OBSTETRIC EMERGENCIES

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Objectives

By the end of these lessons, the student will be able to describe emergency obstetric conditions under the following sub-headings:-

1. Definition
2. Predisposing factors
3. Causes
4. Pathophysiology
5. Diagnosis and Investigations
6. Clinical features
7. Medical and Nursing Management
8. Complications

Introduction

- Obstetrics is a branch of medicine and surgery dealing with pregnancy, labour and the puerperium
- Emergency refers to a sudden crisis requiring urgent intervention
- Abnormal Midwifery is the health care profession in which providers identify potential complications of pregnancy, delivery and postpartum period and offer appropriate, timely interventions.

- Emergency Obstetric Care refers to a set of minimal health care elements, which should be availed to all women during pregnancy and delivery.
- It includes both life saving and emergency measures e.g. caesarean section, manual removal of placenta, etc. as well as non-emergency measures (e.g. use of the partograph to monitor labour, active management of the third stage of labour, etc.)

- Emergency Obstetric Care is a component of Safe Motherhood Initiative (SMI) and its functions are generally categorized as Basic Emergency Obstetric Care (BEmOC) and Comprehensive Emergency Obstetric care (CEmOC).
- **Basic** Emergency Obstetric Care includes:
  1. Administration of IV antibiotics.
  2. Administration of magnesium sulphate (anticonvulsants).
  3. Administration of parental (IV) oxytocics.
  4. Performing manual removal of the placenta.
  5. Performing removal of retained products.
  6. Performing assisted vaginal delivery (e.g. by vacuum extraction).
  7. Performing newborn resuscitation

- **Comprehensive** Emergency Obstetric Care includes all the seven above, PLUS:
  8. Performing surgery (Caesarean Section), including provision of emergency obstetric anaesthesia.
  9. Administration of blood transfusion.

Examples of Obstetric Emergencies

1. Cord Presentation/ Cord Prolapse
2. Antepartum Haemorrhage (APH)
3. Postpartum Haemorrhage (PPH)
4. Raptured Uterus
5. Amniotic Fluid Embolism
6. Acute Inversion of the Uterus
7. Fetal Compromise or Fetal Distress
8. Maternal Distress or Maternal Fatigue
9. Vasa Praevia
10. Pre-eclampsia
11. Eclampsia
12. Obstetric Shock
13. Shoulder Dystocia

1. Cord Presentation and Cord Prolapse

- **Cord presentation** is a condition whereby a loop of the umbilical cord lies in front of the presenting part and the membranes are still intact. Pressure on cord diminishes O_2 supply.
- **Cord prolapse** is a term used when the loop of umbilical cord lies in front of the presenting part and the membranes are ruptured.
- **Occult cord presentation/prolapse** describes the condition that occurs when the umbilical cord lies alongside/beside, but not in front of, the presenting part and the membranes are either intact (presentation) or have raptured (prolapse).
- Cord presentation and cord prolapse are considered as obstetric emergencies and complications of labour and delivery hence need immediate intervention.

Causes of Cord Prolapse

1) Multiple pregnancy esp. 2nd twin
2) Contracted pelvis: because the membranes may rupture before the head has engaged.
3) High head: the membranes may rupture spontaneously when the foetal head is still high, permitting cord to slip downwards.

   -Artificial rupture of the membranes (ARM) is contraindicated in high head.
4) Low implantation of the placenta,

5) Marginal insertion of the cord and an abnormally long cord.
6) Prematurity, preterm and/or Small for Gestational Age (SGA) babies; there is more room between the small foetal head and the maternal pelvis.
7) Polyhydramnios: the cord is likely to be swept down in a gush of liquor when the membranes rupture spontaneously (or by ARM).

8) Mal-presentation/malposition; any condition in which the presenting part does not fit well into the lower uterine segment will permit the umbilical cord to slip down in front of the presenting part e.g. breech, OPP.
9) Multiparity; presenting part may or may not be engaged when membranes rapture and malpresentation is more common

Diagnosis of Cord Presentation and Cord prolapse during labour

During labour, cord presentation or prolapse can be diagnosed on V.E as follows;

- On vaginal examination, a soft pulsating mass can be **felt** in front of the presenting part behind the fore waters (with the membranes intact) *–cord presentation*
- A cord may be felt in the vagina or **seen** at the vulva either pulsating or not pulsating and the membranes have ruptured *(cord prolapse).*
- Abnormalities of fetal heart rate *(fetal bradycardia)*
- **DDX;** foetal membranes, footling breech, compound presentation.

Management of Cord Presentation

- Assess the cervical dilatation, presenting part, status of the cord i.e. whether pulsating or not and the FHR.
- As soon as you confirm cord presentation on VE, (which is rare), you should remove your fingers, taking care not to rupture the membranes incase they are still intact.
- Reassure the mother and explain to her the situation at hand.
- Ask your assistant to inform the doctor and theatre staff to prepare for emergency caesarean section while you prepare the mother for the section. Provide O_2 if necessary.
- Take the mother to theatre as soon as possible as the membranes may rupture at any minute

Management of Cord Prolapse

- If you diagnose prolapse of the cord while performing a vaginal examination, check for the following factors that determine the method of management;
  a) Pulsation of the cord,
  b) Cord not pulsating,
  c) Stage of labour (cervical dilatation); whether she is in first or second stage,
  d) Adequacy of the pelvis.

NB: If the cord is pulsating you must act very fast in order to save the baby.

Pulsating Cord with Mother in First Stage

- Explain to the mother the situation at hand while you push up the presenting part so as not to compress the cord.
- If the cord is outside the vagina (cord has prolapsed), gently replace it in the vagina to prevent spasm and keep warm *(valium injection can also be given to relieve cord spasms)*.
- Instruct your assistant to inform the obstetrician and the theatre staff, and keep the mother in **knee chest position** without removing your fingers. Continue elevating the presenting part until the patient is in theatre.
- Your assistant should give the mother **oxygen** by facemask while someone else **draws blood** for grouping and cross matching.

Cont'…

- At this point you should fix an intravenous line and keep the vein open by setting up an infusion.
- The mother should be asked when she last had a meal and gastric aspiration should be commenced if necessary.
- Once consent has been taken, the mother should be rushed to the theatre as soon as possible.

NB: If the cervical os is fully dilated and the pelvis is adequate, you should encourage the mother to push.

Cont...

- Tell the assistant to inform the doctor and prepare for vacuum extraction.
- Continue encouraging the mother to push and give a generous episiotomy to hasten the delivery of the baby.
- Should the doctor arrive before the baby is out, he may perform vacuum extraction.
- If there is mal-presentation, the patient should be quickly prepared for caesarean section.

If the Cord is not Pulsating

- The procedure to be followed varies from institution to institution. In some institutions you may be able to personally inform the patient of the situation. Otherwise, you may have to wait until the doctor comes to tell the patient about her dead foetus.
- If the patient is in first stage of labour and the pelvis is adequate, let nature take its course and deliver a fresh stillbirth (counsel the mother).
- If the patient is in the second stage, she is encouraged to push and she will deliver a stillbirth.
- In case there is a contracted pelvis, a caesarean section should be performed in spite of the death of the foetus.

Management of Cord Prolapse

- Perform V.E.?
- Note if the cord is pulsating
- Note cervical dilatation
- If cord is outside, replace in the vagina to keep warm

Is the cord pulsating?

Yes

In first stage of labour, do not remove the fingers;

- Elevate the presenting part
- Reassure the mother
- Position in knee/chest
- Tell your assistant to give oxygen by facemask
- Inform the doctor and theatre
- Prepare for Caesarean Section
- Draw blood for cross matching
- Start IV line
- Prepare the mother within 5 minutes or less and wheel to theatre

Mother is second stage

Cervical os fully dilated

- Explain to the mother the situation to gain her cooperation
- Tell your assistant to inform the doctor and prepare for vaginal delivery

No

It's no longer an obstetric emergency.

- Explain to the mother the situation
- If mother is in first stage of labour, and pelvis is adequate, nature is allowed to take its course and deliver a stillbirth

If mother is in second stage of labour;

- Explain the situation and reassure her
- Exclude cephalo-pelvic disproportion
- Encourage her to push

Mother either first or second stage with contracted pelvis

- Explain the situation
- Prepared for extraction of a stillbirth

Complications of Cord Presentation and Cord Prolapse

- Asphyxia Neonatorum or
- Respiratory Distress or
- Neonatal Death or Still Birth due to cord compression
- Hypofibrinogenemia
- Puerperal Sepsis

ANY QUESTIONS SO FAR ?

2. Antepartum Hemorrhage (APH)

- **Definitions**

1. Bleeding from the genital tract in late pregnancy after 24 weeks of gestation and before the onset of labour.
2. Per vaginal bleeding during pregnancy whose cause may be placenta praevia or placenta abruption usually presenting in the last trimester of pregnancy

- **NB:** this is an obstetric emergency and major complication of pregnancy hence immediate interventions are required to save the live of both the mother and the foetus.
- **DDX:** labour (bloody show), cervical erosion, cervicitis, cervical polyp, carcinoma, trauma, uterine rapture.

Causes of APH

1. Placenta Praevia
2. Placenta Abruption *(Abruptio placenta)*

1. APH due to Placenta Praevia

-placenta praevia refers to an abnormal implantation of placenta in the anterior or posterior lower uterine segment; at or near the internal cervical os causing bleeding.

-four types (grades) of placenta praevia;

a) **Grade I**-only a tip of placenta in lower segment
b) **Grade II** –placenta partially located in the lower uterine segment near the internal cervical os.
c) **Grade III** – placenta located completely over the internal cervical when closed upto 4cm dilated
d) **Grade IV** – placenta is centrally located over the internal cervical os until full dilatation.

Signs and Symptoms of Placenta Praevia

i. Painless vaginal bleeding whose onset is at rest and sudden; blood is scanty or heavy bright red in colour. Bleeding tends to monitor degree of shock.
ii. Pale looking patient, the degree of which corresponds to the amount of blood loss.
iii. The presenting part may be high or abnormal due to mal-presentation and/or mal-position
iv. No tenderness in the abdomen.
v. Soft and relaxed uterus
vi. Foetal parts are easily palpable
vii. Foetal heart sounds are usually present.

Management of Placenta Previa

- Bed rest if mother responds to conservative mgt.
- Do surfactant test if mother has not gone into labour at 38/40
- If bleeding starts after 38/40, examine in theatre under GA for c/s
- If placenta not felt in anterior part, do ARM and start syntocinon drip to induce labour
- If bleeding is due to type ii, iii, and iv posterior, do c/s
- If bleeding is followed by premature labour, 2 doses of dexamethasone is given to promote lung maturation
- Consider giving IVF if bleeding is severe (D5% alternate with N/S)
- Give analgesics to relieve pain e.g. pethidine 100mg or morphine 15mg
- Transfuse mother once blood is ready
- If severe, prepare the woman for Em c/s and be prepared to receive an asphyxiated baby.

2. APH due to Placenta Abruption

-Placenta abruption refers to premature or early separation of the normally implanted placenta usually occurring after 28 weeks gestation; resulting to retro-placental bleeding which can be of revealed, concealed or mixed type.

Predisposing factors to Placental Abruption

- Placental insufficiency incase of anaemia, hypertension
- Trauma to the abdomen
- Polyhydramnios
- Multiparity due to laxed uterine muscles
- External cephalic version

Causes of Abruptio Placenta;

- Toxaemia,
- Trauma,
- Sudden uterine decompression,
- Abnormally short umbilical cord due to traction

Signs and Symptoms of Abruptio Placenta

i. Painful per vaginal bleeding
ii. Constant abdominal pain
iii. Tender abdomen
iv. Woody hard and tense uterus
v. Foetal sounds are absent in severe cases

Types of Placenta Abruption

a) **Revealed type**

-All the blood lost is seen dripping down from the cervix to the vaginal canal

b) **Concealed type**

-The bleeding occurs at the placental site and remains trapped between the uterine wall and the placenta forming a clot. No vaginal bleeding is seen.

c) **Mixed type**

-Has features of revealed and concealed type.

Comparison between placenta praevia and placenta abruption

	Placenta Praevia	**Placenta Abruption**
Bleeding	Typically present after 22 weeks gestation; may be precipitated by intercourse	Typically present after 22 weeks gestation; may be retained in the uterus.
Pain	Painless	Constant abdominal pain
Abdomen	Non-tender on palpation	Tender on palpation
Uterus	Soft and Relaxed	Tense and woody hard.
Foetal condition	Normal (Fetal parts easily palpable, fetal heart sounds usually present)	Abnormal (Fetal distress or absent/abnormal fetal heart sounds)
Shock	Sometimes present	Sometimes present

Management of *abruptio placenta*

- Determine the *gestational age*, *maternal* and *foetal* condition, and *amount of bleeding*;

  -If gestation is below 36/40, and the maternal and foetal condition is satisfactory (i.e. normal FHR, no shock, bleeding is controlled), conservative management is given and continue with bed rest at home.

  -If gestation is above 36/40, do ARM to induce labour. In case bleeding is severe with more than 2 litres blood loss, **treat the woman for shock**. Consider IVF and blood transfusion if the bleeding is severe.

