fetal death of a co-twin , artery-to-artery, and vein-to-vein anastomosis.(as high as 20% risk in surviving co-twin)
 - Prompt delivery following the death of a co-twin has not been shown to prevent neurological injury
 - Delivery for the purpose of preventing injury should, therefore, be weighed against the risks of premature delivery.

- Increased perinatal mortality (10-20%):
 - More in monozygotic twins.
 - It is mainly related to low birth weight.
 - It may be due to
 - preterm delivery
 - IUGR with PIH(*Pregnancy Induced HTN*)
 - hypoxia (placental or cord accident)
 - operative manipulation: Birth trauma and CP
 - congenital malformation.
- Increased low body weight:
 - Neonates are lighter [due to preterm or IUGR],
 - More in monozygotic and with increased fetal number



Intrauterine growth Retardation:



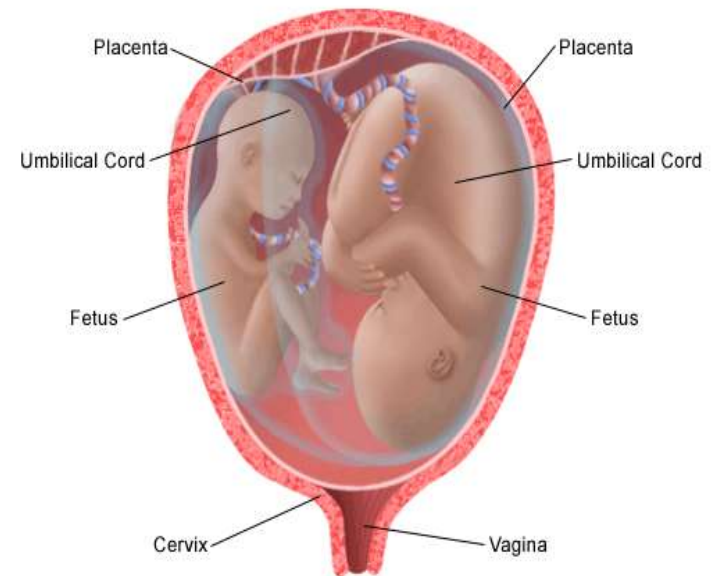
- Birth weights of twins, triplets, etc. are smaller than weights of corresponding singletons.
- Most of the deficit of birth weight occurs in the final 8-11 weeks of pregnancy.
- Due to unmet nutritional needs or due to other placental complications.

Twin to Twin transfusion

(10% of monozygotic twins),



- Vascular communication between 2 fetuses, mainly in monochorionic ;MA or DA placenta
- Twins are often of different sizes:
 - **Donor twin** = small, pallid, dehydrated (IUGR), oligohydramnios (due to oliguria), die from anemic heart failure.
 - **Recipient twin** = plethoric, edematous, hypertensive, ascites, kernicterus (need amniocentesis for bilirubin), enlarged liver, polyhydramnios (due to polyuria), die from congestive heart failure, and jaundice



Hemoglobin concentrations differ by >5g/dL.





- Discordant growth-affects about 15% of twins(due to different genetic potential ,unequal sharing of the placental mass)
 - Is expressed as percentage of the larger twins weight($WL-WS/WL \times 100$)
 - Discordance >20% is abnormal.
 - Risks associated are
 - » low birth weight
 - » increased rate of NICU admission & oxygen requirement
 - » hyperbilirubinemia

Malformations

- Major malformations develop in 2% of twins
 - From twining itself
 - -conjoined twins,
 - cardiac anomaly
 - From vascular interchange(TTTS)
 - Reverse flow with acardia in one twin
 - From crowding-club foot,
 - congenital hip dislocation

Conjoined twins



- Incomplete late division of monozygotic twins produces conjoined twins. Conjoined twins are connected at identical points .
 - o Thoracopagus - Joined at chest (40%)
 - o Xiphopagus/omphalopagus - Joined at abdomen (34%)
 - o Pygopagus - Joined at buttocks (18%)
 - o Ischiopagus - Joined at ischium (6%)
 - o Craniopagus - Joined at head (2%)

Conjoined Twins



- If the division occurred just after embryonic disc formation, incomplete or conjoined twins will occur.

They may be joined

- anteriorly [thoracopagus-commonest],
- posteriorly [pyopagus]
- cephalad [craniopagus] or
- caudal [ischiopagus].



TWIN GESTATION TYPE :-

DC – DA, MC – DA, MC - MA, CONJOINED



Plac No.	2	1	1	1
Amni No.	2	2	1	1
Incidence.	1:300	1:400	1:3000	1:50,000
Time of Cleav.	0- 72 hrs	4 to 8days	9–12 days	> 12 days.
Zygoty.	Diz or Mo	Monozy	Monozy	Monozy
Rela to Ferti Rx.	Yes	No	No	No
Risk of TTTS.	X	Yes	Yes	NA
Cord Entangle	X	X	Yes	NA
Cong Anomaly	Low	Medium	High	NA
Vasa Previa	Low	High	High	NA
Placenta Previa	High	High	High	High
Pre term Delivery	High	High	High	High

Notes:- DC (Dichorionic), DA(Diamnionic), MC(Monochorionic),
MA(Monoamnionic),

DC- DA :- may be Dizygo if diff gender; Same gender Di or Monozygo

Maternal Complications



- Increased **maternal mortality**.
- Increased **pregnancy risks**:
 - Anemia (15%): due to iron deficiency or folic acid deficiency
 - Preeclampsia- eclampsia: 3x(fetal number & placental mass involved in the pathogenesis)
 - Glucose intolerance.
 - Threatened or actual abortion.
 - Polyhydramnios (12%)
acute: more in monozygotic than dizygotic twins.
Chronic: not related to type.

Maternal complications contd...

- Mechanical effects: with the uterus larger than period of amenorrhea; it may be associated with dyspnea, dyspepsia, pressure on ureter with increased UTI, supine hypotension syndrome, increased varicosities and lower limb edema.
- Premature rupture of membranes
- Antepartum hemorrhage: both abruption (due to PIH and folic acid deficiency) and placenta previa (due to large placenta).
- Psychological: problem of caring, prolonged rest and hospitalization.
- Malpresentation and malposition



- Increased **labor risks**:

- Preterm labor (50%): which may be spontaneous or induced Uterine dystocia.
- Abnormal fetal presentation.
- Twins entanglement and locked twins
- Vasa Praevia (due to vilamentous insertion of the cord).
- Postpartum Hemorrhage
- Puerperal Sepsis

Diagnosis



- 25% of antenatal diagnosis of twin is missed .
- Twin should be suspected by history and examination
- It should be confirmed by U/S (as early as 10 wks).
- To decrease PNM, it should be early diagnosed, properly assessed antenatally and properly managed intranatally.

History...



- Patient profile:
 - Etiological factors; with positive past history and family history specially maternal.
- Early pregnancy: Hyperemesis, bleeding.
- Mid-pregnancy:
 - Greater weight gain than expected,
 - abdominal size > period of amenorrhea,
 - early PIH symptoms, persistent fetal activity.
- Late pregnancy:
 - Pressure symptoms (dyspnea, dyspepsia, UTI, piles, edema and varicose veins in LL).

Examination



- **General:**
 - An early increase weight gain,
 - Pallor
 - Less mid-trimesteric fall blood pressure
 - Early PIH
 - Early edema, and varicose veins in LL.
- **Abdominal:**
 - Fundal level > amenorrhea especially in mid-pregnancy
 - exclude other causes.
 - Palpation: Multiple fetal parts – 3 poles, 2 heads, small head in relation to uterine size, fetal movement all over abdomen.
 - identify presentations.
 - Auscultation of FHS:
 - 2 different recordings by 2 observers and a difference > 10 bpm a Gallop between 2 points[Arnoux sign]
 - ECG.
- **Pelvic:** Specially during the course of labor
 - small presenting part compared to abdominal size

POSITIVE SIGNS



- Palpation of multiple fetal parts.
 - Two FHS at same time by two diff. observer with difference of 10 beats /mt in rate.
 - U S G in early or late preg, more than one sac or fetus. Or X-ray in late preg. Rule out :- Molar preg, Hydramnious, and Tumor
- Biochemical parameters:-
 - Beta H C G Raised but not more than 100,000miu/ml
 - MSAFP Twice high level than singleton preg.

-

Ultrasonography

- Confirm fetal number [2 sacs or 2 fetal heads in perpendicular planes].
- Two gestation sacs each containing a yolk sac (≥ 5.5 weeks)



Diagnosis contd....

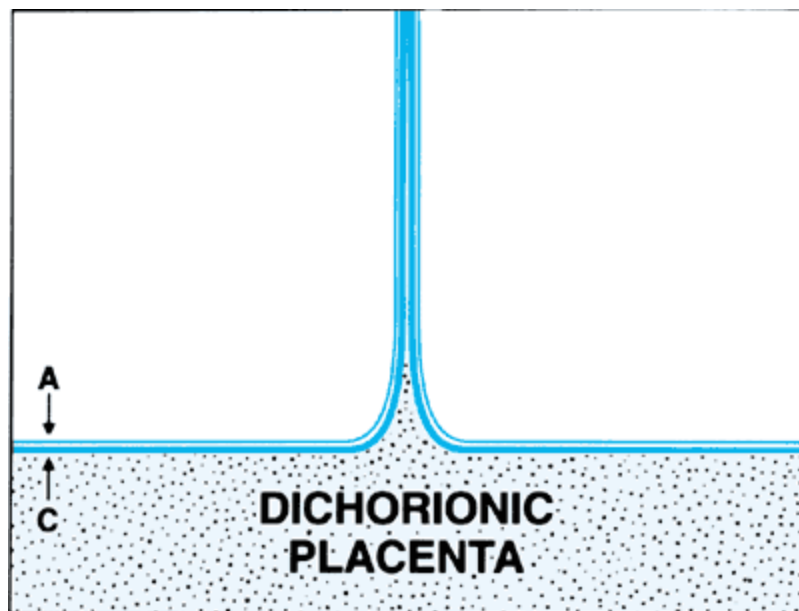


- Two fetus with heart beats(≥ 6 weeks)
- Diagnosis of vanishing twin syndrome
 - . disparities of gestational sack diameter or CRL by $>3\text{mm}$,
 - .disappearance of one gestational sack.

Diagnosis contd...



- Diagnose type:
 - Mono- vs. dizygotic twins.
 - In all dizygotic and in 1/3 of monozygotic twins, the dividing membrane between two sacs in twins comprises a double layer of chorion and amnion from each sac (dichorionic - diamniotic), separated by a triangle-like tongue of decidua extending from the fetal surface of the placenta.
 - This is known as twin peak (or Lambda sign) which is pathognomonic for dichorionic placentation



B

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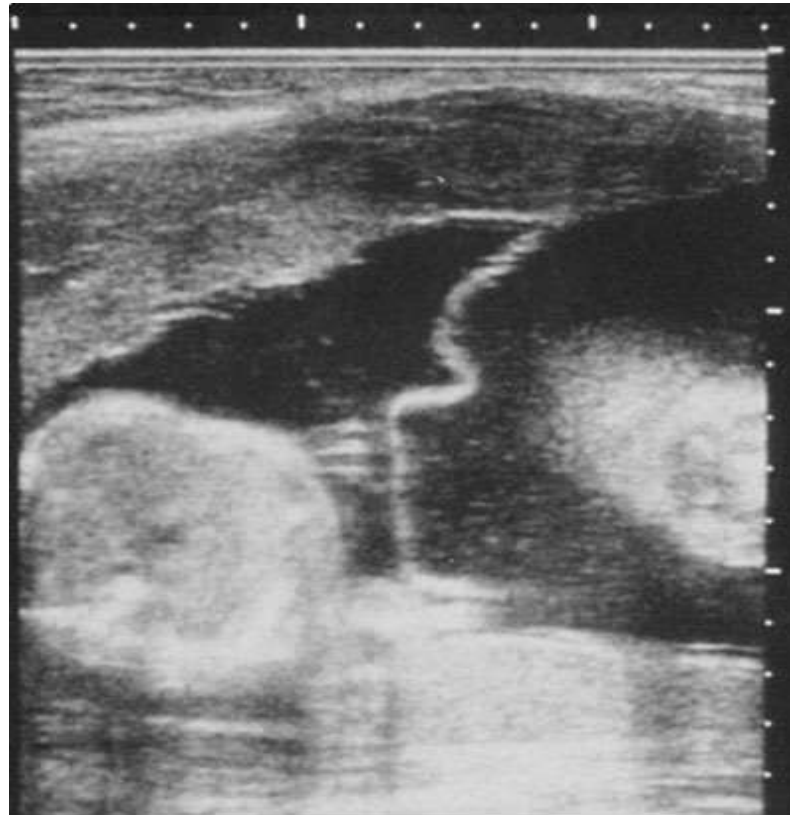
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Diagnosis contd...



- In monozygotic pregnancy, the dividing thin membrane of the two sacs (made of 2 layers of amnion only) is inserted perpendicular to the fetal surface of the placenta.
- This is known as the **Tau sign**.

The tau sign ,US finding i monochorionic twin.



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Diagnosis contd...



- **Conjoined Twin :-**

- Multiple fetus but single heart.
- Longitudinal lie but not engaging.
- Similar parallel lie (Vx- Vx , Breech- Breech).
- An abnormal fetal attitude
- Diagnosis : U S , X ray plane film, Amniography

MANAGEMENT :-



- **ANTEPARTUM :-** Care as high risk pregnancy.

- **Nutritional counseling.**

Normal weight (BMI **18.5 to 24.9 kg/m²**)-

weight gain **16.8 to 24.5 kg** decreased risk of preterm birth and higher birth weights

- Iron & Folic acid supplementation

supplementation-**60-100mg**/day of Fe & 1mg of
acid is recommended

folic

Antipartum management contd



- More ANC Visits

- Extra rest & Early work leave
- Counsel on danger signs of high risk preg.
- Monitor for PIH / PE. Glucose intolerance
- Counsel about headache, vision disturbance and epigastric pain.
- Clinical / US Fetal monitoring for wellbeing.
- More frequent BPP/NST when indicated.
- Fetal mov counts. Not reliable
- Preterm labor: tocolytic agents & steroid inj. <34wks indicated. No VBAC.

Antepartum approach with respect to the complications



Selective Reduction /Selective Termination

- The presence of > 3 fetuses carries the risk of losing them all (preterm delivery).
- The number is reduced to twins only by injecting potassium chloride intracardiac under U/S guidance (about 1.5 ml of 15% solution).
 - *Potassium chloride may diffuse and affect other fetuses.*



TTTS - Amniotic septostomy

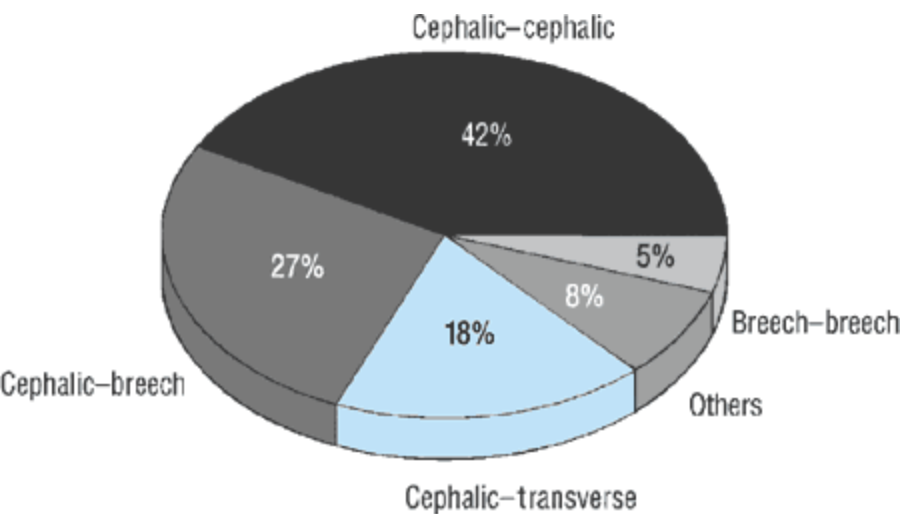
- serial reduction amniocentesis
- laser ablation of the anastomoses
(using endoscopic laser)

INTRAPARTUM CARE

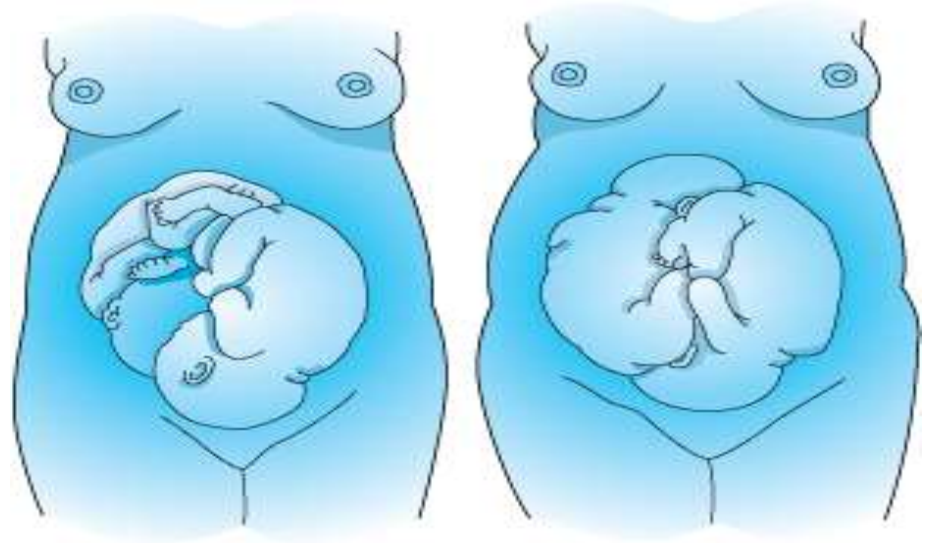


- **Following arrangements to be made :-**
 - **To have arrangements in health set up.**
 - Obstetric Perinatology care**
 - Obstetric anesthesia services &**
 - Neonatology care**
- **Labor & Delivery : Confirm fetal numbers and presentations, fetal weight, and placental loc**
- **Arrange minimum 2 units of blood**
- **Electri fetal monitoring during labor if available**
- **Secure I V line with crystalloid.**

possible combinations of fetal positions



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Route of Delivery



vaginal-

- If both Twins are by Vertex: Vaginal delivery.
- Twin A-Vertex / Twin B Non vertex :-
 - Twin a vaginal
 - Twin B Options are :
 - ECV, Assisted breech extraction, Total breech extraction,
 - Internal Podalic version & breech ext..

cesarean delivery:

- conjoint twin, triplets and so on
- Monoamniotic twins .
- If first twin not cephalic and twins are viable :-

C.S. for Multiple Pregnancy:



Indications of C.S.

