Exam Preparatory Manual for Undergraduates Obstetrics and Gynecology

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1

Infertility

Q. Define infertility. What are the causes of infertility?

Q. Causes of female infertility.

Q. Causes of male infertility.

DEFINITION

Infertility is defined as the failure to conceive after **one year** of **regular unprotected** intercourse

Incidence

10–20% of reproductive ages couples.

Types of Infertility

- *Primary:* Patient has never conceived
- *Secondary:* Previous pregnancy but failure to conceive subsequently (irrespective of outcome of that pregnancy).

Causes of Infertility

- Male factor: 30–40%
- Both Male + Female Factors: 20%
- Female factor: 35–50%
- Unexplained: 10%

Female Factors

- Ovarian: 30–40%
- Tubal and peritoneal factors: 30–40%
- Uterine: 5–10%
- Cervical: 5%
- Unexplained: 10–15%.

Worldwide ovarian factors are the most common cause of female infertility, but in India tubal factors are equally or more common.

Ovarian Factors

- **Anovulation** or oligo-ovulation: For examples, **PCOS**, ovarian failure (primary or secondary), thyroid dysfunction, adrenal dysfunction, hyperprolactinemia.

WHO Category for Anovulation
- I: Hypothalamic pituitary failure
- II: Hypothalamic pituitary disturbance/PCOS
- III: Ovarian failure
- IV: Hyperprolactinemia.

- **Diminished ovarian reserve/premature ovarian failure:**
  Increasing age leads to diminished ovarian reserve. Ovarian reserve means the quality and quantity of oocytes in the ovary.

  Causes of premature ovarian:
  - Chromosomal etiology
  - Iatrogenic causes
  - Radiation
  - Chemotherapy
  - Surgical alteration of ovarian blood supply
  - Savage syndrome
  - Infections
  - Autoimmune disorders
  - Galactosemia
  - Cigarette smoking
  - Idiopathic.
- **Luteal phase defect (LPD):** Inadequate function of corpus luteum leading to progesterone deficiency which hinders implantation. LPD is mainly due to defective folliculogenesis. PCOS, ovulation induction, thyroid dysfunction, Hyperprolactinemia, endometriosis, decrease in FSH and/or Luteinizing hormone are important causes.
- **Lutenized unruptured follicle (LUF):** Ovum is trapped inside follicle which gets lutenized. It is a/w hyperprolactinemia, endometriosis.

Tubal and Peritoneal Factors

Tubal obstruction/blocks due to:
- PID (chlamydia, gonococci, etc.)
- Endometriosis
- Pelvic adhesions.
- Genital tuberculosis
- Previous tubal surgery or sterilization

Uterine Factors: (Prevent Implantation)

- Fibroids (Submucous and intramural which distort the cavity)
- Polyps
- Endometritis especially tuberculosis
- Synechiae (Asherman's syndrome)
- Uterine hypoplasia
- Uterine anomalies (septate uterus, unicornuate uterus).

Cervical

- Cervical stenosis
- Prolapse

- Scanty cervical mucus
- Viscous or purulent discharge (chronic cervicitis)
- Antisperm antibody cervical mucus.

Vaginal

- Vaginal atresia
- Transverse vaginal septum.

Male Factors

- Pretesticular (hypothalamic-pituitary disorder): 1-2%
- Testicular disorder: 30-40%
- Post-testicular disorder (sperm transport problem): 10-20%
- **Idiopathic: 40–50% cases.**

Pretesticular	Testicular	Posttesticular
Hypogonadotropic hypogonadism	Varicocele, orchitis, trauma, torsion	Obstruction (infection)
Idiopathic	Heat/irradiation/chemo-therapy	Kartagener syndrome/Young syndrome
Kallmann syndrome (deficient GnRH secretion associated with anosmia)	Bilateral cryptorchidism	Post-vasectomy
Erectile dysfunction/ejaculatory failure	Klinefelter syndrome, Yq 11 microdeletion	Congenital bilateral absent vas deferens (associated with cystic fibrosis)
	Idiopathic	Inguinal hernia repair (accidental damage to vas deferens)

Idiopathic variety is considered to be the MC cause of male infertility.
Varicocele is the MC surgically correctable cause of male infertility.

Causes

I. Congenital

- Undescended testes: Spermatogenesis is affected because the scrotal temperature should be 1–2 °F lesser than the body temperature.
- Congenital absence of vas deferens.
- Kartagener syndrome: Loss of ciliary function and sperm motility.
- Epispadias/Hypospadias: Failure to deposit sperms in the vagina.

II. Thermal factor

Scrotal temperature is raised in conditions such as varicocele, big hydrocele, filariasis, tight undergarments, working in hot atmosphere.

III. Infection

- Mumps orchitis after puberty
- Chronic systemic illnesses such as bronchiectasis
- Mycoplasma or chlamydia trachomatis or viral infection of seminiferous tubules or prostate depresses sperm count.

IV. General factors

Chonic debilitating illnesses, malnutrition, heavy smoking, alcohol (inhibit spermatogenesis by suppressing Leydig cell function and gonadotropin levels).

V. Endocrine

- Kallmann's syndrome: Deficient GnRH Secretion, hypogonadotropic hypogonadism a/w Anosmia
- Sertoli-cell-only-syndrome: FSH is raised in idiopathic testicular failure with germ cell hypoplasia.
- Hyperprolactinemia: Associated with impotence.

VI. Genetic

- Klinefelter's syndrome (47 XXY)
- Yq 11 microdeletion.

These would lead to azospermia or severe oligospermia.

VII. Iatrogenic

Radiation, cytotoxic drugs, cimetidine, Beta blockers, antihypertensives, anticonvulsants and antidepressants can hinder spermatogenesis.

VIII. Immunological

Antibodies against spermatozoal surface antigenà clumping of spermatozoa after ejaculation.

IX. Obstruction of efferent ducts

Due to TB, gonococcal infection, surgical trauma (herniorrhaphy, vasectomy), congenital (Young's syndrome).

X. Failure to deposit sperms high in the vagina (Coital problems)

- Erectile dysfunction
- Hypospadias
- Ejaculatory defect- premature, retrograde or absence of ejaculation.

XI. Errors in seminal fluid

- Low fructose
- High prostaglandin content
- High viscocity.

Factors Affecting Both Sexes

- Environmental and occupational factors
- Excessive radiation damages the germinal cells. Exposure to lead, other heavy metals, and pesticides has also been associated with male infertility
- Smoking and other recreational drugs and alcohol have been associated with infertility in both males and females
- *Exercise:* Compulsive heavy, overstrenuous exercise is deleterious and leads to ovulatory disorders and luteal phase dysfunction in females and oligospermia in males
- Inadequate diet associated with extreme weight loss or gain:
  - Weight has an impact on fertility at either extreme
  - Weight loss associated with anorexia nervosa or bulimia induces hypothalamic amenorrhea
  - Obesity may be associated with anovulation
  - In men, obesity has been associated with decreased sperm quality.

Q. Methods of diagnosing ovulation.
Various methods used to detect ovulation are:

Indirect methods:

1. Menstrual history
2. Evaluation of end organ changes:
   - BBT
   - Cervical mucus study
   - Vaginal cytology
   - Hormonal estimation
     - Sr progesterone
     - Sr estradiol
     - Sr luteinizing hormone
     - Urinary luteinizing hormone
   - Endometrial biopsy
3. USG Follicular Study (TVS preferred)

Direct

- Laparoscopy

Conclusive

- Pregnancy

Indirect

Menstrual History

The following features are strong evidence of ovulation:

- Regular cycles
- **Midmenstrual pain (ovulation pain-Mittelschmerz)** or excessive mucoid vaginal discharge or spotting
- Features of primary dysmenorrhea or PMS.

Evaluation of End Organ Changes

- BBT: Basal body temperature: **Rarely done nowadays**
- **Biphasic pattern** of temperature variation in ovulatory cycle
- **In anovulatory cycle there is no rise of temperature throughout the cycle.**

Principle

Progesterone and norepinephrine both are thermogenic and therefore there would be rise in temperature following ovulation.

Procedure

The patient takes daily oral temperature in morning before rising out of bed.

Interpretation

The temperature is raised by 0.5–1 °F (0.2–0.5 °C) following ovulation and remains high throughout the second half of cycle and falls about 2 days prior to the next period-'**biphasic pattern**'.

There maybe a drop in temperature of about 0.5 °F before the rise and that almost coincides with luteinizing hormone surge or ovulation.

It helps in determining ovulation and helps the couple to determine the most fertile period.

Limitations

- BBT indicates ovulation retrospectively and cannot predict precisely with time
- Rarely ovulation has been observes though BBT is monophasic.

Cervical Mucus Study

From 7th to the 18th day of the menstrual cycle, a fern—like pattern of dried cervical mucus is seen. Disappearance of this fern like pattern beyond 22nd day of the cycle is suggestive of ovulation.

The fern pattern, is due to sodium chloride. Progesterone causes dissolution of sodium chloride crystals and hence ferning will not occur.

Also following ovulation, there is **loss of spinnbarkeit** present in the midcycle.

Vaginal Cytology

Rarely done nowadays.

Maturation index shifts to left from preovulatory phase to the secretory phase due to the effect of progesterone. Single smear on day 25/26 reveals effect of progesterone if ovulation has taken place.

Hormonal Estimation

- **Sr progesterone:** Done on day 8 (<1ng/ml) and **day 21 (>6ng/ml) indicates ovulation**
- Sr estradiol attains a peak rise about 24 hours prior to luteinizing hormone surge

- Sr luteinizing hormone: Daily estimation at midcycle period can detect the luteinizing hormone surge. Ovulation occurs 36 after the onset of luteinizing hormone surge and 12 hours after the luteinizing hormone peak
- **Urinary luteinizing hormone:** Luteinizing hormone kits are available. The patient does the test at home on daily basis. It is started 2 to 3 days before the expected surge. Ovulation occurs 14–26 hours of detecting urinary luteinizing hormone.

Endometrial Biopsy:

Rarely done nowadays for the purpose of detecting ovulation.

- Endometrial sampling can be done on OPD basis with pipple
- D/C reserved for cases if more sample needed (cases of suspected endometrial TB)
- **Done on 21st–23rd day of cycle** (contraception used in the cycle to prevent pregnancy)
- Evidence of secretory endometrium (progesterone action on estrogen primed endometrium) indicates ovulation
- **Subnuclear vacuolation is the earliest evidence (36–48 hours following ovulation).**

Sonography (very commonly done)

- **Serial sonogrpahy (TVS, follicular study) can measure the Graafian/dominant follicle just prior to ovulation (18–20 mm)**
- *It is very useful following ovulation induction for timing of IUI/planned relations and also for ovum pick up in IVF*
- **Collapsed follicle and free fluid in POD are features of recent ovulation**
- Also, the endometrium in proliferative phase has triple stripe echotextural pattern and following ovulation under progesterone influence **endometrium becomes more hyperechoic and homogeneous, and continues to thicken during the luteal phase.**

Laparoscopy

Laparocopic visualization of recent corpus lutem or microscopic detection of ovum from aspirated fluid from POD is the direct evidence of ovulation.

Pregnancy is the surest and conclusive evidence of ovulation.

Q. What is ovarian reserve? What are the tests for ovarian reserve?

INTRODUCTION

Female reproductive aging is a process in which over time, oocytes decrease in quantity and quality and do not regenerate.

Factors such as genetics, lifestyle, environment, and medical issues, including endometriosis, ovarian surgery, chemotherapy, and radiation, can influence the quantity and quality of a woman's oocytes.

Definition

The concept of 'ovarian reserve' defines a woman's **reproductive potential** as a function of the **number and quality of her remaining oocytes.**

The general purpose of ovarian reserve testing is to assess the quality and quantity of the remaining oocytes in an attempt **to predict reproductive potential.**

Tests

Available tests of ovarian reserve include biochemical markers (i.e. FSH, estradiol, antimüllerian hormone, and inhibin B) and ovarian ultrasound imaging (i.e. antral follicle count and ovarian volume). These screening tests have been best studied as predictors of IVF outcome: oocyte yield from ovarian stimulation and rate of pregnancy.

Test	Cutpoint
FSH (International units/L)	10–20
AMH (ng/mL)	0.2–0.7
AFC (n)	3–10
Inhibin B (pg/mL)	40–45
CCCT, day 10 FSH (International units/L)	10–22

Ovarian reserve testing should be performed for women **older than 35 years** who have not conceived after 6 months of attempting pregnancy and women at higher risk of diminished ovarian reserve, such as those with a history of **cancer treatment** with gonadotoxic therapy, pelvic irradiation, or both; those with medical conditions who were treated with gonadotoxic therapies; or those who had **ovarian surgery** for endometriomas and smokers.

Basal Follicle-Stimulating Hormone (Day 3 FSH)

With advancing reproductive age, basal serum FSH concentrations increase on days 2–4 of the menstrual cycle.

High values (greater than 10–20 International units/L) are associated with diminished ovarian reserve and poor response to ovarian stimulation.

Basal Estradiol

Estradiol is released from the ovary during follicular development. The estradiol level is usually low (less than 50 pg/mL) on days 2–4 of the menstrual cycle. However, an elevated value (greater than 60–80 pg/mL) in the early follicular phase can indicate reproductive aging and hastened oocyte development. Through central negative feedback, a high estradiol level can suppress an elevated FSH concentration into the normal range, so the value of obtaining an estradiol level is that it allows the correct interpretation of a normal basal FSH level. Basal estradiol has low predictive accuracy for poor ovarian response and failure to conceive; therefore, this test should not be used in isolation to assess ovarian reserve.

AMH

Antimüllerian hormone is a glycoprotein hormone that is produced by the **granulosa cells of primary, preantral, and antral follicles 2–6 mm in diameter; thus, it reflects the size of the primordial oocyte pool.**

- **AMH test can be done on any day of a woman's cycle** unlike FSH level test, which has to be done on day 2 or 3 of the menstrual cycle
  - As the number of ovarian follicles decreases with age, a concomitant decrease in AMH levels occurs, which reflects this age-related oocyte depletion
  - Undetectable and **low AMH levels (0.2–0.7 ng/mL) indicate diminished ovarian reserve.**

Inhibin B

Inhibin B is a glycoprotein hormone that is secreted primarily by preantral and antral follicles. The serum concentration of **inhibin B decreases with the age-related decrease in the number of oocytes.** Inhibin B has central negative feedback that controls FSH secretion; therefore, a decrease in inhibin B levels leads to increased pituitary FSH secretion and higher early follicular FSH levels.

Clomiphene Citrate Challenge Test

- The clomiphene citrate challenge test is performed by measuring serum FSH on cycle day 3, administering 100 mg clomiphene citrate daily on cycle days 5–9, and again measuring serum FSH on cycle day 10.
- After taking the last dose of clomiphene, **the FSH level should return to a normal level by the next day – menstrual cycle day 10.**
- If the ovaries are not functioning normally, **the FSH level will still be elevated on day 10.**
- In women with a reduced number of ovarian follicles, lower estradiol and inhibin B production leads to less central negative feedback of FSH secretion and an elevated FSH level after clomiphene stimulation.
- Therefore, an elevated FSH level on day 10 of the clomiphene citrate challenge test is suggestive of diminished ovarian reserve.

Ultrasound Evaluation of Ovarian Reserve

Antral Follicle Count

The antral follicle count records the number of visible ovarian follicles (2–10 mm mean diameter) that are observed during transvaginal ultrasonography in the early follicular phase (cycle days 2–5). The number of antral follicles correlates with the quantity of remaining follicles. **A low antral follicle count is considered 3–6 total antral follicles and is associated with poor response** to ovarian stimulation during IVF.

Ovarian Volume

Ovarian volume on TVS decreases with progressive follicular loss. Low ovarian **volume < 3 ml** predicts poor response to ovarian stimulation during IVF.

Q. Tests for tubal patency.

INTRODUCTION

- Tubal and peritoneal factors account for 30–40% cases of female infertility.
- At least one fallopian tube should be structurally and functionally normal to achieve a pregnancy.

The anatomical patency of the fallopian tubes can be assessed by following tests:

1. Rubins's test (obsolete now)
2. Hysterosalpingography (HSG)
3. Saline infusion sonography (Sonohysterosalpingography)

4. Hysterosalpingo contrast sonography (HyCoSy)
5. Laparoscopy and chromopertubation
6. Falloposcopy
7. Salpingoscopy.

1. *Rubin's test/insufflation test: Obsolete now, not done*

Principle: Entry of air or CO_2 into peritoneal cavity when pushed transcervically under pressure gives evidence of tubal patency.

When to do? In postmenstrual phase, 2 days after stoppage of bleeding.

Observation

Pressurized CO_2 is pushed through cervix into uterus via cannula.

The test is considered positive and the tubes are patent:

- Fall in pressure when raised beyond 120 mm Hg
- Gas passes out of the tubes into the abdomen to produce hissing sounds that can be heard on ascultation.
- If it causes referred shoulder pain
- It gives false negative findings due to cornual spasm in one third cases
- **It cannot detect the site and side of block.**

2. *HSG (short note)*

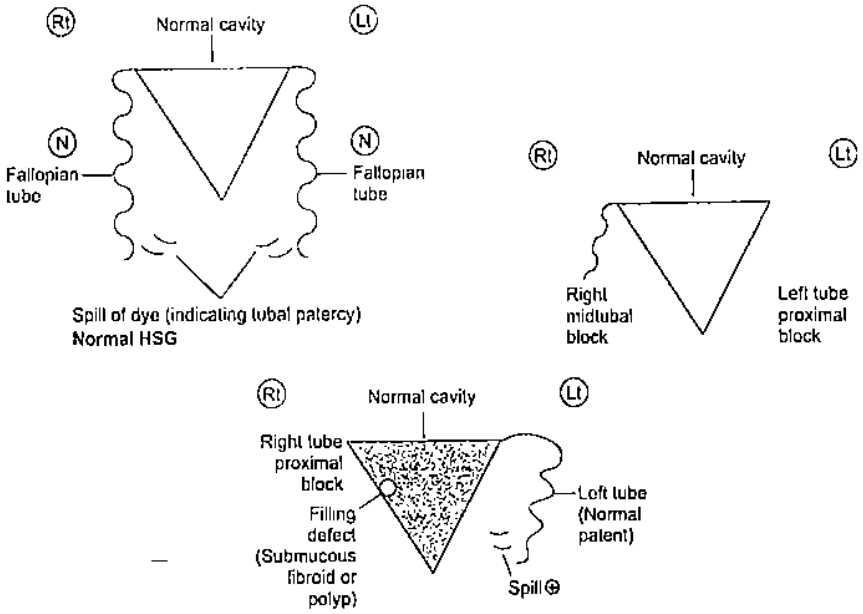


Fig. 1.1

HSG is a radiographic diagnostic study of the uterus and fallopian tubes to assess the uterine cavity and tubal patency.

Considered as the **first line/initial investigation** of choice for tubal factors.

Indications

HSG is helpful in detecting:

- **Tubal patency** in cases of infertility or following tuboplasty surgery. **It can detect the side and site of block**
- It can detect tubal pathologies like:hydrosalpinges, salpingitis isthmica nodosa (SIN), and peritubal adhesions
- Uterine cavity pathologies like polyps, submucosal leiomyomas (filling defects)
- Asherman's syndrome (Intrauterine synechiae) multiple filling defects
- Müllerian anomalies like unicornuate/bicornuate/septate uterus or any variation in cavity shape like 'T' shaped uterine cavity
- After metroplasty operation (septal resection/lateral metroplasty for T shaped uterus) to verify the success of surgery.

When is it done?

The procedure should be performed on **day 8 or day 9** of the menstrual cycle to ensure that the patient is not pregnant and to prevent false-positive intrauterine filling defects and proximal tubal occlusion due to endometrial thickening and to avoid radiation to the ovum.

Pre-procedure

- Antibiotic prophylaxis: Doxycycline, 100 mg orally twice daily, beginning the day before the HSG and continuing for 5 days
- Consent taken
- Pre-procedure atropine given. Antispasmodics/pain killer advisable.

Steps

- HSG is performed by instilling radio opaque contrast into the uterine cavity while using fluoroscopy
- Performed in radiology department **without anesthesia** (major advantage or laparoscopy)
- The anterior lip of cervix is held with vulsellum or tenaculum and cervix is cannulated with Rubins or Calwins cannula
- Radio opaque dye: Water soluble meglumine diatrizoate or oil based dye ethiodized oil is pushed with syringe
- 2 views are taken: One showing the filling of uterus and second the tubes and the peritoneal spill of dye indicating the tubal patency.

Advantages of water-soluble dye

- Provides better detail of the uterine cavity and mucosal folds of the ampullary portion of the tube
- More quickly eliminated

- No granuloma formation
- Negligible peritoneal irritation
- No risk of embolization if extravasation.

Advantages of oil based dye

- Significantly higher post-HSG pregnancy rate (17-23% with water-soluble contrast and 24-38% with oil-based contrast). This could be due to improvement in endometrial receptivity after oil-based contrast exposure and better flushing of tubes)
- Less cramping pain.

Contraindications

Absolute contraindications to the procedure.
- Known contrast allergy
- Pregnancy, and
- Active pelvic infection.

Complications (rare)

- Pelvic pain
- Vasovagal reaction with bradycardia and hypotension, potentially resulting in syncope
- Flair up of pelvic infection
- Allergic reaction to the dye
- Extravasation of dye may result in embolism with an oil contrast agent.

HSG has sensitivity and specificity of **65% and 85%**, respectively, for fallopian tube assessment and sensitivity and specificity of approximately **80%** in correctly identifying uterine cavity pathology with both false-positive and false-negative rates approximately 10–20%.