  -Monitor maternal BP and Pulse regularly; take her for C/S and prepare to receive an asphyxiated baby.

  -In case foetus is dead, and pelvis is adequate, don't take the mother for c/s but allow vaginal delivery since it causes uterine contractions that reduce bleeding.

Complications of APH

1) Puerperal Sepsis incase it was concealed type
2) Anaemia due to excess bleeding
3) Kidney failure
4) PPH due to hypofibrinogenemia or few living ligatures
5) Asphyxia neonatorum
6) Preterm/SGA babies
7) Still birth
8) Neonatal death as an outcome
9) Maternal death

3. Postpartum Hemorrhage

Learning Objectives;

- By the end of the lesson, the learner should be able to;
  - *a) Define the term Postpartum Haemorrhage (PPH)*
  - *b) Differentiate between primary and secondary PPH*
  - *c) List and explain four causes of post-partum hemorrhage*
  - *d) Identify predisposing factors to PPH*
  - *e) Outline the clinical features of PPH*
  - *f) Explain the diagnosis of PPH*
  - *g) Discuss the management of PPH depending on the causes,*
  - *h) List five complications of PPH*

Definition of Postpartum Haemorrhage;

- PPH refers to excessive bleeding from the genital tract (per vaginal) of an amount exceeding 500ml soon after delivery up to the end of puerperium (6weeks to max.12 weeks postpartum).
- If it occurs within 24 hours, it is called primary PPH, but if it occurs after 24hours of delivery and up to about 12 weeks postpartum, it is called secondary PPH.
- It is an obstetric emergency and a major complication of puerperium hence immediate interventions required to save the life of the mother.

Predisposing Factors to PPH

i. Previous history of PPH or retained placenta
ii. High Parity resulting in uterine scar tissue
iii. Presence of fibroids
iv. Maternal Anaemia
v. Ketoacidosis
vi. Multiple Pregnancy

Causes of PPH (?4Ts)

a) ***Atonic uterus*** (T-Tone);
b) ***Retained products of conception*** **(**T-Tissues);
c) ***Trauma*** to the genital organs (T-trauma)
d) ***Coagulation disorders*** (T-Thrombin);
e) ***Infections*** *e.g. 2^{o} PPH may be due to puerperal sepsis and uterine sub-involution*

NB: The most important way of managing PPH is to determine the cause of the bleeding then manage the condition as per the cause.

a) Atonic uterus and retained products of conception

- Atonic uterus refers to the failure of the uterus to contract and retract after delivery of the baby.

Causes of atonic uterus

1) Precipitate Labour or Prolonged labour
2) Mismanagement of labour
   - ➢ High dose of analgesics during first stage of labour
   - ➢ Full bladder interferes with the contraction of the uterus
   - ➢ Rapid expulsion of big babies such that the uterus has no time to retract effectively
   - ➢ Retained blood clots and membranes
   - ➢ Mishandling the fundus e.g. kneading produces irregular contractions
   - ➢ Pulling the placenta before separation
3) Grand multi-parity due to laxity of uterine muscles

Causes of atonic uterus (Cont'…)

4) Over-distension of the uterus muscles during pregnancy due to e.g. polyhydramnios or multiple pregnancy,
5) Placenta praevia; when lower uterine segment cannot contract effectively to control bleeding
6) Placenta abruption
7) Placenta accreta (placenta is adherent with chorionic villi penetrating into myometrium and interfere with contraction
8) Uterine blocks e.g. uterine fibroids
9) Endocrine disorders e.g. failure to produce oxytocin

Signs and symptoms of atonic uterus

- Visible bleeding starts a few minutes after birth of the baby and gushes out like a spring
- Uterus fills big, soft and doesn't contract; may be filled with blood or blood clots.
- Uterus becomes flabby and may not be palpable
- **Signs of shock** set in (low BP, rapid weak pulse, cold clammy skin, pallor of conjuctiva, palm, altered level of consciousness etc. evidenced by mother becoming restless or drowsy).

Management of Atonic Uterus and Retained Products of Conception

(Myles 2nd Ed. 2010, reprinted 2014 pg.543)

i. *When placenta is in situ;*

- Ensure bladder is empty,
- Give ergometrine to enhance contractions
- Massage the fundus of the uterus until it hardens
- When the uterus is contracted, check for signs of placental separation and if present try CCT
- If placenta delivery is not successful yet the bleeding continues, give 2nd dose of ergometrine and prepare for bimanual removal of the placenta under GA

ii. ***When placenta is out;***

- Massage the uterus for contraction but take care not to cause inversion.
- Empty the bladder or cathetherize as appropriate
- Expel any clots in the uterus
- Repeat dose of ergometrine or syntometrine
- Start syntocinon 5-20 i.u. in 500ml 5% dextrose to run at rate of 40 drops/min and check the effect after 2 hours
- Check whether the blood is clotting or not to rule out coagulation disorder

Cont'…

- Start IVF dextrose 5% alternate with N/S
- If bleeding continues despite well contracted uterus, examine the birth canal for obvious trauma
- Examine the placenta to rule out succenturate lobe
- Take blood samples for GXM
- Give oxygen by mask to increase the oxygen content in blood
- Administer analgesic e.g. pethidine to induce rest and allay anxiety.
- Estimate blood loss and transfuse when the blood is ready

b) PPH due to Trauma

- This is diagnosed when bleeding starts immediately after birth of the baby and continues even when the uterus is well contracted.
- There is obvious trauma at uterus, cervix, vaginal wall, vulva or the perineum.

Management of PPH due to Trauma

- Management depends on the location of the trauma, perineal tears or vaginal tear - *ligate and repair*
- Tears in the upper vaginal wall and cervix are repaired in theatre under GA
- If rapture of the uterus – *repair in theatre or do hysterectomy*
- If uterus is inverted, *do manual replacement* and *start syntocinon drip to contract the uterus*.

c) PPH due to Coagulation Disorder

- Diagnosis is made when there is no trauma and the uterus is well contracted.
- It is mainly due to hypofibrinogenemia which maybe as a result of amniotic fluid embolism or sepsis
- It is managed by giving IVF with plasma expanders and fibrinogen preparations
- It may also need transfusion

Secondary PPH *(PPH ≥ 24 hrs)*

Definition

-Secondary PPH refers to excessive per vaginal bleeding >500ml after 24 hours of delivery extending up to the end of puerperium (6-12 weeks postpartum).

- It occurs when there are retained products of conception, or intrauterine infections.
- Presents with persistent red lochia and excessive hemorrhage.
- It is best prevented by observing aseptic technique during labour and examining the placenta and membranes well.
- All clients who have PROM should be put on broad-spectrum antibiotics

Specific Management of Secondary PPH

- Call for help, inform doctor,
- Reassure mother and significant others
- Massage uterus if still palpable
- Expel any clots
- Encourage mother to empty bladder
- Give uterotonics e.g. ergometrine i.m or i.v.
- Assess all pads/linen soaked with blood to assess the volume of blood lost.
- If bleeding persists, reassure mother, discuss treatment options and prepare her for theatre.

ANY QUESTIONS SO FAR?

4. Raptured Uterus

Learning Objectives;

- By the end of the lesson, the learner should be able to;
  - *a) Define the term raptured uterus*
  - *b) Explain two categories of raptured uterus*
  - *c) Identify nine predisposing factors to rapture of uterus*
  - *d) List the causes of uterine rapture.*
  - *e) Describe the pathophysiology of raptured uterus.*
  - *f) Outline the signs of impending rapture and actual signs of raptured uterus*
  - *g) List the early and late signs of scar rapture.*
  - *h) Discuss the management of raptured uterus*
  - *i) List at least six complications of raptured uterus*

Definition of Rapture of the Uterus;

- The rapture of the uterus is a tear in the wall of the uterus due to trauma, pregnancy related complications e.g. atonic uterus or infection.
- It should not be confused with acute inversion of the uterus or abnormal uterine action.
- Raptured uterus is a serious complication which should not occur in today's obstetric care where there is good antenatal and intra partum care.
- This tear is divided into two categories.
  a) **Complete or Intra Peritoneal**
  b) **Incomplete or Extra Peritoneal**

a) Complete or Intra Peritoneal

- This is a tear in the wall of the uterus, which involves all the three muscle layers of the uterus and extends to the peritoneum, i.e. the rapture of endometrium, myometrium and perimetrium or peritoneum.

b) Incomplete or Extra Peritoneal

- This is the tearing of the uterus, which involves only two muscle layers of the uterus i.e. the endometrium and myometrium.
- Tears can occur prenatally, during labour or delivery and may endanger the lives of both mother and foetus.

Predisposing Factors to Uterine Rapture

1) Polyhydramnios and multiple pregnancy
2) Pregnancy occurring within six months post caesarean section with the placenta situated on the scar
3) Obstetric manipulation or operations such as during internal cephalic version
4) Previous operation of the uterus
5) Foetal pelvic disproportion
6) Myomectomy-removal of myoma e.g. uterine fibroids

Cont'…

7) Grand multi-parity
8) Placental implantation abnormalities e.g. placenta accreta, increta, and percreta)
9) Trauma; uterine rupture may also occur after a dramatic increase in intrauterine pressure as seen in severe cases of blunt abdominal trauma, such as motor vehicle crashes.

Causes of Uterine Rapture

- **Prenatally**, a ruptured uterus may occur due to a weak scar.
- **During labour and delivery** or when not in labour a ruptured uterus may occur as a result of:
  i. Obstructed labour, for example in malpresentation, cephalopelvic disproportion (CPD), contracted pelvis
  ii. Intrauterine manipulation, for example, internal cephalic version of second twin
  iii. Forceps delivery and vacuum extraction
  iv. Shirodkar stitch in labour
  - *('Shirodkar' was an Indian Obstetrician, the 'stitch' refer to nylon suture used to close internal os of cervix due to cervical incompetence and failure to recognize or remove the stitch before term or onset of labour may lead to uterine rapture).*

Cont'…

v. Rigid cervix
vi. Breech delivery
vii. Multi-parity, due to the degeneration of the uterine muscles
viii. Previous trauma or classical C/S scar
ix. Manual removal of placenta
x. Perforation of uterus
xi. If myomectomy was done,
xii. Abuse/excessive or injudicious use of oxytocin drugs e.g. induce when there is uterine scar or CPD
xiii. Previous dilatation and curettage

Pathophysiology of Uterine Rapture

- A distinction is made between uterine rupture and dehiscence. **Uterine dehiscence** is myometrial separation at a site of a uterine scar from previous surgery, and the uterine serosa remains intact. The vertical scar from a classic cesarean section greatly weakens the muscular active segment of the uterus. Increase in uterine pressure may result in tearing at these areas of weakness.
- Uterine rupture, in contrast, involves the entire thickness of the uterine wall, resulting in communication between the uterine and peritoneal cavities

Pathophysiology Cont'…

- The placenta and fetus may then be extruded into the peritoneal cavity.
- Bleeding usually occurs from the edges of the defect, but it may vary from minimal to massive, depending on the size and relative vascularity of the defect and whether the defect involves the placenta or extends into uterine or vaginal blood vessels.
- In complete uterine rupture, the defect may originate from a previous surgical scar or, less commonly, it may occur spontaneously in an unscarred uterus.

Clinical Presentation of Raptured Uterus

i. Simple uterine dehiscence at the site of a previous low transverse cesarean section may be asymptomatic and discovered incidentally at the time of repeat cesarean delivery or manual uterine exploration after vaginal delivery.
ii. Often local tenderness is reported.
iii. A sudden onset of pain may be seen with an increase in uterine irritability
iv. Cessation or disappearance of established uterine contractions in a labouring patient.

Clinical Presentation Cont'...

v. Fetal heart rate abnormality is often the earliest sign of uterine rupture in the laboring patient.
vi. Fetal movements may be absent; FH may be present or absent depending with site of rapture
vii. Palpable abnormalities on abdominal examination, recession of the fetal presenting part, and loss of fetal heart tones are seen in massive rupture
viii. Per Vaginal bleeding is variable and rarely reflects total blood loss. Simultaneous bleeding into the abdominal cavity is common, and signs of fetal distress, maternal hypovolemia, or shock may be seen with only minor vaginal bleeding

Signs of Impending Rapture

- Rapid Pulse
- Vaginal Bleeding
- Tenderness of lower abdomen
- Lack of descent and strong contraction
- Sharp pain at lower abdomen
- Rapture due to previous scar has no warning signs (silent).

Actual Signs of Scar Rapture

- During labour a classical scar is more likely to rupture than the lower segment scar.
- Actual signs of scar rapture are divided into two categories; early and late signs;

Early Signs of Scar Rupture

- *Constant lower abdominal pain* that worsens during a contraction.
- There is *fresh bleeding*, which may be mistaken for show.
- Contractions may continue but the *cervical os fails to dilate*.
- *Pulse rate is raised* due to shock and tends to increase slowly.