More than 2 viable fetuses, if:

- weight < 2 kg,
 - discordant growth (i.e.; IUGR or twin-twin transfusion, or disproportionate twins, twin B larger than A (BPD > 2 mm),
 - twin A: is non-vertex.
 - Conjoined Twins
- Single amniotic cavity (as diagnosed by U/S or amniogram).
 - Previous Uterine scar.
 - During Labor: if delayed progress, fetal distress, or if twin B transverse position and cervix is thickened (retained second twin).
 - Associated pregnancy complication i.e.; severe PIH, placenta previa.
 - Contracted Pelvis
 - Lack of expertise in internal podalic version

• **AFTER DELIVERY OF FIRST TWIN :-**

- Cut cord as far out side vagina as possible & clamp.
- Hand over delivered fetus to assistant.
- Confirm lie and presentation of 2nd twin, look for cord.
- Uterus contracting and presenting part at inlet-
Do ARM on second fore bag. If uterine inertia sets in start Oxytocin drip following amniotomy and deliver fetus.
- If fetal compromise detected and vaginal delivery not eminent then emergency CS .
- Delivery interval between two should not be **>30min.**
- Active management of third stage.
- Placenta is delivered after 2nd twin.
- Examine placenta in detail for Zygoty determination.
Monozygotic – dividing membrane **2 layers**
Dizygotic – septa has **five layer**, 2amnion,2chorion,decidua in between.



Retained Twin B



- The usual time interval between delivery of twin A and B is 15-20 minutes and should not be more than 30 minute.
- If there are facilities for proper monitoring this interval may be increased
- Indications of CS for Twin B
 - Transverse lie
 - Fetal Distress
 - Contracted cervix
 - Prolapsed cord
 - Premature Breech
 - Failed Extraction

POST PARTUM CARE :-

- Oxytocin drip should start after delivery of placenta
Methyl ergometrin or inj. Prostaglandin should be available if needed for managing PPH.
- COMPLICATIONS :
 - PPH
 - Delayed delivery of 2nd twin.
 - Discordant twins : diff. of 20% or more in Fetal wt
of twins as % of wt of larger twin.
 - Twin to Twin Transfusion Syndrome:-
 - Placental vascular connection, Hgb diff >5gm/dl.
 - Birth wt diff >20% , Plethoric and IUGR Fetus,
.Two Vessel cord/ single umbilical artery
(7% especially monozygotic) is associated with congenital anomalies.



RH - ISOIMMUNIZATION

Introduction

- > 15 blood group systems are recognised :
 - *ABO, Rh, Kell, Duffy, MN, P, Lewis, Lutheran, Xg, Li, Yt, Dombrock, Colton, Public antigens & Private antigens.*
- Most of these blood group antigens have been found to be associated with hemolytic disease.
- However– ABO & Rh account for 98%
- RH blood group system was first demonstrated by testing human blood with rabbit anti sera against red cells of Rhesus monkey & classifying Rh negative & Rh positive
- Incidence of Rh negative varies in different races:
 - Mongoloids- **nil**, Chinese & Japanese- **1-2%**, Indians-5%, **Africans-5-8%**, Caucasians-**15-17%** & **Basques-30-35%**.

The Basics Of Blood

W.B.C. & Platelet

R.B.C.

Plasma

ANTIGEN

>400 Agglutinogens on the cell membrane

ANTIBODY

*Natural & Immune Agglutinins/
Isoantibodies*

Antigen-Antobody reaction on the
cell surface → **HEMOLYSIS**

The Basics Of Blood; contd...

- ***Natural antibodies*** are formed against most of the major group antigens & present in almost all individuals when the antigen is absent
 - Mostly of them are **IgM type**.
 - Usually do not **cross placenta**.
- ***Immune antibodies:-*** In contrast the immune or isoantibodies are IgG.
 - Best react at body temp. & **readily cross placenta**.
 - Most antibodies are complement binding

ABO System & Pregnancy

- Majorities of hemolytic diseases are due to **ABO incompatibility**
- There is a 20% chance of ABO incompatibility of mother & foetus
- Only 5% chance of developing hemolytic disease only in **type A & B** infants of **type O mothers**, that too only of milder forms
- Anti-A & B produced in the mother being natural are IgM molecules & so do not cross placenta.

Rhesus Blood Group System

- The genotype is determined by the inheritance of 3 pairs of closely linked allelic genes situated on chromosome 9 & named as D/d, C/c, E/e (Fisher- Race theory)
- The foetus inherits one gene from each group as a haplotype such as sets of Cde, cde etc from each parent
- 12 sets of combinations & 78 genotypes are possible. Most frequent genotypes are –
 - Cde/cde(33%), Cde/cDe(18%), Cde/cDE(12%) cDE/cde(11%), cde/cde(15%), cdE/cde(1%), Cde/cde(1%)

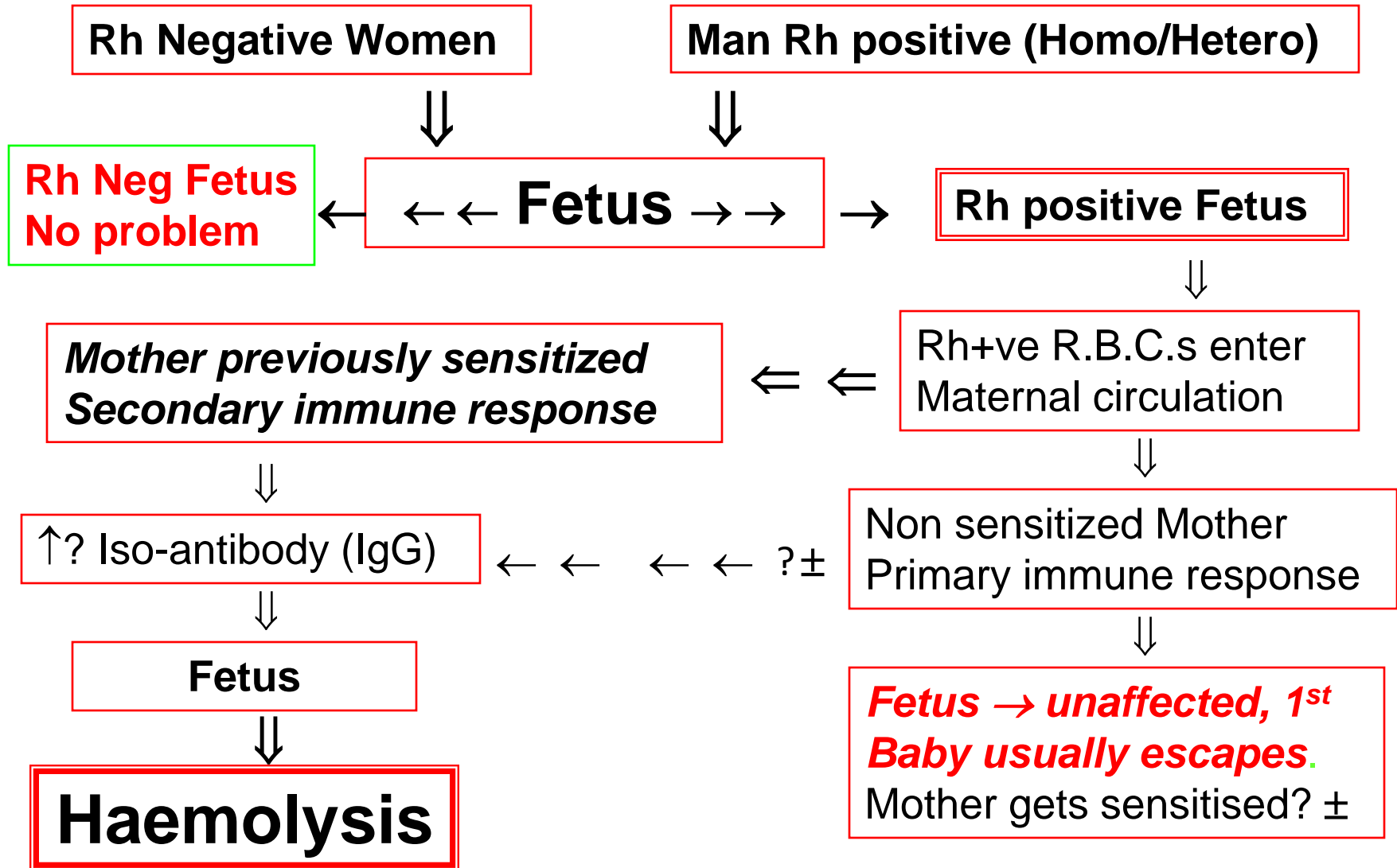
Rhesus Blood Group System; contd...

- The antigenic determinants form an intrinsic part of the red cell membrane protein structure
- **‘D’** is by far the most immunogenic in the Rh system excepting those that have the natural antibodies.
- There is a rare type of Rh negative called Rh null who lack all known Rh antigens.
- The precise function of Rh-antigen unknown; but probably in maintaining RBC membrane integrity.

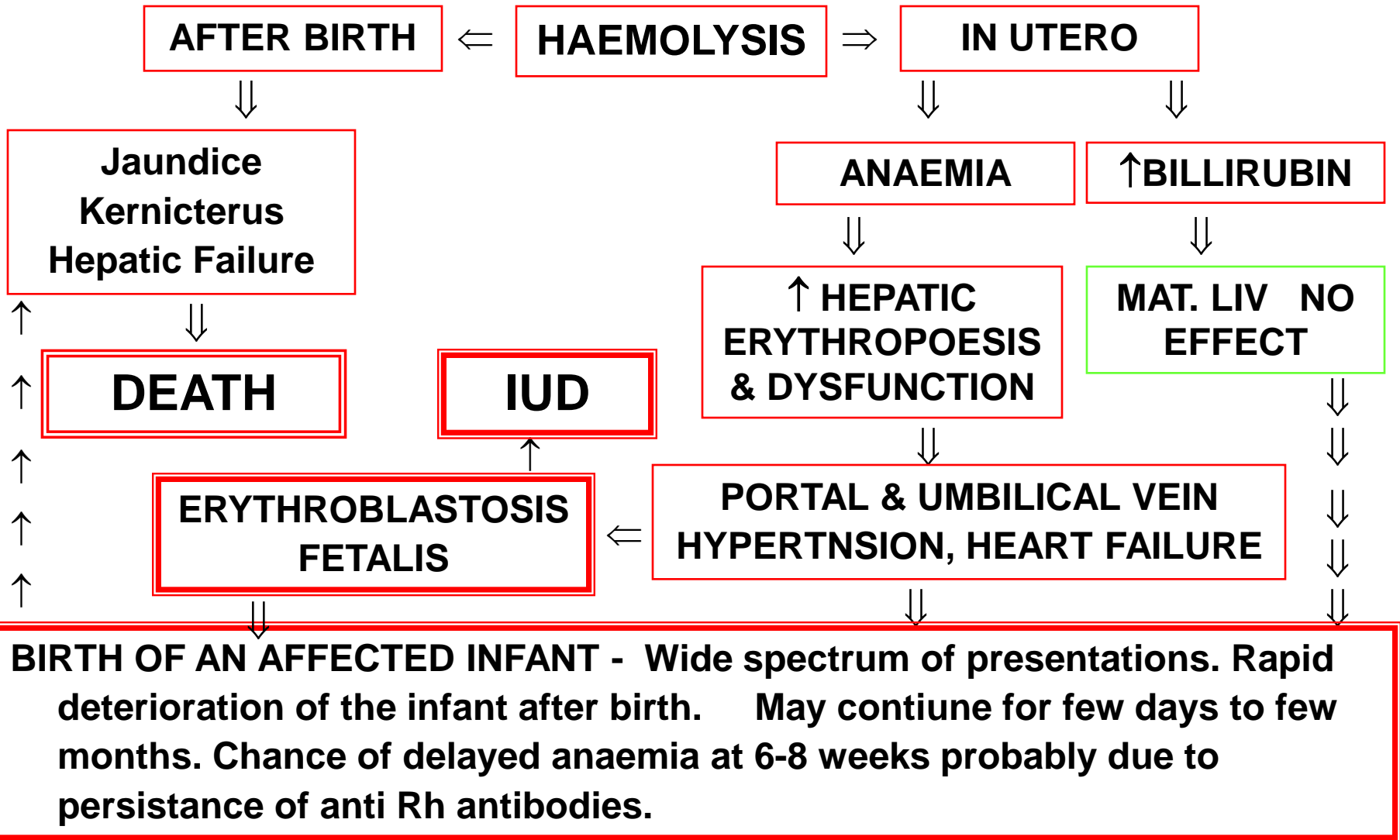
Pathogenesis Of Rh Iso-immunisation

- 3 preconditions for alloimmunization to occur:
 - *Rh-negative mother and Rh-positive fetus.*
 - *Crossing of sufficient number of fetal RBC.*
 - *Maternal immunogenic capacity.*
- Chances of T.P.H/F.M.H. are only **5% in 1st trimester** but **47% in 3rd trimester**, many conditions can increase the risk.
- Chances of primary sensitization during 1st pregnancy is only **1-2%**, but **10 to 15%** of patients may become sensitized after delivery
- ABO incompatibility and Rh non-responder (in as many as 30% Rh-negative mothers) status may protect.
- Amount of antibodies that enter the fetal circulation will determine the degree of haemolysis

Pathogenesis Of Rh Iso- immunisation



Pathology Of Iso-immunisation



Prevention of Rh Incompatibility

- Premarital counseling? Ambitious?
- Blood grouping must for every woman, before 1st pregnancy.
- Avoid Rh+ve Blood transfusion
- Proper management of unsensitised Rh negative pregnancies.

Management of Unsensitised Pregnancy

- Blood typing at 1st visit,
 - If negative → husband's typing.
 - If husband is also negative → no treatment
- If husband is positive, if possible, Homo/Hetero?
- Do Indirect Coomb's test of mother – and if negative repeat at 28 weeks:-
 - If negative, 300 g of Rh immunoglobulin (RhIgG) is given.
 - If Positive → **Sensitised**
- At birth- cord blood for ABO/Rh typing, direct coombs test
 - Baby Rh negative – Be happy
 - Baby Rh positive - 300mcg anti-D immunoglobulin be given within 72 hours after delivery (shown to be effective in preventing isoimmunization if given up to 28 days after delivery).

Management of Unsensitised Pregnancy

- In Abortion, Ectopic, CVS-
 - Pregnancy < 12 weeks- **50mcg Anti D**
 - Pregnancy >12 weeks- **300mcg Anti D**
- APH, IUD, Amniocentesis, Abdominal trauma, Foetal-maternal hemorrhage → **300mcg Anti D**
- If Rh positive- Test mother's blood for ICT & Infant's for DCT
- ?Prophylactic Anti D administration during antenatal period to all negative mothers at 28weeks and again at 34 / 36 weeks.

Management of Sensitized Pregnancy

- Causes of sensitization-
 - Misinterpretation of maternal Rh type
 - Rh +ve blood transfusion
 - Unprotected preg. & labour
 - Inadequate dose / improper use of IgG on previous occasions
 - Immunization to cross-reacting antigen
- Knowledge of maternal antibody titer to the specific antigen
- Sensitized = antibody titer of $\geq 1:4$
- Careful planning during antepartum, intrapartum & neonatal period
- Intrauterine foetal monitoring with repeated ultrasound examination, cordocentesis / amniocentesis
 - Fetus Rh Negative: - Observation

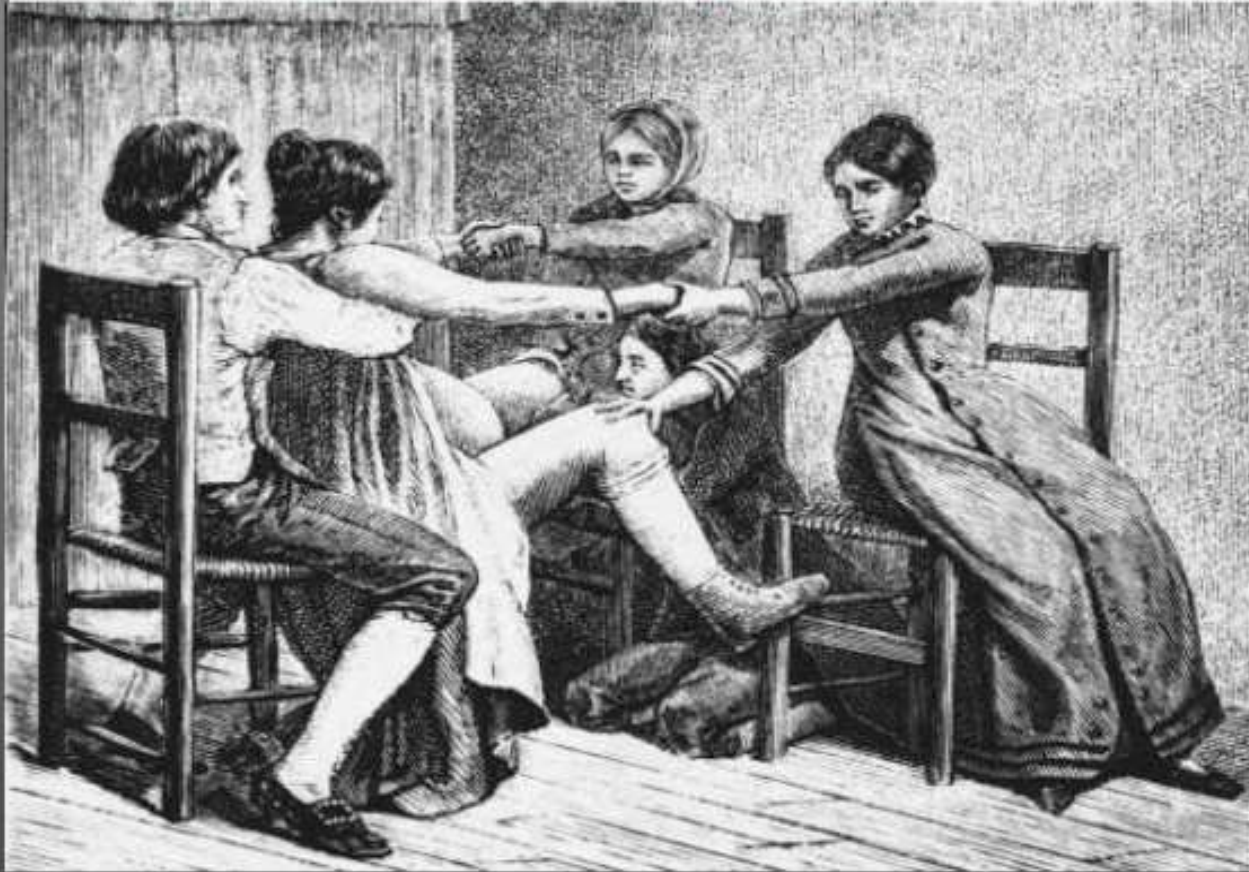
Management of Sensitized Pregnancy

- Fetus Rh +ve: - AF analysis for Liley's chart follow-up.
 - **Mildly Affected Fetus** (falls into **zone 1**)
 - Is considered to unaffected or mildly affected.
 - Testing repeated every 2–3 weeks, and
 - Delivery near term (after pulmonary maturity achieved).
 - **Moderately Affected Fetus** (falls into **zone 2**);
 - Should be tested more frequently, every 1–2 weeks.
 - Delivery may be prior to term (as pulm. maturity is reached)
 - **Severely Affected Fetus** (falls into **zone 3** on the Liley curve),
 - Has frank evidence of hydrops (eg, ascites, pleural or pericardial effusion, subcutaneous edema).
 - Consider medical termination of pregnancy.