3. *Saline Infusion Sonography*

Steps

- A transcervical catheter with balloon/pediatric Foley's catheter is placed in cavity and balloon inflated at the level of os.
- Saline is injected under ultrasonographic visualization.
- Longitudinal and transverse views of the cavity are evaluated for filling defects.
- Finally, a small amount of air bubbles are injected to assess tubal patency.
- USG can follow the fluid through the tubes into the POD indicating tubal patency.

When to Do?

The SIS should be performed during cycle days 6–10 (like HSG) so that the endometrium is thin, allowing better detection of intrauterine lesions. In addition, this ensures that an undiagnosed pregnancy is not disrupted.
- Antibiotic prophylaxis: Same as HSG
  If the patient has a history of genital tract infection or pelvic inflammatory disease, antibiotics may be given before the procedure. If hydrosalpinges are noted, antibiotics are given after the procedure.

Advantages

- It provides a simple and inexpensive means by which to evaluate the uterine cavity and assess tubal patency.
- It is well-tolerated by patients and can be done in the OPD.
- Additionally, it **eliminates the risks associated with the use of dye and radiation required by the HSG.**
- SIS has been shown to reveal a substantial percentage of infertile patients with intracavitary abnormalities like synechiae or polyps (superior to HSG) and uterine anomalies.

While the SIS can confirm tubal patency, it does not provide information about the contour of the tubes. Thus, if a patient has a history of endometriosis or other tubal disease, an HSG would be preferred

4. *Hysterosalpingocontrast Sonography (HyCoSy)*

- It is an effective tool for tubal patency and uterine cavity evaluation.
- This investigation is considered safe, well tolerated, rapid, easy to perform and inexpensive.
- HyCoSy is a transvaginal sonography in which a galactose solution containing galactose microbubbles is injected into the uterine cavity using a cervical catheter.
- Several studies have shown that HyCoSy displays high specificity and sensitivity in tubal patency and uterine cavity assessment.

5. *Laparoscopy and Chromopertubation (with a methylene blue dye)*

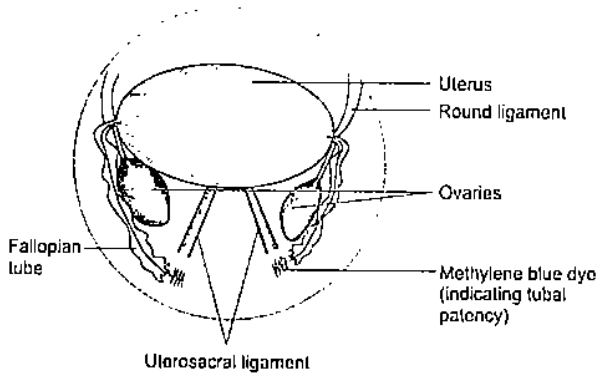


Fig. 1.2: Laparoscopy and chromopertubation

It is the **gold standard (definitive) method** for evaluation of tubal and peritoneal factors for infertility.

It is also the investigation of choice for diagnosing endometriosis.

It is both diagnostic and therapeutic.

Generally combined with hysteroscopy to evaluate the uterine cavity.

Steps

Generally done postmenstrually.

- Laparoscope is introduced into the peritoneal cavity to view the pelvic structures (uterus, tubes, ovaries, POD)
- Methelene blue dye is injected into the uterus through the cervix. The dye passes through the tubes into the peritoneal cavity and can be seen coming out of the fimbrial end indicating tubal patency (spill of the dye)

Thus, the side and the site of the block is visualized.

In infertile patients, laparoscopy (diagnostic) can evaluate:

- Tubes: Tubal blocks, tubal patency (with chromopertubation), peritubal adhesions, hydrosalpinges, genital tuberculosis
- Uterus: Uterine anomalies, fibroids
- Peritoneal factores: Endometriosis, adhesions ,PID
- Ovaries: PCOD, Chocalate cyst of endometriosis.

Therapeutic: In the same sitting

- Tubes: Adhesiolysis, tuboplasty, TL reversal
- Uterus: Myomectomy
- Ovary: Cystectomy,oophorectomy,cyst aspiration, LEOS for PCOS
- Endometriosis.

Indications

- Infertility with advanced age (>35 years)
- Abnormal HSG
- Failure to conceive after reasonable period (6 months) of normal HSG
- Unexplained infertility
- Suspected endometriosis, chronic PID.

6. *Falloposcopy*

- A flexible micro endoscope (through the hysteroscope) is inserted into the tube through its uterine opening to study the lumen of entire length of tube.
- Initially a hysteroscope is introduced into the uterus to identify the proximal tubal ostium. Once the ostium is identified the falloposcope can be inserted through the operative channel of the hysteroscope and advanced.
- The patient needs general anesthesia or conscious sedation.
- Obstructions, adhesions, polyps within the tubal canal, and debris can be identified.
- Perforation of a fallopian tube is a known complication.

7. *Salpingoscopy*

Tubal lumen is studied introducing a rigid endoscope through the fimbrial end of the tube. It is performed through the operative channel of a laparoscope.

Q. Evaluation of uterine factors of infertility.

INTRODUCTION

Uterine factors are responsible for around 10% cases of female infertility.

Uterine factors (prevent implantation) which can lead to infertility or recurrent pregnancy loss are:

- Fibroids (submucuous and intramural which distort the cavity)
- Polyps
- Endometritis especially tuberculosis
- Synechiae (Asherman's syndrome)
- Uterine hypoplasia
- Uterine anomalies (septate uterus, unicornuate uterus).

Evaluation

- USG

Ultrasonographic imaging is an effective, easy to use, safe, and readily available noninvasive method. 3D USG is also now available.

It can detect **fibroids, polyps, uterine anomalies.**

Also, it can measure **endometrial thickness (ET) and vascularity** which is very important factor for implantation.

Endometrium in proliferative phase has **triple stripe echotextural pattern** (estrogenic action) and following ovulation under progesterone influence endometrium becomes more hyperechoic and homogeneous, and continues to thicken during the luteal phase.

- HSG: As in previous answer
- SIS: As in previous answer
- Hysterosalpingocontrast sonography (HyCoSy) As in previous answer
- Laparoscopy can evaluate the uterus from outside and is helpful in cases of intramural fibroids, uterine anomalies (unicornuate/bicornuate uterus). **It cannot diagnose intracavitatory pathology and septate uterus.**
- **Hysteroscopy:**
  - **Most useful investigation to evaluate any intracavitatory pathology**
  - It is both diagnostic and therapeutic
  - Increases accuracy in diagnosing the cause of intrauterine filling defects detected on HSG
  - Intracavitary lesions (fibroids, septum, and adhesions) implicated as causes of infertility can be visualized and treated
  - In unexplained infertility, hysteroscopy may be performed simultaneously with laparoscopy to evaluate the uterine cavity.

Indications of Therapeutic/Operative Hysteroscopy

For all intracavitatory pathologies **hysteroscopy is the gold standard for treatment.**

- **Polyps and fibroids:** Endometrial polyps and submucous fibroids are well known to cause infertility and can be removed with hysteroscopy (polypectomy, myomectomy).
- Intrauterine adhesions: (Asherman syndrome).
- The intrauterine adhesions are often associated with amenorrhea or infertility.
- **Hysteroscopy is the gold standard used to diagnose and treat these adhesions.** Benefits include visually directed lysis of adhesions.
- **Lateral wall Metroplasty** in cases of T-shaped uterus and **septal resection** in cases of septum.
- It can also help in taking endometrial biospy for genital TB (histopathology and TB PCR).

Q. Evaluation of male partner in an infertile couple.

INTRODUCTION

Infertility is defined as the failure to conceive after one year of regular unprotected intercourse.

Male is directly responsible in 30–40% of cases and both male and female could be responsible in about 20% cases.

Detailed History

- Age, duration of marriage, any previous marriage and proven fertility to be enquired
- Medical history including history of STDs, infections such as **Mumps orchitis** after puberty, TB, recurrent chest infection **(Young's Syndrome)**, diabetes
- *Any recent febrile illness* (can depress spermatogenesis upto 6 months)
- *H/O surgeries, e.g.* ***Herniorrhaphy,*** *operation on the testis, genital area (orchidopexy, scrotal, urethral surgey, retroperitoneal surgery)*
- Undescended testes, testicular trauma/torsion
- *Occupational History:* To note if there is excessive exposure to heat/radiation or h/o chemotherapy or radiotherapy
- Sexual history- Erectile/ ejaculatory dysfunctions, frequency of coitus
- *Impotence, loss of libido, diminution in beard growth and decrease frequency of shaving are suggestive of* ***testosterone deficiency***
- Use of any antiandrogen drugs (cimetidine, spironolactone) or anabolic steroids
- Headache, visual disurbances, galactorrhea (pituitary tumours, hyperprolactinemia) and anosmia (Kallmann's syndrome)
- H/O addictions; smoking, alcohol.

Physical Examination

General

- Body proportions
- Beard growth
- Anosmia
- Gynecomastia.

Penile Examination

- Urethral meatus

- Ulcers/Discharge
- Scars.

Scrotal/Testes

- Testes volume **(Prader orchidometer). Average testicular volume =18 ml (range 12–30 ml)**
- Small testes indicative of pretesicular or testicular causes
- In pretesticular causes testes size generally is 5–12 ml with soft consistency
- Consistency of testes (in **KF syndrome small, firm testes, < 3 ml each)**
- Position
- Presence of varicocele.

Epididymal

- Nodules/ irregularity
- Tenderness
- *Palpable cystic distention of caput epididymis* ***(Bayle's sign)*** *indicates epididymal obstruction.*

Vas Deferens

- Presence/absence
- Nodules/irregularity
- Tenderness.

Investigations

1. *Routine tests:* Blood sugars, TSH, urinalysis
2. *Semen analysis:* (Asked as a separate short note) **The first and the most important investigation.**

Methods of semen collection include:

- *Masturbation,* directing the sample into a sterile container. This is the most common way to collect a semen sample.
- *Sexual intercourse* in a special type of *condom* known as a *collection condom.* Collection condoms are made from silicone or polyurethane, as *latex* is somewhat harmful to sperm. Such samples are inferior to the ones collected by masturbation in clean cup.
- *Coitus interruptus* (withdrawal). With this technique, the man removes his penis from his partner near the end of intercourse and ejaculates into a wide-necked cup or bottle.
- Sample sent to laboratory as soon as possible so that examination is performed within 2 hours.
- Penile vibratory stimulation and electroejaculation are two other alternatives for men with *anejaculation* due to *spinal cord injury.*

The ideal specimen for examination is after **3–5 days of abstinence.** More prolonged period does not yield better results.

Accepted reference values for semen analysis (WHO, 2010)

	Reference value
Ejaculate volume	1.5 ml or more
pH	7.2–7.8
Sperm concentration	15 million /ml (20 million/ml is the old criteria)
MOTILITY (within 1 hour of collection)	
Total motility (progressive + nonprogressive):	40 % or more (50% or more is old criteria)
Progressive motility	32% or more
Normal Morphology	4% or more (70% or more old criteria)
Vitality (live spermatozoa):	58 % or more
Leukocytes	Less than 1 million/ml
Viscosity	< 2 cm thread post liquefaction

Abnormal semen analysis (Nomenclature)

- Aspermia: Absence of semen
  - Azoospermia: Zero sperm count
  - Asthenospermia: Less than 40% motile spermatozoa
  - Oligozoospermia: Count less than 15 million/mL
  - Teratospermia: Less than 4% normal forms
  - Necrozoospermia: Dead or immobile sperms
  - Oligoasthenoteratospermia: Disturbance in all 3 variables.

3. Hormonal evaluation

Serum FSH, Luteinizing hormone, Testosterone, Prolactin, TSH

- Testicular dysfunction causes an increase in FSH and Luteinizing hormone
- Sr FSH level estimation helps determine the site of pathology:
  - i. A **very high FSH** would indicate a **testicular cause**
  - ii. A very low FSH would indicate pretesticular (hypothalamic/pituitary) cause
  - iii. A **normal FSH** would indicate a **post-testicular cause**
  - Low FSH and Luteinizing hormone are seen in hypogonadotrophic hypogonadism
  - Low testosterone, high Luteinizing hormone are noted in Leydig cell dysfunction
  - Elevated Prolactin may be seen in pituitary adenoma leading to impotency.

4. ***Fructose content in seminal fluid:*** Absent fructose suggests congenital absence of seminal vesicles/ part of ductal system or both.
5. ***Testicular Biopsy:*** Done in cases of azoospermia to differentiate testicular failure from Post-testicular pathology. The tissue to be sent in **Bouin's solution** and not in formol saline.
   - The testicular tissue may be cryopreserved for future ICSI.
6. ***TRUS (transrectal ultrasound):*** To visualize the seminal vesicles, prostate and ejaculatory ducts obstruction

Indications for TRUS

- Azoospermia
- Severe oligospermia with normal testicular volume
- Abnormal digital rectal examination
- Ejaculatory duct abnormality (cysts, dilatation).

Distention of seminal vesicles on TRUS is suggestive of ejaculatory duct obstruction.

7. *Scrotal ultrasonography*

Scrotal ultrasonography is used to evaluate the anatomy of the testis, epididymis, and spermatic cord. It is a useful adjunct for evaluating testicular volume, testicular and paratesticular masses, and colour Doppler for the presence or absence of varicoceles.

8. *Vasogram*

Radiographic study to evaluate ejaculatory duct obstruction. Mostly replaced by TRUS.

9. *Chromosome and genetic analysis*

- To be done in cases of azospermia with testicular causes(raised FSH)
- **Klinefelter's syndrome** (XXY) is the commonest abnormality
- **Yq11 microdeletions** (microdeletions in long arm of Y chromosome) can also be detected (male children of these men will carry same microdeletions and exhibit infertility in adulthood)

10. *In selected case, biochemical tests of creatine phosphokinase and reactive oxygen species are done as sperm function tests. CPK helps in sperm transport and ROS interfere with sperm function.*

Q. Various treatment options for male infertility.

Q. ART (IUI, IVF, ICSI).

INTRODUCTION

Male is directly responsible in 30–40% of cases of infertility and both male and female could be responsible in about 20% cases.

The treatment will depend on the cause and the semen analysis report.

The treatment is often difficult and unsatisfactory.

General

Lifestyle Modification

- **Improvement in general health, weight loss in cases of obesity,** and patients should be encouraged to **stop smoking cigarettes** and alcohol and to limit environmental exposures to harmful substances and/or conditions.
- **Control of sugars in cases of diabetes mellitus and correction of thyroid disorders if present.**

- Stress-relief therapy and consultation of other appropriate psychological and social professionals may be advised.
- Infections should be treated with appropriate antimicrobial therapy.

Specific

- **Impotence:**
  - Psychosexual treatment may help
  - Dopamine agonist **(cabergoline, bromocriptine)** in cases of hyperprolactinemia
  - **Sildenafil 50–100 mg or tadafil (10–20 mg)** is recommended one hour before sexual activity
  - In unresponsive cases IUI can be done.
- **Retrograde ejaculation:**
  - Phenylephrine (alpha agonist) to improve the tone of internal urethral sphincter
  - **Postejaculatory urine sample** can be used to recovery the sperms and **IUI** can be done.
- **In hypogonadotropic hypogonadism:**
  The following is tried with varying success:
  - **Injection HCG** 5,000 IU, IM given once or twice a week (to increase endogenous testosterone production)
  - **Injection HMG or FSH** (75–150 IU) twice or thrice a week is added if no response
  - **Pulsatile GnRH** therapy is also helpful especially in cases of Kallman's syndrome. It is given by minipumps infusion
  - **Clomiphene citrate:** 25 mg once a day for 2–3 months increases FSH, Luteinizing hormone and testosterone and can increase the sperm count.
- **In hypergonadotropic hypogonadism/testicular failure:**
  - Medical treatment has no role
  - Treatment options include (depending on the sperm count)
    - IUI
    - IUI-DONOR (AID = artificial insemination donor)
    - IVF/ICSI
    - Adoption.
- In cases of genetic abnormality (KF syndrome,Yq11 microdeletions), sertoli-cell-only-syndrome, testicular atrophy only options are:
  - AID
  - Adoption.
- **In cases of oligoasthenospermia due to idiopathic causes:**

Various medical management can be tried with variable success.

The following are thought to improve sperm count/motility:
1. Antioxidants like astaxanthin
2. Multivitamin (specially vitamin E and C)
3. Coenzyme Q
4. Levocarnitine

5. Zinc, selenium
6. Glutathione
7. L-arginine.

Clomiphene citrate: 25 mg once a day for 2–3 months increases FSH, Luteinizing hormon and testosterone and can increase the sperm count .

IUI or IVF (depending on the count) will be needed in cases where the medical managemen fails.

- **Surgeries**

Varicocele

- **Varicocele is the MC surgically correctable cause of male infertility.**
- Successful varicocelectomy results in improvement in semen parameters in 60–70% o patients.
- The repair also typically halts further testicular damage and improves Leydig cell function
- Semen analysis may show improvement as early as the 3-month follow-up visit.

In cases of obstruction of vas or epididymis (normal FSH and azoospermia, i.e. post tesiticular pathology):
- Microsurgery: Vasoepididymostomy or vasovasostomy
- Patency can be obtained in upto 80% cases and pregnancy rate is 50%.

Transurethral Resection of the Ejaculatory Ducts

Patients with a known or suspected obstruction of the ejaculatory ducts may be eligible fo transurethral resection of the ejaculatory ducts (TURED), which durably improves semer quality in patients with ejaculatory duct obstruction.

- ***ART (Assisted Reproductive Technology)***

ART encompasses all procedures that involve manipulation of gametes and embryos outsid the body for treatment of infertility.

It mainly includes:
1. IUI
2. IVF-ET
3. ICSI
   GIFT, ZIFT, POST, and SUZI ARE NO LONGER DONE.

1. *IUI (Intrauterine Insemination)*

Mainstay treatment and most commonly used treatment for male infertility.

Indications

- Male factor infertility (sperm counts between 5 and 15 million/mL). **If sperm count is less than 5 million/mL, IUI is ineffective**

- Unexplained infertility (treatment of choice is superovulation + IUI)
- Antisperm antibody in cervical mucus
- Erectile dysfunction/impotency
- Semen deposition problem (epispadias/hypospadias/penile deformities)
- Vaginismus
- Retrograde ejaculation (Immediate postcoital urine is collected. Semen is then separated from urine).
  - **Patent fallopian tube is prerequisite.** Fallopian tubes have to be patent for IUI to be successful. If fallopian tubes are blocked, IUI should not be done.
- In IUI, the semen sample is washed/prepared (swim-up technique/swim-down technique/density gradient technique)
  - The dead sperms/debris and immotile sperms are removed; only highly motile good-quality sperms are taken in the catheter and
  - 0.5-0.7 mL washed sample is injected into the uterine cavity at the time of ovulation
  - Follicular study is done and injection HCG 5000 IU, IM is given when the dominant follicle is **18-20 mm**
  - IUI is generally done 36 Hours after HCG injection.

The success of IUI in one cycle is about 15-20%.

Total 3-6 cycles may be needed (50-60% success).

IUI with Donor semen is done in cases of:

- Genetic disorders
- Azospermia due to testicular causes (sperms not available at all for ICSI)
- Oligoasthenospermia requiring IVF/ICSI in nonaffording patients
  - The donor should be healthy with same blood group and ethinic group as the husband
  - Free from venereal diseases including HIV and hepatitis
  - Consent of both the partners is required.

If the sperm count is less than 5 million/mL:

The options are:

- IVF/ICSI
- IUI-D (donor) in patients not affording IVF/ICSI
- Adoption.

2. ***In vitro fertilization and embryo transfer (IVF-ET)***

Indications

- Tubal pathology/blocks
- Male factor: Count less than 5 million/mL
- ≥ 6 IUI failures
- Unexplained infertility
- Ovarian failure (Donor Oocyte IVF)
- Surrogacy.

Basic Steps of IVF

- Ovarian stimulation with gonadotropins and follicular monitoring
- Oocyte retrieval (ovum pickup) done through TVS—guided needle
- Fertilization: 50,000 sperms are put on each oocyte retrieved

- Embryos kept in incubator for 48-72 hours
- ET done on day 2 or day 3 (48-72 hours) after oocyte retrieval
- Generally 2-4 embryos are transferred in the uterine cavity via catheter and deposited 1 cm below the fundus
- **Success rate of IVF per cycle is 30-35%.**

3. ***Intracytoplasmic Sperm Injection (ICSI) (Micromanipulation)***

Indications:

- Severe oligoasthenoteratospermia
- Azoospermia (provided testes are producing sperms)
- Repeated fertilization failure in IVF.

The steps are identical to IVF (oocyte retrieval and embryo transfer), but for fertilization, one sperm is mechanically injected into one oocyte.

Success rate of ICSI per cycle is 30-35%.

Sperm retrieval techniques in case of azoospermia before doing ICSI:

- PESA = percutaneous epididymal sperm aspiration
- MESA = microscopic epididymal sperm aspiration
- TESA = testicular sperm aspiration
- TESE = testicular sperm extraction (testicular biopsy).

Q. Treatment of female infertility.

INTRODUCTION

Female factor accounts for 35-50% cases of infertility and in 20% cases both partners have a problem.

TREATMENT: GENERAL AND SPECIFIC

General

Lifestyle Modification

- **Improvement in general health, weight loss in cases of obesity,** and patients should be encouraged to **stop smoking cigarettes** and alcohol and to limit environmental exposures to harmful substances and/or conditions.
- **Control of sugars in cases of diabetes mellitus and correction of thyroid disorders if present.**
- Stress-relief therapy and consultation of other appropriate psychological and social professionals may be advised.
- Infections should be treated with appropriate antimicrobial therapy.