Some Late signs associated with scar rupture:

- Mother is *dehydrated*, shows ketosis and is in *severe pain*
- *Rapid pulse* and
- *Pyrexia* (T >38°C)
- *Poor urinary output*, concentrated with ketosis and often blood stained
- *Uterus gets moulded round the foetus*
- *Strong uterine contraction*, which *does not relax* between contractions
- A *Bandl's ring* may be seen
- On vaginal examination, the *vagina is hot and dry*

Late Signs of Scar Rapture Cont'…

- *Presenting part is high*, wedged and immovable
- There is *over lapping of foetal bones* and big caput succedaneum
- The *mother is exhausted before the rupture*, and she will probably cry out during the rupture and complain of a *sharp pain* in the lower abdomen
- She feels something has given way and soon presents with *shock*

Management of Uterine Rapture

- Combat shock by putting up an intravenous drip of saline and elevate foot of the bed; remove pillows and leave woman in flat position to avoid compression of lungs and diaphragm that may cause severe dyspnoea. Alternate D5% and N/S.
- Inform the doctor or theatre staff to prepare for an emergency caesarean section
- Take blood for grouping and cross matching (GXM)
- Reassure the mother and prepare her physically and psychologically for theatre (laparatomy, repair of uterus or hysterectomy); get informed consent for the procedure.

Cont'…

- The options to perform a hysterectomy or to repair the rupture depend on the extent of the trauma and the mother's condition;
  - *A hysterectomy is done if the rupture is beyond repair.*
  - *If the rupture is repaired, you should stress to the mother the importance of not conceiving until at least a year after the operation.*
- Administer analgesics e.g. pethidine PRN,
- The baby is removed in theatre from peritoneal cavity; antibiotics are infiltrated into the cavity and secretions drained to reduce puerperal infection

Cont'...

- Post operative care should be given just like in the case of caesarean section with special attention to the drainage tubes,
- If hysterectomy was not done, couples should be counselled to delay next pregnancy and attend ANC early.
- Emphasize that all subsequent pregnancies should always be delivered by elective caesarean section and there should be no trial of labour whatsoever.
- If hysterectomy was done, explain the consequences e.g. no more conception and if the couple wishes to have children, they should adopt.

Complications of Uterine Rapture

Among the several complications of ruptured uterus to the mother; these include:

1. Paralytic ileus,
2. Peritonitis,
3. Puerperal Sepsis,
4. PPH due to rapture,
5. Septicaemia
6. Urinary tract infection
7. Renal failure
8. Increase maternal morbidity and mortality rate

ANY QUESTIONS ?

5. Amniotic Fluid Embolism (AFE)

- This is a situation in which amniotic fluid gets into the maternal circulation through the sinuses of the placental site forming an embolus which obstructs coronary and pulmonary arteries usually occurs towards the end of the first stage of labour when the membranes have ruptured and there are strong uterine contractions.
- Amniotic fluid is rich in thromboplastin thus when it enters maternal circulation it utilizes fibrinogen in the blood giving rise to blood coagulation disorder (DIC). The emboli so formed obstructs one of the pulmonary arteries and coronary arteries and death may occur within one hour.

Cont…

- This is a very rare catastrophic condition which can occur at any gestation, but it is **most common at the end of the first stage of labour**.
- The body responds in two phases;
  a) In **the initial phase**, the pulmonary artery goes into vasospasm causing hypoxia.
  b) In **the second phase,** there is left ventricular failure, haemorrhage, and blood coagulation disorders followed by pulmonary oedema,

Predisposing factors to AFE

i. Hypertonic uterine action or overstimulation of the uterus by use of excessive oxytocic drugs.
ii. Multi-parity associated with early separation of the placenta (*Placenta abruptio*), where the barrier between maternal circulation and amniotic sac is breached and the placenta bed is disrupted
iii. Uterine trauma e.g. external version, uterine rapture
iv. Rapid precipitate labour (rapid contractions forces amniotic fluid into the maternal circulation).
v. Manual removal of the placenta.

Predisposing factors Cont'…

vi. Procedures like insertion of intrauterine catheter
vii. Rupture of membranes
viii. Caesarean section
ix. Inter-uterine manipulation, for example, internal podalic version

Signs and Symptoms of AFE

- Sudden onset of maternal respiratory distress
- Severe dyspnoea
- Hypotension due to shock
- Cyanosis
- Haemorrhage due to DIC
- Tachycardia
- Chest pain
- Pulmonary Oedema
- Convulsions and/or cardiac arrest (collapse) which may occur immediately after one hour.

Management of AFE

- Call the emergency response team because this is an acute emergency
- Prop up the patient to prevent lung compression and enhance air entry,
- Check SPO_2 and administer oxygen if SPO_2<94%
- Treat shock with IVF and maintain intake and output chart checking on urinary output
- Give *aminophylline* slowly to reduce bronchospasms,
- Administer *prednisolone* to counteract the effect of amniotic fluid in the lung tissue

Management of AFE Cont'…

- *Transfuse* with packed cells, plasma or give fresh whole blood or fibrinogen to combat hypofibrinogenaemia
- Give *clotting factors* e.g. Vit K. to counteract DIC.
- Incase of cardiac arrest, commence *resuscitation* at once
- *Assist the delivery* by vacuum extraction or forceps,

In most cases the prognosis is poor.

- The mother should be transferred to the intensive care unit.
- You should always remember that there are several complications associated with the condition and you should be on the look out for them.
- Also note that perinatal mortality and morbidity are high if amniotic fluid embolism occurs before the birth of the baby.

Complications of AFE

1. PPH due to hypofibrinogenemia
2. Acute renal failure due to excess blood loss
3. Disseminated intravascular coagulation (DIC),
4. Haemorrhage,
5. Cardiac Arrest (cardiovascular collapse)

ANY QUESTIONS SO FAR ?

6. Acute Inversion of the Uterus (AIE)

Learning Objectives;

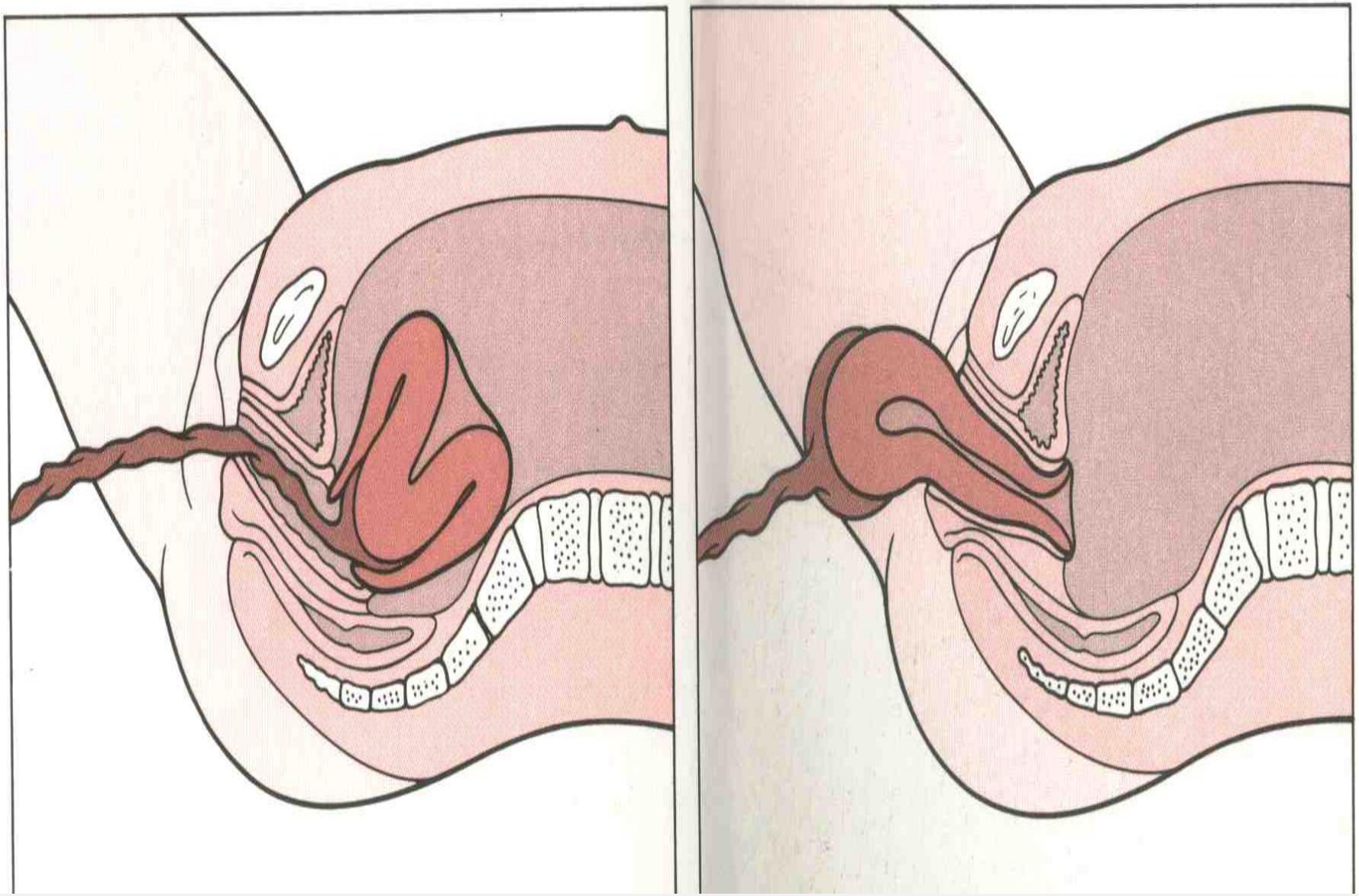
- By the end of the lesson, the learner should be able to;
  a) *Define and classify/ acute inversion of the uterus*
  b) *Identify the predisposing causes of acute inversion of the uterus*
  c) *Explain how to make a diagnosis of acute uterus inversion*
  d) *Outline the signs and symptoms of acute inversion of the uterus*
  e) *Discuss the management of acute inversion of the uterus*
  f) *List complications of acute inversion of the uterus*

Definitions

a) Inversion of the uterus refers to the situation where the uterine body is turned inside out partially or completely.

b) It is also used to refer to prolapse of the uterine fundus to or through the cervix and uterus is in effect turned inside out partially or completely.

- **NB:** This is a serious complication of third stage of labour and should be well diagnosed not to be confused with abnormal uterine action and/or uterine rapture.



Classification of Uterine Inversion:

i. **1st degree** (mild); where the fundus bulges into the internal os of the uterine cavity but does not protrude into the cervix. There is a dimple on the fundus on palpation; it may not be palpable abdominally since it is at the level of internal os.

ii. **2nd degree** (moderate); where the fundus/corpus of the uterus is inverted to the internal os and protrudes through the cervix and the uterus is left inside the vagina. It presents with abdominal pain, *signs of shock* (rapid pulse, low BP, shallow respiration and hemorrhage).

iii. **3rd degree** (severe); where both the uterine fundus, cervix and vagina are inverted, passing through the internal and external os and are visible at the vulva or vaginal orifice.

Cont'…

- In 3rd degree inversion of the uterus, there is severe abdominal pain due to traction of the ovaries, oviduct, and broad ligaments. There is severe bleeding and the signs of shock are evident *[i.e. cold clammy skin, pallor, dry mouth, low BP, rapid pulse, reduced urinary output]*.

NB: Classification of uterine inversion is also based on the time they occur;

- **Acute** refers to immediate prolapsed within 24hrs after delivery while the placenta is still attached.
- **Subacute**; refer to an inversion, which happens after 24 hrs up to 4 weeks;
- **Chronic** (>4 weeks) uterine inversion with or without detachment of the placenta.

The Predisposing Causes of Acute Inversion of the Uterus:

1. *Mismanagement of **third stage** of labour*
   - Combining fundal pressure and controlled cord traction to deliver placenta when the uterus is relaxed, (the hand pushes the relaxed muscle sac inside out).
   - Forceful use of fundal pressure before separation of placenta and when the uterus is atonic.
2. *Placenta abnormalities e.g. accreta, abruption,*
3. *Unknown cause, such as when the prolapse happens spontaneously (which is rare).*
4. *Sudden emptying of the gravid uterus*
5. *Short cord*

How to Diagnose Acute Inversion of the Uterus

- **Inspection;** If inversion is partial, the fundus will not be visible per vagina.
- **Palpation;** No fundus is palpable abdominally
- **VE;** On vaginal examination a mass may be felt
- **Physical Examination** reveals **Signs of Shock** especially pain, which is caused by the stretching of peritoneal nerves and the ovaries being pulled
- **Haemorrhage** between 800ml – 1,880ml, which depends on the degree of placenta adherent on the uterine wall

Management of Uterus Inversion

- Call for help from other midwives.
  - Remember that in an emergency you need effective teamwork. The more medical personnel, the quicker the work is done.
  - The faster the inversion is reversed, the less the risk to the mother.
- Inform the mother what has happened and reassure her.
- Once the other team members have arrived, do your part and if possible, be the one to give instructions as you all work together as a team.