N.B: Intrauterine transfusion of 'Rh Neg' blood as indicated

Abnormal labor – Dystocia

Abnormal Labor - Dystocia



Definition.

- **Dystocia**

- Literal - Difficult labor.

- Abnormalities that prevent or **deviate** from normal course of labor.
 - Characterized by abnormally **slow** progress of labor
 - **Failure** to progress of labor.
 - **Ineffective Labor**

- **Precipitate labor**

- Abnormal labor & the opposite to dystocia.

Normal Effective labor:

- Dynamic process.
- Characterized by uterine contractions that keeps increasing with time in :
 - Regularity
 - Intensity and
 - Duration.
- Causing progressive effacement and dilatation of the cervix.
- Safely acceptable presentation & position of the fetus
- Permit descent of the fetus through the birth canal:
cardinal movements of labor
- Should be completed within a safe period of time!!

Dystocia is the consequence of four distinct abnormalities that may exist singly or in combination:

1. **Abnormalities of the expulsive forces**, either uterine forces insufficiently strong or inappropriately coordinated to efface and dilate the cervix—uterine dysfunction—or inadequate voluntary muscle effort during the second stage of labor.
2. **Abnormalities of fetus** - presentation, position, or development of the fetus.
3. **Abnormalities of the maternal bony pelvis** —that is, pelvic contraction.
4. **Abnormalities of soft tissues** of the reproductive tract that form an obstacle to fetal descent

These abnormalities can be mechanistically simplified into three categories.

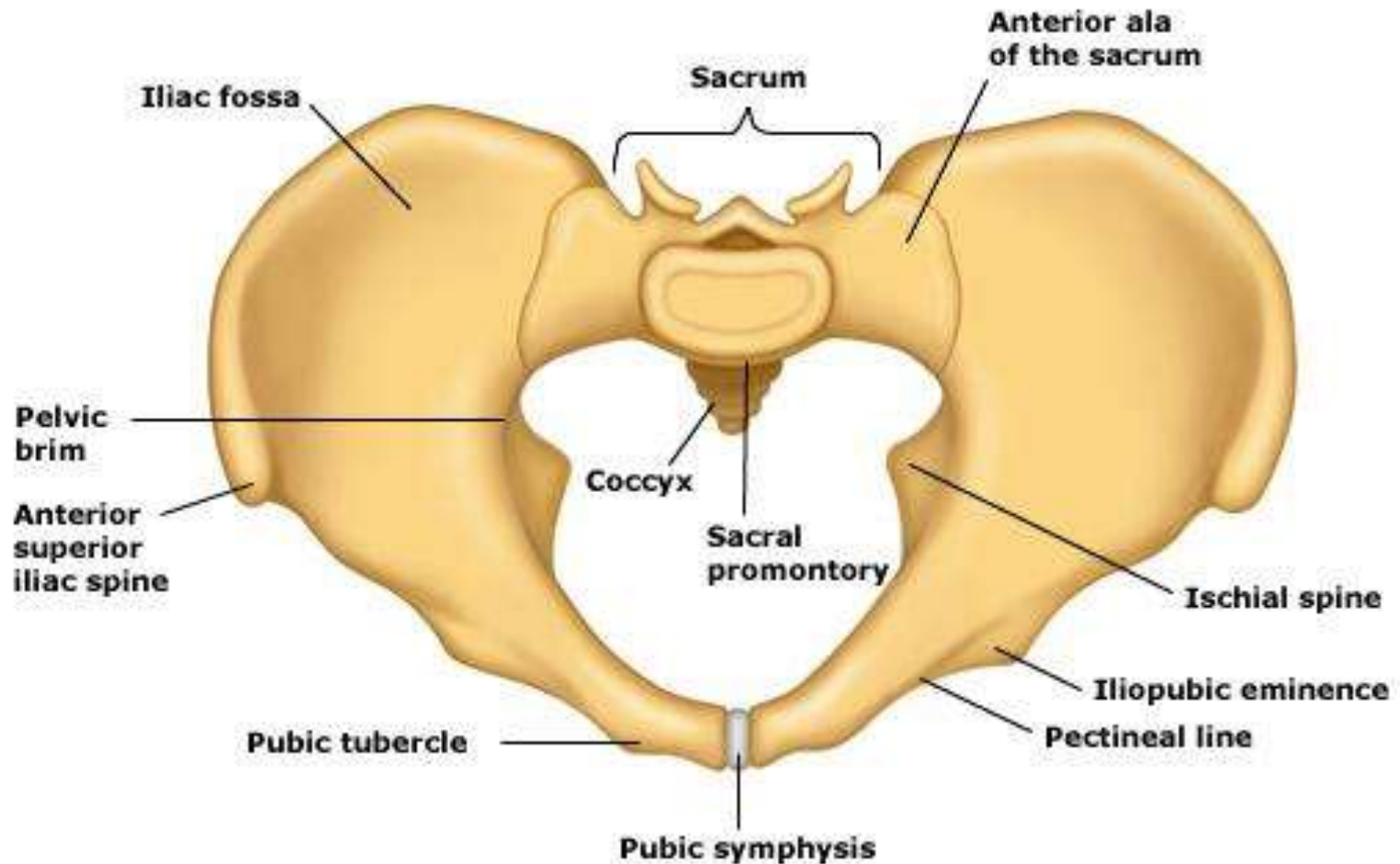
Three general categories – 3 P's

- Passage - Contracted pelvis, Anomalies, Scarring of birth canal, Pelvic masses, Placenta previas.
- Passenger – malpresentation, excessive fetal size.
- Powers – Uterine dysfunction.
 - Poor maternal effort

Abnormalities of the passage.

- **Important pelvic anatomic marks:**
 - 1- inlet: AP & transverse diameter
 - 2- mid pelvis: sidewalls, ischial spines, sacrum, sacro-spinal ligaments
 - 3- outlet pelvis: subpubic angle, coccyx, inter-tuberous diameter

Female pelvis – Superior/Anterior view



Abnormalities of the passage.

- **Pelvic dystocia: - Boney pelvis:**

- a) contracted inlet pelvis:*

- AP diameter of pelvic inlet
(obst. Conjugate) < 10 cm
 - Transverse diameter of inlet < 12 cm
 - Clinically measured Per Vaginal Exam:
"Diagonal conjugate – 1.5cm"
 - **Pelvic dystocia causes:** Unengagement,
Malpresentation, Malposition, PROM,
Cord prolapse, etc.

b) Contraction of mid-pelvis:

interischial spinous diameter <10cm

c) Contraction of pelvic outlet:

Posterior sagittal diameter:

- < 8 cm and extends from the tip of the sacrum to a right-angle intersection with the line between the ischial tuberosities.

Three antero-posterior diameters of the pelvic inlet

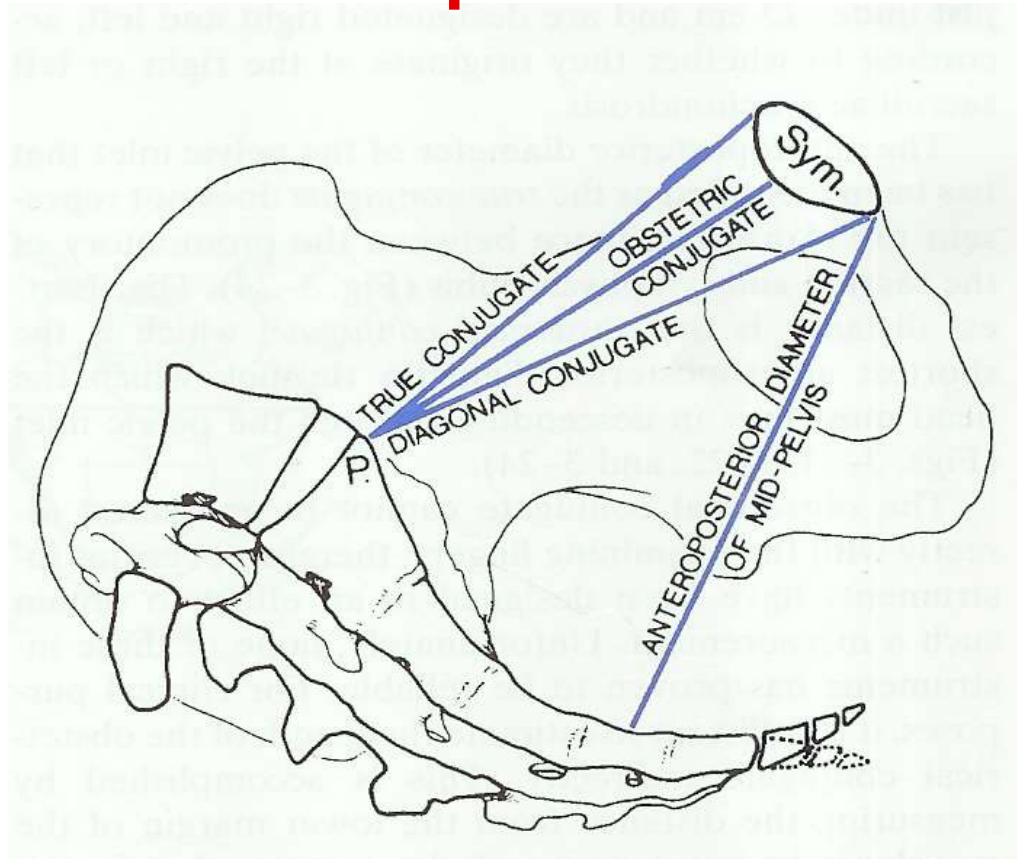
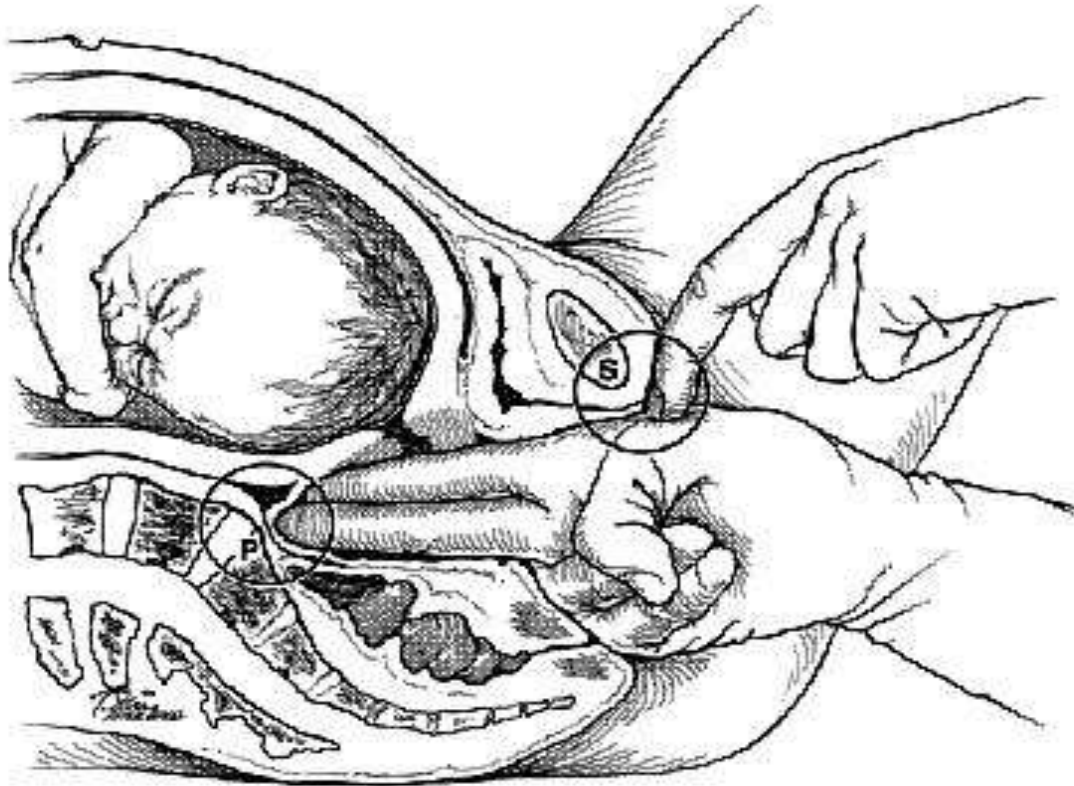


FIGURE 3-24. Three anteroposterior diameters of the pelvic inlet are illustrated: the true conjugate, the more important obstetrical conjugate, and the clinically measurable diagonal conjugate. The anteroposterior diameter of the midpelvis is also shown. (P = sacral promontory; Sym = symphysis pubis.)

Clinical measurement of Diagonal conjugate



Diameter of the inlet and midpelvis

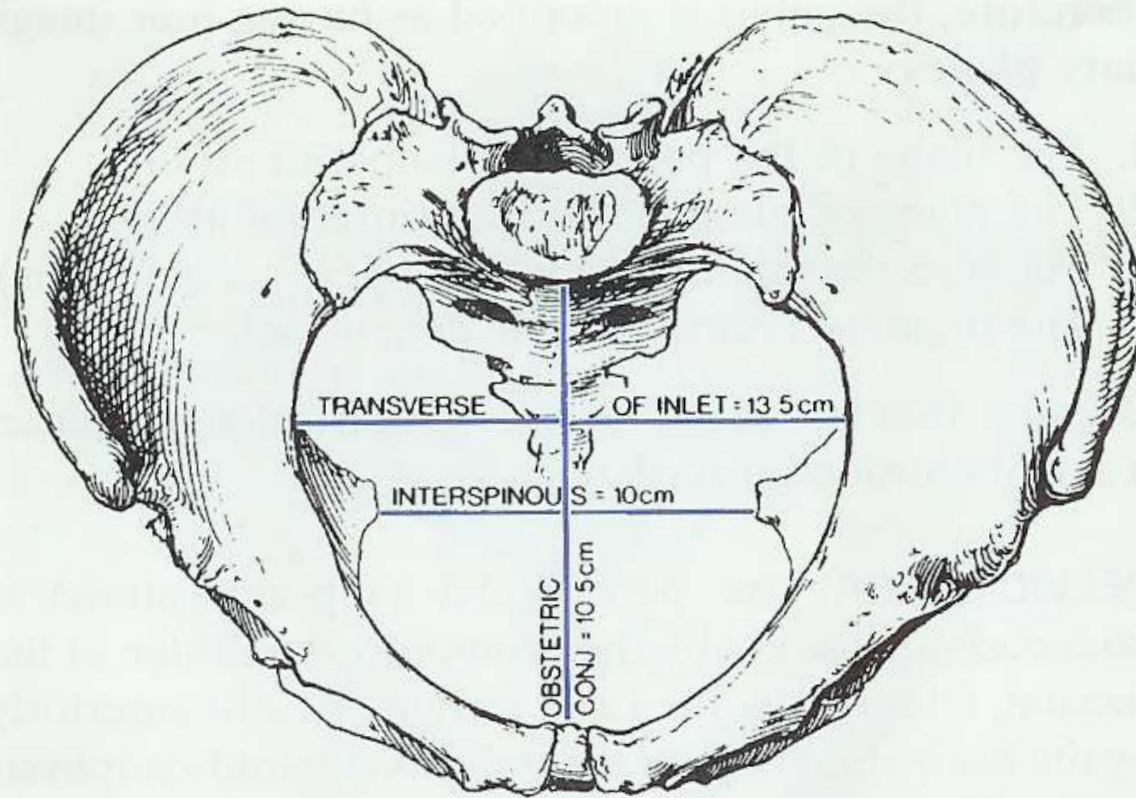
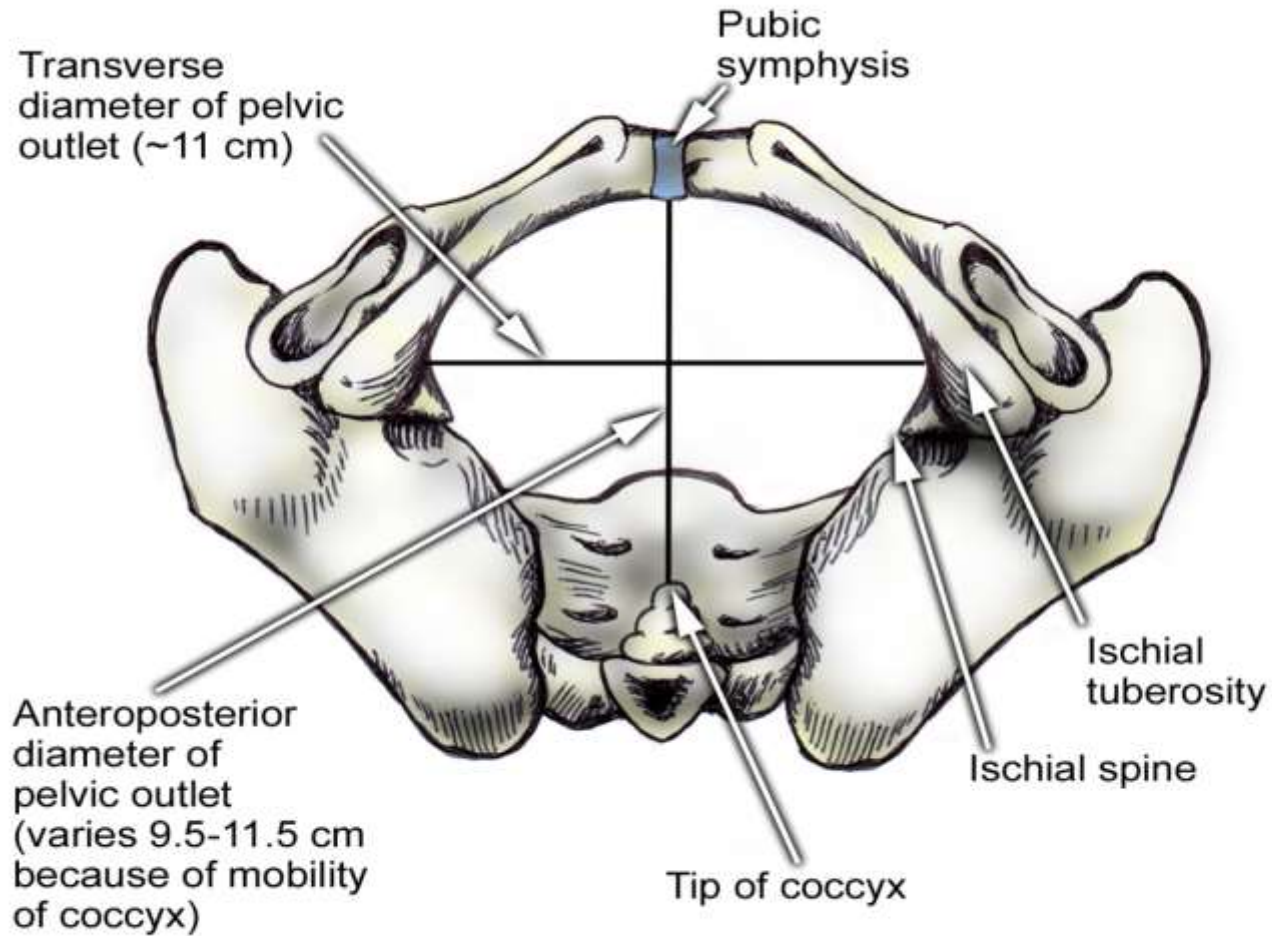


FIGURE 3-25. Adult female pelvis demonstrating anteroposterior and transverse diameters of the pelvic inlet and transverse (interspinous) diameter of the midpelvis. The obstetrical conjugate is normally greater than 10 cm.