Specific

Specific treatment depends on the cause of infertility.

Anovulation

- Ovulation Induction (Medical and Surgical): **Refer to PCOS answer**

Diminished Ovarian Reserve (DOR)

Options are:

1. **Dehydroepiandrosterone (DHEA) 25 mg three times a day for 3 months** has been reported to improve pregnancy chances in patients with DOR which occurs either as a consequence of premature ovarian aging (POA) or female aging.
   DHEA is a naturally existing hormone that the female body converts into androgens, mainly testosterone. Even though androgens are male hormones, they're present in both sexes and are essential in the female body for the production and development of healthy eggs.

   DHEA's beneficial effects on female fertility include:
   - Increased IVF pregnancy rates
   - Increased chance of spontaneous conceptions
   - Shortened time to pregnancy
   - Increased quality and quantity of eggs and embryos
   - Decreased risk of miscarriage and chromosomal abnormalities in embryos
   - Improved cumulative pregnancy rates in patients under fertility treatment.
2. IVF: as in previous answer.
3. IVF with donor oocyte.

LPD

Progesterone gels or natural micronized progesterone 100–200 mg, 2 to 3 times a day vaginally or orally can be used from day of ovulation for 10–14 days.

If the patient conceives it should be continued till 10 weeks of gestation.

Endometriosis

Refer to Answer of Endometriosis

Tubal Factor

- Tuboplasty
- IVF (as in previous answer) if tuboplasty fails or is unsuccessful
- Adoption.

Tuboplasty operation	
Adhesiolysis (Salpingo-ovariolysis)	Separation or division of adhesions
Fimbrioplasty	Separation of the fimbrial adhesions to open up the abdominal ostium
Salpingostomy	That creates a new opening at the abdominal ostium in a completely occluded tube. It is called terminal or **'cuff' salpingostomy.** The eversion of the neo-ostium is maintained by few stitches of 6–0 Vicryl
Tubotubal anastomosis	When the segment of the diseased tube following tubectomy operation is resected and end to end anastomosis is done
Tubocornual anastomosis	When there is cornual block, the remaining healthy tube is anastomosed to the patent interstitial part of the tube

Cornual cannulation: 10–20% of all tubal blocks are in the proximal part of the fallopian tub

Management with IVF or microsurgical tubocornual anastomosis is more expensive, invasi and less successful.

Cornual cannulation can be one under fluoroscopy, ultrasongraphy or hysteroscop guidance.

With the introduction of operative hysteroscopy, **cannulation of fallopian tube ostia is no the procedure of choice to treat proximal tubal blocks.**

Hysteroscopic proximal tube cannulation can be performed for proximal blocks and is gene ally accompanied with laparoscopy so that any distal tubal disease if present can be evaluate and simultaneously the tubal patency can be confirmed with chromopertubation.

Advantages

- Laparotomy and IVF are avoided
- High success rates
- Day care surgery
- Safe and highly cost effective.

Results

Hysteroscopic tubal cannulation can result in a **patency rate of up to 90%** in at least 1 tub and a pregnancy rate in the range of **50–60%.**

Reversal of Tubal Ligation

The remaining length of the tube is one of the most important factors influencing reversa The more the length, the more successful the results. **Minimum length of reconstructed tub should be 4 cm and the ampullary part should be at least 2 cm.**

Ishtmo-isthmic anastomosis has maximum chance of success.

Most suitable for reversal is clips followed by silastic bands.

The patient should be informed of the 10 times higher chance of ectopic pregnancy (risk o ectopic pregnancy is 3–9%), with danger to the life of the woman herself, following the reversa procedure.

Results of microsurgical reconstructive surgery after sterilization procedures

Sterilization procedure	Term pregnancy (Range %)
Spring-loaded clip	88 (75–100)
Ring occlusion (silastic bands)	75 (44–95)
Pomeroy ligation	59 (45–70)
Electrocoagulation	43 (26–58)

Uterine Factors

For all intracavitatory pathologies hysteroscopy is the gold standard for treatment.

- Polyps and fibroids: Endometrial polyps and submucous fibroids are well known to cause infertility and can be removed with hysteroscopy (**polypectomy, myomectomy**).
- Intrauterine adhesions: (Asherman syndrome).

  The intrauterine adhesions are often associated with amenorrhea or infertility.

  Hysteroscopy is the gold standard used to diagnose and treat these adhesions. Benefits include visually directed lysis of adhesions.

  After adhesiolysis an IUCD or pediatric Foley's catheter is inserted to keep the cavity distended to prevent recurrence. OC pills or estrogen and progesterone tablets are used for 1-3 months to regenerate the endometrium.
- **Lateral wall metroplasty** in cases of T-shaped uterus and **septal resection** in cases of septum.

Cervical Factors

- Cervical mucus quality can be increased by estrogen supplementation
- Also **n-acetyl cysteine** makes cervical mucus thin and improves sperm penetration
- Infections if any are to be treated
- **IUI** is very commonly used.

Unexplained Infertility

- **Super Ovulation + IUI** treatment of choice
- IVF in cases where the above treatment fails.

Immunological Factors

In cases of antisperm antibodies in cervical mucus: Steroids can be given to female and male partner can use condoms .However the benefit is limited and so IUI is the preferred treatment.

In Cases of Normal Ovaries and Absent Uterus

Surrogacy (gestational surrogacy = IVF surrogacy)

Examples

- Congenital absence of the uterus (RMKH SYNDROME)
- Postpartum hysterectomy
- Hysterectomy done for any reasons (cancer, menorrhagia, etc.)
- TB endometritis with dense intrauterine adhesions.

2

Infections

Q. Describe the pathogenesis, clinical features management and treatment of genital tuberculosis.

Q. Genital tuberculosis.

INCIDENCE

Genital TB is seen in 1% of outpatients in developing countries. 5-10% of patients with infertility have genital TB.

Pathogenesis

- Causative organism: Mycobacterium tuberculosis (human type)
- Genital TB **is almost always secondary** to primary infection in extragenital sites like lungs (most common), lymph nodes, urinary tract, bones and joints
- **In the genital tract, the falloplan tubes are usually the first to get infected.**

Mode of Spread

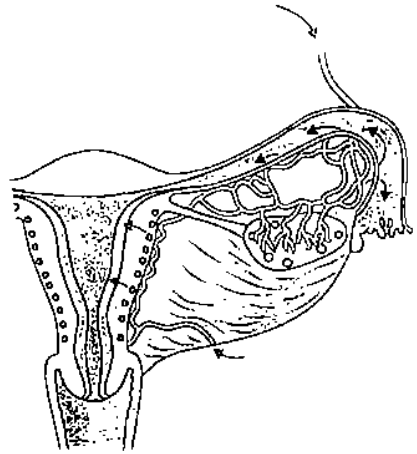


Fig. 2.1: Mode of spread in tubercular infection through bloodstream

- *Blood stream:* In 90% of cases, the pelvic organs are involved by hematogenous spread from any primary site. If the postprimary hematogenous spread coincides with the growth spurt of puberty, the genital organs are affected. If the spread is earlier than the growth phase, the genital organs are spared. The infection remains dormant for 4-6 years before clinical features appear.
- *Lymphatic/Direct:* The genital organs are involved directly or by lymphatics from infected organs like peritoneum, bowel or mesenteric lymph nodes.
- *Ascending (very rare):* Sexual transmission from a male with urogenital TB is possible in vulval, vaginal or cervical lesions.

Pathology of Pelvic Organs

Fallopian Tube (100% cases)

- **Most common organ involved.**
- **Ampullary part** most commonly affected.
- The initial site of infection is the **submucosal part** of the ampullary part of the tube.
- The infection spreads medially along the wall destroying the muscles which are replaced by fibrous tissue.
- The walls get thickened at times in segments, calcified or ossified.
- The infection may spread inwards; the mucosa gets swollen and destroyed.
- The fimbria are everted. The elongated and distended distal tube with the patent abdominal ostium causes the appearance of '**tobacco pouch**'. At times, adhesions may cause occlusion of the ostium.
- The tubercles burst pouring caseous material inside the lumen producing tubercular pyosalpinx which many adhere to the ovaries and other adjacent structures. The infection may spread outwards producing **perisalpingitis** with exudation causing dense adhesions with the surrounding structures, thus forming a **tubo-ovarian mass (TO Mass).**
- At times, miliary tubercles may be seen on the serosal surface of the tubes, uterus, peritoneum or bowels. These are often seen with TB peritonitis.
- At times, the tubes may look normal or nodular at places. These nodules may be present in the isthmic part of the tubes due to proliferation of the tubal epithelium within the hypertrophied muscle layer. This is known as salpingitis isthmica nodosa (SIN). It is seen radiologically as a small diverticulum. SIN is also seen in pelvic endometriosis.

Uterus (80% cases)

- 2nd MC organ involved.
- The infection may spread from the tubes by lymphatic spread or directly through proximity.
- **Cornual endometrium** are commonly affected due to their dual blood supply as well as their anatomical proximity to the tubes.
- Infection starts from the basal endometrium and reaches the surface premenstrually and gets shed off with menses. Reinfection again starts from the basal layer of the endometrium.
- Endometrial ulceration may result in adhesions - *'Asherman's Syndrome'*. This may lead to secondary amenorrhea, infertility and recurrent abortion.
- Rarely the infection spreads to the myometrium and if caseation occurs it may lead to pyometra.

Cervix (20% cases)

- Not commonly affected
- May be ulcerative or nodular type
- It may present as contact bleeding
- On gross appearance, it may appears like CA cervix.

Vulva/Vagina: Rare (1%)

- The lesion may be ulcerative with undermined edges
- Rarely hypertrophic variety.

Ovary (30% cases)

May present as surface tubercles, adhesions, capsular thickening or caseating abscess.

Pelvic Peritoneum (40–50% cases)

- TB peritonitis may be wet or dry
- Wet peritonitis presents with ascites (straw colored fluid) and tubercles on peritoneal surfaces
- In the dry variety, there are dense bowel adhesions due to fibrotic sequelae when the wet variety heals.

Microscopy

Granuloma consists of infiltration of **Langhan's cells**, chronic inflammatory and epithelioid cells surrounding a central area of **caseation necrosis.**

Clinical Features

80% of the affected patients are of childbearing age (20–40 years). Genital TB occurs in 10–20% of patients who have pulmonary TB in adolescence.

Symptoms

- Many patients are **asymptomatic** and accidently detected during infertility work up.
- Infertility:
  - Most common presenting feature, present in 70–80% cases
  - It may be primary or secondary
  - Its mainly due to tubal blockage
  - Adhesions in endometrial cavity or ovulatory dysfunction also contribute.
- Menstrual abnormality:
  Menorrhagia → oligomenorrhea → secondary amenorrhea.
  - In early stages, menorrhagia due to ovarian involvement, pelvic congestion or endometrial proliferative lesion. It is a rare cause of puberty menorrhagia
  - In later stages, oligomenorrhea and then secondary amenorrhea are common. This may be due to **endometrial destruction due to uterine synechiae formation** or ovarian suppression due to tuberculin toxin.
- Chronic pelvic pain: In 20-30% cases. Often a/w TO MASS.

- Vaginal discharge: Cervical or vaginal TB a/w postcoital bleeding or blood stained discharge
- Constitutional symptoms such as weight loss, fever, anorexia.

Signs

General: General health usually unaffected. May be low grade fever, anemia. There may be evidence of active/healed tubercular lesion.

Per abdomen: Negative findings or doughy feel due to matted intestines. TB ascites when encysted resembles an ovarian cyst.

Per vaginum: Negative findings in 50% cases.
- Vulval/vaginal ulcers with undermined edges
- Palpable thickened tubes felt through lateral fornices
- Pelvic masses
- Nodules through posterior fornix.

Investigations

- Blood: Increased ESR, leukocytosis.
- Mantoux Test.
- Chest X-ray for healed/active pulmonary TB.
- Dilatation and curettage: **Preferably one week prior to menses.**
  Endometrium to be sent in formalin for **histopathology** to detect the giant cell and caseation and in normal saline for: AFB microscopy by **Ziehl-Neelsen's stain, culture in Lowenstein Jensen media.**
- **PCR:** Sample also send for detection of M. tuberculosis by polymerase chain reaction (PCR) for nucleic acid amplification. PCR is more sensitive (85–95%) than microscopy and culture. It can detect < 10 organisms in specimens compared to 10000 needed for smear positivity.
- Menstrual blood- collected with a pipette and sent AFB stain/culture/PCR.
- Sputum and urine culture for TB bacillus.
- Biopsy from lymph nodes, lesions in the cervix, vagina or vulva.
- **In a proven case of tuberculosis, HSG is contraindicated** for the risk of reactivation and spread. But HSG done for routine work up of infertile female may reveal the following suggestive features (Fig 2.2):

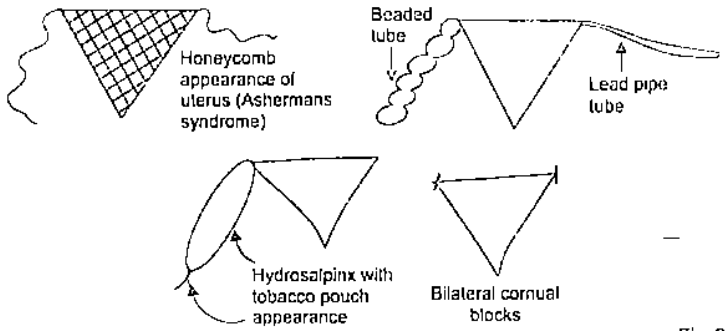


Fig. 2.2 Contd...

Fig. 2.2 Contd...

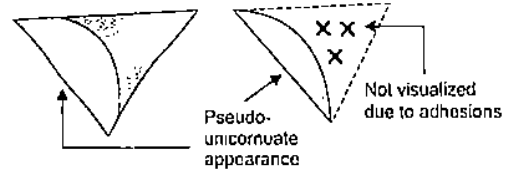


Fig. 2.2: Various HSG findings in case of genital TB

  - Lead pipe tubes
  - Tobacco pouch appearance
  - Beaded tubes
  - Hydrosalpinx
  - Cornual blocks
  - Intravasation of the dye
  - Golf club tube
  - Sperm head tube
  - Uterus—honeycomb appearance (Asherman syndrome)
  - Pseudo-unicornuate appearance
- USG/CT scan in case of pevic mass/ascites
- Laparoscopy: If no endometrial evidence, laparoscopy may be done. This may reveal tubercles and beaded tubes and TO masses. Biopsy can be taken from peritoneal tubercles. Peritoneal fluid may be sent for culture **and ADA (ADENOSINE DEAMINASE) Levels.**

Differential Diagnosis

- Pyogenic tubo-ovarian mass
- Pelvic endometriosis
- Adherent ovarian cyst
- Chronic ectopic pregnancy.

Treatment

General

Hospitalization only for active infection. Correction of anemia and healthy diet to improve resistance is essential. The husband should use a condom during active infection to prevent possibility of contracting urogenital TB.

Chemotherapy

AKT IS THE TREATMENT OF CHOICE

Genital tuberculosis falls in category 1. The treatment is for minimum 6 months.

A. Initial phase: Four drugs are used for 2 months to reduce bacterial load. The drugs are as below:

Drug	Daily adult dose	Type	Toxicity	Points to note
Isoniazid	5 mg/kg max 300 mg	Bactericidal	Hepatitis, peripheral neuropathy	Add pyridoxine 50 mg/day, check LFTs
Rifampicin	10 mg/kg max 600 mg	Bactericidal	Hepatitis, orange discoloration of urine, febrile reaction	Check LFTs, Avoid OCPs
Pyrazinamide	20–25 mg/kg max 2 gm	Bactericidal	Hepatitis, Hyperuricemia, GI upset, arthralgia	Check LFTs, Active against intracellular dividing forms of mycobacterium
Ethambutol	15–20 mg/kg max 2.5 gm	Bacteriostatic	Optic neuritis, visual disturbances	Ophthalmoscopy prior to therapy

B. Continuation phase:
Isoniazid and Rifampicin are continued for 4 more months.
After 1 year of treatment, endometrium should be sampled. If histological/bacteriological examination is positive, the treatment must be continued further. If negative, it should be rechecked after 6 months. A patient may be considered cured if atleast 2 histological reports come negative.

For Infertility

- IUI should not be done as tubes are damaged
- **IVF after completion of AKT** is the treatment for infertility (provided the uterine cavity is normal)
- If the endometrium is cicatrized, then **IVF and surrogacy should be recommended.**

Surgery: **Not routinely indicated.**

Indications

- Unresponsive active disease despite adequate chemotherapy
- TB pyosalpinx, ovarian abscess or pyometra
- Chronic pelvic pain.

Surgery for restoration of fertility (corrective tuboplasty) is contraindicated in genital TB.

Contraindications

- Active TB in extragenital site
- Good response to AKT with decrease in size of pelvic mass
- Incidental finding of tubercular tubo-ovarian mass on laparotomy in young patient. After taking tissue for biopsy, the abdomen should be closed.

Surgery

- Total abdominal hysterectomy with bilateral salpingo-oophorectomy
- In young women, at least one ovary should be preserved
- In selected cases, isolated excision of tubo-ovarian mass, drainage of pyometra or fistula repair may be done.

Prognosis

Pregnancy is rare (5–10%). If the patient conceives, there is 40% chance of ectopic pregnancy and high probability of spontaneous abortion. Higher pregnancy rates are noted with the help of assisted reproductive technology after completion of AKT (provided the uterine cavity is normal). If the endometrium is cicatrized, then IVF and surrogacy should be recommended.

Q. Describe the pathogenesis, clinical features and management of acute pelvic infection.

Q. Acute PID: Clinical features and treatment.

DEFINITION

Pelvic inflammatory disease (PID) is a spectrum of infection and inflammation of the upper genital tract involving the endometrium, fallopian tubes, ovaries, pelvic peritoneum and surrounding structures.

Incidence

The incidence is on rise due to rise in sexually transmitted diseases.
1–2% per year among sexually active women, 85% of these are spontaneous infections in sexually active females and the remaining 15% are due to procedures that favor ascending infection (e.g. D and C, IUCD insertion, HSG, etc). Two thirds of cases are seen in young women less than 25 years of age.

Risk Factors

- Multiple sexual partners
- Menstruating teenagers
- IUCD users
- Previous history of acute PID
- High prevalence of STDs
- Surgical procedures like D/C, HSG.

Protective Factors

- Contraception
  - Barrier methods like condom and spermicidals containing nonoxynol-9 (bactericidal and viricidal)
  - OC Pills - produce thick mucus plug preventing sperm ascent and bacterial penetration, also decrease in duration of blood flow, create shorter interval of bacterial colonization of the upper tract
    - Women with monogamous partner who has had a vasectomy

- Pregnancy
- Menopause
- Azoospermic husband.

MICROBIOLOGY

Polymicrobial Ascending Infection

Primary organisms-sexually transmitted:
- N. gonorrhoeae 30%,
- Chlamydia trachomatis 30%,
- Mycoplasma hominis 10%

Secondary organisms:
- Aerobic: Nonhemolytic streptococcus, E coli, group B streptococcus and staphylococcus
- Anaerobic: Bacteroides fragilis and bivius, peptostreptococcus and peptococcus.

Mode of Affection

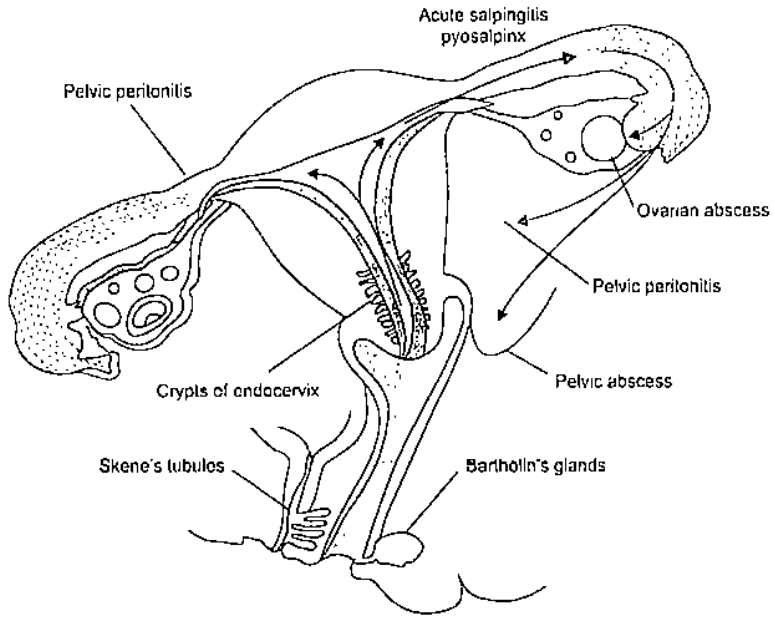


Fig. 2.3: Mode of spread of gonococcal infection

- Gonococcus ascends up to the tubes through mucosal contiguity and continuity, facilitated by vectors such as sperms and trichomonads
- Reflux of menstrual blood with gonococci into the fallopian tubes
- Mycoplasma hominis spreads across the parametrium to affect the tube
- Secondary organisms affect the tube through lymphatics
- Direct infection from the gut.

Pathology

- Fallopian tubes are involved bilaterally
- The process initiated in endosalpinx
- Damage of cells and cilia and also invades in all layers of tubes
- Exudate pours in lumen, both the openings of tube are closed due to congestion and adhesions
- Exudate may be watery producing hydrosalpinx or purulent forming pyosalpinx
- Occasionally the exudate pours into abdomen → pelvic peritonitis/abscess or affects ovary → ovarian abscess.
- A tubo-ovarian mass/abscess is thus formed.