Give instructions as follows:

- One of your assistants should call the doctor
- The other assistant to elevate the foot of the bed to facilitate blood supply to the brain hence helps to manage shock

- Fix a cannula, collect blood for GXM, then fix an intravenous infusion to prevent dehydration.
- Administer analgesics and sedatives e.g. pethidine 100mg to relieve pain; antibiotics due to risk of infection
- Clean the protruding uterus with antiseptic lotion e.g. hibitane,
- Repeat *ergometrine/syntocinon* to control hemorrhage if any

Cont'…

- Replace the uterus into the vagina and maintain it in position with perineal pad.
- If the placenta has not separated, leave it in situ until the uterus is replaced,

NB: The replacement should be done immediately because the uterus becomes oedematous making it larger and harder to replace.

Methods of Replacing the Uterus

a) **Last out, First in,**

- The part that came out last is replaced first e.g. the lower uterine segment is replaced first and fundus last through dilated cervix with the fingers of the hand in the vagina. Pressure is applied on an area near the cervix and gradually the corpus (body of uterus) is replaced. The fundus goes in last by pressure with palm of the hand. During replacement counter pressure is maintained on the abdomen to prevent the uterus from being pushed too far. Following replacement, *ergometrine 0.25mg* is given to make the uterus contract and control bleeding thus preventing subsequent inversion. While the hand remains there, strip the placenta unless it is placenta accreta.
- Continue with IVF and transfuse once blood is ready.

b) Use of Hydrostatic Pressure;

- A giving set is inserted into the vagina and the vagina douched with warm several litres of normal saline or hibitane through the giving set then vaginal orifice blocked or sealed by one hand.
- After pouring several litres of fluid, the pressure exerted by the fluid will build up in the vagina and distend the vagina pushing the inverted uterus back thus restores it to its normal position.
- Give *ergometrine 0.25mg i.v.* to induce contractions that will push the fluid out.

Cont'…

- The placenta can then be delivered by using control cord traction.
- If the doctor comes before you finish the procedure, they can take over.
- In a health centre you should refer the mother to the hospital for further management if you do not succeed.

c) Use of **Johnson's manoeuvre** as a way of replacing the uterus;

- Try to push the fundus using the palm of your hand. Direct the fundus to the vagina and towards the posterior fornix. Lift the uterus towards the membranes with steady pressure and return it to position. Once the uterus is in position, instruct your assistant to give oxytocin while you hold the uterus until you feel a hard contraction.

Medical Management of Inverted Uterus

- If inversion is not possible manually, it may be due to a cervical constriction ring.
- The doctor will prescribe a **relaxant** to relax the cervical os and facilitate the replacement of the inversion

Complications of Inverted Uterus

1. Severe Shock
2. Puerperal Sepsis
3. Recurrent in subsequent deliveries
4. Infertility due to blocked tubes
5. Intestine damage
6. Uterine appendages.
7. PPH

ANY QUESTIONS SO FAR ?

7. Foetal Compromise (Foetal Distress)

- **Learning Objectives;**
  - *a) Define fetal distress/compromise*
  - *b) Highlight the causes of fetal distress*
  - *c) Describe pathophysiology of fetal distress*
  - *d) Outline the clinical features of fetal distress*
  - *e) Explain the diagnosis of fetal distress*
  - *f) Discuss the management of fetal distress*
  - *g) List five complications of fetal distress*

Definition;

- Fetal compromise refers to foetal hypoxia and acidosis that occurs in utero as response to stress resulting to FHR abnormalities
  - Slowing of FHR below 100b/min is an indication severe fetal compromise.
  - If there are abnormal heart rates after contraction observed 3 times, action should be taken immediately to deliver the fetus

Causes of Fetal Compromise

1. *Cord presentation or prolapse;* the cord may be pressed by the presenting part and cut oxygen supply to the fetus. It may also make knots around the neck of the fetus causing hypoxia
2. *Placental insufficiency;* there is a constriction of blood vessels reducing blood supply. This may occur in conditions like placenta praevia, abruption, PET (Pre-eclamptic Toxaemia of pregnancy or pre-eclampsia).
3. *Abnormal uterine contractions* especially hypertonic uterus reduces blood supply to the fetus leading to hypoxia
4. *Prolonged labour* especially if the membranes have raptured

5. *Prematurity* in which case the fetus is not fully developed
6. *Drugs* e.g. opioid analgesics (pethidine), GA depress respiratory centre
7. *APH due to placenta abruption;* i.e. premature separation of the placenta

Signs and Symptoms of fetal distress

i. Abnormalities in fetal heart rate patterns (CTG);
   - Baseline or basal rate abnormalities e.g. tachycardia (FHR ≥160 b/min) or bradycardia (FHR ≤110 b/min)-this is a sign of severe asphyxia
   - Variability abnormalities e.g. increased or decreased variability
   - Periodic change abnormalities e.g. accelerations or decelerations (early, late or variable decelerations) – see Myles for details!

ii. Meconium stained liquor (MSL) - due to lack of oxygen that leads to poor muscle tone making the anal sphincture to relax.
   - NB: Meconium alone is not significant because during second stage in breech it could be passed due to compressions of the abdomen

iii. Convulsive foetal movements which may cause IUFD

Management of Foetal Compromise

- Position the woman should lie in left lateral position to prevent occlusion of inferior vena cava and to relieve pressure on the cord.
- Raise the foot of the bed to improve venous return and prevent hypotension
- Put the mother on complete bed rest
- Prolonged and obstructed labour should be avoided
- Any client with CPD, malpresentation, malposition should be advised to deliver through elective C/S
- Inform obstetrician and paeditrician in anticipation for asphyxiated baby. Have resuscitation tray ready.
- Start IVFs to restore arterial pressure and increase uterine blood flow

Management Cont'…

- Stop any syntocinon infusion if it has been running as this may cause fetal death due to hypertonic contractions. Consider use of *tocolytics* e.g. salbutamol to decrease hypertonicity of uterus
- Do VE to rule out cord prolapse and assess cervical dilatation
- Observe the amniotic fluid for any meconium staining and report on the type e.g. thick MSL indicates severe fetal distress
- Give oxygen by mask to raise maternal-fetal oxygen
- If near 2nd stage of labour, deliver her as soon as possible by giving an *episiotomy* to hasten delivery and encourage her to push
- If the mother cannot push the baby out, *forceps delivery* or *vacuum extraction* can be done.

Complications of Fetal Compromise

- Asphyxia Neonatorum
- Mental Retardation
- IUFD (intrauterine foetal death)
- Still Birth

8. Maternal Distress/Maternal Fatigue

Learning Objectives;

- By the end of the lesson, the learner should be able to;

a) Define maternal distress
b) List the causes of maternal distress
c) Outline the clinical features of maternal distress.
d) Explain the diagnosis of maternal distress
e) Discuss the management of maternal distress
f) List five complications of maternal distress

Definition;

- Maternal distress or fatigue refers to a situation where the mother's condition has deteriorated due to stress during labour.
- It occurs when the stress and strain of labour is so much that the mother cannot bear any more.
  - **NB:** Labour should be terminated as soon as the woman gets into maternal distress

Causes of Maternal Distress

- Prolonged labour
- Obstructed labour
- Malpresentation
- Malposition
- Pre-eclampsia
- Eclampsia
- CPD
- Cardiac disease in pregnancy and labour
- Severe anaemia

Signs and Symptoms of Maternal Distress

- Increased pulse rate (HR>90b/min)
- Increased temperature above 37°C
- Restlessness and fatigue
- Weakness
- Sweating
- Ketone bodies in urine
- Smell of acetone
- Oliguria
- Vomiting
- Dehydration
- Anxiety

Management of Maternal Distress

- *Manage the underlying cause* of distress
- *Sedate* the mother to promote rest
- *Give IVFs* e.g. D5% to provide energy and prevent dehydration
- *Administer oxygen* by mask to prevent foetal hypoxia
- Closely *observe the FHR* and *uterine contractions*
- If cervical dilatation is < 6 cm, *prepare for C/S*
- If the mother is in or approaching 2nd stage, an *episiotomy* is given and when cervix is fully dilated, *vacuum extraction* is done

ANY QUESTIONS SO FAR?

9. Vasa Praevia

Definition

- Vasa praevia refers to bleeding from fetal umbilical vessels as they traverse the placental membranes.
  - The *bleeding is purely fetal in origin* and therefore is an unusual cause of upper genital tract bleeding in that it poses almost no maternal risk, but may rapidly lead to fetal compromise and death.

Pathophysiology of Vasa Praevia

- Fetal umbilical vessels usually insert centrally onto the placenta
- In vasa praevia, a lateral or velamentous insertion of the umbilical cord occurs onto the chorionic plate of the placenta or when there is an extra (succenturiate) lobe.
- With these potentially dangerous variants, fetal vessels traverse within the placental membranes prior to their insertion

Cont...

- If these fetal vessels cross the lower uterine segment and present in advance of the fetus, they are then vulnerable to rupture or laceration with rupture of the placental membranes.
- Because the circulating blood volume of a fetus is small (approximately 300 to 500 ml), relatively unimpressive amounts of vaginal bleeding may easily lead to severe fetal compromise and fetal exsanguination.
- Spontaneous or artificial rupture of placental membranes or descent of the fetal presenting part may cause rupture of these vessels. Painless vaginal bleeding as well as the rapid occurrence of fetal compromise occur soon thereafter.

Clinical Presentation of Vasa Praevia

- Painless Vaginal bleeding and/or
- Rapid occurrence of fetal heart rate abnormalities are the hallmarks of ruptured vasa previa. Sudden fetal bradycardia may be seen.
- Occasionally, the aberrant fetal vessels are detected prior to their rupture at the time of elective cesarean section, by ultrasound as an incidental finding, or by palpation of fetal vessels overlying the presenting part during vaginal examination. Unfortunately, however, vasa previa is seldom recognized prior to vessel disruption.

Management of Vasa Praevia

- If vasa previa is diagnosed prior to vessel disruption, delivery by cesarean section is indicated.
- In ruptured vasa previa, if the fetus is viable, delivery should be accomplished by the most expeditious route possible, usually cesarean section.
- The need for intensive neonatal resuscitation, including transfusion therapy, should be anticipated.

ANY QUESTIONS SO FAR ?

Case Scenario

- Mrs.T Para 2+0 is admitted to the antenatal ward at 32 weeks of gestation with a diagnosis of severe pre-eclampsia.
  a) Outline the classification of pre-eclampsia (3 marks)
  b) Explain the pathophysiology of pre-eclampsia (4 marks)
  c) State five cardinal signs of Impending Eclampsia (5 marks)
  d) Describe the specific management of Mrs. T until she reaches term (6 marks)
  e) List the effects of pre-eclampsia;
     i. two to the mother (1 mark)
     ii. two to the fetus (1 mark)

10. PRE-ECLAMPSIA

Specific Learning Objectives;

- By the end of the lesson, the learner should be able to;

i. *Define pre-eclampsia*
ii. *List predisposing factors to pre-eclampsia*
iii. *Explain the causes of pre-eclampsia*
iv. *Explain the pathophysiology of pre-eclampsia*
v. *Describe the diagnostic investigations and findings of pre-eclampsia*
vi. *Outline the classification of pre-eclampsia*
vii. *Outline the clinical features/presentation of pre-eclampsia*
viii. *List the effects of PET to the mother and the fetus*
ix. *Discuss the medical and nursing management of pre-eclampsia*
x. *List complications of pre-eclampsia*

Definition of Pre-eclampsia

- Pre-eclampsia refers to hypertensive disorder of pregnancy characterized by onset of *elevated BP (acute hypertension)* and *proteinuria, oedema* (face, hands) or both newly diagnosed during pregnancy after 20 weeks' of gestation in a patient known previously to be normotensive.
  - Exceptions are in the cases of trophoblastic disease or multiple gestation, when pre-eclampsia may appear before 20 weeks' gestation
  - Pre-eclampsia is a disorder of unknown aetiology peculiar to the human pregnancy.
  - It is the most common medical complication of pregnancy, and a major cause of maternal morbidity and mortality worldwide

Predisposing factors to Pre-eclampsia

- Age-maternal age < 20 years or age > 40 years;
- Gravidity –preeclampsia is a disease of young primigravida accounting for 85% of cases
- Nulliparity; woman who has never held a pregnancy to viability state
- Genetics-chromosomal abnormalities
- Multiple pregnancy
- Polyhydramnios
- Essential Hypertension or Pregnancy aggravated HTN
- Chronic (Pre-existing) Hypertension in older women
- Family History of pre-eclampsia
- Mother with past history of pre-eclampsia
- Underlying renal disease and UTI

Predisposing Factors Cont'…

- Race (Black Race)
- Obesity
- Diabetes
- Hydatidiform mole
- Non-immune hydrops fetalis
- Collagen vascular disease
- Vitamin D deficiency
- Periodontal disease
- Antiphospholipid syndrome
- Low socio-economic status

Causes of Pre-eclampsia (Theories)

- No known cause,
- The following factors are associated theories;

a) ***Immunological mechanisms***; supported by fact that:
   - more common in first pregnancy or
   - multiparous women who have changed spouses.

b) ***Genetic Predispositions;***
   - Occurrence in some families more than others.
   - Single gene theory has been identified.

c) ***Dietary deficiencies vs Increased Metabolism***

- ? Dietary protein (high pre-pregnancy body weight predisposes to PET)
- ? Calcium or zinc deficiency where supplementation has reduced the incidence of PET.
- ? Obese mother accumulates fats that lead to hypertension

d) ***Vasoactive compounds***

- Endothelins - (vasoconstrictor)
- Nitric oxide (vasodilator) low levels or absence may play a role in PET

Pathophysiology of Pre-eclampsia

- In normal early pregnancy, the muscular walls and endothelium of the spiral arteries are eroded and replaced by trophoblast.
- The second phase of invasive process occurs between 16 and 20 weeks of gestation when the trophoblast erodes the spiral arteries in the myometrium.
- In pre-eclampsia, the trophoblastic invasion of the spiral arteries is thought to be inhibited resulting in decreased placental perfusion which may ultimately lead to early placental hypoxia and oxidative stress.