Out let pelvis



Caldwell and Moloy classified the 4 major types of adult pelvic types:

- | | |
|--------------|-----------------|
| 1. Gynecoid, | 3. Anthropoid, |
| 2. Android, | 4. Platypelloid |

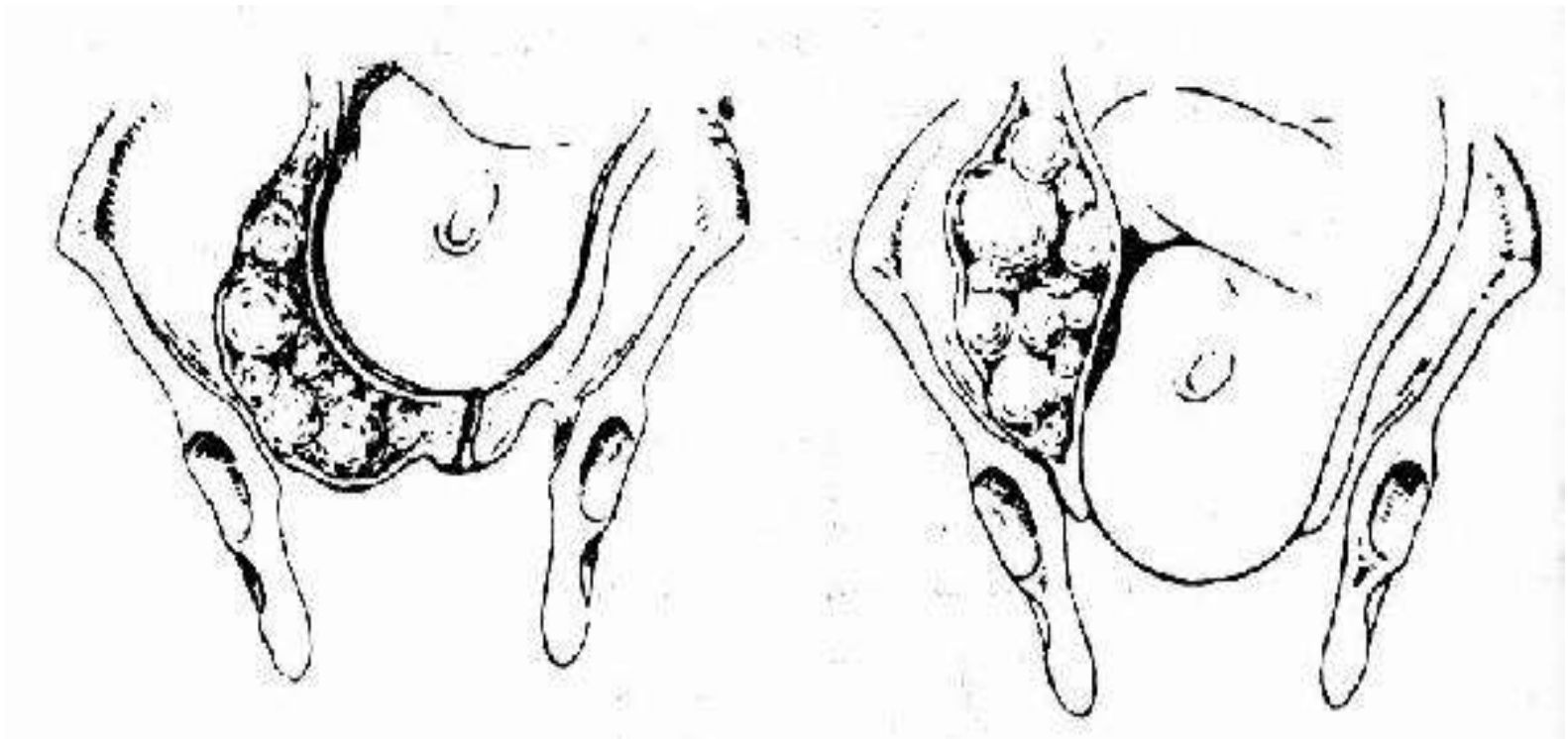
The **gynecoid** pelvis:

- is considered the most typically "female" type
- the most favorable for uncomplicated vaginal delivery
- Found in approximately 50% of all women,
- the pelvic inlet has an oval configuration with a
- transverse diameter slightly greater than the anteroposterior diameter.
- Pelvic side walls are straight,
- the ischial spines are not prominent,
- the subpubic arch is wide, and the sacrum is concave

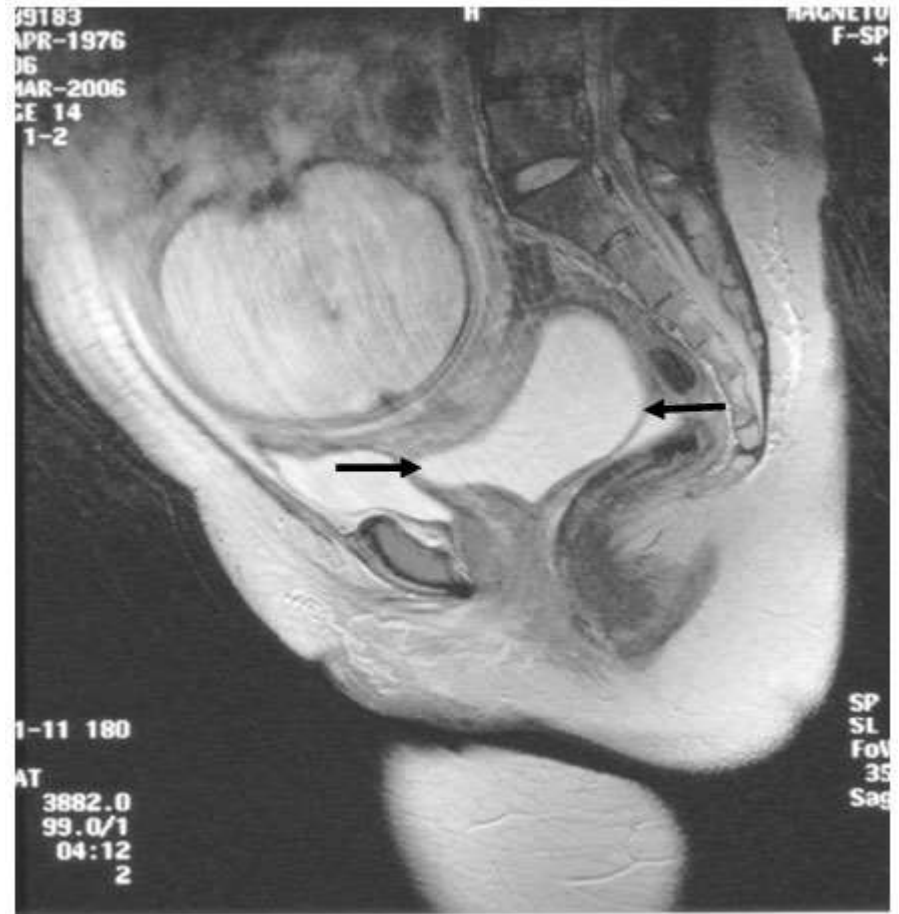
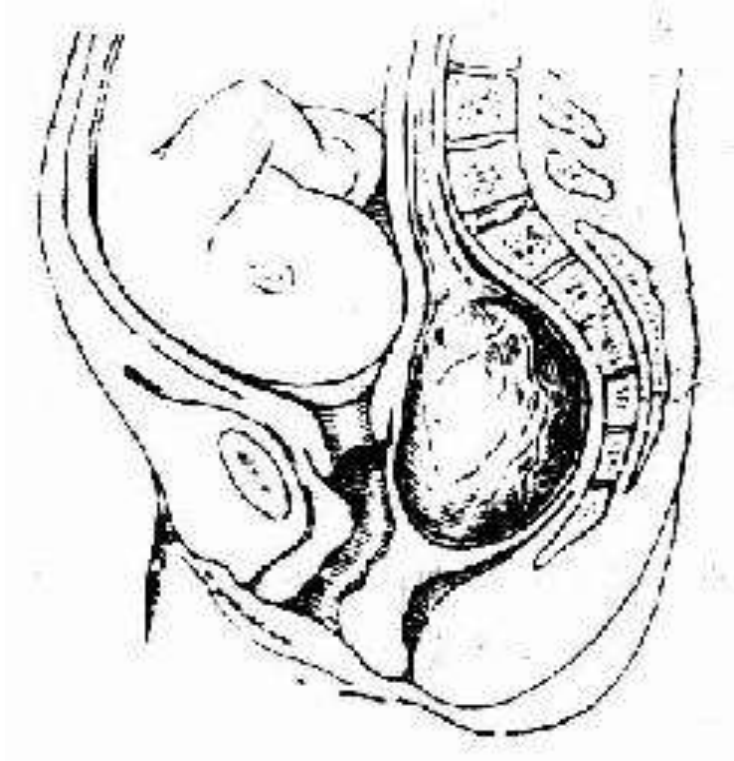
Soft tissue Dystocia

Examples of Abnormalities of soft tissues of the reproductive tract that form an obstacle to fetal descent –
Tumor Previa (Masses), Vaginal Septum, Tighter Perineum,
etc.