Clinical Features of Acute PID

Symptoms

Wide range of nonspecific symptoms
- Symptoms usually appear at and immediately following menses
- Bilateral lower abdominal and pelvic pain, rapid and acute in gonococcal infection (3 days) than in chlamydial infection (5–7 days)
- Fever
- Irregular excessive vaginal bleeding (a/w endometritis)
- Purulent/copious vaginal discharge (due to associated lower tract infection)
- Nausea, vomiting
- Dyspareunia
- Pain in right hypochondrium due to perihepatitis (Fitz-Hugh-Curtis syndrome). 5–10% cases of acute salpingitis liver is affected by transperitoneal/vascular spread.

Signs

- Rise of temperature > 38°C
- Lower abdominal tenderness, liver may be enlarged and tender
- Purulent vaginal and cervical discharge, congested cervix
- Congested external urethral meatus or opening of Bartholin's duct, through which pus may be seen
- Tenderness on movement of the cervix (CMT+)
- Bilateral tenderness on Fornix palpation
- Adnexal mass felt through fornices.

Investigations

- Gram staining and culture (aerobic and anaerobic) of discharges.

Discharges collected from:
1. Urethra, Bartholins glands
2. Cervical canal
3. Fallopian tube (laparoscopy), cul de sac.

- *Blood:* Leukocytosis >10,000/mm^3 and CRP raised, ESR raised >15 mm/hour
- Serological test for syphilis in both partners

- *Laparoscopy:* Most reliable but reserved only in nonresponding cases or in cases where differential diagnosis is acute appendicitis or ruptured ectopic or torsion/hemorrhage/rupture of ovarian cyst
  - The tubes would appear edematous and congested
  - Hydrosalpinx or pyosalpinx or TO mass may be seen
  - Exudates can be collected from fimbrial ends and POD for studies
  - **VIOLIN STRING adhesions** in pelvis and around liver suggest chlamydial infections
- Culdocentesis with purulent fluid having white cell count >30,000/mL
- *Sonography:* Dilated tubes, fluid in POD and to mass are sugesstive of PID
- *Male partner:* Smears and cultures from urethral secretions.

Stages of PID (Galnesville)

Stage 1: Acute salpingitis without peritonitis

Stage 2: Acute salpingitis with peritonitis

Stage 3: Acute salpingitis with tubal occlusion or tubo-ovarian complex

Stage 4: Ruptured tubo-ovarian abscess

Stage 5: Tubercular salpingitis.

Differential Diagnosis

1. Acute appendicitis
2. Ruptured ectopic
3. Torsion/hemorrhage/rupture of ovarian cyst
4. Endometriosis
5. Diverticulitis
6. UTI.

Complications

Immediate

- Pelvic peritonitis, pelvic abscess
- Septicemia, septic shock.

Late

- Chronic PID, pelvic adhesions and formation of tubo-ovarian mass
- Chronic pelvic pain
- Infertility (12% with one episode, 25% with 2 episodes and 50% with 3 episodes)
- Ectopic pregnancy (6–10 fold increase risk)
- Fitz-Hugh-Curtis syndrome
- Dyspareunia.

Treatment

- Adequate rest, analgesics and anti-inflammatory drugs to be prescribed
- Sexual partner to also be treated appropriately
- Antibiotics started even before the reports are available.

CDC GUIDELINES FOR TREATMENTS OF PELVIC INFLAMMATORY DISEASES

Outpatient Treatment

Regimen A

- Ofloxacin, 400 mg orally two times daily for 14 days or
- Levofloxacin, 500 mg orally once daily for 14 days with or without metronidazole, 500 mg orally two times daily for 14 days.

Regimen B

- Cefoxitin, 2 g intramuscularly, plus probenecid, 1 g orally concurrently, or
- Ceftriaxone, 250 mg intramuscularly, or
- Equivalent cephalosporin
  Plus:
  - Doxycycline, 100 mg orally two times daily for 14 days
  - With or without metronidazole, 500 mg orally twice a day for 14 days.

Inpatient Treatment

Regimen A

- Cefoxitin, 2 g intravenously every 6 hours or
- Cefotetan, 2 g intravenously every 12 hours
- Plus: Doxycycline, 100 mg orally or intravenously every 12 hours

Regimen B

- Clindamycin, 900 mg intravenously every 8 hours
  Plus: Gentamicin, loading dose intravenously or intramuscularly
  (2 mg/kg of body weight) followed by a maintenance dose (1.5 mg/kg) every 8 hours

Indications of Inpatient Antibiotic Therapy

- Suspected pelvic abscess
- Severe illness, temperature >38°C
- Uncertain diagnosis—where surgical emergencies, for example, ectopic pregnancy cannot be excluded
- Unresponsive to outpatient therapy for 48 hours
- Intolerance to oral antibiotics
- Coexisting pregnancy
- Patient is known to have HIV infection.

Indications for Surgery

- Generalized peritonitis
- Pelvic abscess (which does not respond to drainage and antibiotics)
- TO abscess (which does not respond to antibiotics).

To Prevent Reinfections

- Patient education (avoid multiple partners, use of condoms)
- Partner treatment.

The only unequivocal proof of successful treatment after salpingitis is an intrauterine pregnancy.

Q. Bacterial vaginosis/vaginitis (BV).

BV is a common vaginal infection. **The term vaginosis is preferred over vaginitis as there is no vaginal inflammation.**

Etiology

BV is caused by an imbalance and decrease in the naturally occurring bacteria of the vagina.

In BV the number of the normal lactobacilli (Doderlein's bacilli) decreases with simultaneous increase in concentration of other types of bacteria, especially anaerobic bacteria.

The causative organisms which act synergistically are:
- ***Gardnerella vaginalis***
- *Ureaplasma urealyticum*
- Mycoplasma hominis
- Peptococcus and mobiluncus.

Clinical Features

- Moderate, malodorous, grayish white homogenous discharge, adherent to vaginal wall
- No evidence of vaginal inflammation.

Diagnosis

- **AMSEL'S criteria** are used.
- **Any three out of four** should be present.
  1. Grayish white discharge
  2. Vaginal pH > 4.7 (alkaline)
  3. **Drop of discharge mixed with 10% KOH = Fishy odor** (due to release of amines, acridine, and putredine) = **Whiff test**
  4. Smear prepared with normal saline: **Clue cells** (vaginal epithelial cells covered with *coccobacilli and the cells appear as stippled or granular*). Clue cells are diagnostic of BV.

Complications of BV in Pregnancy

- Preterm labor
- PROM
- Choriomanionitis.

Treatment

- **Metronidazole** 400 mg thrice a day for 5-7 days is the drug of choice
- Locally clindamycin and metronidazole gel can be used

- **Probiotics** to be given to increase the number of lactobacilli in the vagina
- **Lactic acid wash** to maintain the pH of vagina to prevent recurrence.

Q. Trichomoniasis

Vaginal trichomoniasis is the most common and important cause of vaginitis in child bearing age period.

Causative Organism

Actively motile parasite Trichomonas vaginalis, a pear shaped parasite, 20 ×10 microns.

Mode of Transmission

- Predominantly by sexual contact
- Also possible by toilet articles from one woman to another or rarely through examing gloves
- Incubation period 3-28 days.

Pathology

- The favourable vaginal pH for trichomonads to thrive is **5.5-6.5**
- In 25% women the parasite is present in vagina in asymptomatic state
- During and after menses, after sexual stimulation and following illness, the pH is raised to 5.5-6.5
- In 75% cases the organism can be isolated from urethra, skene's tubercles or Bartholins glands.

Clinical Features

- Profuse offensive vaginal discharge (often dating from last menstruation)
- Itching and irritation
- Dysuria and frequency.

On Examination

- **Greenish-yellow frothy** offensive discharge
- Vulval and vaginal inflammation
- **Strawberry appearance** of cervix and vagina (multiple punctate hemorrhagic spots on cervix and vagina).

Diagnosis

- Flagellate motile organism **(Hanging drop preparation)**
- The confirmation is by culture of discharge in **Kupferburg media or Feinberg-Whittington media.**

Treatment

- Both partners need to be treated
- **Metronidazole** 400 mg thrice a day for 5-7 days is the drug of choice for both husband and wife. A second course may be required

- To use barrier contraception until wife is cured
- *To prevent recurrence:* Both partners, metronidazole for 7 days, following menstruation for 3 consecutive cycles
- Locally clindamycin and clotrimazole pessaries and metronidazole gel can be used
- Probiotics to be given to increase the number of lactobacilli in the vagina
- **Lactic acid wash** to maintain the pH of vagina to prevent recurrence.

Q. Monilliasis/candida vaginitis.

It is a very common vaginal infection.

It is caused by *Candida albicans, gram positive yeast like fungus.*

Etiopathology

The organism thrives on carbohydrates and likes an acid medium (pH 4.0–5.5). Hence, candidal vaginitis is associated with a pH of < 4.5.

Risk Factors for Candidiasis Include

- Oral contraceptive/steroids use.
- Young age at first intercourse
- Diabetes (increased glycogen and glucosuria)
- HIV or other immunocompromised states
- Broad spectrum antibiotic use (destroy the good lactobacilli)
- Pregnancy (increased glycogen and renal glycosuria).

During and following menses the vaginal pH is elevated and there is relief of symptoms.

Clinical Features

- Vaginal discharge
- Intense pruritus, out of proportion to discharge
- Dyspareunia due to local soreness.

On Examination

- Vaginal erythema with adherent thick, **curdy white or cottage-cheese-like vaginal discharge**
- Cervix usually appears normal
- Vulva may be red and swollen
- Removal of white flakes reveals multiple oozing spots.

Diagnosis

- Direct smear of the discharge = pseudohyphae and spores
- Culture media for *Candida* = Sabouraud's agar or Nickerson's media
- In cases of repeated attacks diabetes mellitus to be ruled out. FBS and PLBS to be done.

Treatment

- Both partners need to be treated
- To use barrier contraception until wife is cured
- Nystatin or clotrimazole pessaries for local use. One pessary to be inserted in vagina hig up for 7–14 days
- Oral: Fluconazole or itraconazole is also highly effective
- Husband should also be treated with clotrimazole or nystatin ointments and or fluconazole if needed.

Q. Leukorrhea.

It is strictly defined as excessive normal vaginal discharge. It is inappropriate to include vaginitis as a cause of leukorrhea.

CRITERIA

- Excess secretion is evident from persistent vulval moistness or brownish yellow stains o drying or need to use pads
- Nonpurulent
- Nonoffensive
- Nonirritant
- Never causes pruritus.

Etiology

The excess secretion is due to:
- Physiologic excess
- Cervical cause
- Vaginal cause.

Physiologic Excess

A/w increase in estrogen
- During puberty: Increased endogenous estrogen may also lead to cervical erosion
- Menstrual cycle
  - Around ovulation: Peak in estrogen
  - Premenstrual pelvic congestion: Increase mucus secretion from cervix and endometria glands
- Pregnancy: Increase in estrogen and vascularity. Increased vaginal transudate and cervica gland secretions
- During sexual excitement: Secretions from Bartholin's glands.

Cervical Cause

Noninfective lesions
- Cervical erosions
- Chronic cervicitis
- Mucous polyps
- Ectropion.

Vaginal Cause

- Increased pelvic congestion → vaginal transudation
- Prolapse
- Chronic PID
- Birth control pills
- ILL health: Excess exfoliation of superficial cells.

Diagnosis

- The discharge is nonpurulent, nonoffensive and, nonirritant
- General examination may reveal ill health (leukorrhea does not lead to ill health, reverse is true)
- Vulval examination reveals white or creamy discharge and no evidence of pruritus
- Bimanual and speculum examination: Normal or conditions mentioned above causing cervical and vaginal leukorrhea
- To rule out infections the discharge is subjected to microscopic examination for pus cells
- If pus cells detected further investigations like hanging drop preparation, gram stain, culture, etc. to be done.

Treatment

- Improve general health
- To have sympathetic attitude and anxiety state should be removed
- Cervical lesions may require electrocautery, cryosurgery or trachelorrhaphy
- Appropriate treatment for lesions producing vaginal leukorrhea
- Pill users may require to stop the pill temporary
- Local hygiene.

3
Menstrual Disorders

Q. Cryptomenorrhea.

Q. Imperforate hymen.

INTRODUCTION

Crypto=hidden

It is a condition in which there is periodic shedding of endometrium (menstruation) but the blood fails to come out of the genital tract due to outflow tract obstruction.

CAUSES

Congenital

- *Imperforate hymen:* It is due to failure of disintegration of the central cells of Mullerian eminence that project into urogenital sinus
- Transverse vaginal septum
- Atresia of vagina, cervix.

Acquired

Cervical stenosis (following surgeries like conization, amputation or cancer).

PATHOLOGY

The blood fails to come out of hymen → distends the vagina → hematocolpos → then uterus distends (hematometra) → hematosalpinx.

Clinical Features

- *In congenital variety:* The girl is around 14 to 16 years of age, presents with primary amenorrhea
- In acquired cases there would be secondary amenorrhea
- Periodic lower abdominal pain
- Urinary complaints like frequency, dysuria or retention of urine (hematocolpos leads to elongation of urethra).

Examination

- Abdominal examination reveals globular mass (distended uterus or full bladder) in supra-pubic region
- *Vulval examination:* Tense bulging hymen with bluish coloration
- Rectal examination reveals bulged vagina and uterine mass.

Management

USG makes the diagnosis of hematometra and hematocolpos.

- *Imperforate hymen:* **Cruciate incision on hymen**
- Antibiotics
- Head end elevated post surgery.
- Dilatation of cervix in cases of cervical stenosis
- Reconstructive surgery in cases of transverse septum of vagina, and vaginal and cervical atresia.

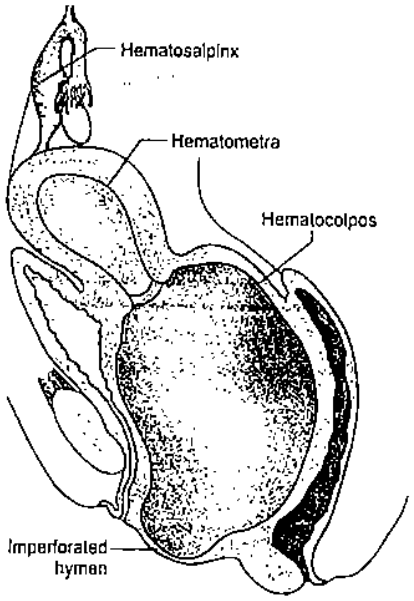


Fig. 3.1: Hematocolpos and hematometra due to imperforate hymen

Q. Define menorrhagia. Causes of menorrhagia.

Definition

Menorrhagia is defined as menstruation at regular and normal cycle intervals but with **excessive flow (> 80 ml)** and/or duration (> 7 days) and is one of the most common gynecologic complaints.

A normal menstrual cycle is 21 to 35 days in duration, with bleeding lasting an average of 5 days and flow measuring 25 to 80 mL (usually 30–50 ml).

In practice, measuring menstrual blood loss is difficult. Thus, the diagnosis is usually based upon the patient's history.

Causes

- *Organic:* Pelvic, systemic, endocrinal, hematological
- *Functional:* DUB (Dysfunctional uterine bleeding) due to disturbed HPO axis
- *Iatrogenic:* Use of anticoagulants, steroids, improper use of hormones.

Pelvic (Due to increased surface area or hyperplasia of the endometrium)

- Fibroids
- Polyps
- Adenomyosis, endometriosis
- IUCD in utero
- Early phase of tuberculous endometritis, pelvic inflammatory disease
- Granulosa cell tumor of ovary
- *Pregnancy complications:* Miscarriage or ectopic pregnancy should always be ruled out in reproductive age group
- *Cancer:* Rarely, uterine cancer, ovarian cancer and cervical cancer can cause excessive menstrual bleeding.

Systemic

- Liver failure
- Renal failure.

Endocrinal

- Hypothyroidism
- Hyperthyroidism.

Hematological

Inherited bleeding disorders such as Von Willebrand's disease, ITP.

Important Causes of Menorrhagia in Different Age Groups

Puberty	Reproductive age	Perimenopausal
HPO axis immaturity	Pregnancy-related complication (incomplete abortion)	Endometrial hyperplasia, carcinomas
Dysfunctional uterine bleeding (DUB)	Fibroids, polyps, adenomyosis, endometriosis	DUB, infrequent ovulation
Coagulation defects (ITP, von Willebrand disease)	DUB	Endocrine problems
Endocrine abnormalities	Endocrine abnormalities	Fibroids, polyps, adenomyosis, endometriosis

Q. Pathophysiology of DUB.

Q. Investigations in case of DUB.

Q. Management of DUB.

Q. Medical management of DUB.

DEFINITION

Dysfunctional uterine bleeding (DUB) is abnormal uterine bleeding that occurs in the **absence of any clinically detectable organic, systemic and iatrogenic cause (pelvic pathology like fibroids, etc. and pregnancy excluded)**. It is considered a diagnosis of exclusion.

This condition usually is associated with anovulatory menstrual cycles but also can present in patients with oligoovulation (dysfunction of HPO axis: Endocrine origin).

Incidence

Dysfunctional uterine bleeding is a common diagnosis, making up 5 to 10% of cases in the outpatient clinic setting. It is more prevalent in extremes of reproductive period: puberty and perimenopause.

About 20% of affected individuals are in the adolescent age group, and 50% of affected individuals are aged 40 to 50 years.

PATHOPHYSIOLOGY

Types

- Ovulatory (10%)
- Anovulatory (90%).

Ovulatory

Polymenorrhea: Rare: condition occurs following childbirth, abortion or during adolescence and premenopausal period. Short follicular phase.

Oligomenorrhea: Very rare during adolescence and premenopausal period. Prolonged follicular phase.

Menorrhagia: Uncommon.
Two varieties.

*Irregular shedding of endometrium (**Halban's disease**):* Persistent corpus luteum → persistent progesterone.

The desquamation is continued for a variable period with simultaneous failure of regeneration.

Histology reveals mixture of secretory and proliferative endometrium done on day 5 or 6 of cycle.

Irregular ripening of endometrium: Inadequate function of corpus luteum/luteal phase defect → progesterone deficiency. **Premenstrual spotting** occurs prior to start of proper flow.

Serum progesterone levels are < 5 ng/ml and histology shows patchy area of secretory changes amidst proliferative endometrium.

Anovular Menorrhagia

DUB (menorrhagia) due to anovulation in premenopausal women is called as **Metropathia hemorrhagica or Schroeder's disease.**

Anovulatory cycles are associated with a variety of bleeding manifestations. Estrogen withdrawal bleeding and estrogen breakthrough bleeding are the most common spontaneous patterns encountered in clinical practice.

Anovulatory cycles have no corpus luteal formation. Progesterone is not produced. The endometrium continues to proliferate under the influence of unopposed estrogen leading to a phase of amenorrhea for about 6 to 8 weeks.

Eventually, the estrogen levels falls or the endometrium outgrows its blood-supply and this out-of-phase endometrium is shed in an irregular manner that would be prolonged and heavy. Also there is no vasoconstrictor effect of PGF2α resulting in heavy bleeding.

There is myohyperplasia with symmetrical enlargement of uterus to size of about 8 to 10 weeks.

The endometrium looks thick, congested, and polypoidal.

Microscopy

- Hyperplasia of all endometrial components
- Cystic glandular hypertrophy
- **Swiss cheese pattern**
- Empty glands lined by columnar epithelium
- Absence of secretory changes.

MANAGEMENT

History

- Most patients are adolescents or are older than 40 years
- The history of excessive bleeding is confirmed by asking about the number of pads soaked/day and passage of clots and duration of bleeding. Keeping a menstrual calendar is helpful
- Typically, the usual premenstrual symptoms that accompany ovulatory cycles will absent
- Rule out the presence of signs or symptoms indicative of bleeding disorders. History of easy bruising, bleeding gums, epistaxis, and excessive bleeding episodes during childbirth, surgery, or dental procedures maybe useful
- Rule out iatrogenic causes of bleeding like IUCD, steroidal hormone intake
- Patients who report irregular menses since menarche may have polycystic ovarian syndrome
- Patients with adrenal enzyme defects, hyperprolactinemia, thyroid disease, or other metabolic disorders also might present with anovulatory bleeding.

Examination

- The physical examination can elicit several anatomic and organic causes of abnormal uterine bleeding

- Suspect dysfunctional uterine bleeding (DUB) when a patient presents with unpredictable or episodic heavy or light bleeding despite a normal pelvic examination
- A complete physical examination should begin with assessment of hemodynamic stability and proceed with evaluation of the following:
  - Obesity (BMI)
  - Signs of androgen excess (hirsutism, acne)
  - Thyroid enlargement or manifestations of hyperthyroidism or hypothyroidism
  - Galactorrhea (may suggest hyperprolactinemia)
  - Ecchymosis, purpura (signs of bleeding disorder)
  - Signs of anemia or chronic blood loss
  - A careful gynecologic examination (PS and PV), including Papanicolaou test (Pap smear) is warranted except in cases of puberty menorrhagia (virgins). Rule out the presence of uterine fibroids or polyps, any adnexal mass, forniceal and cervical movement tenderness.