Pathophysiology Cont'…

- This first stage of the disease process (pre-eclampsia) occurs early in pregnancy and is difficult to detect clinically.
- The second stage follows when oxidatively stressed placenta triggers the release of one or more factors e.g. endothelins that damage the endothelial cells in maternal circulation

  (endothelins are endothelial cells from the endothelium, which lines the cardiovascular system and serous cavities of the body).

Pathophysiology Cont'…

- The subsequent maternal systemic inflammatory response and endothelial cell dysfunction results in the clinical signs of pre-eclampsia seen after 20 weeks gestation. Damage to the endothelial cells with widespread inflammation results.
- The combined effect of these events will cause;
  - Vasospasm, vasoconstriction and increased BP
  - Abnormal coagulation and thrombosis
  - Increased permeability of the endothelium leading to oedema, proteinuria and hypovolaemia

    (*these are the clinical features of pre-eclampsia*)

Diagnostic investigations and findings of pre-eclampsia

- **History;** (see *predisposing factors*), headache, visual disturbances, epigastric pain (upper quadrant pain)
- **Inspection;** oedema of face, hands, ankle or lower limbs
- **BP measurements**; elevated BP ≥140/90mmhg on two occasions 6 hours apart but not more than 7 days apart is an indication of pre-eclampsia
- **Lab Invx;**
  - Renal Function Tests (RFTs) e.g. urinalysis for amount (may show oliguria), proteinuria (>300mg in 24 hours is common), raised serum creatinine levels;
  - Full blood count (FBC); shows thrombocytopenia,
  - Liver function tests (LFTs); elevated transaminases

Classification of Pre-eclampsia

A. Mild pre-eclampsia
B. Moderate pre-eclampsia
C. Severe pre-eclampsia
D. Superimposed pre-eclampsia
E. Postpartum pre-eclampsia

A. **Mild pre-eclampsia;** detected when after rest, the *BP is ≥ 140/90 mmhg (systolic pressure is 13-20mmhg above basal BP recorded in early pregnancy after 20 weeks' gestation, while diastolic pressure is above 80-90 mmhg of baseline BP)* measured on two occasions at least 6 hours apart but no more than 7 days apart. *Proteinuria > 300 mg in a 24-hour urine collection* or a score of +1 but <2 (30 mg/dL) on at least two random urine dipstick tests collected 6 hours apart but no more than 7 days apart

B. **Moderate pre-eclampsia;** diagnosed when there is marked *rise is systolic BP* between 140-160mmhg and diastolic of100mmhg; *proteinuria* of 0.3-0.5g/l with no evidence of UTI

C. **Severe pre-eclampsia**; diagnosed when on bed rest, the patient's *BP is ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic,* measured on two occasions at least 6 hours apart but no more than 7 days apart, and/or *Proteinuria greater than 5 g in a 24-hour collection (or >3-4+ on dipstick in at least two random clean-catch sample)* even if associated with BPs in the mild range.

- <u>BP alone is not always a dependable indicator of severity of pre-eclampsia</u>
- Severe pre-eclampsia is diagnosed when the elevated BP is accompanied by any of the following;

i. **Oliguria** (urine <500ml in 24 hours);
ii. **Cerebral or visual disturbances**, including altered consciousness, headache, scotomata, blurred vision, or some combination of these,
iii. **Pulmonary edema** or **cyanosis**;
iv. **Epigastric or right upper quadrant pain** associated with impaired liver function without a known cause, indicated by **elevated serum liver transaminases**;
v. **Thrombocytopenia** (a platelet count lower than hundred thousand per microlitre - $<100,000/mm^3$);
vi. **Fetal growth restriction**
vii. **Increased serum creatinine** (>1.2 mg/dl)

D. **Superimposed pre-eclampsia**; development of pre-eclampsia in women with pre-existing hypertension and/or pre-existing proteinuria
E. **Postpartum pre-eclampsia**; increased BP, edema (of lower legs, ankles, feet) and proteinuria accompanied with headache, epigastric pain or vomiting developing postnatally (after delivery) without having had any associated antenatal problems.

Classification of Pre-eclampsia and comparison with Eclampsia

Findings	Mild Pre-eclampsia	Moderate Pre-eclampsia	Severe Pre-eclampsia	Eclampsia
Diastolic BP (mmhg)	absolute level is > 90 but <100	Absolute level is >100 but < 110	absolute level is >110	As in severe pre-eclampsia plus fits/convulsions in the absence of other medical conditions that predispose to convulsions.
Systolic BP (mmhg)	>120-140	140-160	≥160	
Proteinuria	Trace or +1 (>300mg)	1 or 2 (1+)	2+ or greater (>5g)	
Generalized oedema including face and hands	Absent	Present	Persistently present	
Headache	Absent	Present (mild)	Severe headache and convulsions	
Visual disturbance	Absent	Present (mild)	Present	
Epigastric or Upper abdominal pain	Absent	Present (mild)	Present	
Oliguria	Absent	Present (mild)	Present	
Diminished foetal movements	Absent	Present	Present	

Classical Characteristics/Presentation of Pre-eclampsia

Signs and Symptoms develop late hence the importance of prenatal care with early detection and management. These include;

- **Hypertension** (diastolic BP ≥90mmhg that persists). Constitutes Pregnancy-induced hypertension (PIH) with *proteinuria* or *generalized oedema* *after 20th week of gestation*
- **Proteinuria;** this is defined as *proteinuria of 300mg per 24hrs or more of urinary protein at least 2 random urine specimens 6 hours apart*. This is an important sign of PET; its absence may rule out diagnosis of PET
- **Oedema;** is pathological and not just dependent. It usually involves the face and hands and persists even after arising.
  - Tight ring finger is a good indicator of edema.

- **Weight gain;** sudden increase of weight may precede development of PET. Weight increase of 500g per week is normal. This is due to fluid retention that precedes signs of edema.
- **Headaches;** in more severe cases, headache is often frontal but may be occipital. It is resistant to ordinary analgesics.
- **Epigastric pains;** often a sign of severe PET or eminent convulsions, being due to hepatic ischaemia or stretching of the hepatic capsule by edema and haemorrhage
- **Visual disturbances;** partial or unilateral if retinal detachment and total if cortical blindness

Systemic effects of PET

a. Maternal effects
b. Fetal effects
c. Fetal-maternal (placenta) effects

a) Maternal Effects of PET

- **Cardiovascular changes**
  - *i). Haemodynamic changes*
    - high systemic vascular resistance
    - hyperdynamic ventricular function
  - *ii) Blood volume*
    - Reduced blood plasma volume,
    - Haemo-concentration due to generalized vasoconstriction and increased vascular permeability
  - *iii). Hematological changes.*
    - thrombocytopenia
    - reduction in clotting factors
    - abnormal erythrocytes- rapid haemolysis
    - Elevated fibrinogen levels

Note: neonatal thrombocytopenia may occur in neonates of mothers with PET due to hypoxia, acidosis or sepsis.

- **Kidney and Urinary System;**
  - reduced renal perfusion and GFR due to glomeruloendotheliosis (swelling of capillary endothelium)
  - increased plasma uric acid
  - increased plasma creatinine 2-3 times the non - pregnant state.
  - Proteinuria; proteinuria is necessary to diagnose PET-eclampsia. Since it develops late, some women may not have proteinuria; therefore it is important to measure the 24-hour urine specimen.

- **Liver;**
  - ***HELLP Syndrome*** (haemolysis, elevated liver enzymes, low platelet count) with severe right upper quadrant pains and hyperbilirubinaemia.
- **Brain;**
  - cerebral oedema,
  - thrombosis,
  - haemorrhage,
  - headache, convulsions, coma (rare)
- **Eyes;**
  - visual disturbances, retinal detachment and resultant blindness
- **Respiratory System;** Pulmonary edema;

b) Fetal effects of PET

- Neonatal Thrombocytopenia,
- Fetal distress
- Oligohydramnios
- Prematurity
- High morbidity leads to high mortality
- IUGR (Intrauterine Growth Restriction)
- IUFD (Intrauterine Fetal Death)

c) Placental (fetal-maternal) effects of PET

- Impairment of placental perfusion from vasospasm is responsible for the high perinatal morbidity and mortality associated with PET.

MANAGEMENT OF PRE-ECLAMPSIA

- The principles of management of PET aim to achieve the following objectives:
  i. *Early detection of predisposing factors and warning signs*
  ii. *BP control and fetal growth monitoring*
  iii. *Termination of pregnancy with the least trauma to the mother and fetus.*
  iv. *Delivery of infant who would thrive*
  v. *Complete restoration of health of the mother.*

The Principles of Management include:

1. **Early detection** of predisposing factors and warning signs
2. **Bed rest** (hospitalization)
3. **Drug therapy** (anti-hypertensives)
4. **Monitoring fetal growth and well-being**
5. **Termination of pregnancy**
6. **Avoid maternal complications** e.g. cerebral, liver, pulmonary or visual complications

1. **Early detection of predisposing factors and warning signs;**
   - Careful observation at appropriate intervals so as to identify *predisposing factors* that include familial history of pre-eclampsia, multiple fetuses, diabetes, chronic vascular disease, renal disease, hydatidiform mole, fetal hydrops
   - *Warning signs* or *predictors of PET* include *rapid weight gain* in the later half of pregnancy and *upward trend in diastolic blood pressure*. These can only be picked from well-planned antenatal program and the symptoms and signs of PET, reported and/or detected.
   - **Aspirin;** Low dose aspirin *(75-100mg daily)* may be effective in some women in preventing the development of PET.

NB: *Diuretics* and *Sodium restriction* have no value in the management of pre-eclampsia hence are contraindicated.

2. Hospitalization (bed rest)

- Bed rest (as outpatient) for those with *mild PET*
- Those with *worsening PET* are hospitalized to monitor signs of severe PET
- BP monitoring
- Daily weighing
- Daily Renal function tests (RFTs) e.g. urinalysis (24hr urine) for proteinuria, dip stick test, uric acid
- Monitor plasma serum creatinine, hematocrit,
- LFTs (liver transaminases) and platelet count
- Monitor fetal growth (size) with ultrasound, fetal kicks charts, BPP (biophysical profile 1-2 times a week); amount of liquor
- Delivery at term - 37 weeks of gestation

3. **Drug Therapy**

a) ***Mild and moderate hypertension***

- Phenobarbitone 30mg t.i.d or Valium 5mg t.i.d for mild PET
- Aldomet 250mg t.i.d or 750mg q.i.d
- Hydralazine 25-50mg t.i.d
- Nifedipine 20mg b.d

b) ***Severe PET***

- Treat as eclampsia

4. Monitoring of fetal growth and well-being

- *Fetal movements;*
  - fetal kick chart *(10 kicks in 24 hours.)*
- *Assessment of;*
  - Fundal height
  - Amount of liquor
  - Contraction stress test
  - Biophysical profile.
- *Admit patient and do Bishop score,*
  - If cervix is favorable, induce labour with syntocinon.
  - If cervix is unfavorable, ripen the cervix with PGE_2 pessary then induce labour

5. Termination of pregnancy

- Delivery is the definitive management in PET and it is recommended if gestation is >37 weeks in the interest of the mother with total disregard of fetal state (regardless of fetal state)
- Patient is admitted and Bishop score done,
  - If cervix is favorable, labour is induced with syntocinon.
  - However if cervix is unfavorable, ripen the cervix with PGE_2 pessary then induce labor.

Indications for termination of pregnancy

i. If there is no fetal growth
ii. Severe oligohydramnios
iii. Deteriorating maternal conditions in terms of renal, CNS, hepatic functions.
iv. Good chances of survival of the infant.
v. Uncontrolled B.P despite maximum doses of anti-hypertensives (diastolic blood pressure confirmed to be consistently higher than 110 mmhg).
vi. Rising serum creatinine;
vii. Persistent or severe headache;

viii. Abnormal liver function tests;
ix. HELLP syndrome;
x. Epigastric pain;
xi. Thrombocytopenia,
xii. Pulmonary edema,
xiii. SGA fetus with failure to grow on serial ultrasound examinations.
xiv. Eclampsia,

NB: Conservative Management of Severe Pre-eclampsia.