Pelvic mass



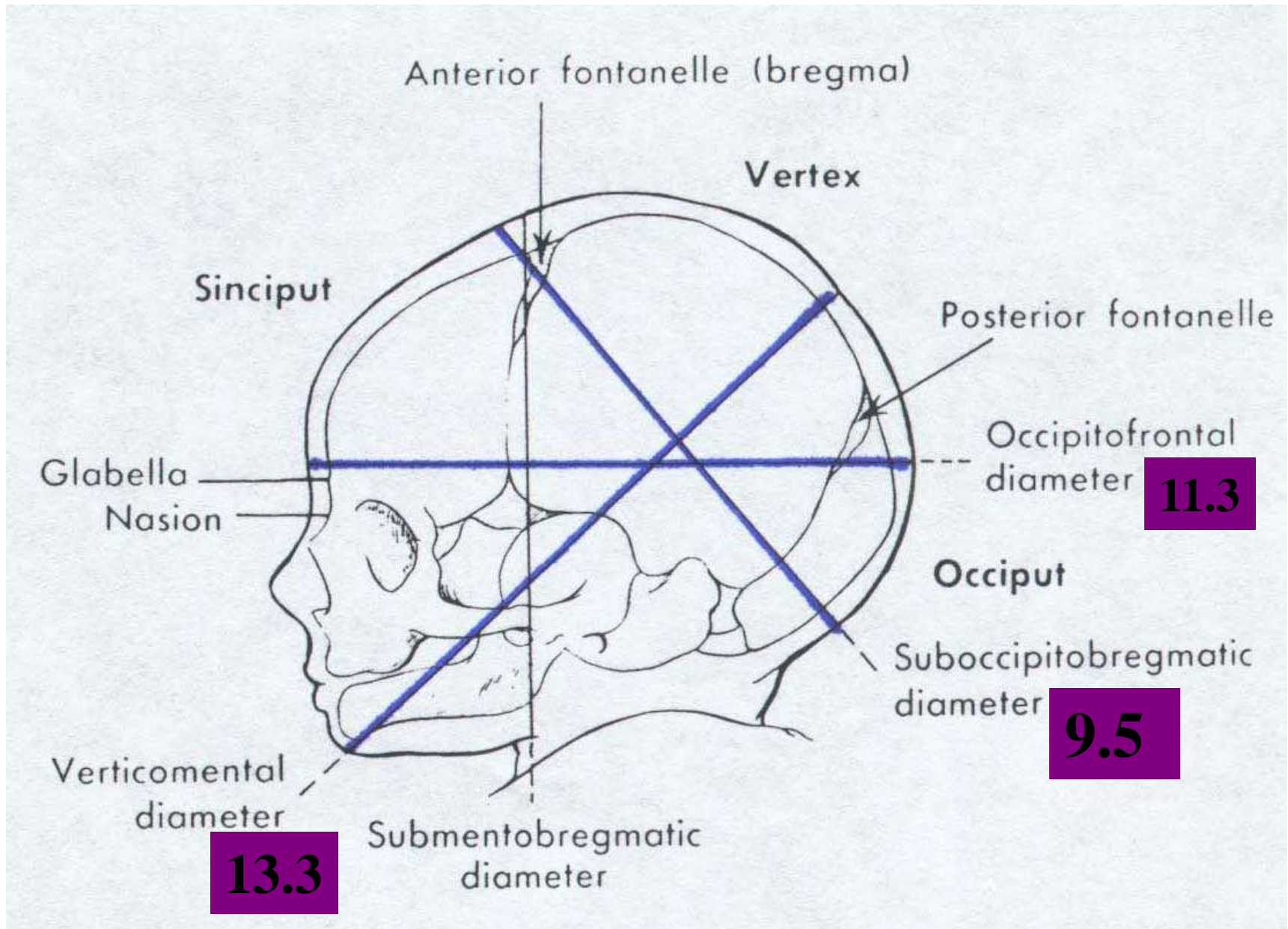
Pelvic mass

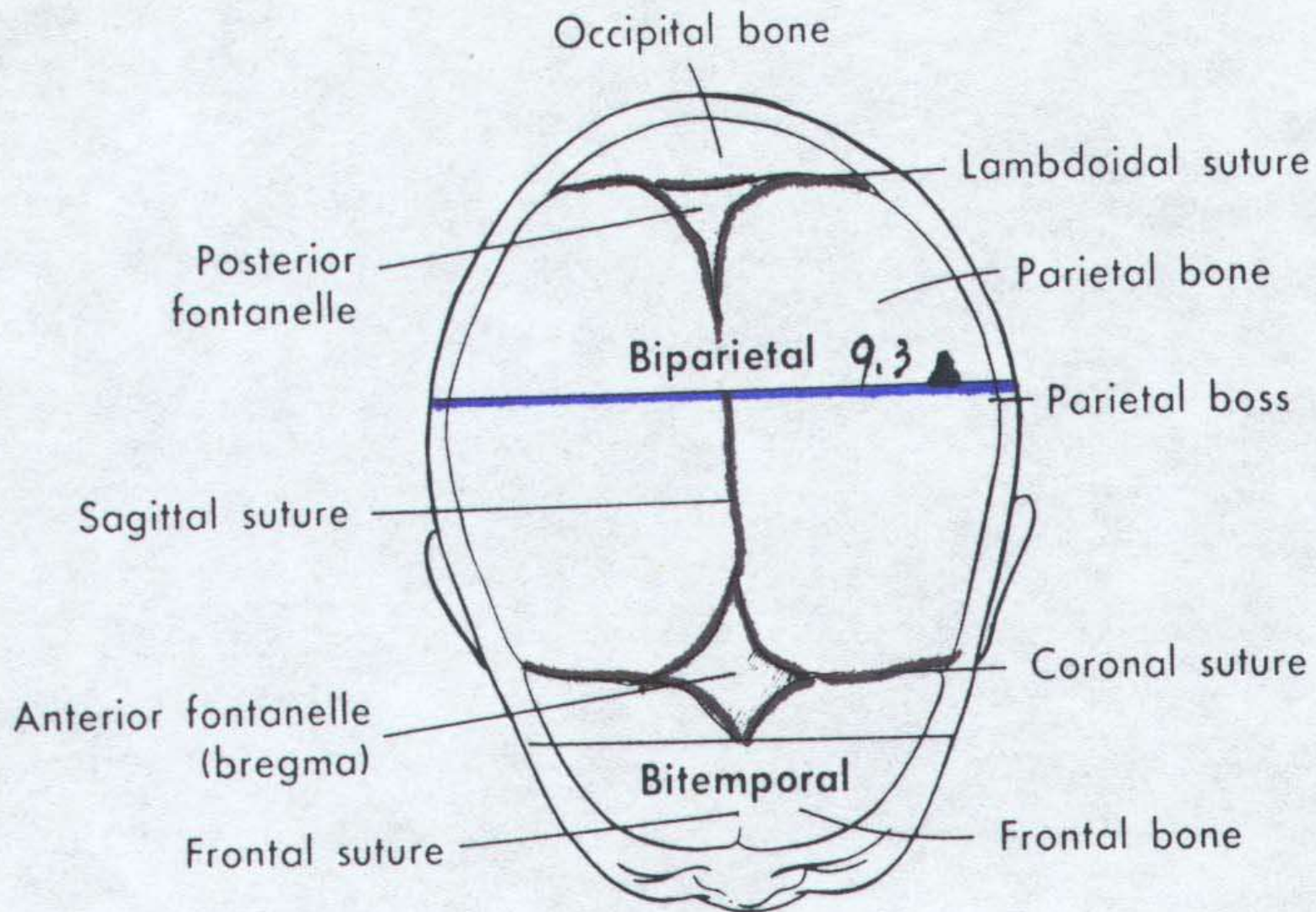


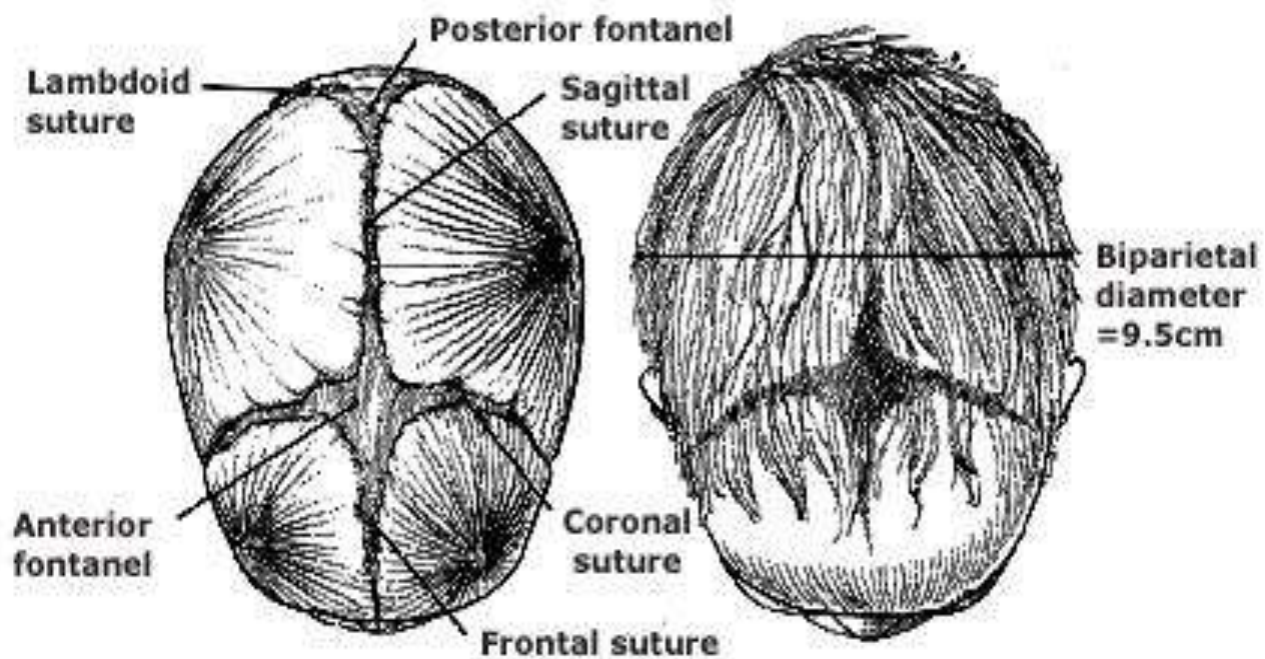
Abnormalities of the passenger

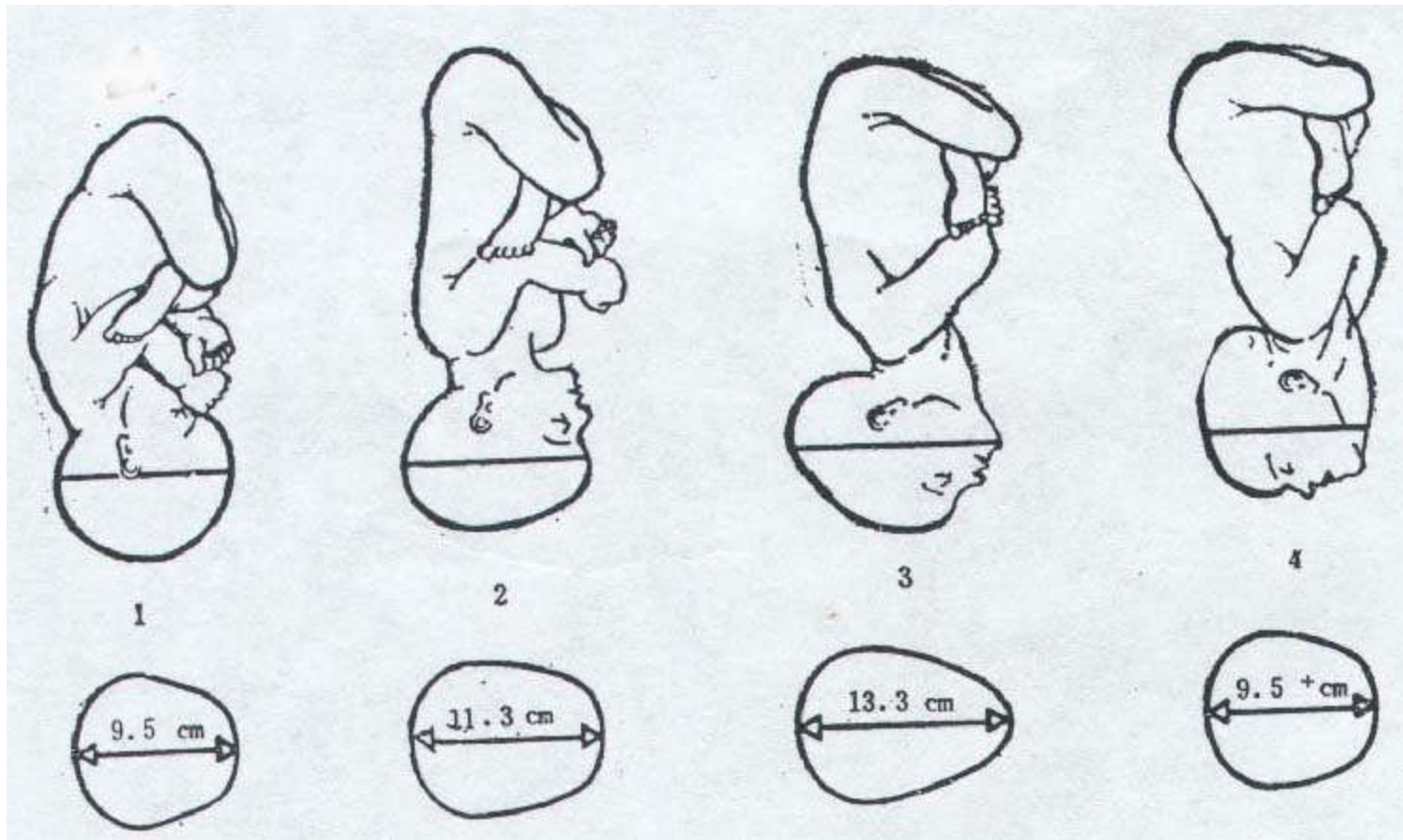
- **Fetal dystocia:**
 - Excessive size: fetal macrosomia, hydrocephalus, shoulder dystocia
 - Malpresentations: transverse lie (shoulder presentation), oblique lie, breech, face, brow, compound, asynclitism.
 - Malpositions: persistent occipito-posterior, deep transverse arrest, face mento- posterior
 - Congenital anomalies
 - Multiple gestation – locking twin, conjoining, etc.

Fetal diameters









Cephalic position and the diameter through pelvis

**occiput
presentation**

**parietal
presentation**

**brow
presentation**

**face
presentation**

Abnormalities of the powers

- **Uterine Dystocia:**

- Hypotonic uterine dysfunction.
- Hypertonic or in-coordinated uterine dysfunction:
- Combination of the two above.
- Lack of voluntary expulsive effort in 2nd stage of labor.

Abnormalities of the powers

Physiology of normal contraction:

- There is gradient of myometrial activity
- Greatest & lasting longer at fundus then body the lower segment.
- The excitation waves start at the cornual area to fundus then body then lower segment.
- Results in contraction of upper segment & retraction of lower segment of the uterine musculature

Characteristics of the **normal power**

- Intensity is greater in the fundus – fundal dominance
- Basal resting pressure
12-15mmHg
- Average 24mmHg
- Well synchronized
- Frequency 3 - 5
- Duration 60s
- Regular
- Rhythmic and forceful

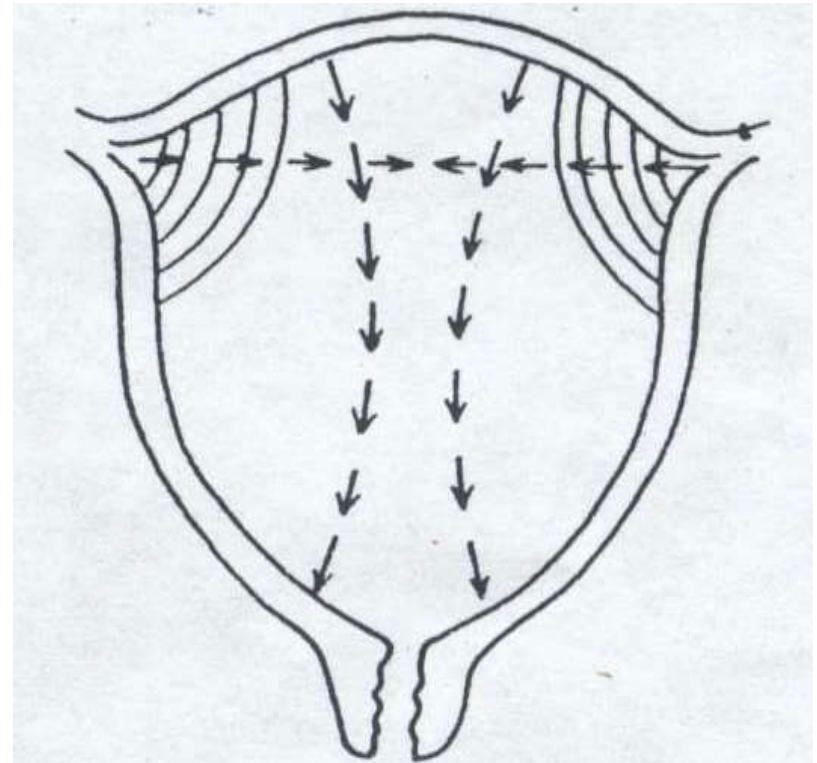


图 6-2 子宫收缩的对称性

Hypotonic dysfunction

- **Insufficient**
- **Regular, i.e.** normal gradient pattern
(synchronous)
- **Infrequent**
 - **Response well to oxytocin**
 - **Most in primigravidas in
active phase labor**

Hypertonic and uncoordinated dysfunction

- Resting tone ↑
- Dyssynchronous – inhibits the contraction-retraction effect of labor.
- Frequent intense contraction
- Constriction ring (see next slide)

Rx Options:

- Tocolysis – stopping labor
- Decrease oxytocin
- Cesarean section
- Sedation

Pathological retraction ring

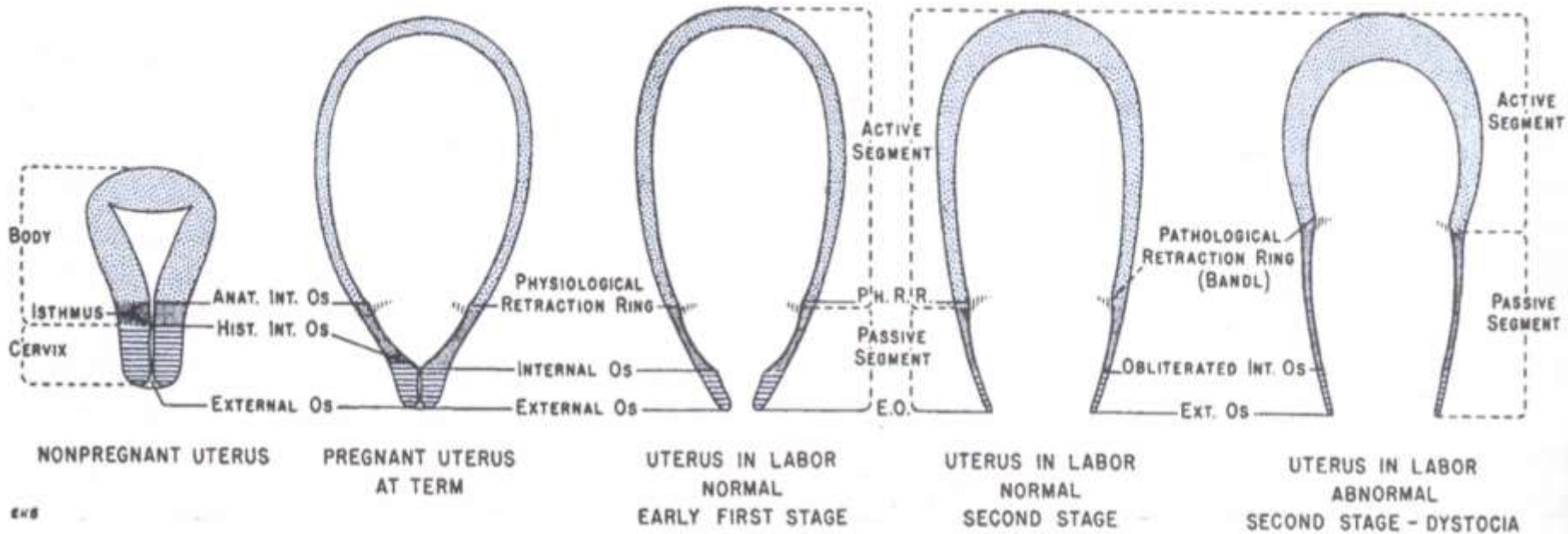


FIGURE 11-2. Sequence of development of the segments and rings in the uterus in pregnant women at term and in labor. Note comparison between the uterus of a nonpregnant woman, the uterus at term, and the uterus during labor. The passive lower segment of the uterine body is derived from the isthmus; the physiological retraction ring develops at the junction of the upper and lower uterine segments. The pathological retraction ring develops from the physiological ring. (Anat. Int. Os = anatomical internal os; E.O. = external os; Hist. Int. Os = histological internal os; Ph.R.R. = physiological retraction ring.)

Incidence of Dystocia.

- Difficult to know exact incidence
 - ambiguities in definitions
 - uneventful outcomes not reported
- Nulliparas, 25% of all labors.
- Multiparas, 10% of all labors
- 40% of the indications for C/S. (USA, current figure).
 - 50% of primary C/S.
 - 21% of repeat C/S.

Maternal risk factors.

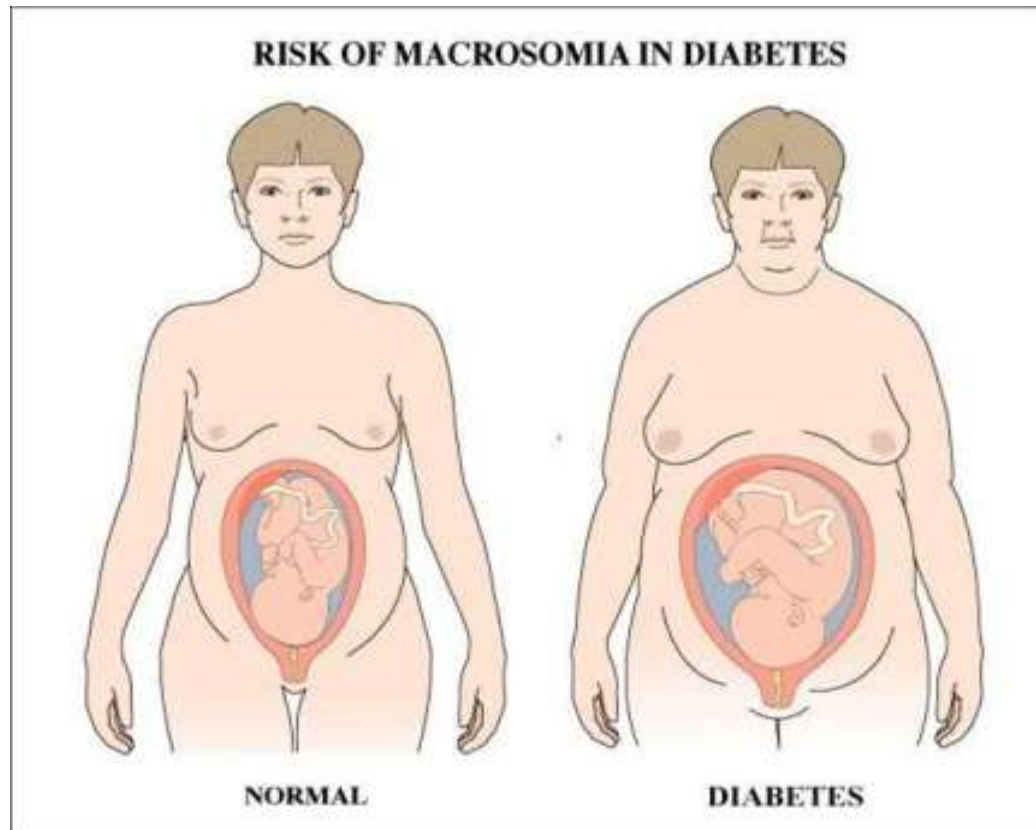
- No antecedente vaginal delivery.
- Previous history of dystocia.
- Pelvis not gynecoid.
- Contracted pelvis.
- Concomitant uterine lesion.
- DM, especially GDM!