Investigations

- Laboratory studies for patients with dysfunctional uterine bleeding (DUB) include:
  - Exclude the diagnosis of pregnancy first by doing UPT or Beta HCG. The most common cause of abnormal uterine bleeding during the reproductive years is abnormal pregnancy. Rule out threatened abortion, incomplete abortion, and ectopic pregnancy
  - CBC with platelets
  - TSH and prolactin
  - Pap smear
  - Liver functions
  - Coagulation studies (BT, CT, PT, aPTT) in cases of **puberty menorrhagia.**
- **Endometrial sampling:** (the aim is to rule out hyperplasia and endometrial cancer):
  - Perimenopausal age group
    - **Histopathological diagnosis (D&C/endometrial biopsy/hysteroscopy and biopsy) should always be made first** to rule out endometrial hyperplasia/cancer before proceeding with any treatment
    - **Hysteroscopy and biopsy are preferred** to blind D&C (Dilatation & Curettage).
  - Puberty age group
    - D&C is used as the last resort **only when all the medical methods fail** to control bleeding
    - It is both diagnostic and therapeutic.
  - Reproductive age group
    - In cases of DUB, three cycles of hormonal manipulation is given (OC pills or cyclical progesterone)
    - If the menorrhagia persists then histopathological diagnosis (D&C/endometrial biopsy/hysteroscopy and biopsy) should be made.

USG

- Ultrasound can be used to examine the status of the endometrium. Endometrial hyperplasia, endometrial carcinoma, endometrial polyps, and uterine fibroids can be identified easily by this technology.

- Saline-infusion sonohysterography is also very useful in evaluating for intracavitar (submucosal) fibroids and endometrial polyps
- *Hysteroscopy:* It is preferred over D/C. It helps in better evaluation of the lesion and als helps in taking the biopsy. **Hysteroscopy is also therapeutic in cases of polyps an submucous fibroids**
- *Laparoscopy:* Needed if adnexal pathology/endometriosis/PID is suspected.

Treatment

General and Specific (medical and surgical).

General

- To correct anemia with hematinics, diet, and blood transfusion in severe cases.
- *Medical:* **It is considered first line treatment for DUB.**

Various drugs used are:

Hormones	Nonsteroidal anti-inflammatory drugs (NSAIDs)	Antifibrinolytics
COC	Mefenamic acid	Tranexamic acid
Progestins (Norethisterone, MPA, Dydrogesterone)		EACA
CEE (Conjugated equine estrogen)		
Androgens, Danazol		
LNG-IUD (MIRENA)		
GnRH analogs		
Desmopressin		

Progesterones

- MPA 10 mg or norethisterone 5 mg three to four times a day for four to seven days can b used to control the acute bleeding
- Cyclical progesterone can be used from day 5 to day 25 or from day 20 to 25 of the cycle
- Progestins inhibit estrogen receptor replenishment and activate 17-hydroxysteroi dehydrogenase in endometrial cells, converting estradiol to the less active estrone
- Synthetic progestins have an antimitotic effect, allowing the endometrium to becom atrophic if administered continuously. These drugs are very effective in cases of endometri hyperplasia
- **Treatment with a progestin for 10 to 12 days/month will allow for controlle predictable menses and will protect the patient against the development of endometri hyperplasia**
- Medroxyprogesterone acetate is the drug of choice for patients with anovulatory DUB
- DMPA works by causing progesterone induced amenorrhea.

COC

- Contraceptive pills containing estrogen and progestin have been advocated in patien with DUB

- It is preferred in women who desire contraception
- Therapy also used to treat acute hemorrhagic uterine bleeding. In long-term management of DUB, combination oral contraceptives are very effective
- Low dose and very low dose OC Pills are preferred now.

Estrogen

- The role of isolated estrogen is limited
- Estrogen alone in high doses is indicated in certain clinical situations
- Prolonged uterine bleeding suggests the epithelial lining of the cavity has become denuded over time. In this setting, a progestin is unlikely to control bleeding. Estrogen alone will induce return to normal endometrial growth rapidly
- It is effective in controlling acute, profuse bleeding. Exerts a vasospastic action on capillary bleeding by affecting the level of fibrinogen, factor IV, and factor X in blood, as well as platelet aggregation and capillary permeability
- Estrogen also induces formation of progesterone receptors, making subsequent treatment with progestins more effective
- IV estrogen (not available in India) or CEE tablets can be used.

MIRENA/LNG-IUD

- It release 20 mcg LNG/day and causes a state of **progesterone induced amenorrhea**, induces endometrial atrophy. Up to 97% reduction in blood loss is achieved
- It can be used for up to 5 years and is hysterectomy can be avoided.

Danazol

Very rarely used because of androgenic side effects.

Gestrinone

2.5 mg orally twice a week for 3 months is also effective.

GnRH Analogs

- Work by reducing concentration of GnRH receptors in the pituitary via receptor down regulation and induction of postreceptor effects, which **suppress gonadotropin release**
- **This form of medical castration** is very effective in inducing amenorrhea
- Because prolonged therapy with this form of medical castration is associated with osteoporosis and other postmenopausal side effects, its use is often limited in duration
- Because of the expense of these drugs, they usually are not used as a first line approach but can be used to achieve short-term relief from a bleeding problem, particularly in patients with renal failure or blood dyscrasia.

Desmopressin Acetate (DDAVP)

It has been used to treat abnormal uterine bleeding in patients with coagulation defects. Transiently elevates factor VIII and von Willebrand factor.

Surgical

Most cases of DUB can be treated medically. Surgical measures are reserved for situations when medical therapy has failed or is contraindicated.

1. D/C
2. Endometrial ablation
3. Hysterectomy

- D&C is an appropriate diagnostic step in a patient who fails to respond to hormonal management
- The addition of hysteroscopy will aid in the treatment of endometrial polyps or the performance of directed uterine biopsies
- Therapeutic D&C alone for DUB has not been shown to be very efficacious
- Laparoscopic, abdominal or vaginal hysterectomy might be necessary in patients who have failed or declined medical management and who experience a disruption in their quality of life from persistent, unscheduled bleeding
- **Also in cases of hyperplasia with atypia hysterectomy is preferred if the family is complete**
- **Ovaries should be preserved during hysterectomy**
- Endometrial ablation is an alternative for those who wish to avoid hysterectomy or who are not candidates for major surgery.

UTERUS CONSERVING SURGERIES FOR DUB

(Endometrial Ablation/Resection)
The various surgeries are:

1. Transcervical resection of endometrium (TCRE) in which the basal endometrium is removed using diathermy loop
2. Roller ball endometrial ablation
3. Laser (Nd YAG) endometrial ablation
4. MEA (microwave of 9.2 GHz used for endometrial ablation)
5. Uterine thermal balloon in which hot saline (85 degree C for 10-15 minutes) is circulated within the balloon after it is placed inside the uterus
6. Hydrothermablator in which heated saline in circulated within uterine cavity

In a D/C, only superficial endometrium is removed which grows back, but in above minimally invasive surgeries **the basal endometrium is destroyed** so that it does not regenerate back.

Prerequisites

- Patient's family should be complete
- *Histopathology*: There should be no evidence of malignancy.

Advantages

- Day care procedure
- Major surgery such as hysterectomy is avoided.

Results

- Forty percent patients will become amenorrheic
- Forty percent will have hypomenorrhea
- Only 20% will require hysterectomy.

Q. Dysmenorrhea.

DEFINITION

Dysmenorrhea is defined as painful menstruation so as to incapacitate day to day activities.

Types

- *Primary (spasmodic):* In absence of any pelvic pathology
- *Secondary (congestive):* In presence of pelvic pathology.

Primary dysmenorrhea is mostly confined to adolescent, more common among affluent society, generally with a positive family history.

The pain always occurs in ovulatory cycles and is usually cured after pregnancy and vaginal delivery.

The various theories for primary dysmenorrhea are:
- Uterine myometrial hyperactivity, junctional zone (JZ: subendometrial layer of myometrium) hyperplasia, dysperistalsis and hyperactivity of JZ
- Overactivity of sympathetic nerves → hypertonicity of circular fibers of isthmus
- PGFα → is more in ovulatory cycles which causes ischemia of myometrium
- Vasopressin, endothelins, leukotrienes, and platelet activating factor are all increased which cause uterine hyperactivity, hyperperistalsis, dysrhythmic contractions, ischemia, and pain
- Psychosomatic factors, anxiety lower pain threshold.

Clinical Features

- The pain begins few hours or just before the menses
- The pain last for few hours to 24 hours, rarely beyond 48 hours
- Spasmodic in nature, lower abdomen and may radiate to back and medial aspect of thigh
- May be a/w nausea, vomiting, diarrhea, tachycardia, pallor, cold sweats
- Fainting, syncope in severe cases
- Clinical examination does not reveal any abnormalities
- USG helps in ruling out any pelvic pathology.

Treatment

Assurance, counseling, encourage normal activities.

Drugs

- NSAIDs: Given for 1 to 3 days, for 3 to 6 cycles
- Mefenamic acid 250 to 500 mg tds
- Ibuprofen (400 mg) tds or naproxen (250 mg) qds
- Indomethacin (25 mg) tds
- Newer selective COX-2 inhibitors may also be used
- Glycerin trinitrate transdermal patches are also used
- OC pills (suppresses ovulation and hence used) 3 to 6 cycles

- The patients wanting contraception, those with associated menorrhagia and patients unresponsive to NSAIDs or in whom NSAIDs are contraindicated are good candidates for OC pills
- Dydrogesterone (from day 5–25) has also been tried
- TENS (transcutaneous electrical nerve stimulation) has also been used to relieve the pain

Surgery (very rarely required).

Laparoscopy maybe very rarely needed to rule pelvic causes especially endometriosis.
- Laparoscopic Uterine Nerve Ablation (LUNA) has not been found to be very beneficial
- Laparoscopic Presacral Neurectomy (LPSN) to cut down sensory pathways (T11-T12) from uterus
- Cervical dilatation very rarely done. May lead to incompetence in future.

Secondary Dysmenorrhea

Etiology

- Endometriosis
- Pelvic inflammatory disease (PID)
- Ovarian cysts and tumors
- Cervical stenosis
- Adenomyosis
- Fibroids
- Uterine polyps
- Intrauterine adhesions
- Congenital malformations (e.g. bicornuate uterus)
- Intrauterine contraceptive device (IUCD) in utero
- Transverse vaginal septum
- Pelvic congestion syndrome
- Allen-Masters syndrome.

Clinical Features

The patient is usually in her thirties.
- The pain usually appears **3 to 5 days prior to the period and relives with start of bleeding**
- The pain is dull, situated in back and front. The onset and duration of pain depends on the pathology producing the pain
- There is no systemic discomfort unlike primary dysmenorrhea
- There maybe other symptoms related to the underlying pelvic pathology
- Clinical examination would reveal the pathology
- The underlying pathology maybe detected by USG, laparoscopy or hysteroscopy.

Treatment

Treatment is aimed at underlying cause (e.g. myomectomy, polypectomy, adhesiolysis, chocolate cystectomy, hysterectomy, etc.) and depends on the patients age and parity.

Q. Define primary amenorrhea. Differentiate between MRKH syndrome and CAIS.

Definition

Primary Amenorrhea

- In absence of secondary sexual characters, no menses till the age of 14 years or
- In presence of secondary sexual characters, no menses till the age of 16 years.

Causes

- *MC cause of primary amenorrhea is ovarian dysgenesis/Turner syndrome.*
- Mullerian agenesis (Mayer-Rokitansky-Küstner-Hauser or MRKH syndrome) is the second MC cause and androgen insensitivity syndrome or testicular feminizing syndrome (AIS/TFS) is the third MC of primary amenorrhea.
- *Each and every case of primary amenorrhea karyotyping should be done.*
- In the *entire gynecology, these are only two conditions in which there is primary amenorrhea and absent uterus:*

	Mullerian agenesis (RMKH)	Complete androgen insensitivity syndrome (CAIS)
Karyotype	XX	XY
Gonads	Ovaries	Testes (inguinal)
Axillary/pubic hair	Present	Absent/sparse
Associated anomalies	Renal and skeletal/vertebral defects and deafness may be present	Absent
Reproduction	Possible with surrogacy as ovaries function normally (they can have their own biological child)	Not possible but gonadectomy, vaginoplasty, and ERT are required

Breasts are well-developed in both the above cases. In CAIS breast development is due to peripheral aromatization of testosterone to estrogen.

Key Points to Remember About CAIS

- They do not have ambiguous genitalia at birth. The external genitalia look like females
- Testes secrete both testosterone and anti-Mullerian hormone/Mullerian inhibiting factor (AMH/MIF), but testosterone functions are absent (as receptors are insensitive)
- Since the testes have a risk of developing gonadoblastoma/seminoma, orchidectomy should be done
- Vaginoplasty should be done for sexual activity and estrogen replacement therapy (ERT) given for bone protection and maintenance of secondary sexual characters
- Patients of CAIS should be continued to be reared as females.

4

Fibroids

Q. Etiology and clinical features of fibroids.

Q. Signs and symptoms and management of fibroids.

INTRODUCTION

Fibroids are benign smooth muscle tumors arising from the myometrium.

They are the **MC benign tumors of uterus, and they are also the MC pelvic tumors in females.**

Incidence

Incidence increases with the age of patient. It is estimated that at the age of 30 years around 20% of women will have fibroids. Prevalence is highest between 35–45 years of age. By the age of 45 years it is estimated that every third or fourth women would have a fibroid.

Etiology

- It is **predominantly an estrogen-dependent tumor:**
  - A/W early menarche, late menopause
  - Associated anovulation and PCOS
  - Grows in size during pregnancy, and following menopause there is cessation of growth.
- Nulliparity (a uterus which does not bear a baby consoles itself by having a fibroid). Multiparity is protective.
- Deletions in chromosome 7 and t (12, 14) are associated with fibroids.
- More common in colored races/black women.
- *Infertility:* Fibroids can cause infertility and infertile women are more prone to develop fibroids.
- *Obesity:* EGF, IGF1 and TGF stimulate the growth of fibroid either directly or via estrogen. **Smoking is protective for fibroids.**

CLINICAL FEATURES

- The patients are usually nulliparous or have primary infertility or having a long duration o secondary infertility.
- Prevalence is highest between 35–45 years of age.

Symptoms

- **Majority (75%) of the fibroids remain asymptomatic**
- They are discovered accidently during USG/laparoscopy/laparotomy or during pelvic examination
- **Symptoms depend on the location and size of the fibroid.**

Menstrual Abnormalities

They are mainly a/w submucus and intramural fibroids.

Menorrhagia

Menorrhagia is the classic and MC symptom.

It is due to:
- Increased surface area of endometrium
- Endometrial hyperplasia due to hyperestrogenism and anovulation
- Interference with normal uterine contractility.
- Congestion of subjacent endometrial venous plexus and pelvic congestion
- Relative deficiency of TXA2.

Metrorrhagia

Metrorrhagia may be due to ulceration of submucuous fibroid.

Dysmenorrhea

Congestive/secondary dysmenorrhea is due to pelvic congestion or associated endometriosis.

Infertility

It is due to:
- Submucus or intramural fibroid distorts the cavity and thereby makes the uterus unsuitable for implantation
- Congestion of endometrial plexus and ulceration of endometrium over submucous fibroid may also prevent implantation
- Also, elongation of the cavity and prevention of rhythmic uterine contraction may impair sperm transport
- Cornual fibroid may block the fallopian tube
- Menorrhagia and dyspareunia may also contribute to infertility.

Lump in Abdomen

- Subserous fibroids or large intramural fibroids are most likely to cause this
- The patient may feel lump/heaviness in lower abdomen without any other symptoms.

Pressure Symptoms

- It can press the bladder causing frequency, dysuria or retention. It can press the rectum causing constipation
- Broad ligament fibroid can press the ureter causing **hydroureter and hydronephrosis.**

Pregnancy Complications

Effects of fibroids on pregnancy:
- Recurrent abortions
- Impacted posterior fibroid can lead to retroverted gravid uterus and urinary retention
- Malpresentations
- Preterm labor
- IUGR
- Prolonged labor/obstructed labor
- Cervical dystocia (due to cervical or broad ligament fibroid)
- Abruption
- Atonic PPH
- Increased risk of obstetric hysterectomy.

Effects of Pregnancy on Fibroids

- Red degeneration
- Increase in size (due to increase in vascularity, edema and hypertrophy and hyperplasia o fibromuscular tissue)
- Torsion of pedunculated subserosal fibroid
- Infection and polypoidal changes (more in puerperium).

Pain in Abdomen

Fibroids are usually painless. Pain is due to some complications (like degeneration, torsion or associated pathology like endometriosis.

Signs

- Pallor (due to menorrhagia)
- **Abdominal examination:** If the uterus size is less than 12 weeks, it is not palpable o abdominal examination.
- If uterus is enlarged to 12–14 weeks or more the following features are noted:

Palpation

- Feel is **firm to hard**, may be cystic in cases of cystic degeneration
- Well defined margin, lower pole cannot be reached suggesting of pelvic origin
- **Generally bosselated, nodular surface**, may be uniformly enlarged in case of single fibroi
- Mobility is restricted from above downwards but can be moved from side to side.
- Swelling is dull on percussion.

PV Examination

- Bimanual examination reveals irregularly enlarged uterus
- The mass/swelling is **not felt separately from the uterus**
- **The cervix moves with the movement of the mass felt per abdomen** and movement c the mass moves the cervix.

Investigations

- To confirm the diagnosis.
- **Preoperative evaluation:** Blood group, CBC, Blood sugars, ECG, Chest X-ray, etc.
- **USG is the imaging modality of choice** in the detection and evaluation of uterine fibroids
  - Uterus is enlarged and distorted.
  - Uterine fibroids most often appear as **concentric, solid, hypoechoic masses.** These solid masses absorb sound waves and therefore cause a variable amount of acoustic shadowing.
  - Fibroids may vary in their degree of echogenicity; they can be heterogeneous or hyperechoic, depending on the amount of fibrous tissue and/or calcification. Fibroids may have anechoic components resulting from necrosis.
  - The echogenic endometrial stripe may be displaced by a fibroid. Calcifications are hyperechoic, with sharp acoustic shadowing.
  - Also, hydroureter and hydronephrosis can be detected by USG.
  - Doppler ultrasound shows vascularity of the uterus and fibroids. Besides it can differentiate between the fibroid and localized adenomyosis. **The blood flow surrounds a fibroid, but diffuses through adenomyosis.**
  - USG has a sensitivity of 60%, a specificity of 99%, and an accuracy of 87%.
  - **3D USG** is also available nowadays and can locate the fibroids accurately.
- **Saline infusion sonography** is very helpful to **detect submucous fibroid or polyp.**
- **MRI** is more accurate for fibroid mapping but is expensive and not routinely used.
- **CT scanning** also has a limited role in the diagnosis of uterine fibroids. On CT scans, fibroids are usually indistinguishable from healthy myometrium unless they are calcified or necrotic. Calcifications may be more visible on CT scans than on conventional radiographs.
- **Hysteroscopy** not only recognizes a submucous fibroid, but also allows its excision under direct vision.
- D & C is required to rule out endometrial cancer in a woman complaining of menstrual disorders, irregular bleeding and postmenopausal bleeding. Histopathology of endometrium gives clue to its etiology and rules out endometrial cancer.
- **Laparoscopy:** Can be diagnostic as well as therapeutic.

Myomectomy or hysterectomy can be done through laparoscopic route. Associated PID and endometriosis can be detected. Also the tubal patency can be established in infertile patient.

DD

1. Pregnancy
2. Full bladder
3. Adenomyosis
4. Ovarian tumor
5. Myohyperplasia (in cases of DUB)
6. TO mass.

TREATMENT

- General and specific
- **All fibroids do not require treatment but all symptomatic fibroids require treatment.**

General

To correct anemia: Iron supplements and even blood transfusion may be needed in cases of severe anemia.

Specific

Medical and Surgical.

Medical Management

- To control menorrhagia and dysmenorrhea: NSAIDS, low dose oc pills, progesterone
- To decrease the size and vascularity of fibroids:
  Drugs which decrease the size of fibroids (never for permanent treatment, as the fibroid grows back to its usual size after the action of drug is over; they are **mainly used preoperatively**):
  - GnRH analogs (MC used)
  - Danazol
  - Progesterone (DMPA/Mirena/POP/low-dose OC pills)
  - Mifepristone (RU-486)
  - Gestrinone
  - Anastrozole (aromatase inhibitor)
  - Asoprisnil and Ulipristal acetate (selective progesterone receptor modulator—SPRM).

  GnRH analogs are used preoperatively:
  - Decrease the vascularity and blood loss during surgery
  - To induce amenorrhea to build up hemoglobin in cases of anemia
  - May facilitate laparoscopic or hysteroscopic surgery
  - May help to convert an abdominal surgery to a vaginal surgery.

Surgical Management

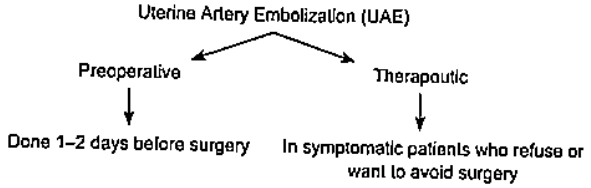
It is the main stay of treatment.
Myomectomy: Laparotomy, laparoscopy, hysteroscopy, vaginal
Hysterectomy: Laparotomy, laproscopic, vaginal.

- The type of surgery depends on the patient's age, parity, desire for pregnancy, symptoms and number of fibroids
- The route of surgery is decided by location of fibroid, size of the fibroid and the uterus and the surgeon expertise
- Fibroids causing infertility, or recurrent abortions or symptomatic fibroid in a young patient = myomectomy
- **For submucous fibroids hysteroscopic myomectomy is the gold standard**
- For subserous and intramural fibroids laparoscopic surgery is preferred today over laparotomy
- Old patients/family complete = hysterectomy preferred.