- Patients who are eligible (most will require delivery in the next 2 weeks) should receive the following care:
  - Bed rest
  - Seizure prophylaxis for the first 24 hours of hospitalization
  - BP measurement every 4 hours.
  - Daily examination to assess weight, review systems, check for edema, and check deep tendon reflexes
  - Daily monitoring of 24-hr fluid status, CBC with platelet count; and measurement of AST, LDH, and bilirubin levels
  - Daily 24-hour urine protein
  - Daily fetal surveillance including fetal movement counts and NST or biophysical profile

ANY QUESTIONS?

Case Scenario

- Mrs. Chepkwarkwaran, 36 year old para 1 + 0 gravida 2 at 34 weeks gestation has been brought by relatives to the accident and emergency department presenting with sudden onset of tonic-clonic seizures. Medical history reveals no past significant chronic illness. Physical exam shows bilateral limb swellings especially ankles. Investigations done and repeated 6 hours thereafter during management indicate BP of 160/100 mmhg and significant proteinuria.
- **Quiz**:
  a) *Give the specific diagnosis of Mrs. Chepkwarkwaran*
  b) *Discuss the management of condition in (a) above*

11. ECLAMPSIA

Session Objectives;

- By the end of the lesson, the learner should be able to;
  - *a) Define the term eclampsia*
  - *b) List the causes of eclampsia*
  - *c) Describe four stages of eclamptic fits*
  - *d) Outline the clinical features of impending eclampsia*
  - *e) Explain the differential diagnosis of eclampsia*
  - *f) Discuss management of eclampsia to include the use of magnesium sulphate and diazepam; the mode of delivery of the baby and post-natal care of both mother and baby.*
  - *g) List five complications of eclampsia*

Definition of Eclampsia;

1) Severe hypertensive disorder of pregnancy characterized by **very high BPs** (BP >160/110mmhg), **proteinuria, oedema** (occult or clinical) and convulsions/fits or coma in the absence of other medical conditions predisposing to convulsions.
2) Severe hypertensive disorder of pregnancy characterized by **convulsions/fits** and **coma**, in the absence of other medical conditions predisposing to convulsions, in a woman with **pre-eclampsia**.
   - **Impending eclampsia** means that eclamptic fits are likely to occur very soon, usually in a woman with severe pre-eclampsia.

Signs of Impending Eclampsia

- A *sharp rise in blood pressure* (BP >160/100 on two occasions 6 hours apart
- *Oliguria* (decrease urinary output <400ml/24 hours).
- *Proteinuria* (Increase protein +++, or 1gm/l/24 hours)
- *Cerebral edema* presenting with *mental confusion*, irritability, *drowsiness*, photophobia, *convulsions* and *severe frontal headache*.
- *Visual disturbances* due to retinal oedema (e.g. blurred vision, flashes of flight)
- *Epigastric pain*
- *Nausea/vomiting*.
- *Hyper-reflexia*

Prevention of Eclampsia

- *Early detection and treatment of signs of eminent eclampsia*
- *Antihypertensives* to control BP to 140/90 mmhg e.g. hydralazine 10mg iv slowly for 15 min then 25 mg in 500ml dextrose
- Give *sedatives*; valium bolus dose followed by 20-40mg of valium in 500ml dextrose to run at 60 drops/min until sedation is achieved then maintain at 15drops/min
- Give *diuretics* e.g. Lasix 40mg (only when indicated)
- Give *mannitol* to prevent renal failure
- If the mother is at risk of infection, give *prophylactic antibiotics*
- NB: if patient doesn't respond to the above management, *terminate the pregnancy irrespective of the gestation*

Differential Diagnosis (DDx) of Eclampsia

- Eclampsia must be differentiated from other conditions that may be associated with convulsions and coma, e.g.
  - Epilepsy,
  - Cerebral malaria,
  - Meningitis, Encephalitis
  - Head injury,
  - Cerebrovascular accident,
  - Cerebral tumuor
  - Intoxication (alcohol, drugs, and poisons),

Differential Dx of Eclampsia Cont'…

- Drug withdrawal,
- Metabolic disorders,
- Water intoxication,
- Encephalitis,
- Hypertensive encephalopathy,
- Hysteria.

➢ **Note:** All women with convulsions (fits) in pregnancy should be assumed to have eclampsia until another cause is determined.

Characteristics of Eclamptic fits:

- *Convulsions* may occur regardless of the severity of hypertension, are difficult to predict and typically occur in the absence of hyper-reflexia, headache or visual changes.
- Convulsions are tonic-clonic and resemble grand-mal seizures of epilepsy
- Seizures may recur in rapid sequence as in status epilepticus, and end in death.
- Convulsion may be followed by coma that lasts minutes or hours, depending on the frequency of seizures.
- 25% of eclamptic fits occur after delivery of the baby.

Stages of Eclamptic fits

- An eclamptic fit is similar to an epileptic fit, and has the following stages:

***A) Premonitory stage* (lasts 10-20 seconds);**

- Characterized by twitching of facial muscles, rapid eye movements, restlessness and loss of consciousness

***B) Tonic stage* (lasts 10-20 seconds);**

- The body is rigid or stiff with muscular spasms, eyes are bulging and staring and teeth are tightly clenched together.
- Skin colour becomes blue or dusky (cyanosis)
- The back may be arched and respiration may stop due to spasms of the diaphragm.

Cont'…

C) Clonic Stage

- This stage lasts 1- 2 minutes and is marked by:
  - Violent/forceful contraction and relaxation of the muscles leading to convulsions,
  - Rapid movement of jaws which may lead to biting of the tongue by the violent action of the jaws
  - Increased saliva or "foaming" at the mouth which may lead to inhalation of mucous or saliva
  - Deep noisy breathing
  - Face looks congested (filled with blood) and swollen

Cont'…

D) Coma stage

- This may last **minutes or hours**. during this time;
  - There is a deep state of unconsciousness
  - Breathing is noisy and rapid
  - Cyanosis fades, but the face remains congested and swollen
  - Convulsions continue and breathing becomes difficult.
  - Further fits may occur before the woman regains consciousness

Management of Eclampsia:

- Call for help
- Maintain open airway by putting patient in *semi-prone position* to allow drainage of saliva.
- Do suctioning and insert airway to aid respiration.
- Administer oxygen to correct severe hypoxia and acidosis
- Start IV fluids but restrict fluid intake to avoid pulmonary and cerebral oedema. Maximum of 30 drops per minute.
- Control fits/convulsions with i.v. Valium or i.v. $MgSO_4$ and ensure safety of patient to prevent falling during convulsions. Observe and record frequency, strength and duration of fits. Consider delivery after control of fits.
- BP control and monitoring quarter hourly
- Catheterize patient for continuous emptying of bladder, take urine sample for testing protein, acetone and sugar then closely monitor fluid intake and urine output

Management of a fitting patient

- Patient should be put in semi prone position so that mucous and saliva can drain out
- Tight fitting dresses around the neck should be loosened or removed
- No attempt should be made to insert any instrument into the mouth
- Administer anticonvulsants e.g. *magnesium sulphate* (or *diazepam*) as per regime to control fits
- Aspirate secretions from the mouth and nostrils as necessary

Cont'…

- Give Oxygen continuously during fit and for 5 minutes after each fit (if available) to prevent severe hypoxia.
- Fitting should be allowed to complete its course without restraining the patient
- Observe and record the frequency, strength and duration of each fit.
- Start IVF to hydrate the woman but strictly monitor input and output chart.
- Privacy and dignity of patient must be observed - pull screens around her

Anticonvulsants:

i. Magnesium Sulphate:

- This is the recommended drug of choice to treat and prevent eclampsia rather than diazepam or phenytoin.
- This works by antagonizing calcium channels of smooth muscle. It is also thought to aid vasodilation thereby reducing cerebral edema and prevent seizures.
- Administer IV/IM for seizure prophylaxis in preeclampsia.
- Use IV for quicker onset of action in true eclampsia

Magnesium Sulphate schedules for severe pre-eclampsia and eclampsia

Loading Dose

- **4g of 20%** $MgSO_4$ Solution given ***I.V*** *over 5-10min.* Follow promptly with **10g of 50%** MgSO4 solution over 10-15 min; 5g in each buttock as deep ***I.M*** injection with 1mL 2% lignocaine in the same syringe
- Ensure that aseptic technique is practiced when giving magnesium sulphate deep IM injection. Warn the woman that a feeling of warmth will be felt when magnesium sulphate is given.
- If convulsions occur after 15 minutes, give 2g magnesium sulphate (50% solution) I.V over 5 minutes

Cont'...

Maintenance Dose;

- Give *5g of 50% $MgSO_4$ solution* + 1 mL lignocaine 2% I.M every 4 hours into alternate buttocks or *I.V 5g/500ml NS*.
- Continue treatment with *$MgSO_4$* for 24 hours after delivery or the last convulsion, whichever occurs last.
- **NB:** If 50% solution is not available, give *1g of 20% $MgSO_4$* solution IV every hour by continuous infusion

Closely monitor pt for signs of toxicity;

- **Withhold or delay drug if:**
  - Respiratory rate falls below 16 per minute
  - Patellar reflexes are absent
  - Urinary output falls below 30ml per hour over the preceding 4 hours

Keep antidote ready: [Calcium Gluconate]

- In case of respiratory arrest:
  - Assist ventilation (mask and bag, anaesthesia apparatus, intubation)
  - Give Calcium Gluconate 1g (10-20mL of 10% solution) IV slowly until begins to antagonise the effects of magnesium sulphate and respiration begins

Cont'...

- In the absence of MgSO4, **Diazepam** may be given **IV;**

Diazepam schedules for severe pre-eclampsia and eclampsia;

Loading dose;

- Diazepam *20mg IV slowly over 2 minutes*
- If convulsions recur, **repeat loading dose**

Maintenance dose;

- Diazepam *40mg in 500ml IV fluids* (NS or RL) titrated to keep the woman sedated but can be aroused
- Maternal respiratory depression may occur when dose exceeds 30mgs in 1 hour. Assist ventilation (mask and bag, anaesthesia apparatus, intubation), if necessary
- Do not give more than 100mg of diazepam in 24 hours!!!

Cont...

Rectal Administration of Diazepam;

- Give Diazepam rectally when IV access is not possible.
- The loading dose is 20mg in 10ml syringe. Remove the needle, lubricate the barrel and insert the syringe into the rectum to half its length. Discharge the contents and leave the syringe in place, holding the buttocks together for 10 minutes to prevent expulsion of the drug. Alternatively, the drug may be instilled into the rectum through a catheter.
- **If convulsions are not controlled within 10 minutes administer an additional 10mg per hour or more, depending on the size of the woman and her clinical response.**

BP Control

- Take BP every 5 minutes until diastolic is 90mmhg then maintain it at this level by titrating IVF infusion of hydralazine.
- The goal is to lower BP to prevent cerebrovascular and cardiac complications while maintaining utero-placental blood flow.
  - Control of mildly increased BP does not appear to improve perinatal morbidity or mortality, and, in fact, it may reduce birth weight.
- Antihypertensive treatment is indicated for diastolic blood pressure above 105 mm Hg and systolic pressure above 160 mm Hg, though patients with chronic hypertension may tolerate higher values.

Cont...

- The goal is to maintain diastolic blood pressure between 90 and 100 mm Hg and systolic pressure between 140 and 155 mm Hg.
- First-line medications are;
  - Labetalol, given orally or IV;
  - Nifedipine, given orally or IV; or
  - Hydralazine IV.

  NB: *Atenolol, ACE inhibitors, ARBs, and diuretics should be* ***avoided***
- Monitor FHR frequently
- Do VE to rule out APH

Delivery:

- *Delivery via SVD or C/S* should take place as soon as the woman's condition has been stabilized, preferably within 6-8 hours from first convulsion, *when BP has dropped* or within 12 hours of admission.
- Delaying delivery to increase foetal maturity will risk the lives of both the woman and the foetus.
- Delivery should occur regardless of the gestational age, but **Eclampsia alone is not an indication for C/Section.**
- Get skilled anaesthetist' help early; this will also aid the management of hypertensive crises

Mode of Delivery

i. ***Vaginal delivery is recommended:***

- If the cervix is favourable (soft, dilated, effaced), rupture the membranes and induce labour using oxytocin
- If there is no absolute indication for C/section
- If safe anaesthesia is not available for C/section or if the foetus is dead or too premature for survival,
- If the cervix is unfavourable (firm, thick, closed), ripen the cervix using prostaglandins or a Foley catheter

Cont'...

ii. ***Caesarean Section should be done:***

- If vaginal delivery is not anticipated within 8 hours (for eclampsia) or 24 hours (for severe pre-eclampsia), deliver by C/section,
- When BP has dropped.
- If there are foetal heart rate abnormalities (<100 or >180 beats/minute)
- If the cervix is unfavourable (firm, thick, closed) and the foetus is alive,

Postnatal Care:

- Continue anticonvulsive therapy for 24 hours after delivery or last convulsion, whichever occurs last.
- Continue antihypertensive therapy as long as the diastolic pressure is 110 mmHg or more.
- Continue to monitor urine output. If urine output is less than 500 ml in 24 hours, limit the amount of fluid intake to 500 mls per 24 hour + an amount equal to the amount of urine passed
- Watch carefully for the development of pulmonary oedema, which often occurs after delivery.
- Life threatening complications can still occur after delivery e.g. *postpartum pre-eclampsia*. Monitor carefully until the patient is clearly recovering.