Fetal risk factors

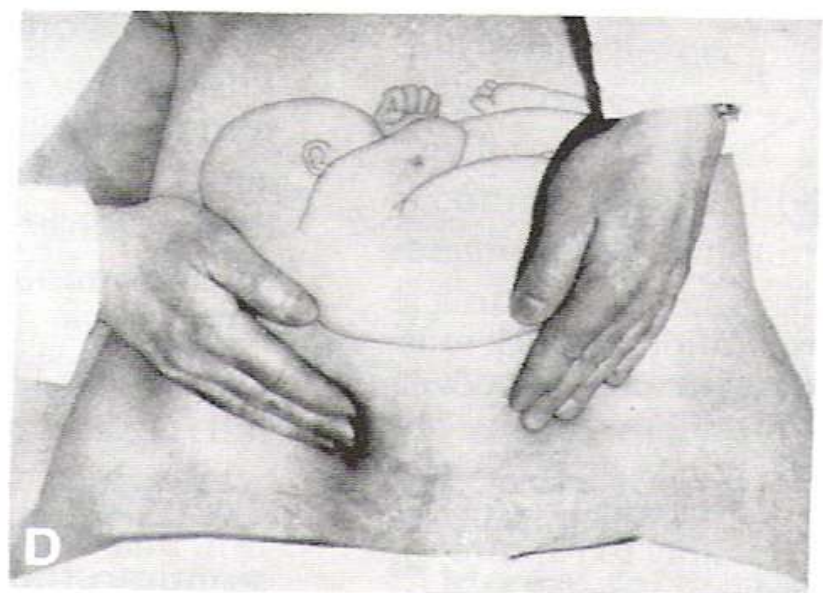
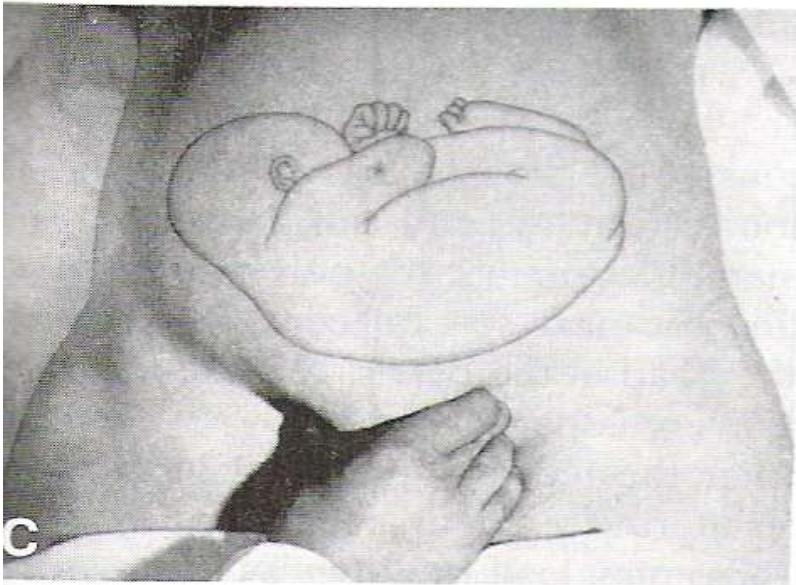
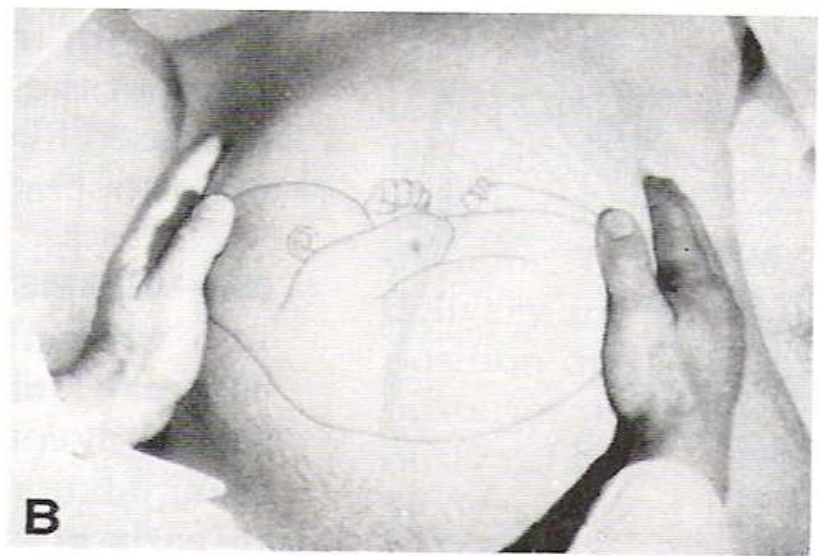
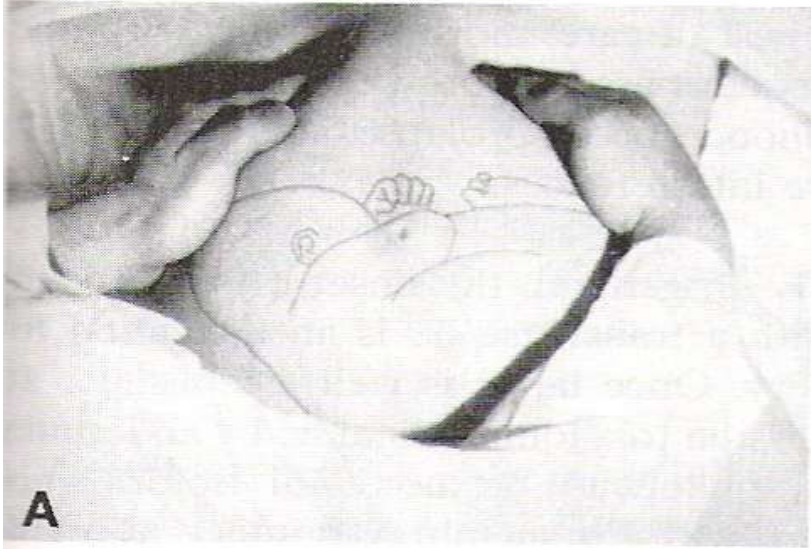
- Weight >4000 g & Postterm fetus
- Non-vertex presentation.
- Shoulder Dystocia
- Fetal malformations.

Fetal macrosomia

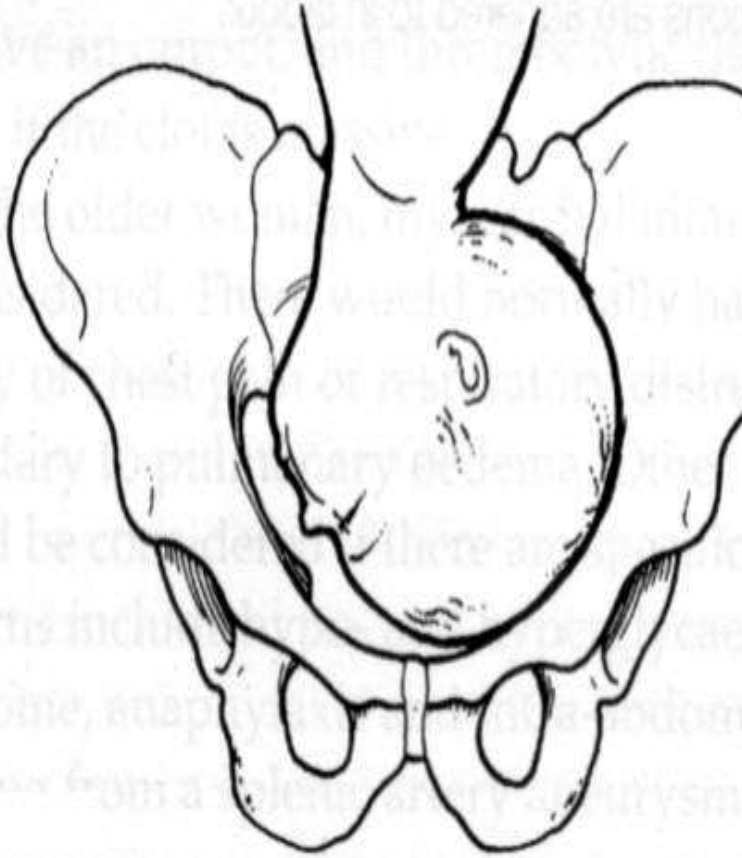
large for gestational age(LGA)
 $\geq 4000\text{g}$



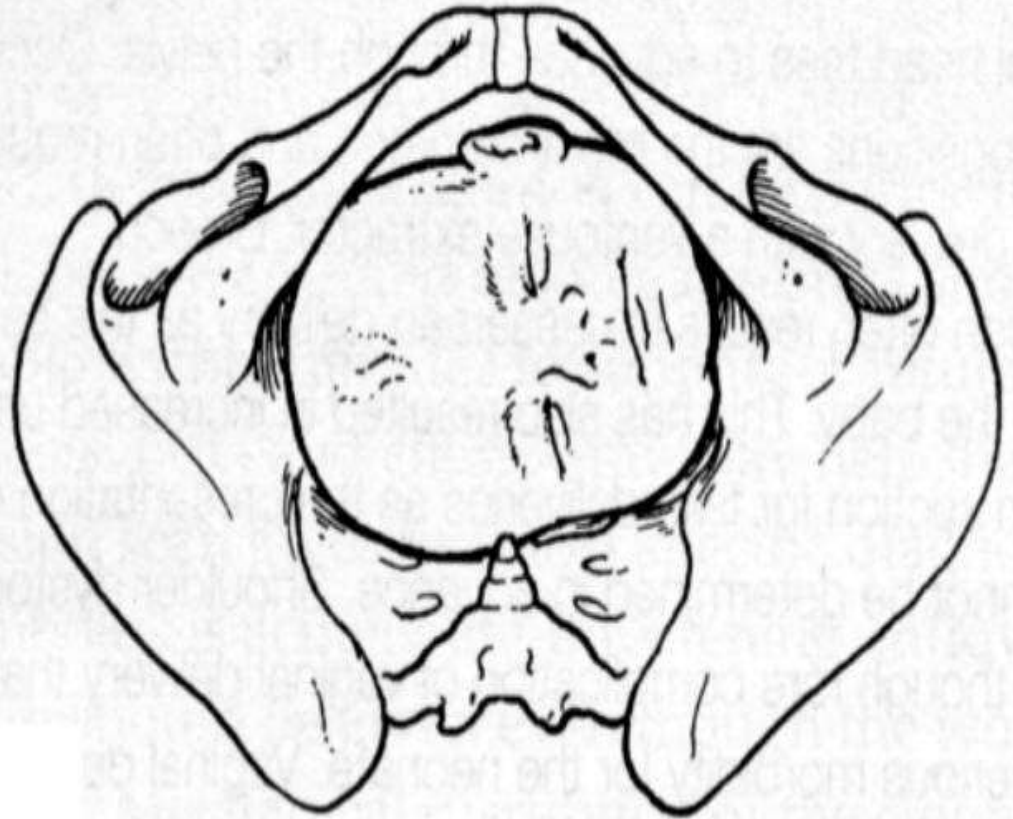
Transverse fetal lie



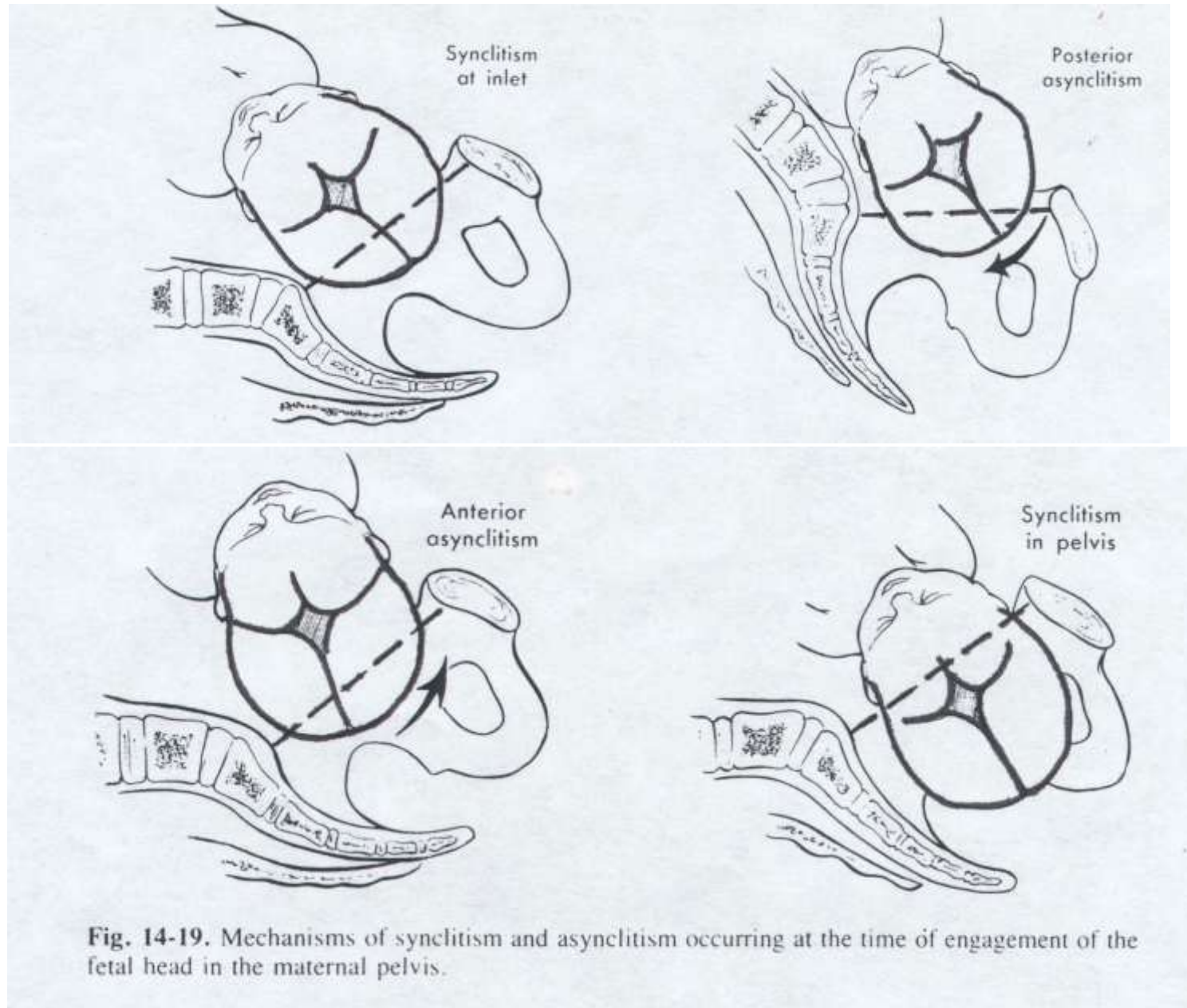
Brow



Face



Asynclitism



Shoulder presentation

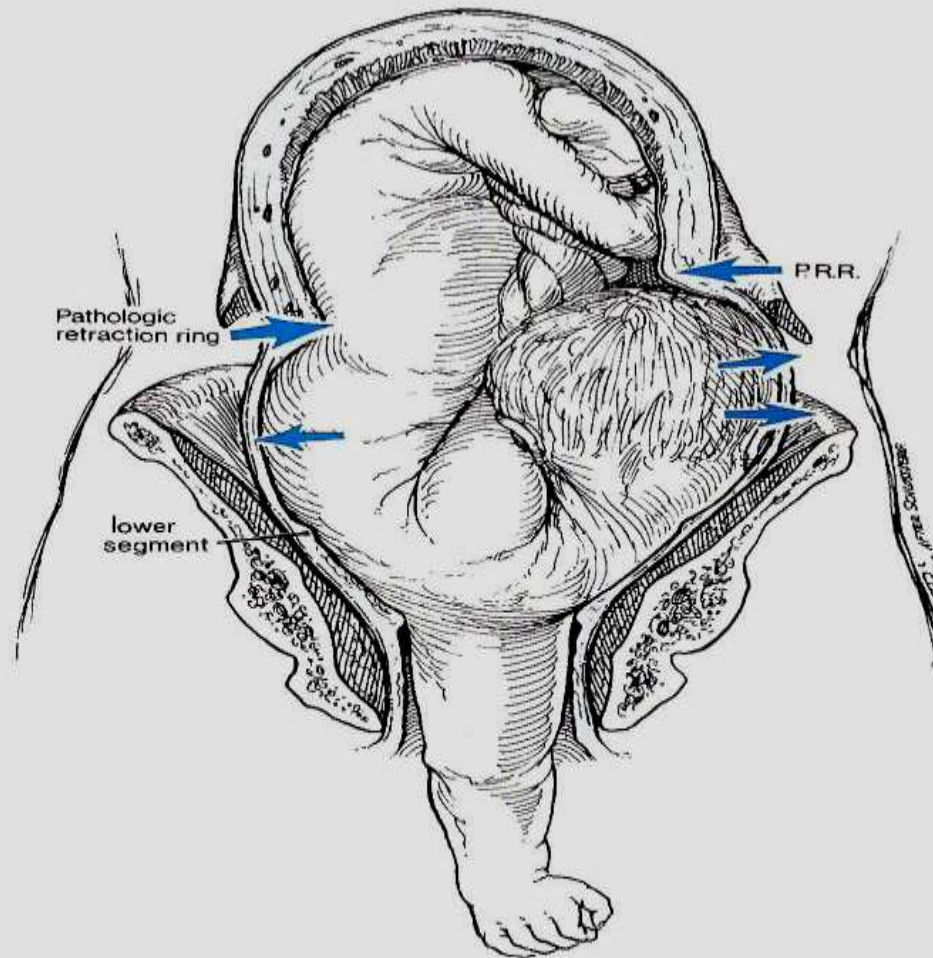


FIGURE 19-7. Neglected shoulder presentation. A thick muscular band forming a pathological retraction ring has developed just above the very thin lower uterine segment. The force generated during a uterine contraction is directed centripetally at and above the level of the pathological retraction ring. This serves to stretch further and possibly to rupture the very thin lower segment below the retraction ring. (P.R.R. = pathological retraction ring.)

Fetal malformation

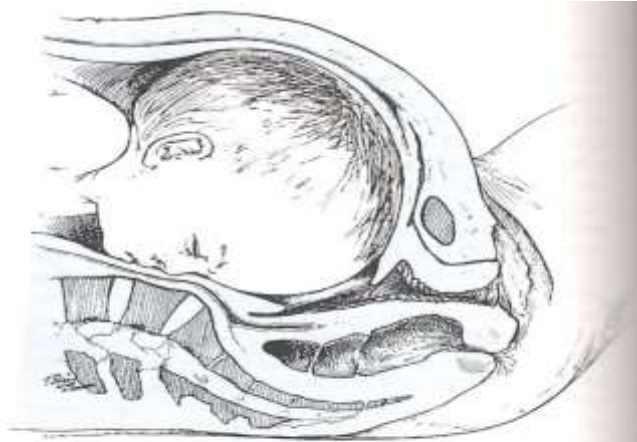


FIGURE 19-14. Severe dystocia from hydrocephalus, cephalic presentation. Note the disparity between the small size of the face and the rest of the cranium.