Indications of Surgery in Asymptomatic Fibroid

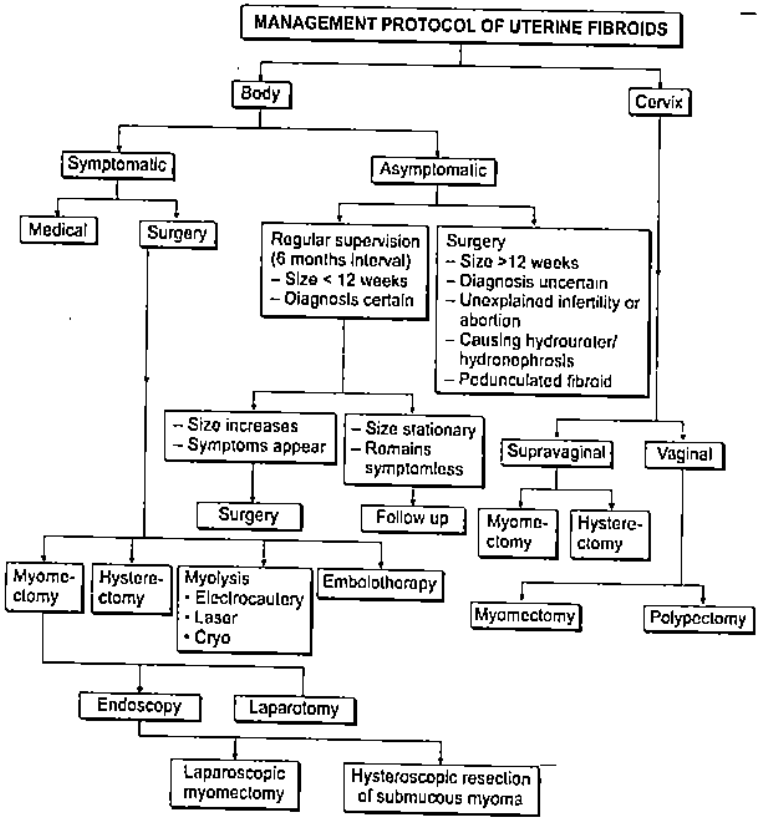
- Size >12 weeks of pregnancy
- Diagnosis not certain
- Fibroid grows during follow-up
- Subserosal pedunculated fibroid (because of risk of torsion)
- Situated in the lower part of the uterus and likely to complicate deliveries in future
- Fibroids compressing ureter and causing hydroureter/hydronephrosis
- Unexplained infertility with distortion of uterine cavity
- Unexplained recurrent abortions.

Flow Chart 4.1: Uterine artery embolization (UAE)



In this procedure, the femoral artery is cannulated, and artificial clot of polyvinyl alcohol is used to block the uterine artery and its branches supplying the fibroids. It decreases the blood loss during surgery. The same technique can also be used as a therapy for symptomatic patients who refuse or want to avoid surgery. After embolization there is 60-65% decrease in size of fibroids over a period of 6-9 months, and so the patient's symptoms may decrease or disappear. If the patient is still symptomatic after 1 year, then surgery should be considered.

Flow Chart 4.2: Management protocol of uterine fibroids



Q. Degenerations in fibroids.

Q. Red degeneration.

DEGENERATIONS/SECONDARY CHANGES IN FIBROIDS

Fibroids can Undergo Various Types of Degenerations

Hyaline degeneration: It is the MC type (65%).

- It can occur in fibroids of all sizes. It is more common in fibroids having more connectiv tissues. The central part of the fibroid (being the least vascular) is the common site. Th fibroid feels soft and elastic.
- On cut section there are irregular homogenous areas with **loss of** whorl-like appearance.
- Microscopy reveals hyaline changes in muscle and fibrous tissue.

Cystic degeneration: More common in interstitial fibroids and usually occurs following men opause. It is formed by liquefaction of areas within hyaline changes.

Fatty degeneration: Found after menopause. There is deposition of fat globules in the muscl cells.

Calcareous degeneration

- In calcareous degeneration, phosphates and carbonates of lime are deposited in th periphery along the course of the vessels. The best examples of calcareous myomas ar those in old patients with long-standing myomas. They have been found as **'womb-stones** in graveyards. Calcareous tumors are easily identified by radiography.

Red degeneration (also known as carneous degeneration)

- Occurs because fibroid overgrows its blood supply **(micronecrothrombosis)**
- Most commonly occurs in **second trimester of pregnancy** followed by in the puerperium
- Cut section: Raw beefy appearance, fishy odor
- Patient presents with acute abdomen, vomiting, fever, and leukocytosis
- D/D: Acute appendicitis, pyelonephritis, and abruption.

Management

Always conservative management (never surgery)

- Hospitalization
- Bed rest
- Analgesics
- IV fluids
- IV antibiotics (SOS).

Atrophy: As a result of diminished vascularity after menopause, there is a shrinkage in the size of the tumor, which becomes firmer and more fibrotic. A similar change occurs in myomas after pregnancy enlargement. Temporary shrinkage by 50% occurs following GnRH therapy, but regrows after stoppage of therapy.

Sarcomatous change: Sarcomatous change in a myoma is extremely rare, and the incidence is 0.1-0.5% of all myomas. Intramural and submucous tumors have a higher potential for sarcomatous change than subserosal tumor. It is rare for malignant change to develop in a woman under the age of 40. It is commonly seen in a postmenopausal woman

when the tumor is noticed to grow suddenly, causing pain and postmenopausal bleeding. To the naked eye, a sarcomatous myoma is yellowish grey in color and hemorrhagic. The consistency is soft and friable, and not firm like a simple myoma. Another important sign is the nonencapsulation of the tumor. Sarcoma is highly malignant and spreads via the blood stream.

Infection: Infection (Streptococal and Bacteriodes) is common in submucous and myomatous polyps if they project into the cervical canal or into the vagina.

- An infected polyp causes blood-stained purulent discharge. This generally happens following delivery or abortion.

5

Prolapse

Q. Supports of the uterus.

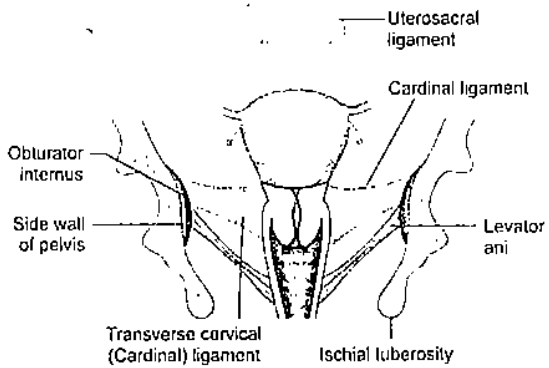


Fig. 5.1: Supports of the uterus

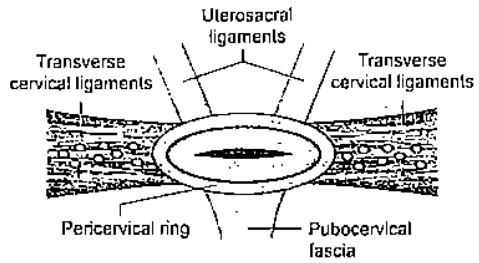


Fig. 5.2: Ligamentous supports of uterus

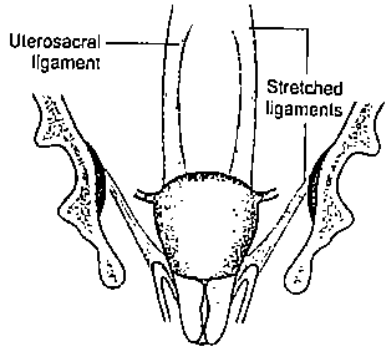


Fig. 5.3: Prolapse of uterus due to weakness of the supporting structures

Note: All the ligaments are stretched

SUPPORTS OF THE UTERUS

Normal Position of Uterus is Anteverted and Anteflexed

The external os is at the level of ischial spine.

The uterus is held in this position by **3 tier support systems.**

I. Upper Tier

- Endopelvic fascia covering the uterus
- *Round ligaments:* A remnant of the gubernaculum extending from the uterine horns to the labia majora via the inguinal canal. It functions to maintain the anteverted position of the uterus
- *Broad ligaments:* This is a double layer of peritoneum attaching the sides of the uterus to the pelvis. It acts as a mesentery for the uterus
- **Broad ligament and round ligament** are considered as false supports of the uterus.

II. Middle Tier
(Strongest support)

- **Pericervical ring:** Collar of fibroelastic connective tissue encircling the supravaginal cervix
- The ligaments (pubocervical,cardinal and uterosacral) are attached anteriorly, laterally and posteriorly to the ring respectively
- **Endopelvic fascia/ligaments/pelvic cellular tissues**
- The endopelvic fascia is condensed at places to form ligaments:
  - **Pubocervical ligament:** From pubic symphysis to anterior part of cervix and pericervical ring

  - **Cardinal/Mackenrodt's/transverse cervical ligament:** Located at the base of the broad ligament, the cardinal ligament extends laterally from the cervix and the pericervical ring to the lateral pelvic walls (condensation of parietal fascia covering the obturator internus). It contains the uterine artery and vein
  - **Uterosacral ligaments:** From the periosteum of 2nd, 3rd and 4th sacral vertebra to the posterolateral part of cervix and the pericervical ring.

This hammock like arrangement of condensed pelvic cellular tissue is the cardinal suppo of uetrus.

III. Inferior Tier

- Levator ani muscle (pelvic diaphragm) and the fascia covering it
- Perineal muscles forming the perineal body
- Urogenital diaphragm.

Delancey's Three Levels of Support of Vagina

- Level 1: The cardinal-uterosacral ligament complex provides apical attachment of th uterus and vaginal vault to the bony sacrum. Uterine prolapse occurs when the cardina uterosacral ligament complex breaks or is attenuated.
- Level 2: The arcus tendineus fascia pelvis and the fascia overlying the levator ani muscle provide support to the middle part of the vagina.
- Level 3: The urogenital diaphragm and the perineal body provide support to the lower pa of the vagina.

Q. Etiology of prolapse.

INTRODUCTION

Prolapse is defined as the displacement of an organ from its normal anatomical position.

Genital prolapse occurs due to weakness of the supports.

Etiology

- Predisposing factors: This could be acquired or congenital
- Aggravating factors.

Predisposing Factors

Acquired

Vaginal delivery with consequent injury to the supports is the single most important factor

Prolapse is unusual in cases delivered by cesarean section.

The injury caused by:

- Overstretching of cardinal and uterosacral ligaments
- Overstreching of perineum
- Overstretching and breaks in endopelvic fascia

This could be due to:
- Premature bearing down efforts prior to full dilatation
- Forceful traction with forceps or vacuum or application prior to full dilatation
- Prolonged 2nd stage of labor
- Precipitate labor
- Undue fundal pressure
- Delivery of large baby
- Repeated frequent childbirths
- Poorly timed episiotomy
- Improperly conducted delivery

- Imperfect repair of perineal injuries
- *Neuromuscular damage of levator ani:* **Internal rotation causes maximum damage** of levator ani muscle
- *Subinvolution of supporting structures:* Ill nourished asthenic women, with early resumption of activities which greatly increase the intra-abdominal pressure.

Congenital

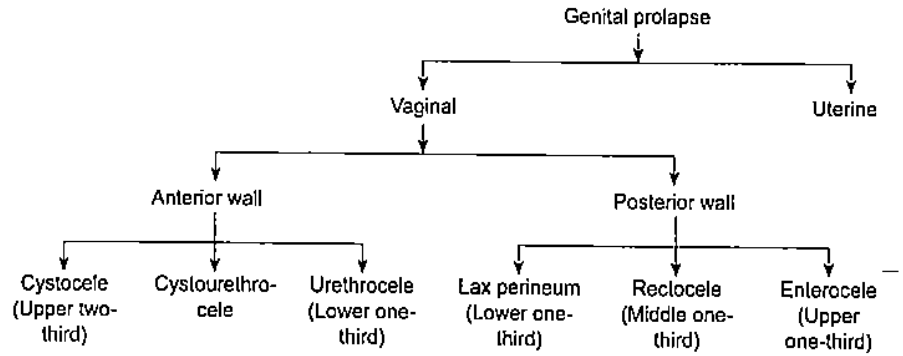
Congenital weakness of supports can cause nulliparous prolapse or prolapse following easy vaginal delivery:
- Connective tissue disorders (Ehlers-Danlos syndrome, Marfan syndrome)
- Neurological anomalies (spina bifida occulta).

Aggravating Factors

- Postmenopausal atrophy **(decrease in estrogen decreases collagen strength)**
- Chronic cough/constipation (increase in intra-abdominal pressure)
- Malnourishment
- Large ovarian tumor, fibroid (can cause pressure on uterus).
- Obesity

Q. Types of prolapse and degrees of prolapse.

TYPES OF GENITAL PROLAPSE



- **Cystocele is the MC type of vaginal prolapse.**

Vaginouterine prolapse (More common)	Uterine/Uterovaginal prolapse (Less common)
Traction variety	Pulsion variety
• Vagina prolapses first and then, due to traction, pulls cervix and uterus • Supravaginal elongation is present • Uterocervical length (UCL) is increased	• Uterus prolapses first and then drags vagina later • Supravaginal elongation not seen • UCL is not increased

Vault prolapse can occur following vaginal/abdominal/laparoscopic hysterectomy

Degrees of Prolapse

- First degree: Descent of cervix into the vagina (external os is at the level of ischial spine in normal anatomical position)
- Second degree: Descent of cervix up to the introitus
- Third degree: Descent of cervix outside the introitus
- Fourth degree (procidentia): Whole uterus (including the fundus) is outside the introitus.

The five stages of prolapse (POPQ = Pelvic Organ Prolapse Quantitative Scoring)
- Stage 0: No prolapse
- Stage I: The most distal portion of the prolapse is >1 cm above the level of the hymen
- Stage II: The most distal portion of the prolapse extends from 1 cm above to 1 cm below the hymen
- Stage III: The most distal portion of the prolapse is >1 cm below the hymen but protrudes no further than 2 cm less than the total length of the vagina
- Stage IV: Complete eversion of the vagina.

Q. Clinical features/signs and symptoms of genital prolapse.

INTRODUCTION

Prolapse is defined as the displacement of an organ from its normal anatomical position.

Genital prolapse occurs due to weakness of the supports.

Symptoms

- Variable
- There may not be any symptoms even with severe degree of prolapse or the following symptoms may be present:

Patient Profile

Generally **postmenopausal** or premenopausal multiparous women with complaints of:
- Something coming out through per vagina/fullness within the vagina
- Backache or lower pelvic dragging pain during walking, working
- The above 2 symptoms are usually **relieved on lying down.**

Vaginal Symptoms

- Sensation of a bulge or protrusion
- Seeing or feeling a bulge
- Pressure
- Heaviness
- Excessive whitish discharge (due to venous congestion) or
- Blood stained discharge due to decubitus ulcer in dependent part.

Urinary Symptoms (In presence of cystocele)

- **Frequency or urgency** may be due to cystitis
- Weak or prolonged urinary stream
- Feeling of incomplete emptying/incomplete evacuation leads to frequency
- **Manual reduction** of prolapse needed to start or complete voiding
- SUI (Stress urinary incontinence, due to bladder neck descent)
- Rarely retention of urine
- Painful micturition due to infection.

Bowel Symptoms (In presence of rectocele)

- Feeling of incomplete emptying
- Straining during defecation
- Digital evacuation needed to complete defecation
- Pushing of posterior vaginal wall needed to start or complete defecation
- Incontinence of flatus, or liquid or solid stool.

Sexual Symptoms

- Dyspareunia or difficulty in coitus.

Clinical Examination

Composite examination—inspection and palpation: Vaginal, rectal, rectovaginal may be needed.

Findings will depend on type of prolapse.

General examination:
- Nutritional status
- BMI
- Signs of myopathy/neuropathy
- Chronic airway disease
- Abdominal mass
- **Abdominal tone (help in planning type of conservative surgery).**

Local

POP is evaluated in dorsal position and standing position if needed.

The patient is asked to strain/valsalva maneuver, which would demonstrate prolapse not seen at rest.

Prolapse of uterus may be a/w prolapse of adjacent organ: Bladder and rectum.

Cystocele

- Bulge of varying degree of anterior vaginal wall, increase on straining may be seen or may be seen on separation of labia and depressing the posterior vaginal wall
- Mucosa over the bulge: Transverse rugosities
- **Impulse on coughing, diffuse margins and reducible.**

Cystourethrocele

- The bulging will include the lower one third of the anterior wall
- There may be associate SUI—leakage of urine on coughing.

Lax Perineum

- Gaping introitus with old scar of perineal tear may be seen
- Lower part of posterior vaginal wall is visible with or without straining.

Rectocele and Enterocele

- Bulging in posterior vaginal wall with transverse sulcus between the two
- The mid vaginal one is rectocele
- The enterocele bulge is in the upper third, close to the cervix and cannot be reached by finger in rectum.

Uterine Prolapse

- In 2nd or 3rd degree prolapse or procidentia: Inspection reveals a mass protruding out of introitus, the leading part of which is external os
- In 1st degree on PS, there would be descent of cervix below ischial spines on straining
- To diagnose 4th degree prolapse palpation is essential. Thumb placed anteriorly and fingers posteriorly above the mass outside the introitus will appose **(pinch test)**
- Degree of prolapse or POPQ should be done
- Evidence of decubitus ulcer or pigmentation may be present
- Bimanual examination: Shallow fornices, with normal length of vaginal cervix and normal size of uterine body
- UCL (uterocervical length) with uterine sound will reveal **increase in length** of uterine cavity which signifies **supravaginal elongation** of cervix seen in vaginouterine variety.
- Levator ani muscle tone to be assessed
- Pubovaginalis is palpated in lower third of vagina for its tone (Patient is asked to squeeze the anus)
- Rectal examination helps to detect deficient perineum.

Q. Decubitus ulcer

It is a trophic ulcer.

It is always found at the dependent part of prolapsed mass lying outside the introitus.

So it will be present **only in 3rd and 4th degree prolapse** and never in 1st and 2nd degree.

Decubitus ulcer is due to **friction, congestion, and circulatory changes** in the dependant part of the prolapse.

↓

Initial surface keratinization

↓

Cracks

↓

Infection

↓

Sloughing

↓

Ulceration and complete denudation of surface epithelium

The diminished circulation is due to constriction of prolapsed mass by vaginal opening and narrowing of uterine vessels by stretching effect.

Ulcer must be treated before operation otherwise:
- Difficulty in incision
- Postoperative healing slowed
- ↑ Bleeding
- Postoperative infection

Management

- Cervical cytology to rule out malignancy
- Reduction of the prolapse into the vagina and
- Daily packing with roller bandage or tampon soaked in **glycerin and acriflavine** heals the ulcer in a week or two.
  - Glycerin: Hygroscopic agent, decrease edema and acriflavine = yellow colored dye that helps in epithelization
- Estrogen cream can also be used in postmenopausal women.

Q. Key points of various surgeries for prolapse.

Q. Surgeries for young/nulliparous patient with prolapse.

Q. Treatment options for old/postmenopausal lady with prolapse.

INTRODUCTION

Prolapse is defined as the displacement of an organ from its normal anatomical position.

Surgical Treatment for Prolapse

Age, parity, type and degree of prolapse are the factors that decide the type of surgery.

Young patients desirous of further childbearing/ menstrual function	For old patients, family complete, postmenopausal women
Vaginal route: • Fothergill's operation • Shirodkar's uterosacral ligament advancement	• Vaginal hysterectomy with or without anterior and posterior colporrhaphy • Le Forte's repair (complete colpocleisis) • Goodell-Powel surgery (partial colpocleisis)
Anterior colporrhaphy Posterior colporrhaphy	Anterior colporrhaphy Posterior colporrhaphy
Abdominal route: SLING Surgery (preferred choice) • Purandare • Shirodkar • Khanna • Virkud (composite sling)	

Anterior Colporrhaphy ('A' repair)

Indications

Cystocoele, Urethrocoele.

Principles

- Separation of anterior vaginal, wall from bladder
- Separation and mobilization of bladder from cervix
- Plication of the pubovesicocervical (uterovesical fascia), thus elevating the bladder base and obliterating the cystocoele
- Excising the redundant portion of vaginal wall thus narrowing the vagina.

Posterior Colporrhaphy (colpoperineorrhaphy) ('P' repair).

Indications

Rectocele, old torn perineum, relaxed vaginal outlet as a part of all vaginal operations of prolapse.

Principles

- Removal of triangular piece of posterior vaginal wall
- Rectocele corrected by suturing pararectal fascia
- Approximation of levator ani by deep suturing **(Young's stitch)**
- Suturing the vaginal edges longitudinally thus narrowing the vaginal opening.

MANCHESTER (FOTHERGILL) OPERATION

(Devised by Archibald Donald and William Fothergill)

Indications

Young patient with second (sometimes even third) degree prolapse with supravaginal elongation of cervix and who wants to preserve menstrual function.

Initially, the operation was thought to preserve the fertility status of the patient.

But as it is associated with a lot of complications (mentioned below), **it is not a preferred option in nulliparous patients.**

Main Step is Amputation of Cervix

Principles

- Anterior colporrhaphy is done
- Cervix is dilated. It helps in passage of sutures, ensures adequate drainage and prevents cervical stenosis. Curettage can be done
- **Amputation of cervix**
- Plication of Mackenrodt's ligaments in front of the cervix. This facilitates their shortening and raising the cervix
- Posterior lip of the amputated cervix is covered by vaginal flap using a **Sturmdorff suture**
- Colpoperineorrhaphy is done.

Various complications include:
- Primary hemorrhage/secondary hemorrhage
- Repeated second trimester abortions due to **cervical incompetence**
- Preterm labor/PROM
- Cervical stenosis
- Cervical dystocia
- Infertility due to cervical factor.