Cont'…

- **Consider referral** of women who have:
  - **Oliguria** (less than 500 ml urine output in 24 hours) that persists for 48 hours after delivery
  - **Coagulation failure** (e.g. coagulopathy or haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome)
  - **Persistent coma** lasting more than 24 hours after a convulsion

NB: Post delivery;

- *Urinary output is the first sign of recovery*
- *Proteinuria and oedema disappear within a week.*
- *B.P returns to normal within 2-weeks after delivery.*

Complications of Eclampsia

- Abruptio placentae with disseminated intravascular coagulopathy (DIC)
- Renal Failure (acute) or Renal insufficiency
- **HELLP Syndrome** (Haemolysis, elevated liver enzyme levels, and low platelet count).
- Cerebral haemorrhage and oedema.
- Confusion and/or Coma (rare)
- Pulmonary oedema
- Myocardial failure
- Liver necrosis
- Hemiplegia

Complications Cont'…

- Bone fractures or injuries
- Thrombosis
- Blindness
- Maternal death
- Foetal distress
- Asphyxia neonatorum
- Prematurity
- IUGR or foetal demise (IUFD)
- Still birth
- Neonatal deaths
- Psychoses – good prognosis

ANY QUESTIONS SO FAR?

ASSIGNMENT

- Discuss eclampsia under the following headings;
  i. Definition,
  ii. Causes,
  iii. Stages of eclamptic fits (4 stages),
  iv. Clinical features of impending eclampsia,
  v. Differential diagnosis of Eclampsia,
  vi. Management of eclampsia to include use of magnesium sulphate and/or diazepam,
  vii. Mode of delivery of the baby and post-natal care of both mother and baby,
  viii. Complications of eclampsia

SHOCKED ?

**HALF CLOSE YOUR EYES…WHAT DO YOU SEE?
HAPPY NOW? NEVER MIND. HIVO NDO POMBE
HUFANYA WALEVI…SAITAN NI SAITAN TU!!!**

12. OBSTETRIC SHOCK

- **Shock** refers to the collapse of the circulatory system, which results in sudden fall of blood pressure, reduction in blood flow and deficient supply of oxygen to the tissues causing dysfunction of organs and cells (especially the vital organs e.g. heart, lungs, brain, kidney).
- In **obstetric shock**, the condition may be due to complications of pregnancy and labour.

Three main types of shock

a) **Hypovolaemic shock,** which is as a result of reduction in intravascular volume after hemorrhage or severe burns.

b) **Cardiogenic shock,** which is shock due to acute heart condition e.g. myocardial infarction that cause inability of the heart to pump blood.

c) **Distributive shock**, which results from a malfunction in the vascular system causing maldistribution of the circulatory systems. This can be caused by septic and anaphylactic shock.

Other types of shock

i. **Allergic or Anaphylactic shock;** shock produced by injection of a protein to which a patient is sensitive
ii. **Neurogenic shock;** shock due to nervous or emotional factors
iii. **Septic shock;** life threatening condition produced by sepsis (infection) in which there is tissue damage and severe fall in blood pressure as a result of septicaemia(a lot of bacteria or large bacterial toxins in blood) e.g. toxic shock syndrome
iv. **Toxic shock;** shock due to poisonous or toxic substances, also called toxic shock syndrome.
v. **Shell shock;** mental health condition caused by stresses of warfare

Causes of Obstetric Shock

i. Haemorrhage during pregnancy, labour and puerperium
ii. Obstetric trauma such as difficult instrumental delivery, forcible breech extraction, manual removal of placenta or caesarean section
iii. Prolonged labour
iv. Fluid loss, for instance, excessive diuresis or hyperemesis gravidarum
v. Supine hypotensive syndrome
vi. Pulmonary embolism, which may dislodge and cause oxygen deprivation
vii. Reactions due to blood transfusion or drugs

Stages of Shock;

The condition of shock develops in several stages;

a) **Initial Stage**

- The venous return to the heart is decreased due to reduction of blood or fluid. There is also a reduction in stroke volume and cardiac output caused by inadequately filled ventricles of the heart. There is a fall in blood pressure, which decreases oxygen supply to the tissue and affects the function of the cells

b) Compensatory Stage

- At this stage the body can compensate for up to 10% of fluid volume.
- When it reaches 20-28% it begins to fall.
- The sympathetic nervous system responds to a drop in cardiac output by constricting vessels in the gastro-intestinal tract, kidney, skin and lungs. This causes the skin to be pale and cool. Peristalsis slows, urinary output is poor and gas exchange in the lungs is impaired. This causes ischaemia and collapse of alveoli, ultimately leading to respiratory failure known as 'adult respiratory distress syndrome'.
- Blood is distributed to the vital organs only.

Compensatory Stage Cont'…

- There is an increased heart rate in an effort to improve cardiac output and blood pressure.
- The pupils dilate and the sweat glands are stimulated causing the skin to be moist and clammy.
- Adrenaline and aldosterone from the adrenal medulla and adrenal cortex are released.
- The posterior pituitary lobe also produces antidiuretic hormone, which causes vasoconstriction in an attempt to improve cardiac output.
- Venous return to the heart increases but cannot be sustained for long unless fluid loss is replaced.

c) Hypovolaemic Stage

- When the compensatory mechanism begins to fail, there is a further fall in cardiac output and blood pressure.
- Vital organs lack perfusion and coronary arteries lack supply. There is poor peripheral circulation.
- The pulse is either weak or absent.
- In the brain, the level of consciousness deteriorates and the mother becomes increasingly unresponsive.
- The renal tubules become ischaemic, leading to kidney failure. Waste products such as urea and creatinine are not excreted, resulting in their increased presence in the blood.

Hypovolaemic stage Cont'…

- The gut's function as barrier fails due to ischaemia and gram-negative bacteria are able to enter the blood stream
- The liver can no longer metabolise drugs and hormones. As the bilirubin can no longer be conjugated, it builds up and jaundice develops.
- With the liver failing to act as a filter, there is a build up of lactic acid and ammonia in the blood due to the failure of waste metabolism. Liver enzymes are released in the blood circulation by dead hepatic cells.

d) Final Irreversible Stage of Shock

- The distractions of the cells are irreparable causing multisystem failure and ultimately death

Signs and Symptoms of Shock

- Low BP <90/60mmhg (hypotension/hypovolemia, hemorrhage) – this is an essential factor!!!
- Rapid, weak (feeble) or absent pulse
- Pyrexia (Acute fever -T ≥ 38°C)
- Severe Dyspnea (Respiratory distress)
- Tachycardia (HR>90b/min) or Bradycardia (HR<60b/min)
- Cyanosis (central or peripheral)
- Cold, clammy, moist skin
- Dry mouth (dehydration), dry mucus membrane
- Pallor of conjunctiva, palms or skin
- Pupil dilation
- Pulmonary oedema

↑

Signs and Symptoms of Shock cont'…

- Severe acute pain
- Kidney failure (oliguria, anuria, deranged UECs, ammonia in blood, ketosis);
- metabolic and/or respiratory acidosis or alkalosis
- Altered/decreased level of consciousness e.g. restlessness or drowsiness
- Emotional distress
- Diaphoresis (excessive sweating or perspiration)
- Excessive thirst
- Rigors or intense shivering (opposite of rigor mortis)
- Convulsions or Coma
- Diarrhoea, Nausea, Vomiting

Outcome of Shock

- Early arrest of the cause and replacement of body fluid will give **full recovery**.
- At times the mother may survive but develop permanent damage to various organs **(morbidity)**. This is referred to as Sheehan's syndrome.
  - *Sheehan's Syndrome is a condition where sudden or prolonged shock leads to irreversible pituitary necrosis characterized by amenorrhea, genital atrophy and premature senility.*
- **Death** as a result of shock is usually due to a *delay in treatment*

Management of Hypovolaemic Shock

- Urgent resuscitation measures should be applied to prevent irreversible damage to the patient.
- The first thing you should do is to maintain a clear airway by turning her on one side. If she is unconscious, insert an airway by turning her on one side and administer oxygen, 40% at the rate of four to six litres/min.
- Find the source of bleeding, whenever possible and try to stop the bleeding. Replace fluid immediately.
- Take blood for a cross match and give blood transfusion as and when ready.

Cont'…

- Meanwhile a plasma expander such as dextran, haemocel or glucose saline (1 litre) should be administered as soon as possible
- When the blood is ready, the first 1,200mls should be given rapidly (within 30 minutes). The doctor should remain with the patient during this exercise.
- Avoid excessive warmth as it will interfere with the constriction of the peripheral blood vessels, which usually occurs in response to shock.

Cont'…

- Elevate the foot of the bed by 30cm. This will raise blood pressure 10mm Hg by gravity. This allows the blood to flow to vital centres in the brain.
- Hydrocortisone 100-500mg is given slowly in cases of suprarenal failure.
- A sedative may be necessary in the case of restlessness to calm an apprehensive patient.

Observations

- The following observations should be made while monitoring the patient:
  - Assess level of consciousness, noting signs of restlessness or confusion
  - Monitor blood pressure continuously, about every 15-30 minutes
  - Cardiac rhythm needs to be monitored continuously
  - Measure urine output hourly by the use of indwelling catheter

Cont'…

- Take hourly temperature and observe the colour of the skin. Improvement to normal body temperature and colour may mean the function of the organs is going back to normal. Persistence of subnormal temperature means the reverse
- The infusion quantity and rate should be maintained accurately by measuring haemodynamic pressure
- Observe further occurrence of bleeding

Septic Shock

- also known as endotoxic or bacteraemic shock.
- The main cause of septic shock is gram negative organism such as *Escherichia coli*, *Bacillus Proteus* or *Pseudomonas pyocyaneus*
- These organisms are commonly pathogenic in the female genital tract. Gram-positive bacteria, viral or fungal infection, do not commonly cause septic shock.
- In 20-30% of cases, the cause of infection is combined organisms, and treatment becomes complex.
- Septic shock can be caused by prolonged rupture of membranes and puerperal sepsis, especially in cases of caesarean section

Management of Septic Shock

- Use quick fluid therapy including glucose, saline, Ringers' lactate
- An injection of dopamine, 20mg per kilogram. (Dopamine-used to increase mean arterial pressure in septic shock)
- Hydrocortisone is given, 100mgs IV stat, followed by 100mg six hourly until the pulse and blood pressure are stabilized.

Cont…

- **Antibiotics** are commenced immediately after the specimens for culture and other investigations are completed. These include:
    - Gentamycin 80mg IV eight hourly
    - Metronidazole 500mg IV eight hourly
    - Ampicillin 500mg IV six hourly
- These should be administered until the bowel sound return.
- You should then continue with Metronidazole 400mg orally eight hourly for 10 days

ANY QUESTIONS SO FAR?

Case Scenario

- Mrs. Erbs, 40 year old grandmultipara with gestational diabetes was admitted in first stage of labour at 42 weeks gestation. The progress of labour was monitored using a partograph progressing well. Few hours later when the signs of second stage became evident, she was immediately transferred to the delivery room. The second stage nurse was very ready to conduct the delivery. Mrs. Erbs (who was very cooperative) was first instructed on how to bear down and then the nurse went on to deliver the baby. Unfortunately, following crowning, extension, restitution and subsequent delivery of the baby's head, any further effort of the mother to bear down and the nurse to support the perineum became fruitless….
  - ***Quiz:*** *Discuss further management of Mrs Erbs*

12. SHOULDER DYSTOCIA

Shoulder dystocia is said to have occurred when there is:

- Failure of the shoulders to traverse the pelvis spontaneously after birth of the head
- Failure of the shoulder to rotate spontaneously into antero-posterior diameter of the pelvic outlet after delivery of the head

  (review the eight steps in the mechanism of labour/movements of the fetus during second stage ***[D-F-I-C-E-R-I-E-L]****).*