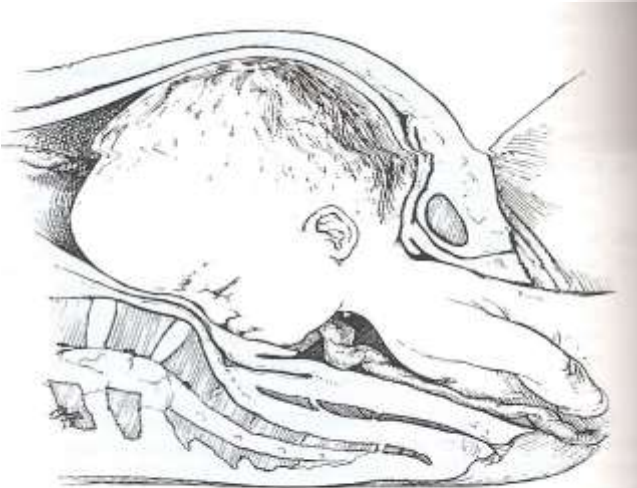
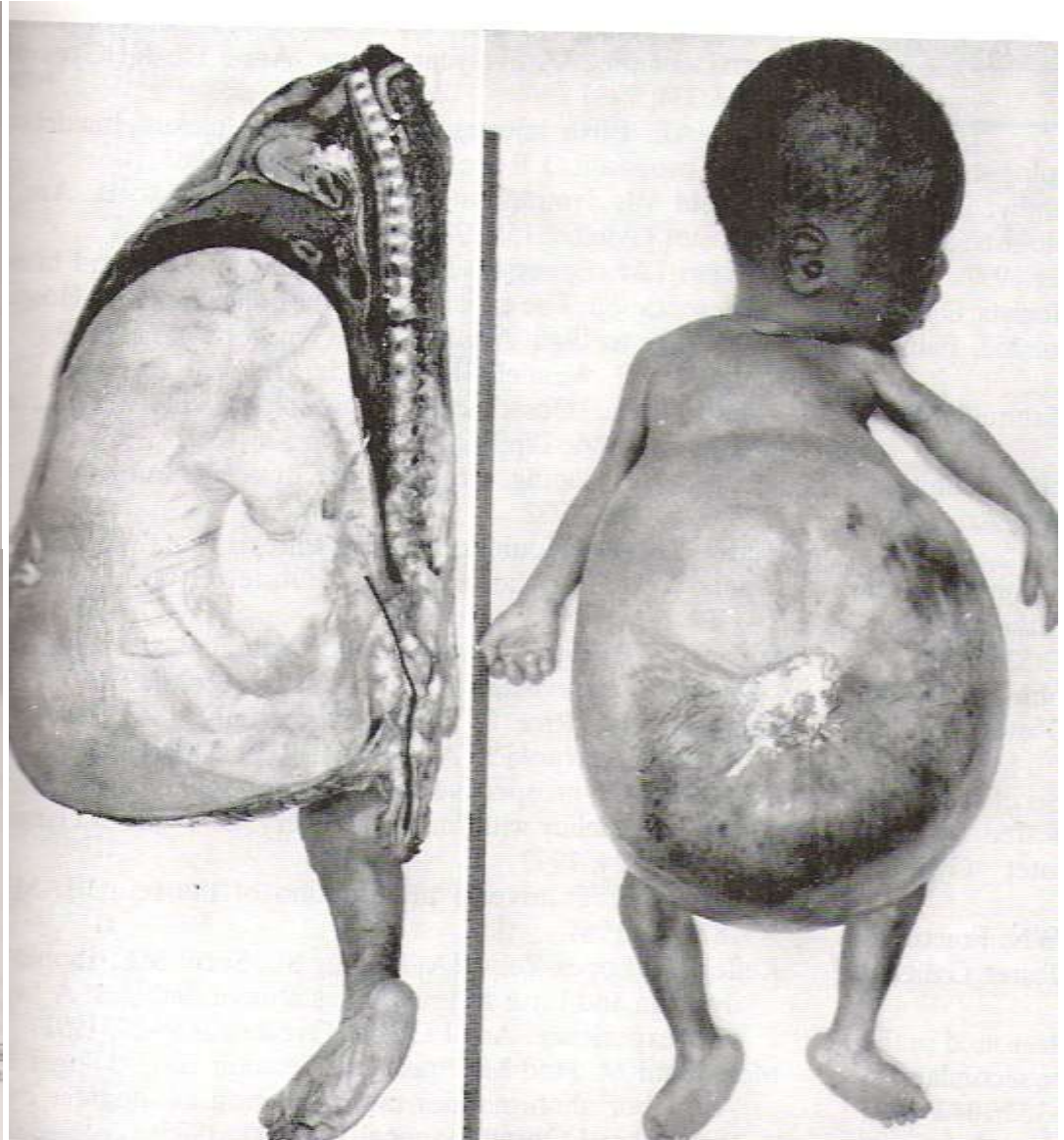


FIGURE 19-15. Severe dystocia from hydrocephalus, breech presentation. Note the distention of the lower uterine segment.



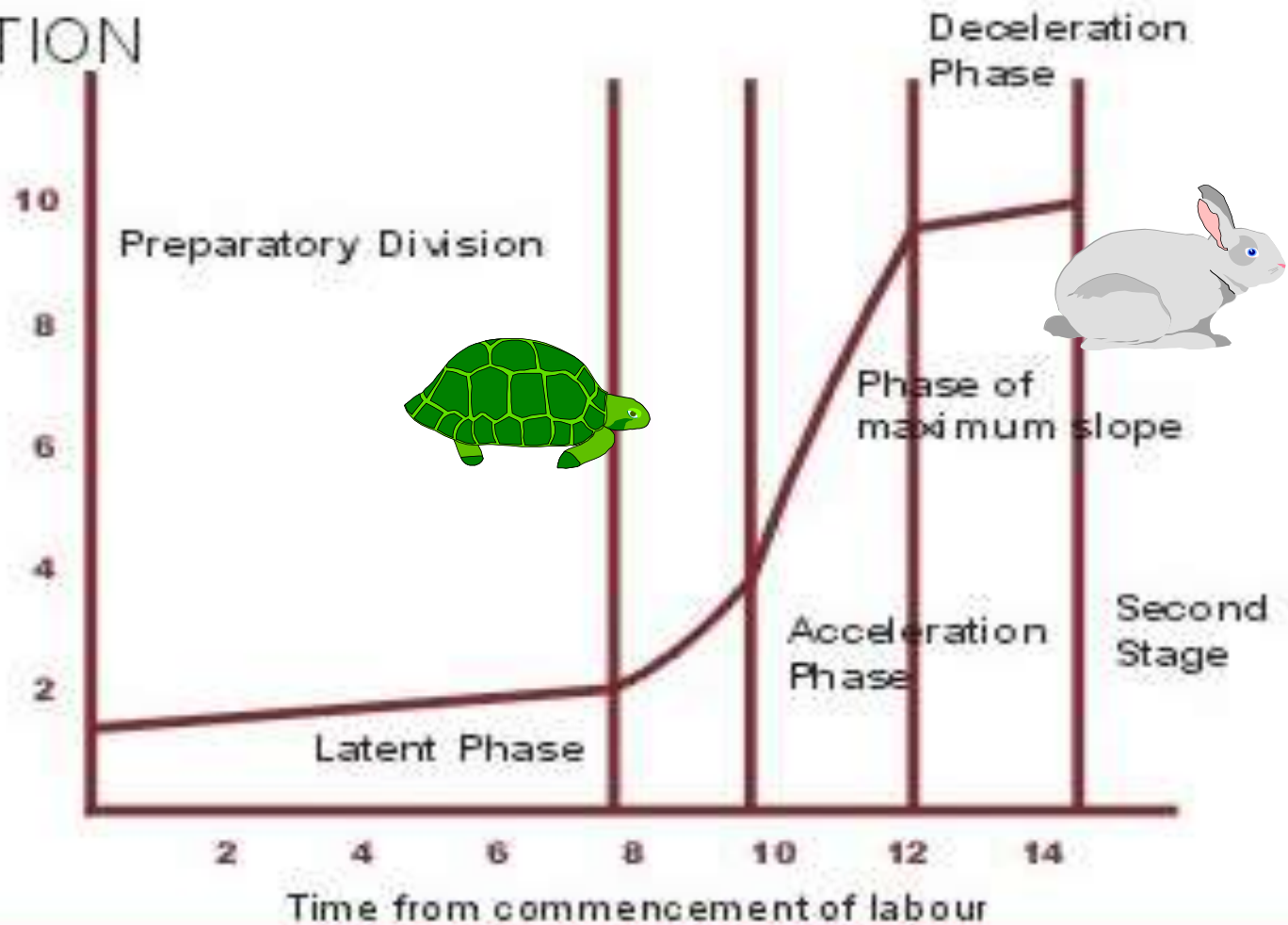
Medical factors.

- Excessive sedation given before the latent phase.
- Use of conduction or regional anesthesia for second stage of labor.

Abnormal Patterns of labor:

1. Prolonged latent phase.
2. Protraction Disorders.
3. Arrest Disorders.
4. Precipitate Disorders.

Cx
DILATATION
(cms)



Friedman's curve showing phase of maximum slope

1. Prolonged latent phase.

- Average normal – Nullipara.-6.4 hr. and multi – 4.8 hrs. fro time of admission.
- Prolonged if, from time of complaint, it is:
 - > 20 hrs for nullipara and
 - > 14 hrs for multipara.
- And from time of Admission Prolonged if it is:
 - > 08 hrs for both nullipara and multipara.

2. Protraction Disorders.

1. Protracted Active Phase Cervical Dilatation.

- <1.2 cm/hr. for nullipara and
- <1.5 cm/hr. for multiparas

2. Protracted Fetal Descent.

- Descent <1 cm/hr. in Nullipara
And less than 2cm/hr in multipara.

3. Arrest Disorders.

1. Prolonged deceleration Phase of Friedman's curve of labor(see next slide)
 - Deceleration phase > 3 hrs. in nullipara
 - > 1 hr. in Multi.
2. Secondary arrest of dilatation.
 - No progressive cervical dilatation for 2 hrs or more.
3. Arrest of descent.
 - Descent fails to progress for > 1 hr in both primi & multi.
4. Failure of descent.
 - Descent fails to occur in the deceleration phase.

4. Precipitate Disorders.

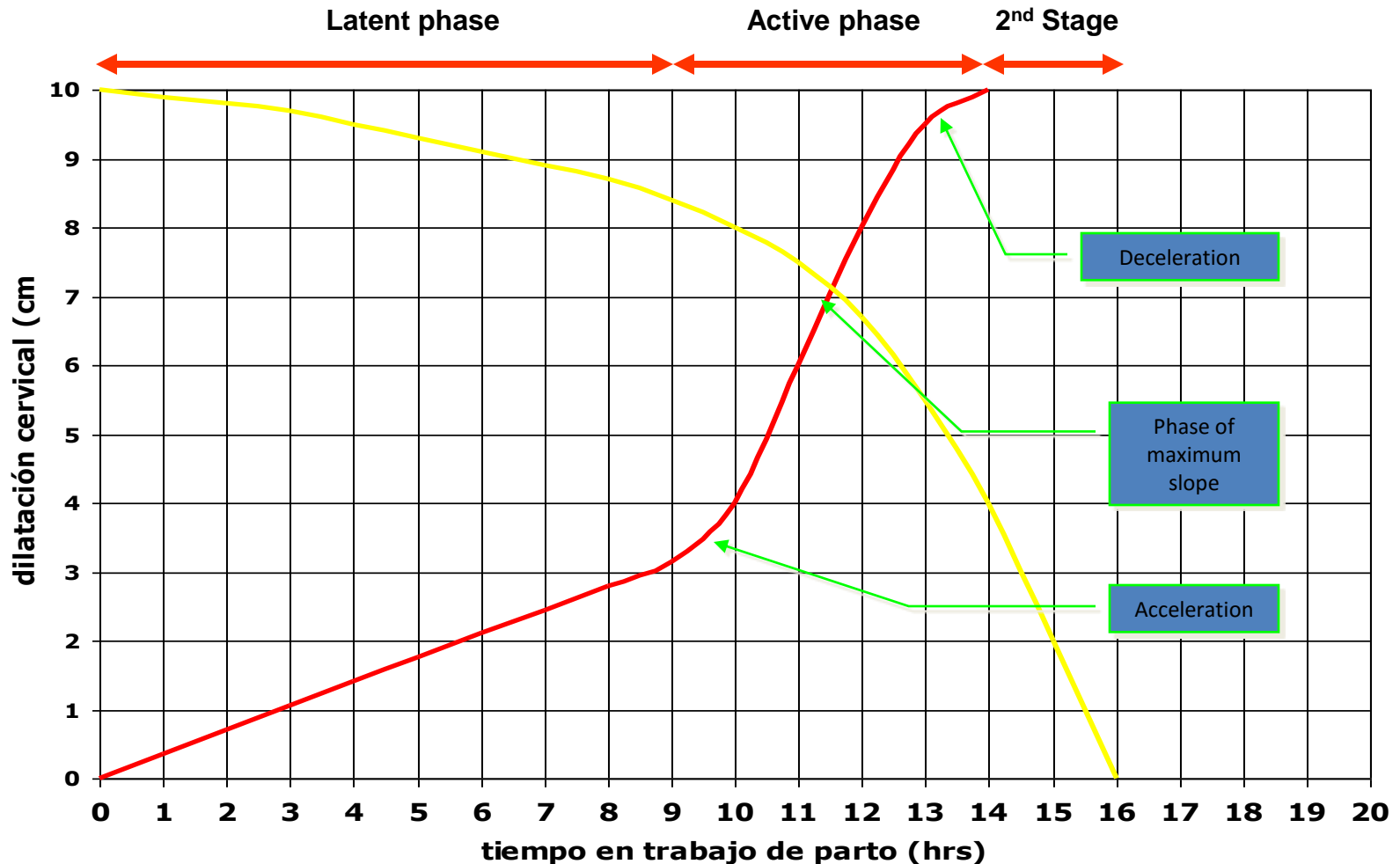
1. Precipitate dilatation.

- Primigravida > 5 cm/hr.
- Multigravida > 10 cm/hr.

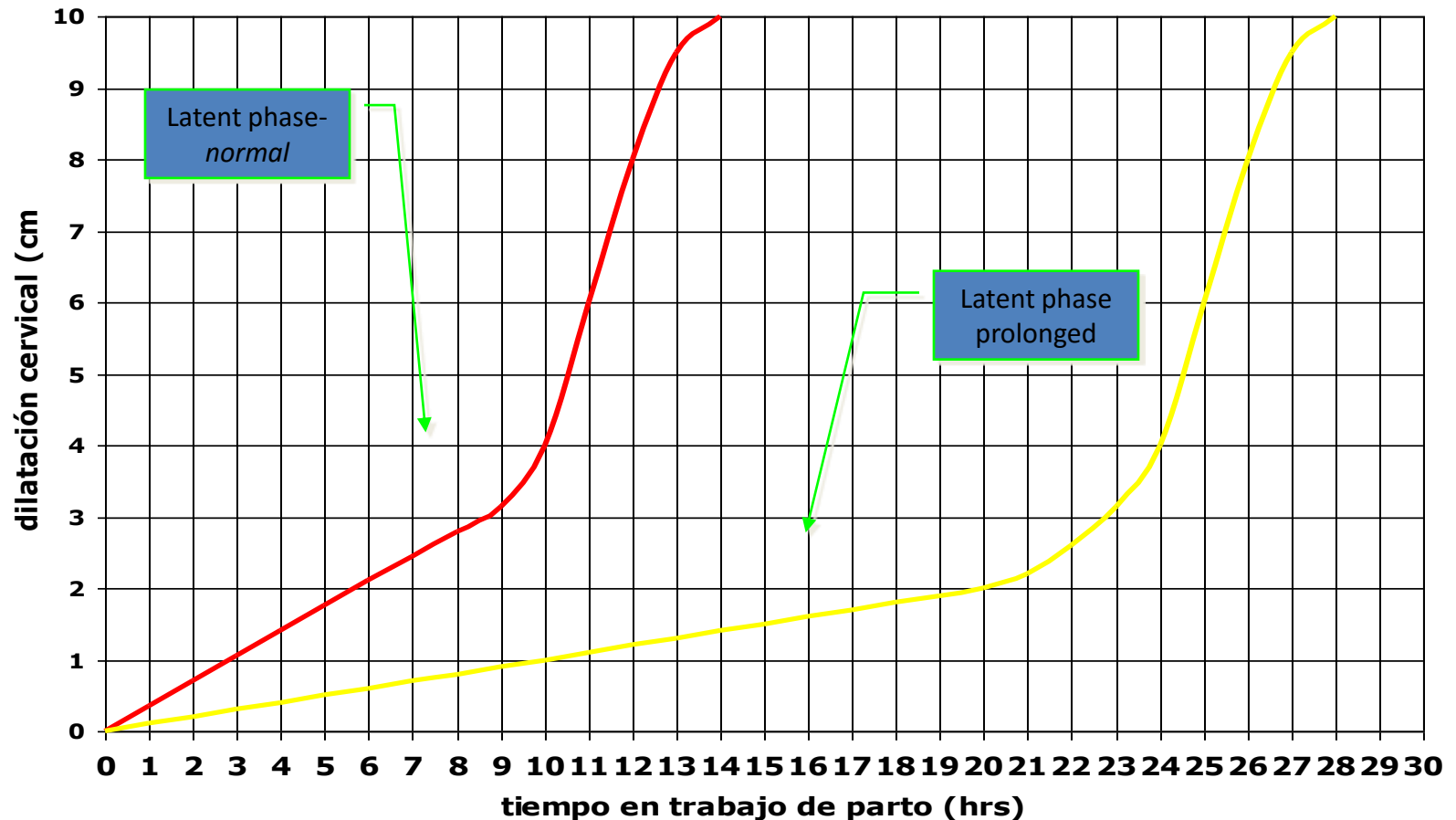
2. Precipitate descent.

- Primigravida > 5 cm/hr.
- Multigravida > 10 cm/hr.

Friedman's curve for nullipara – Cervical dilatation and Descent.



Prolonged latent phase: The Curve.



Prolonged latent phase – Diagnostic Criteria.

- Prolonged latent phase.
 - Nulliparas, >20 hrs.
 - Multiparas, >14 hrs.