SHIRODKAR'S MODIFICATION OF FOTHERGILL'S OPERATION/SHIRODKAR'S UTEROSACRAL LIGAMENTS ADVANCEMENT

Principles

- The vaginal wall is dissected upwards all around the cervix.
- The uterosacrals are dissected, mobilized and brought forwards and sutured in front of the cervix.
- **Cervical amputation is NOT done**
- Posterior repair is done
- The modified Fothergills operation has the advantage that **childbearing function is preserved.**
- This operation is not advisable if the uterosacrals are atrophic

SLING OPERATIONS

Following sling operations **fertility is not impaired** and hence considered **best or most suitable for nulliparous young patients.**

- When the supporting tissues are completely torn or have become atrophic, slings of Mersilene tape or Mesh are used. These synthetic slings produce minimal tissue reaction and remain unabsorbed giving lifelong support.

Types of Sling Operations

- Shirodkar's Sling: Tape is anchored to sacral promontory and posterior aspect of isthmus—STATIC, CLOSED LOOP, POSTERIOR SLING
- Purandare's Cervicopexy: Tape is anchored to anterior abdominal wall and anterior aspect of isthmus DYNAMIC, CLOSED LOOP, ANTERIOR SLING
- Khanna's Sling: Tape is anchored to anterior superior iliac spines and anterior aspect of isthmus—STATIC, OPEN, NEUTRAL SLING
- Virkud's Composite Sling: Tape is anchored to sacral promontory as well as anterior abdominal wall and posterior aspect of isthmus; left uterosacral ligament is also plicated—STATIC SLING + DYNAMIC, OPEN, ANTERIOR + POSTERIOR SLING.

PURANDARE'S SLING OPERATION

- Pfannenstiel incision
- Two strong linen stay sutures are transfixed to the isthmus anteriorly
- Middle portion of a 30 cm Mersilene tape is transfixed to the isthmus anteriorly with the stay sutures
- The left end tape is then passed between left broad ligament, through left internal inguinal ring, piercing the transeversalis fascia and turned medially at linea semilunaris between the rectus muscle and sheath where it is sutured with linen/vicryl/prolene **to rectus sheath**
- **The same is repeated on the other side**
- **Good abdominal muscle tone is prerequisite for this surgery.** If the anterior abdominal tone is poor, this surgery should not be done.

Advantages

- Technically very easy to perform
- Provides dynamic support to uterus.

Disadvantages

- Postsurgery the uterus becomes **retroverted** and the POD becomes deep and hence **enterocele is a long-term complication** of this surgery. It can be prevented by Moskowitz/Halban's surgery in which POD is obliterated
- Since the tape is anchored to the isthmus anteriorly, it may be damaged at subsequent LSCS
- Advancement of bladder on uterus may make exposure of lower uterine segment difficult
- Since it is a closed loop sling; should it become tight there is a risk of bowel loops being trapped between uterus and anterior abdominal wall

KHANNA'S SLING OPERATION

The Mersilene tape is attached to the isthmus anteriorly and it is anchored to the periosteum of the anterior superior iliac spines.

Advantages

- Technically fairly easy to perform
- Does not antevert or retrovert the uterus
- No risk of bowel obstruction.

Disadvantages

- Tape is very superficial and can be very easily felt by the patient and the superficial tape constantly rubs against dress worn tightly at the hip
- If skin wound gets infected, periostitis results which is very painful and there is a risk of the tape getting detached.

SHIRODKAR'S ABDOMINAL SLING OPERATION

- Any cystocele present must be repaired before this operation
- The disc between the fifth lumbar and the first sacral vertebra is exposed. Two strong linen stay sutures are passed through the disc (anterior longitudinal ligament) with the ends kept long
- The left psoas muscle is exposed, a loop of mersilene strip is passed through the muscle belly and ends firmly sewn together to form a loop to avoid obstruction to the rectosigmoid
- A sufficiently long strip of mersilene tape is taken and its middle portion is sutured to the back of the cervix at the level of the uterosacrals
- On the left side, the tape has to pass below the mesentery of sigmoid colon, through the loop to reach sacral promontory
- The psoas loop on the right side is not essential
- The 2 ends of the tape are anchored to the disc (anterior longitudinal ligament) with the linen stay sutures taken previously.

Advantages

- Anatomically it is the **most correct operation** as it maintains the uterus in its correct anatomical position. It provides a strong static bony support
- No tendency to enterocele formation.

Disadvantages/Complications

- Technically very difficult to perform
- Injury to sigmoid colon, mesentery, and ureters
- Hemorrhage from presacral/mesenteric vessels
- Intestinal obstruction
- Injury to genitofemoral nerve (present in psoas muscle).

VIRKUD'S COMPOSITE SLING OPERATION

As the complications of Shirodkar sling are mainly on the left side, in this surgery, on right side the tape is attached to sacral promontory and on left side the tape is attached to rectus sheath **(left-sided Purandare + right-sided Shirodkar).**

- Pfannenstiel incision
- One end of the Mersilene tape (30 cm long) is fixed to the sacral promontory posteriorly with two strong linen stay sutures
- It is then passed subperitoneally on the right side of pelvic wall, then through right broad ligament and fixed to posterior surface of isthmus of cervix with linen stay sutures
- The tape is then passed between left broad ligament, through left internal inguinal ring, piercing the transeversalis fascia and turned medially at linea semilunaris between the rectus muscle and sheath where it is sutured with linen to rectus sheath
- The left uterosacral ligament is then plicated with linen in order to correct the dextrorotation of uterus (this also helps in anteverting the uterus).

Advantages

- Technically the operation is easy to perform
- Provides double support: Bony (sacral promontory) + Dynamic (rectus sheath)
- Uterus remains anteverted
- No tendency to enterocele formation
- No risk of injury to sigmoid mesentery/colon or the genitofemoral nerve
- No risk of bowel obstruction (open sling)
- No difficulty in subsequent LSCS: As tape is posterior.

For old patients, family complete, postmenopausal women

For old patients, family complete, postmenopausal women **who are medically fit** for surgery:

VAGINAL HYSTERECTOMY

With or without anterior and posterior colporrhaphy (pelvic floor repair) is **the best and definitive surgery**

Principles

- Bladder separated and mobilized
- Anterior pouch is opened
- Posterior pouch is opened
- Bilateral uterosacrals and transverse cervical ligaments are clamped, cut and transfixed
- Uterine vessels, clamped, cut and ligated
- Uterus is delivered through anterior and posterior pouch
- Uppermost pedicles of ovarian ligament, tube and round ligament are clamped, cut and ligated, uterus removed
- Anterior colporrhaphy done

- Peritoneum closed by pursestring suture
- The vaginal vault is then closed and **vault suspension is done**
- Posterior colpoperineorrhaphy done.

LE FORTE'S OPERATION (COMPLETE COLPOCLEISIS)

- It is done in very elderly **postmenopausal** women who **are unfit for vaginal hysterectomy** (For example: With medical complications such as heart failure, past history of myocardial infarction, severe hyper tension, etc.)
- This procedure can be performed under **local anesthesia and sedation**
- **Prior to the procedure, PAP smear should be done to rule out cervical dysplasia** and pelvic USG to rule out pelvic pathology
- Sexual function **not** preserved.

Principle

Rectangular pieces of anterior and posterior vaginal walls are excised and edges are sutured to produce colpocleisis.

Goodell & Powel's Modification of Le Forte's Operation.

Same indication as Le Forte's operation (patient medically not fit for vaginal hysterectomy).

Sexual function is preserved.

Principle

Triangular pieces of anterior and posterior vaginal walls are excised and edges are sutured to produce partial colpocleisis

RING PESSARY

It is never curative, **only palliative** (relieves the symptoms).
Additional benefit: Improvement in urinary symptoms.

Indications of Ring Pessary

- Early pregnancy (up to 18 weeks)
- Puerperium
- Patients absolutely unfit for surgery
- Patient refuses surgery
- While waiting for surgery.

6 Polycystic Ovarian Syndrome (PCOS) and Endometriosis

Q. Polycystic ovarian syndrome (PCOS).

Q. Clinical features of PCOS.

Q. Management of PCOS.

INTRODUCTION

PCOS is the **most common endocrinopathy** in women of reproductive age.

First reported by **Stein and Leventhal in 1935** as a syndrome manifested by amenorrhea hirsutism and obesity associated with enlarged polycystic ovaries.

It is however a misnomer as there are no cysts in the ovary (there are multiple small follicles which look like cysts).

DEFINITION

It is a heterogenous syndrome complex characterized by **chronic anovulation** and **hyperandrogenism**, frequently associated with **insulin resistance**, resulting in menstrual irregularity, infertility and hirsutism.

Incidence

It is most important and extremely common disorder affecting 4 to 12% of women of reproductive age. It is **one of the leading causes of infertility.**

Diagnostic Criterias

- **Rotterdam 2003** criteria for diagnosis of PCOS/PCOD—**at least two out of three** should be present:
  - Oligo/anovulation
  - Hyperandrogenism: Biochemical or clinical
  - Twelve or more than 12 follicles 2–9 mm in size present within one or both ovaries on USG(Necklace of pearl pattern) and/or ovarian volume >10 mL
- Obesity is **not required** to make the diagnosis and even the ratio of FSH/ LH = 1/2 or 1/3 not essential to make the of diagnosis of PCOS.

Pathophysiology

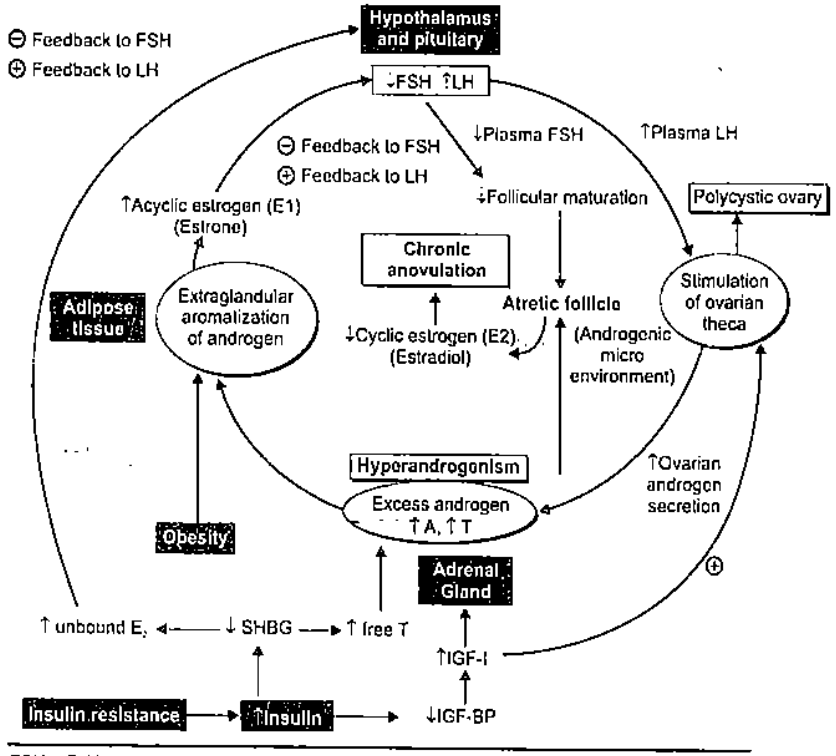
Complex and not yet well understood.

Hyperandrogenism and chronic anovulation with hyperinsulinemia.

- **Hyperthecosis** (increase testosterone from the ovaries due to stimulation of theca cells by persistently elevated LH)
- **Defective aromatization** within the ovaries (hyperandrogenic micro-environment within the ovaries) which prevents follicular maturation
- Normal aromatization in periphery (unopposed estrogenic action) and **no progesterone due to anovulation**
- Insulin resistance (IR) is considered to be the hallmark in pathophysiology of PCOS.

IR through P450c17 enzyme dysfunction causes excessive androgen production from ovary and adrenals.

It also decreases SHBG and thereby increases free testosterone and estrogen levels.



FSH = Follicle stimulating hormone
LH = Luteinizing hormone
E1 = Estrone
E2 = Estradiol
T = Testosterone

A = Androstenedione
SHBG = Sex hormone binding globulin
IGF-1 = Insulin like growth factor 1
IGF-BP = Insulin like growth factor-binding protein
T = Testosterone

Fig. 6.1: Pathophysiology of PCOS

Clinical Features

Signs/Symptoms	Frequency (%)
• Oligomenorrhea	50–90
• Amenorrhea	25–50
• Infertility	55–75
• Obesity	50–60
• Hirsutism	60–90
• Acne	25–30
• Acanthosis nigricans	
• Male pattern alopecia	
• First trimeseter abortions	
• Menorrhagia following prolonged amenorrhea	

PCO patients have **android obesity: Waist to hip ratio >0.85.**

Acanthosis Nigricans

- Diffuse, velvety thickening and hyperpigmentation of the skin
- Present at the nape of the neck, axillae, area beneath the breasts, intertriginous areas, and exposed areas
- Marker of insulin resistance
  - HAIR-AN Syndrome:

  HA = hyperandrogenism, IR = insulin resistance, AN = acanthosis nigricans.
- **Long-term complications** associated with PCOS:
  - Diabetes mellitus (because of IR)
  - Endometrial hyperplasia
  - Endometrial carcinoma (both due to unopposed estrogen stimulation)
- **Metabolic syndrome/syndrome X:** IR, obesity, hypertension, ↑ triglycerides, and ↑ FBS associated with coronary artery disease.

Investigations

Serum Values

- Sr. FSH and LH **(done on day 2 or day 3 of menstrual cycle)**
- **Sr FSH/LH =1:2 OR 1:3.** Raised LH and decreased or normal FSH. (Sr.FSH/ LH = 2:1 = Normal ratio)
- **Sr Testosterone is elevated** but generally less than 150 ng/dl (normal =20–80 ng/dl)
- Androstenedione elevated
- SHBG: Reduced
- Free/Unbound estradiol: Increased
- Estrone is increased
- DHEAS is normal or elevated
- Serum fasting insulin: Increased (>25 micro IU/ml)

- Sr. FBS/ Sr Fasting Insulin < 4.5 indicates insulin resistance
- Sr TSH and PROLACTIN levels must also routinely done
- Lipid profile is also to be done (increase in total cholesterol and LDL and decrease in HDL).

USG

TVS is preferred (100% detection rate).

Necklace of Pearls Pattern

- Twelve or more than 12 follicles 2–9 mm in size present within one or both ovaries and/or ovarian volume >10 mL
- Increase in echogenicity and volume of stroma and increase in stromal blood flow velocity
- USG shows a necklace-of-pearl pattern in 60–80% cases only. **Ovaries can be normal on USG** in a case of PCOS
- Increase in endometrial thickness due to unopposed estrogen stimulation.

Laparoscopy

Laparoscopy (can be diagnostic and therapeutic also)

- Oyster ovaries: Bilaterally **enlarged** (2–5 times of normal size), white, smooth sclerotic ovaries with thickened capsule
- No evidence of corpus luteum or stigma of ovulation.

Treatment

- Individualization of patients
- Treatment depends on patients need and symptoms
- Patient counseled about the disease
- **Lifestyle modification and weight loss** weight loss in obese patients is very important. Weight loss of 5–10% can restore ovulation and fertility in majority of patients and also improves menstrual irregularity and hirsutism
- **Multidisciplinary approach** for weight loss (gynecologist, endocrinologist, dietician, exercise program).

Treatment Options as per Symptoms

- Irregular periods/amenorrhea = regularization of menses with OC pills/cyclical progesterone
- Hirsutism/acne = suppression of androgens with OC pills/antiandrogens/mechanical methods
- Infertility = **ovulation induction with drugs/LEOS/ART (ovulation induction important short note)**
- Obesity = diet/exercise/insulin sensitizers
- Hyperinsulinemia = insulin sensitizsers/weight loss.

PCO → Desires pregnancy → **CC (clomiphene citrate)**

PCO → Desires regular periods and treatment of acne, hirsutism → **COC (containing cyproterone or drosperinone preferred)**

Insulin sensitizers **(metformin, myoinositol)** should be added to the treatment **if there is I**

Infertility/Desirous of Child

Ovulation has to be induced as infertility is due to anovulation.

Ovulation Induction Agents

1. **Clomiphene citrate (CC)** (DOC for anovulatory infertility)

Clomiphene Citrate: It is a racemic mixture of enclomiphene and zuclomiphene. Encl miphene is a more potent isomer responsible for its ovulation-inducing action.

- Dose = 50–250 mg/day for 5 days: From day 2 to day 6 or day 5 to day 9
  However, the US FDA-approved maximum dose for CC is 100 mg
- CC blocks 'E' receptors → increase FSH from pituitary → growth of follicles
- With CC ***Success rate for ovulation is 80% and success for pregnancy is 40%.***

In cases of elevated DHEAS, Dexamethasone 0.5 mg/day at bedtime combined with CC found to be useful.

2. Letrozole, Anastrozole, Tamoxifen
   - Letrozole/Anastrozole/Tamoxifen = aromatase inhibitor blocks conversion of testosterone to estrogen, leading to increased FSH from pituitary.

   Letrozole is now not available for this indication, however Anastrozole, Tamoxifen can b used.
3. Gonadotropins: **HMG** (Human Menopausal Gonadotropin) (from the urine of th menopausal women) and **recombinant FSH**.

With gonadotropins: Success rate for **ovulation is 80–99% and pregnancy rate is 40–70%**

- Follicular study through TVS is done along with ovulation induction to monitor the growt of follicles and when the **dominant follicle is 18–20 mm**, ovulation trigger is given t rupture the follicle
- For ovulation trigger, MC drug used is **Inj HCG 5000–10,000 IU IM** (derived from the urin of pregnant women or by recombinant technology). Recombinant LH is can also be use but is very expensive
- Ovulation occur **36 hours after injecting hCG.**

Side Effects of Ovulation Induction

- *Multiple pregnancies:* 3–8% with CC, 15–30% with Gonadotropins
- *Ovarian hyperstimulation syndrome (OHSS):* Most dangerous complication, more com mon with gonadotropins and very unlikely with CC
- *Increased risk of epithelial ovarian cancers:* Prolonged use of gonadotropins/CC (>6–1 months) can increase the risk of epithelial ovarian cancer.

SURGERY FOR OVULATION INDUCTION IN PCOS

- Laparoscopic ovarian drilling (LOD) or laparoscopic electrocoagulation of ovarian surfac (LEOS)

- In this surgery, monopolar current is passed within the ovary to destroy the ovarian theca
- This surgery is done ***only for infertile patients of PCOS who are resistant to ovulation*** **with gonadotropin** or when very high doses of gonadotropins are required for ovulation
- *Advantages:* No risk of OHSS and multiple pregnancy
- *Disadvantages:* Surgical procedure, risk of premature ovarian failure if excessive ovarian tissue is damaged, and adhesion formation postsurgery.

Success rate for ovulation is 70–90% and pregnancy rate is 40–70%.

ART/IVF

When all the above treatment fails then IVF is used as the last resort for infertile patients.

Insulin Sensitizers

- MC used drug till now was metformin
- Metformin (starting from 500 mg once and can be increased upto 1000 mg twice a day or 850 mg TDS) will help the patient to lose weight and will either cause spontaneous ovulation or *increase the success of ovulation induction drugs.* It reduces fasting insulin,testosterone and BMI
- MC side effects: Nausea/vomiting and bloating (GI upset)
- Most dangerous side effect: Lactic acidosis
- Metformin was thought to be teratogenic, but recent consensus is that metformin **can be continued throughout pregnancy and it decreases the risk of spontaneous abortion and development of gestational DM (GDM)**
- Newer insulin sensitizer **myoinositol** is now available. It is better tolerated than metformin and preferred nowadays.

Desires Regular Periods and Treatment of Acne, Hirsutism

Best treatment is **OC PILLS as it regularizes the cycle and can also suppress acne and hirsutism.**

Oc pills containg cyproterone or drospirenone are preferred.

OCs significantly decrease free testosterone levels. Progestin suppresses LH and estrogen increases SHBG.

Antiandrogens for Hirsutism

- Spironolactone 25–100 mg twice a day.
- Flutamide
- Finasteride
- Cyproterone acetate.

Mechanical methods like plucking/shaving/electrolysis/laser can also be used.

Eflornithine hydrochloride 13.9% cream for local use.

Q. Define endometriosis. Give clinical features and management of endometriosis.

Q. Laparoscopy in endometriosis.

DEFINITION

Endometriosis is defined as the presence of normal functional endometrial mucosa (glands and stroma) abnormally implanted in locations other than the uterine cavity. It was first described by **Von Rokitansky.**

MC Sites in Order of Frequency

- Ovaries (ovarian endometriosis = endometrioma = chocolate cyst of the ovaries)
- POD (Pouch of Douglas)
- Uterosacral ligaments.

Theories for Development of Endometriosis

- Sampson's theory of retrograde menstruation: The most accepted theory
- Ivanoff and Meyer: Celomic metaplasia
- Hematogenous spread
- Lymphatic spread (Halban's theory)
- Direct implantation.

Symptoms

About one third of women with endometriosis remain asymptomatic.

When they do occur, symptoms, such as the following, typically reflect the area of involvement:

- **Dysmenorrhea (50%):** Progressively increasing **secondary dysmenorrhea** (due to PGF_2 and thromboxane)
- Menorrhagia and premenstrual spotting (50–60%)
- **Pelvic pain** or backache due to adhesions,scarring or impingement of nerves
- Lower abdominal or back pain
- **Deep Dyspareunia:** Mostly seen in endometriosis of rectovaginal septum or Pouch of Douglas and with fixed retroverted uterus
- **Infertility:** Around 40–60% patients have infertility due to multiple factors like tubal adhesions, ovarian dysfunction, dyspareunia
- **Dyschezia (pain on defecation):** Often with cycles of diarrhea and constipation, rectal bleeding in cases of involvement of colon and rectum
- Pain on micturition and/or urinary frequency, **cyclical hematuria if bladder is involved.**

Clinical Examination

Abdominal

Examination may be normal. Very rarely, enlarged chocolate cyst or a tubo-ovarian mass may be felt in the lower abdomen arising from the pelvis. The mass is tender with restricted mobility.