Mechanism of Labour in Second Stage

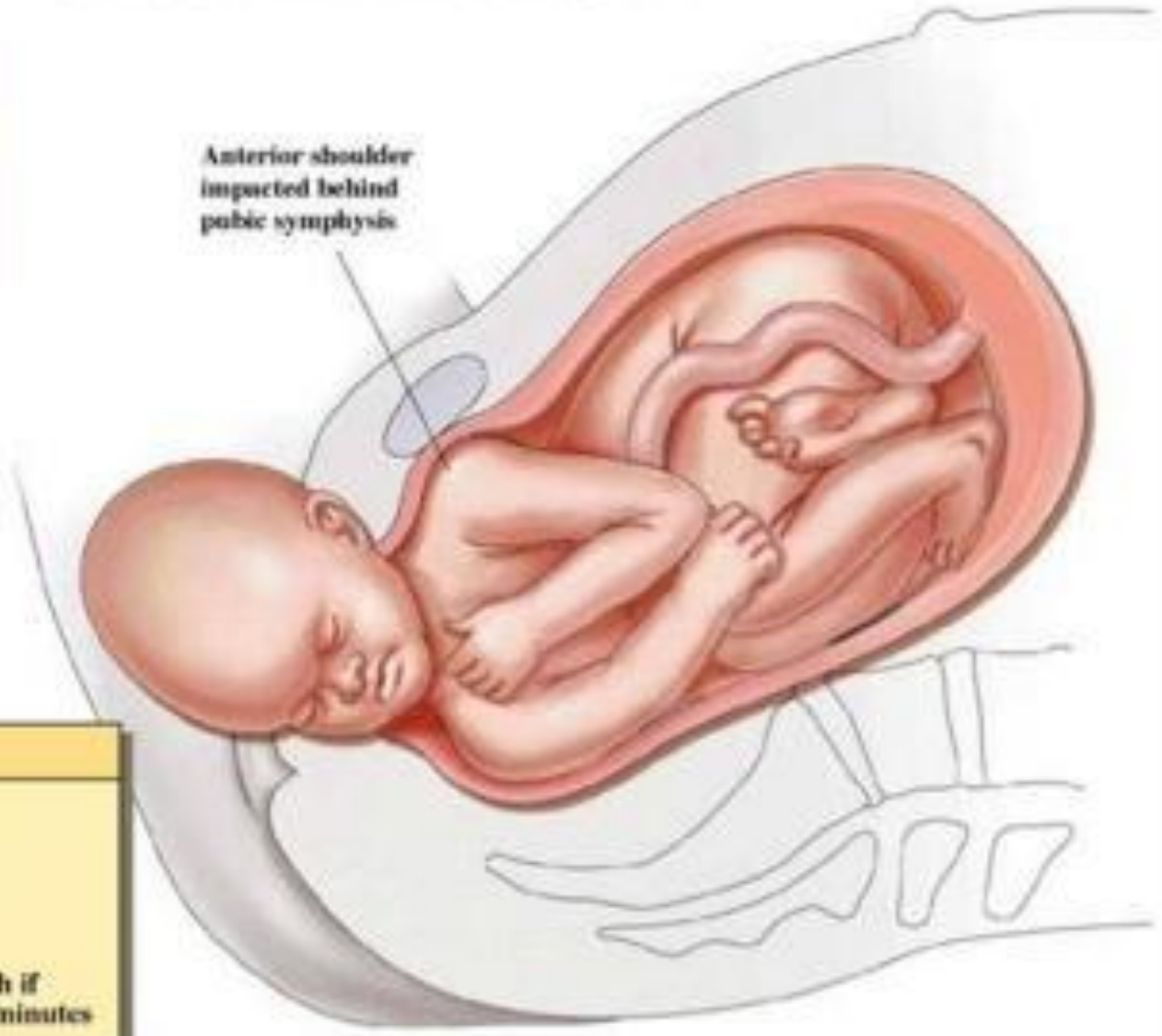
- The foetus undergoes the following movements during second stage of labour;
  i. *Descent of the presenting part,*
  ii. *Flexion of the head*
  iii. *Internal rotation of the head*
  iv. *Crowning of the head*
  v. *Extension of the head*
  vi. *Restitution of the head*
  vii. *Internal rotation of the shoulders*
  viii. *External rotation of the head*
  ix. *Lateral flexion of the body*

Predisposing factors to Shoulder Dystocia

- Unusually large baby,
- Foetal anomalies,
- Post term pregnancy,
- Maternal age of over 35 years,
- High parity,
- Maternal diabetes and gestational diabetes

SHOULDER DYSTOCIA

NORMAL



DANGERS INCLUDE:

- Entrapment of cord
- Inability of child's chest to expand properly
- Severe brain damage or death if child is not delivered within minutes

Diagnosis/Recognition

- **You should watch out for the following warning signs:**
  - There is slow advancement of the head with crowning or failure of the head to rotate externally following restitution
  - Slow crowning of the head
  - There are difficulties in extension of the face during delivery of the head
  - There is slow restitution of the occiput to the lateral position during labour

Management of Shoulder Dystocia

NB: When attempting to deliver the fetus in case of shoulder dystocia, care must be taken not to twist the neck and as far as is possible, excessive traction on the head should be avoided because of the risk to the fetus or damage to the brachial plexus (Erb's palsy).

- As part of the process of management, you should take the following steps;
  - Explain the situation to the mother and reassure her.
  - Position the mother either left lateral with buttocks at the edge of the bed or lithotomy position with buttocks slightly beyond the edge.
  - Ask your assistant to call the doctor.

Management of Shoulder Dystocia Cont'…

- A common treatment algorithm is **ALARMER** or **HELPERR** mnemonics; which stands for;
- **A - Ask for help;** this involves requesting the help of an obstetrician, a paediatrician for subsequent resuscitation of the infant and anaesthesia in case of surgical intervention.
- **L - Leg hyper flexion** (McRoberts' manoeuvre)
- **A - Anterior shoulder disimpaction** by application of supra-pubic pressure.
- **R** for Rubin's manoeuvre
- **M** for Manual delivery of posterior arm
- **E** for Episiotomy
- **R** for Roll over on all fours (Gaskin Manoeuvre)

Cont'...

- Also commonly used is the **HELPERR Mnemonic.** This is a clinical tool that offers a structured framework for coping with shoulder dystocia;
- **H - (Help);** Call for help.
- **E - Evaluate** for episiotomy
- **L - Legs hyperflexion** (McRoberts maneuver)
- **P – (Pressure);** Suprapubic pressure
- **E - Enter maneuvers** (internal rotation)
- **R - Remove** the posterior arm
- **R - Roll** the patient.

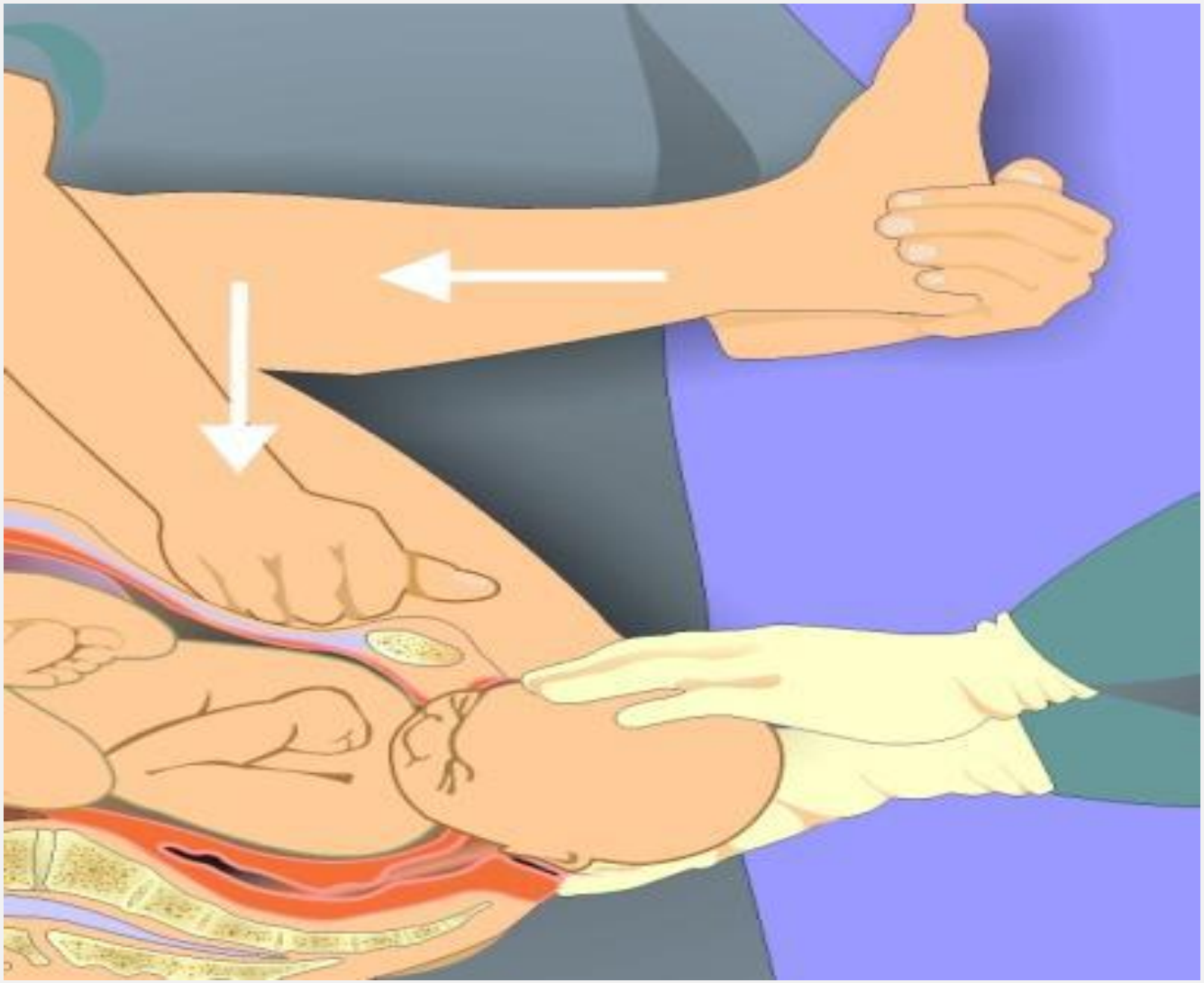
Non - invasive Procedures

- **Any Change in the Maternal Position** may be useful to help release the foetal shoulders as shoulder dystocia is a mechanical obstruction
- **McRobert's manoeuvre** is a useful position;
  - The mother is asked and helped to lie flat on her back and bring her knees as far as possible up to the chest. This manoeuvre has been proved to rotate the symphysis pubic angle posteriorly.
  - The midwife creates pressure gently at the mother's legs and her abdomen
  - The impact of the anterior shoulder is released by this pressure

Cont'...

- **Evaluate for Episiotomy if indicated and consider application of Supra Pubic Pressure if appropriate;**
  - Make an **episiotomy** to enlarge the outlet and reduce pressure at the pelvic floor.
  - While you exert traction to the head downwards and backwards, hook the fingers into anterior axilla and rotate its shoulder forwards.
  - When the shoulders are in anterior posterior, conduct the delivery as usual.

McRobert's Manoevre and Supra-Pubic Pressure/manoevre



All fours position (Gaskin Manoevre)

- The all-fours position (or Gaskin manoeuvre) is achieved by assisting the mother onto her hands and knees; the act of turning the mother may be the most useful aspect of this manoeuvre.
- Here the force of gravity will keep the fetus against the anterior aspect of the mother's uterus and pelvis
- This manoeuvre may be helpful if the posterior shoulder is impacted behind the sacral promontory as this position optimizes space available in the sacral curve and may allow the posterior shoulder to be delivered first

All-fours position (Gaskin Manoeuvre)

Manipulative procedures

- Where non-invasive procedures have not been successful, *direct manipulation of the fetus* must now be attempted;
  - **Performing an episiotomy**
  - Although it does not help release the shoulders per se, episiotomy helps to gain access to the fetus without tearing the perineum and vaginal walls. It enlarges outlet hence reduce pressure at the pelvic floor.

Rubin's Manoeuvre

- Identify the posterior shoulder on vaginal examination.
- Insert two fingers and push the posterior shoulder in the direction of the foetus' chest.
- If the anterior shoulder dislodges, rotate it away from the symphysis pubic and deliver the anterior shoulder.

Wood's manoeuvre

- The midwife inserts her hand into the vagina and identify the fetal chest. Then, by exerting pressure on to the posterior fetal shoulder, rotation is achieved to bring the posterior shoulder forward and make it anterior.
- It is important to hold both the arm and head together to facilitate rotation and reduce the risk of injury

Zavanelli's Manoeuvre

- This manoeuvre is done as a **last option** to save the life of the baby. The obstetrician *reinserts the head into the vagina* by reversing the mechanism and caesarean section is done immediately.

Prevention of Shoulder Dystocia

- Advise the mother on the proper diet to prevent big babies. A proper assessment of the baby's size at 36 weeks by the doctor should enable you to decide the proper mode of delivery.

Complications of Shoulder Dystocia

Maternal complications;

- Two thirds of the patients will have blood loss of more than 1,000mls from injury
- Maternal death may result from uterine rupture

Fetal complications;

- Neonatal asphyxia
- Brachial plexus injury with damage to cervical nerve roots 5 and 6 may result in an Erb's palsy
- Neonatal morbidity may be as high as 42%

ANY QUESTIONS?

Remember !

... in shoulder dystocia, after considering ALARMER or HELPERR especially the need for suprapubic pressure and episiotomy, tell Mc Roberts to instruct the mother to hyperflex legs and hips, Rubin and Woods should only act on rotating baby's shoulders against chest, but Gaskin will have to rotate mother on all fours thereafter, if needed. However, never forget Zavanelli should also be there, though he will only be allowed to come last when everyone else fails *(after all his name begins with the last letter of the alphabet)* and his role would be to reinsert head back to vagina and take mother for immediate C/S.

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THANK YOU