Prolonged latent phases - causes

- Prematurely administered sedation and conduction analgesia
- Poor cervical condition
- Uterine dysfunction
- False labor: Braxton Hicks Contractions

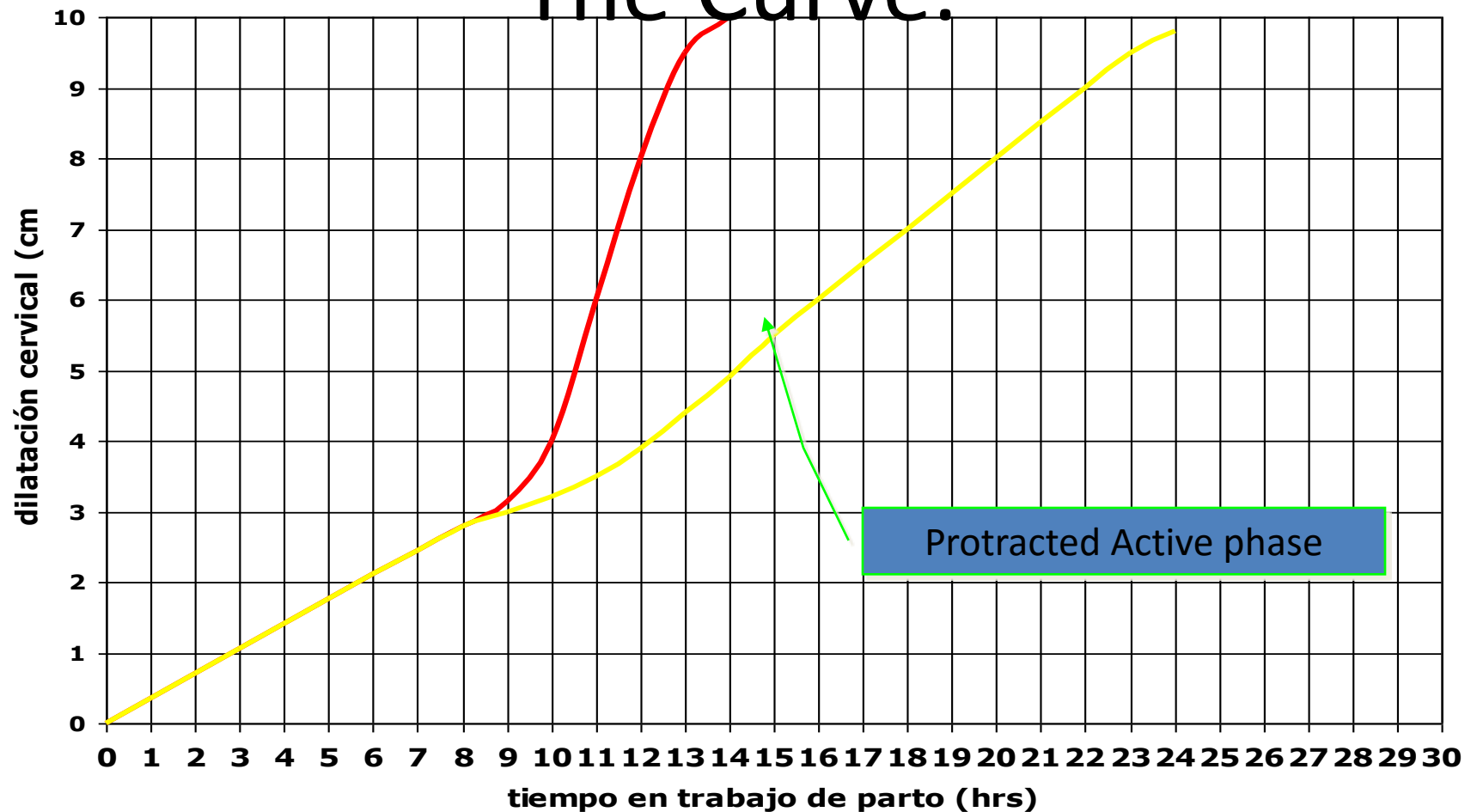
Treatment of prolonged latent phase.

- Expectant
 - 6-12 hrs Bed rest & Hydration : 85% respond.
 - Dx of false labor & home: 10%
 - Narcotic Analgesics.
- Intervention .
 - ARM & Oxytocin infusion: 5% of cases
 - c/s: for feto-maternal compromises.

Protraction Disorders

- Protracted Active Phase Dilatation.
- Protracted Descent.

Protracted Active phase dilatation: The Curve.



Protraction Active phase dilatation— Diagnostic Criteria.

- Protracted active phase dilatation.
 - Velocity of cervical dilation.
 - Nulliparas <1.2 cm/hr.
 - Multiparas <1.5 cm/hr.

Protraction Active phase dilatation -Causes

- Fetopelvic disproportion – one third.
- Inadequate uterine contraction.
- Dysfunctional uterine contraction.
- Malpositions.
- Improperly administered conduction anesthesia >T10 dermatome.

Protraction Active phase dilatation - Rx

- Stimulation with ARM & oxytocin (augmentation)
 - to increase power
 - to correct unsynchrony
- C/S if continuing labor is unsafe!
 - CPD or its cxs, NRFHRP, MSAF,

Protracted Descent– Diagnostic Criteria

- Protracted Descent.
 - It is problem of second stage of labor
 - Diagnosed when velocity of Descent
 - Nulliparas <1.0 cm/hr.
 - Multiparas <2 cm/hr.

Protracted Descent - Causes

- Epidural Anesthesia.
- Motor Block.
- Poor/Inadequate uterine contraction.
- Poor maternal effort

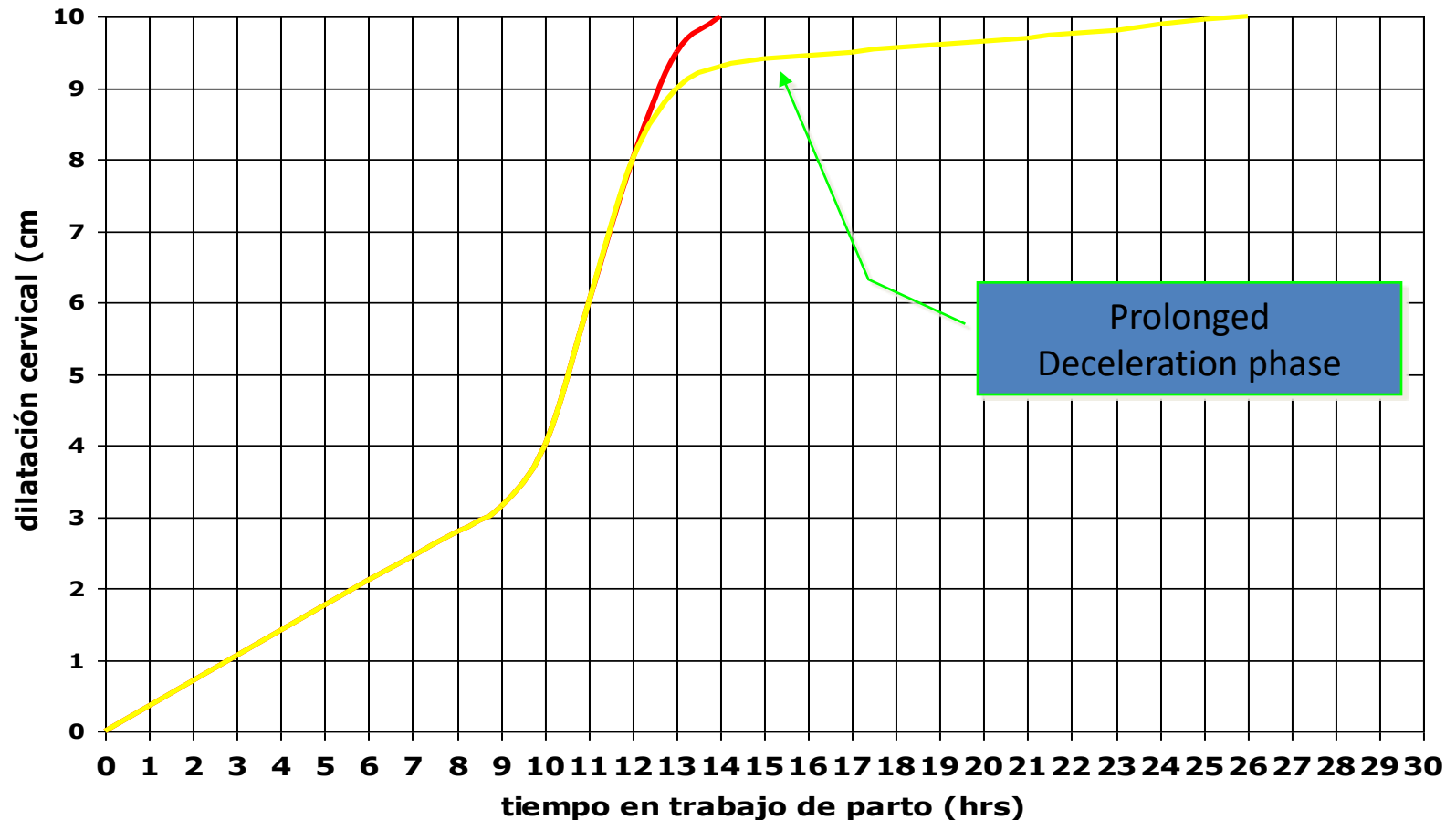
Protracted Descent - Mx

- Normal FHR
 - ARM & Oxytocin stimulation.
- Instrumental Delivery.
- C/S

Arrest Disorders

- Prolonged Deceleration Phase.
- Secondary arrest of dilatation.
- Arrest of Descent.
- Failure of descent.

Prolonged deceleration Phase. Curve.



Prolonged Deceleration phase – Dxtic Criteria.

- Prolonged deceleration phase.
 - Nulliparas, >3 hrs.
 - Multiparas, >1 hrs.

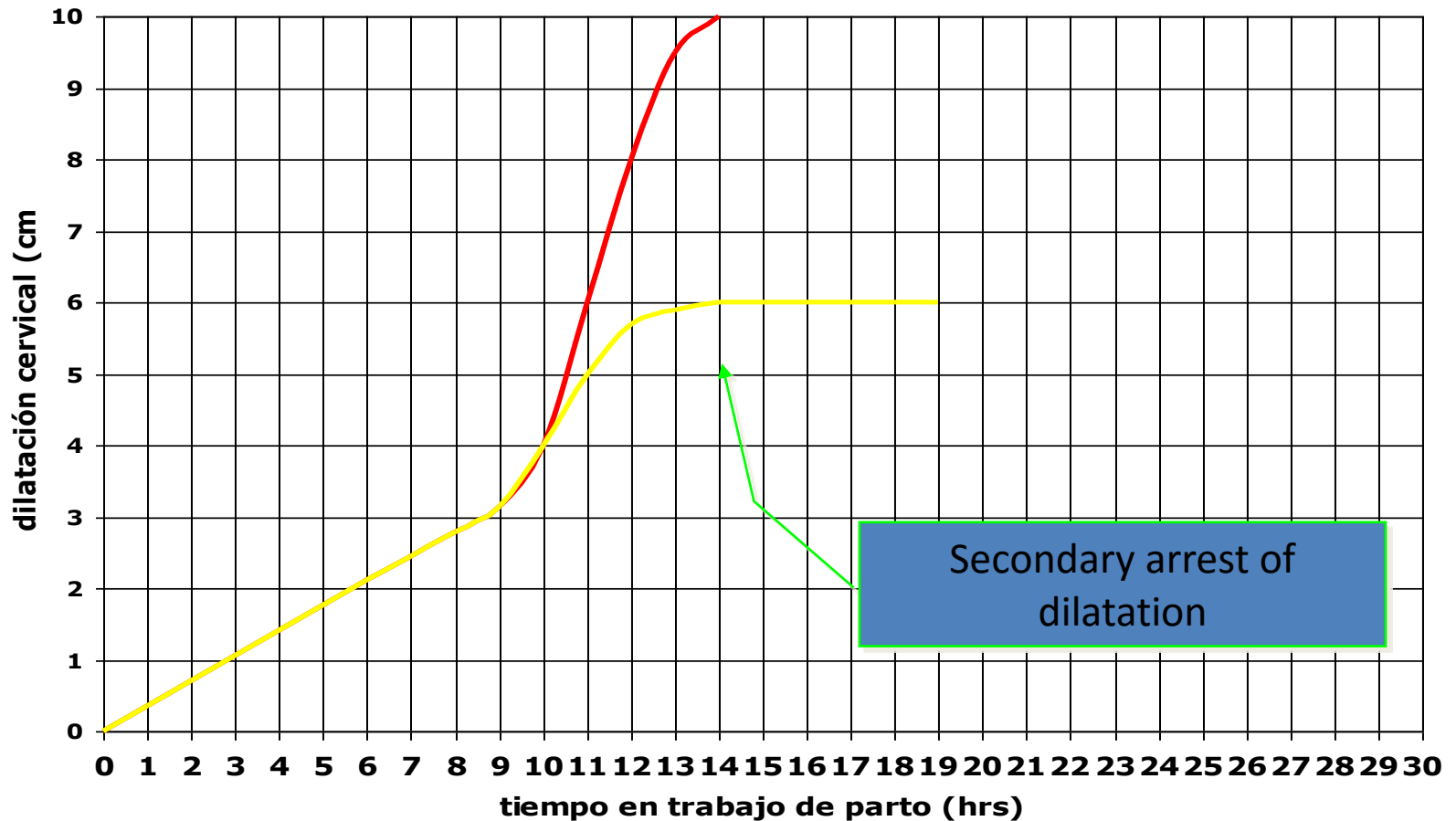
Prolonged deceleration phase - Causes.

- Malposition.
- Fetopelvic disproportion.

Prolonged Deceleration phase - Mx

- ARM & Oxytocin stimulation.
- C/S.

Secondary arrest of dilatation



Secondary arrest of dialation – Diagnostic Criteria.

- No progressive cervical dilatation in active phase of labor.
 - Both Nulliparas and multiparas, >2 hrs.

Secondary arrest of dilatation - Causes.

- Inefficient uterine contraction.
- Feto-pelvic disproportion.

Secondary arrest of dilatation - Mx

- Oxytocin stimulation.
- C/S.

Arrest of Descent – diagnostic Criteria.

- Descent fails to progress.
 - Both Nulliparas and multiparas, >1 hrs.

Arrest of Descent - Causes

- Inadequate uterine contraction.
- Fetopelvic disproportion.
- Malposition.
- Asynclytism.

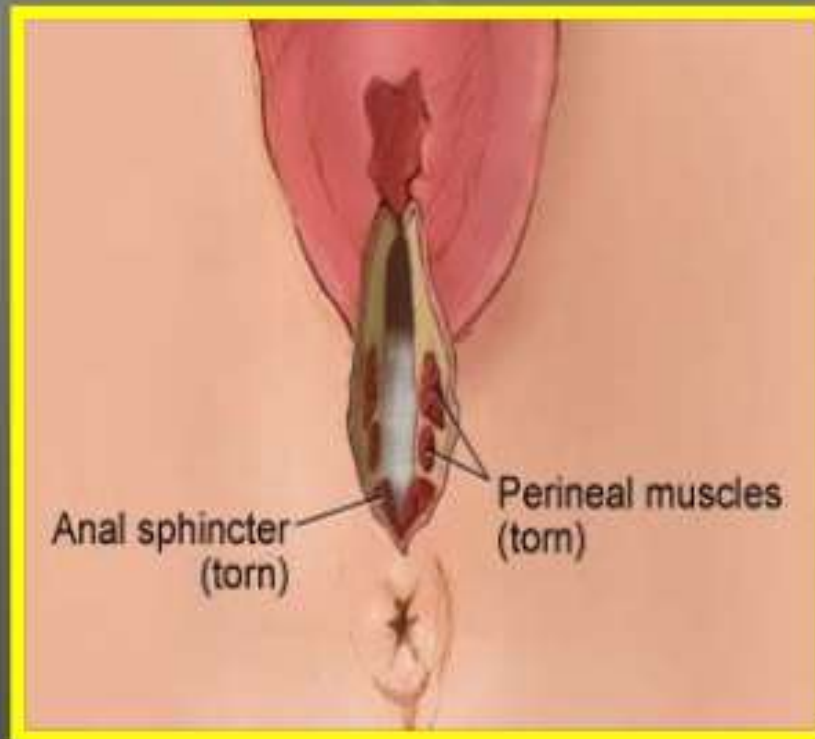
Arrest of Descent - Mx

- Similar to Mx for protracted descent
- Normal fetal heart rate.
 - Oxytocin stimulation.
- Instrumental Delivery,
- C/S

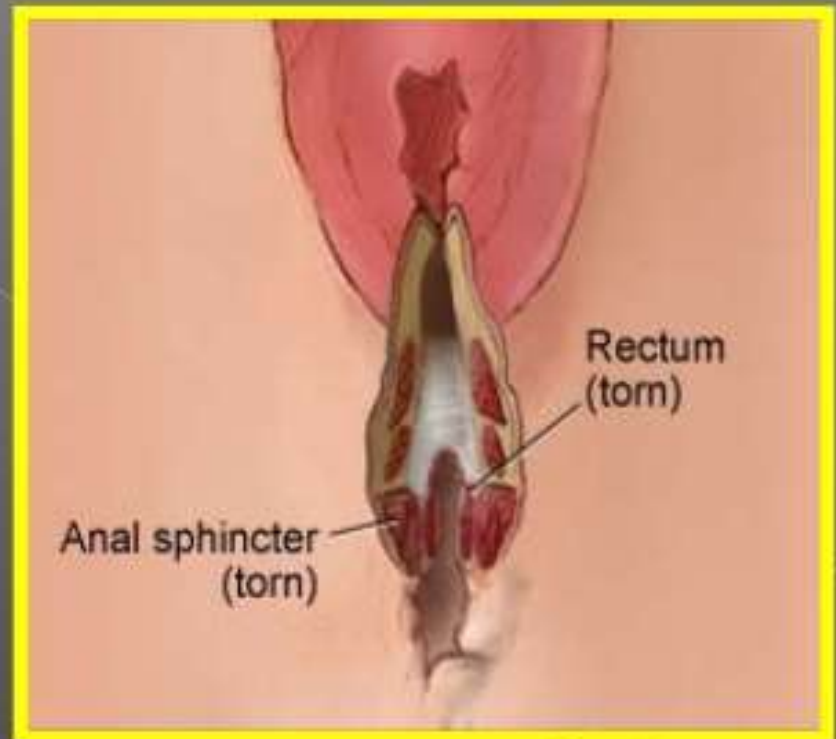
Dystocia: Effect/Fate/Outcome/

1. Intrapartum infections: maternal, fetal
 - fetal sepsis, pneumonia
 - maternal endomyometritis, sepsis
2. Atonic uterus: PPH
3. Obstructed labor
4. Uterine rupture
5. Pelvic floor injury
6. Post partum nerve injury
7. Fistulae

Third degree perineal tear



Fourth-degree Perineal tear



Summary

Diagnosis.

- Clinical History.
- Physical examination.
- Partograph.
- Evaluation of the the 3 P's.

Clinical History.

- Duration of labor and ROM
- Previous Obstetric History.
- History of difficult labor

Physical examination.

- Abdominal Examination.
 - Leopold maneuvers.
 - Estimation of fetal weight.
- Pelvic Examination.
 - Dilatation, Station, Position.
 - Clinical Pelvimetry.

Prolonged Labor - Dystocia (Sxs/Sns/Conditions)

- Maternal dehydrations with its features
- Exhaustion
- Fetal 'distress'
- Sepsis
- Caput and Moulding – if cephalic presentation
- Duration of labor and cx dilation or fetal descent not proportional

Common Clinical Conditions in Women with ineffective Labor

- **Inadequate cervical dilatation or descent**
 - Protracted labor – slow progress
 - Arrested labor - no progress
- **Feto-pelvic disproportion**
 - Excessive fetal size
 - Inadequate pelvic capacity
 - Malpresentation or position of fetus
- **Ruptured membranes without labor**
- **Cord prolapse**

Thank You