Pelvic examination: May be normal or may reveal the following:

- **Fixed retroverted uterus**
- Pelvic tenderness

- **Nodules in the POD**
- Nodularity of the uterosacral ligaments
- Unilateral or bilateral adnexal mass
- Speculum examination may reveal bluish nodules in posterior fornix.

Investigations

Laparoscopy is the Investigation of Choice

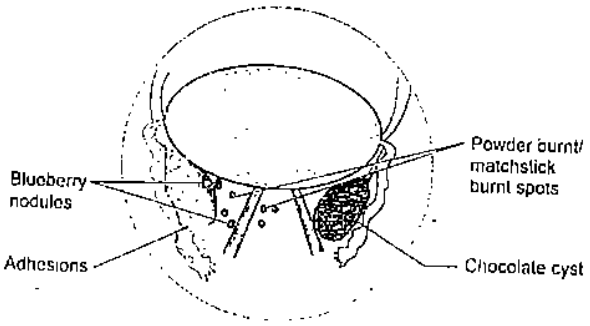


Fig. 6.2: Laparoscopy in endometriosis

Laparoscopy findings are:
- Chocolate cysts
- Powder burn spots
- Matchstick burnt spots
- Blueberry lesion
- Red/purple raspberry lesion
- White lesion
- Red/flame lesion
- Subovarian adhesions
- Subtle peritoneal defects associated with endometriosis is called Allen-Master syndrome.

USG, CT SCAN, MRI may detect ovarian chocolate cysts but can also be normal.

Ca 125 is a non specific marker, may be **moderate elevations** in levels (upto 35 IU/ml is normal).

Treatment

The treatment depends on the patient's age, size and extent of lesion and desire for fertility.
- Medical
- Surgical
- Combined.

Generally surgery is done first followed by medical management or **sandwhich therapy** is done in which medical → surgical → medical treatment is done.

Medical Management

The aim of medical management is **to induce amenorrhea** in the patient thereby causing atrophy of the implants. The treatment is considered suppressive rather than curative as recurrence is high on stopping the treatment.

The medical management provides symptomatic relief from pain,dysmenorrhea, and also decrease the size of lesions:
- **Pseudopregnancy regimen:** OC pills, DMPA POP, and Mirena.
- **Pseudomenopause regimen:** Danazol. (Hardly ever used today because of androgenic side effects).
- **Medical castration: GnRH analogues (most common drug used for medical management).** Treatment is usually restricted to monthly injections for 6 months. Loss of trabecular bone density caused by GnRH is restored by 2 years after cessation of therapy. Other prominent adverse effects include hot flashes and vaginal dryness.

Hormones used in endometriosis

Drugs	Dose	Mechanism
Combined estrogen progestogen (oral pill)	1–2 tablets 6–9 months	Pseudopregnancy
Progestogens		Pseudopregnancy
Oral		
• Medroxyprogesterone acetate	10 mg thrice daily × 6–9 months	
	10–20 mg daily × 6–9 months	
• Dydrogesterone	10–30 mg daily × 6–9 months	
• Norethisterone		
IM		
• Medroxyprogesterone	150 mg 3 months interval × 2	
IUCD		
• Levonorgestrel-releasing-IUCD		
Danazol	400–800 mg orally in 4 divided doses × 6–9 months	Pseudomenopause
Gestrinone	1.25 or 2.5 mg twice a week × 6–9 months	Pseudomenopause
GnRH analogs	• Leuprolide 3.75 mg IM monthly × 6 months • Nafarelin 200 µg intranasally daily × 6 months • Goserelin 3.6 mg depot IM monthly × 6 months	Medical oophorectomy

SURGERY

Surgical care can be broadly classified as conservative when reproductive potential is retained, semiconservative when reproductive ability is eliminated but ovarian function is retained, and radical when the uterus and ovaries are removed. **Age, desire for future childbearing, and**

deterioration of quality of life are the main considerations when deciding on the extent of surgery.

Conservative Surgery

Surgical efforts **(laparoscopy preferred over laparotomy)** are aimed at **removal of the endometrial implants and correction of anatomic distortions.** Implants can be ablated using either laser energy or electrosurgical techniques.

Patients with infertility: **Laparoscopic ovarian cystectomy, adhesiolysis, and electrocoagulation of endometriotic implants** with bipolar current.

Chocolate Cyst/Endometrioma

- Treatment options include drainage/aspiration or cystectomy
- After drainage the cyst wall epithelium is removed or destroyed with coagulation
- This decreases the risk of recurrence but can lead to loss of ovarian reserve
  Pregnancy rates are about 60% in moderate cases and 35% is severe cases
  Maximum rates are observed in first 6 months post surgery
  In severe case IVF-ET is required.

For Pain Relief

Presacral neurectomy is used to relieve severe dysmenorrhea. The nerve bundles are transected at the level of the third sacral vertebra, and the distal ends are ligated.

Nodularity of the uterosacral ligaments may contribute to dyspareunia and low back pain. The transmission of neural pathways is via the Lee-Frankenhäuser plexus. **Laparoscopic uterine nerve ablation (LUNA)** is performed to interrupt the pain fibers.

Radical Surgery

If the family is complete and the patient has severe pain or menstrual complaints: Hysterectomy with bilateral salpingo-oophorectomy with resection of all endometriotic implants.

7 Hysterolaparoscopy

Q. Indications of laparoscopy.

Q. Contraindications and complications of laproscopy.

INTRODUCTION

Laparoscopy is a technique of visualization of peritoneal cavity by means of fiber optic endoscope introduced through abdominal wall.

Laparoscopic surgery, also called minimally invasive surgery (MIS), or keyhole surgery, is a modern surgical technique in which operations in the abdomen and pelvis are performed through small incisions (usually 0.5–1.5 cm) as opposed to the larger incisions needed in laparotomy.

Prior pneumoperitoneum is achieved with the help of CO_2 gas.

The intra-abdominal pressure should be between 10–15 mm Hg.

Indications

- Diagnostic
- Therapeutic.

Diagnostic

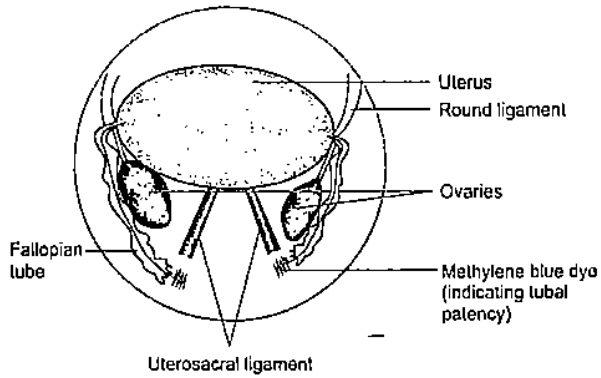


Fig. 7.1: Laparoscopy and chromopertubation

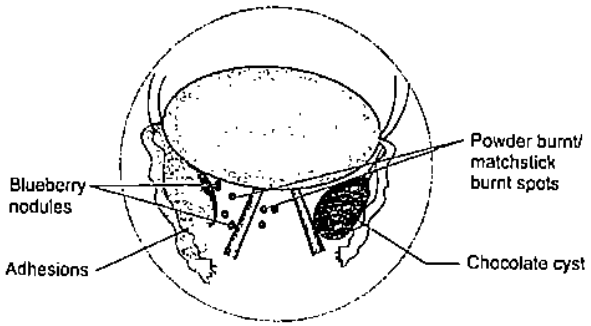


Fig. 7.2: Laparoscopy in endometriosis

- *Infertility:* Tubal factor evaluation, chromopertubation, tubal adhesions, fibroids, endometriosis, etc.
- Chronic pelvic pain
- *Pelvic mass:* Fibroids, ovarian tumors
- Mullerian anomalies
- Uterine perforation
- Patients with primary amenorrhea
- *Acute pelvic lesions:* Ectopic pregnancy, acute salpingitis, etc.

Therapeutic

- *Tubes:* Tubal ligation, adhesiolysis, tuboplasty, TL reversal
- *Uterus:* Myomectomy, hysterectomy (LAVH, TLH)
- *Ovary:* Cystectomy, oophorectomy, cyst aspiration
- *Ectopic pregnancy:* Salpingectomy
- LEOS for PCOS
- Endometriosis
- Urinary incontinence
- Onco surgeries.

Contraindications of Laparoscopy

Contraindications of laparoscopy

- Severe cardiopulmonary disease
- Patient hemodynamically unstable
- Generalized peritonitis
- Significant hemoperitoneum
- Intestinal obstruction
- Extensive peritoneal adhesion
- Large pelvic tumor

Contd...

Contd...

Contraindications of laparoscopy

- Pregnancy > 16 weeks
- Advanced malignancy
- Anticoagulation therapy

Complications

Due to Laparoscopy

- Extraperitoneal insufflation—surgical emphysema
- Omental emphysema
- Trauma/injury to:
  - Blood vessels: Mesenteric, omental, aorta, inferior epigastric vessels
  - Bowel
  - Bladder
  - Ureter —
  - The trauma may be mechanical or thermal by electrical or laser energy
- Electrosurgical complications causing thermal injury
- Gas embolism (CO_2) causing hypotension, cardiac arrhythmia.

Anesthetic Complications

- Hypoventilation: Due to pneumoperitoneum and Trendelenburg position
- Hypercarbia and metabolic acidosis
- Basal lung atelectasis
- Aspiration, cardiac arrest.

Complications Common to Any Surgical Procedure

- Hemorrhage
- Infection
- Wound dehiscence and incisional hernia.

Q. Indications of hysteroscopy.

INTRODUCTION

Hysteroscopy

Procedure to visualize the uterine cavity by means of fiber optic endoscope introduced through cervix.

Indications

- Diagnostic
- Therapeutic

Various indications for diagnostic hysteroscopy are as follows:

- **Abnormal uterine bleeding/menorrhagia/postmenopausal bleeding:** Hysteroscopy ha nearly replaced standard D and C for the management of abnormal uterine bleeding (AUB

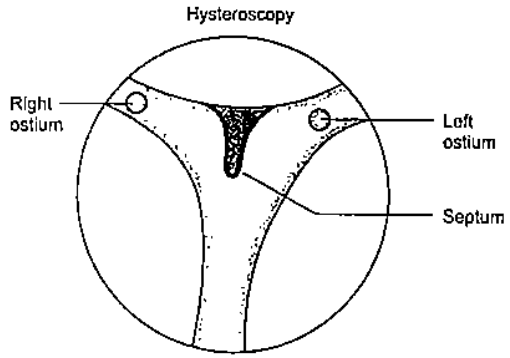


Fig. 7.3: Hysteroscopy in septate uterus

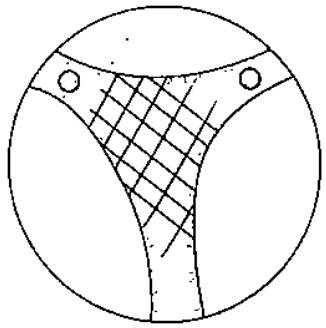


Fig. 7.4: Intrauterine adhesions (Asherman's syndrome)

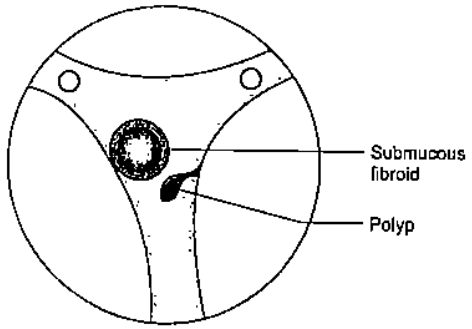


Fig. 7.5: Hysteroscopy detecting submucous fibroid and polyp

as it allows for direct visualization and diagnosis of intrauterine abnormalities (like polyps, submucous fibroids).

- **Infertility:** Hysteroscopy increases accuracy in diagnosing the cause of intrauterine filling defects detected on HSG. Intracavitary lesions (fibroids, septum, and adhesions) are implicated as causes of infertility. In unexplained infertility, hysteroscopy may be performed simultaneously with laparoscopy to evaluate the uterine cavity.
- **Recurrent spontaneous abortions:** Intracavitary lesions (fibroids, septum, and adhesions) are implicated as causes of infertility and recurrent abortions.
- **Misplaced IUCD.**

Indications of Therapeutic/Operative Hysteroscopy

For all intracavitatory pathologies hysteroscopy is the gold standard for treatment.

- **Polyps and fibroids:** Endometrial polyps and submucous fibroids are well known to cause vaginal bleeding and can be removed with hysteroscopy (polypectomy, myomectomy).
- **TCRE and endometrial ablation** can be done in cases of DUB.
- **Intrauterine adhesions:** Asherman syndrome was identified in 1948 as uterine synechiae. These intrauterine adhesions (IUA) are often associated with amenorrhea or infertility. Hysteroscopy is the gold standard used to diagnose and treat these adhesions. Benefits include visually directed lysis.
- **Lateral wall metroplasty** in cases of T shaped uterus and **septal resection** in cases of septum.
- Removal of IUCD which is in the cavity when threads are missing.
- Endometrial biopsy of suspected endometrium under direct vision.
- **Cornual catheterization** in cases of cornual blocks of fallopian tubes.
- **Sterilization:** Essure coil insertion.

8

Oncology

Q. Risk factors for CIN and management of CIN.

Q. Management of CIN III.

INTRODUCTION

Cervical intraepithelial neoplasia (CIN) is a premalignant lesion that is diagnosed by histology as CIN1, CIN2, or CIN3. If left untreated, CIN2 or CIN3 can progress to cervical cancer.

RISK FACTORS FOR CA CERVIX AND CIN

- Young age at first intercourse (<16 years)
- Multiple sexual partners
- Cigarette smoking
- Race
- Early age at first pregnancy
- High parity
- Low socioeconomic status
- **Human papillomavirus (HPV) infection, HSV**
- HIV
- Immunosuppression.

Over 90% cases with CIN and invasive cancer are positive with HPV DNA.

Human Papillomavirus and CIN

- HPV produces CIN in 90% cases

HPV Type	Oncogenic potential	Comment
6,11	Low	• Anogenital warts
31, 33, 35, 51, 52	Intermediate	• CIN 1, 2, 3
16, 18, 45, 56	High	• CIN 2, 3 • Invasive CA

- **HPV-16 is the most common HPV** seen in invasive CA and CIN 2/3 and is found in 50% cases

- HPV-16 is not very specific and is also the most common HPV type in women with normal cytology
- **HPV-18 is more specific than HPV-16** for invasive tumors.

Life cycle of unstable cervical epithelium

Cervical epithelium	CIN I	CIN II	CIN III/CIS
Regression to normal (%)	60	40	30
Persistence (%)	30	35	50
Progression to CIN III/CIS (%)	10	20	–
Progression to invasion (%)	<1	5	20

Diagnosis

- **Exfoliative cytology (Pap smear)** is the gold standard for screening.
  - The Papanicolaou test is a method of cervical screening used to detect potentially precancerous and cancerous processes in the transformation zone. The test was invented by and named after the prominent Greek doctor Georgios Papanikolaou.
  - **Pap test reduces the incidence of cervical cancer by 60–90% and the death rate by 90%.**
  - **As per ACOG guidelines the first Pap smear should be done at 21 years of age or 3 years after vaginal sex.**
  - If first Pap smear is normal then it should be repeated after one year and then again after a year. If three annual Pap smears are normal, then Pap smear should be done every three years **till 65 years of age.**
  - The cells are examined under a microscope to look for abnormalities. The test aims to detect potentially precancerous changes (called cervical intraepithelial neoplasia (CIN) or cervical dysplasia).
  - Traditional Pap tests can be hard to read because cells can be dried out, covered with mucus or blood, or clump together on the slide.
- ***Liquid based cytology:*** Superior to Pap smear and has a better sensitivity and specificity
  - The sample is collected in a similar way to the Pap smear but instead of smearing the sample onto a microscope slide (as in Pap smear), the head of the spatula, where the cells are lodged, is broken off into a small glass vial containing preservative fluid, or rinsed directly into the preservative fluid.
  - The sample is then spun and treated to remove any obscuring material like blood, mucus or pus.
  - A thin layer of the cells is deposited onto a slide. The slide is examined in the usual way under a microscope.
- ***HPV DNA test:***
  The HPV test is a screening test for cervical cancer. The test detects the presence of HPV, the virus that causes cervical cancer. Certain types of HPV—including types 16 and 18—increase cervical cancer risk.
  The sample is taken at the same time as Pap smear.
- VIA (visual inspection with acetic acid)
  Acetic acid is applied on cervix and if there are acetowhite area, colposcopy and biopsy is recommended.

- ***Colposcopy and cervical biopsy:*** An abnormal cytology report warrants further evaluation with colposcopy and cervical biopsy
  - Colposcope is an instrument which gives an illuminated and magnified (6-16 fold) view of the cervix and the tissues of the vagina
  - The main goal of colposcopy is to prevent cervical cancer by detecting precancerous lesions early and treating them
  - Three percent acetic acid is applied on the cervix and biopsy is to be taken from **acetowhite areas** (due to coagulation of nucleic acid)
  - The abnormal findings include:
    - Leukoplakia/acetowhite areas
    - Punctuation (dilated capillaries seen end on)
    - Mosaic pattern
    - Neovascularization/atypical blood vessels and branching (green filter is used).

If colposcopy is not available then Schiller's test directed biopsy can be taken.

Confirmation of Diagnosis is by Biopsy

- Diagnostic cone biopsy: Indications are:
  - If there is a mismatch between cytology and histology (If Pap smear is abnormal but cervical biopsy is normal)
  - If entire TZ is not visualized on colposcopy (unsatisfactory colposcopy)
  - If endocervical currettage is positive.

Treatment

Definitive treatment will depend on:
- CIN I/II/III or CIS
- Patients age
- Parity desire for reproduction
- Facilities available for follow-up (colposcopy and cytology).

The principal treatments for CIN available are:

1. Local ablation:
   (No tissue available for histopathology)
   - Cryotherapy
   - Cold coagulation
   - Electrodiathermy
   - Laser vaporization.
2. Excisional methods **(Tissue is available for histopathology):**
   - Large loop excision of the transformation zone (LLETZ, or LEEP)
   - Cold knife or laser conization.
3. Simple hysterectomy (Not radical).

   - CIN I = Wait and watch and regular follow-up. If it is not possible it can be treated like CIN III
   - CIN II = Follow-up or cryosurgery.

CIN III and CIS

- If patient wants to **conserve the uterus/desirous of further child bearing:**

  Complete destruction of the lesion by any of the following techniques after the following criteria are fulfilled:
  - The entire lesion is visualized within the TZ
  - Exclusion of microinvasion
  - No endocervical glandular involvement.

1. Cryotherapy: Crystallizing intracellular water at -90°C
   Nitrous oxide or CO_2 is used
   Depth of destruction = 5 mm
2. Cold coagulation: Temperature of 100-120°C
3. Electrodiathermy: Unipolar needle electrode is used
   Depth of destruction = 8-10 mm
4. CO_2 laser vaporization
   Depth of destruction = 7 mm
5. Cold knife or laser conization: However **therapeutic conization is reserved for stage IA1 microinvasive cervical cancer in young patients to preserve the uterus.**

Complications

- Hemorrhage
- Infections
- Cervical stenosis
- Cervical incompetence.

6. **Loop electroexcision procedure/large loop excision of transformation zone** (LEEP/LLETZ) **(most commonly done and considered the best conservative treatment for CIN III)**
   - Two to three cm loop of thin stainless steel wire used to excise the TZ
   - Blended current (cutting and coagulation)
   - Depth of destruction = 10 mm
   - Simple and quick procedure under local anesthesia
   - Tissue removed and send for histopathology
   - Minimal complications.

Follow-up

- Post treatment cytology at 6 months till negative and then repeated at 12 months
- Thereafter repeated yearly for 5 years and
- Then 3 yearly.

Hysterectomy (simple abdominal/vaginal or laparoscopic. **NEVER RADICAL)**
- If the family is complete
- If the patient is not ready for regular follow-ups or
- Has associated problems such as prolapse or fibroids
- Cancerphobia.

Prevention

- To delay sexual exposure
- To avoid multiple sexual partners
- To avoid smoking
- Barrier contraception
- Local vaginal and penile hygiene
- **HPV vaccination**
  (highly effective intervention to prevent CIN and CA cervix).
  Bivalent (against HPV types 16, 18) and quadrivalent (against HPV types 6, 11, 16, 18) vaccines are now available in India and recommended for females 9–45 years of age.

Q. Risk factors, pathogenesis and diagnosis of cervical cancer.

Q. Clinical features and management of cervical cancer.

INTRODUCTION

Carcinoma cervix is the MC cancer affecting women in India today, followed by breast cancer. Every 8 minutes a lady dies in India because of cervical cancer. The most common variety of Ca cervix is squamous cell carcinoma (85–90%).

- **Risk factors for CA cervix/CIN:**
  - Young age at first intercourse (<16 years)
  - Multiple sexual partners
  - Cigarette smoking
  - Race
  - High parity
  - Low socioeconomic status
  - Human papillomavirus (HPV) infection
  - HIV
  - Immunosuppression.

The cervix is composed of the columnar epithelium, which lines the endocervical canal, and squamous epithelium, which covers the exocervix. The point at which they meet is called as squamocolumnar junction (SCJ).

- The SCJ rarely remains restricted to external os. Instead, it is a dynamic point that changes in response to puberty, pregnancy, menopause, and hormonal stimulation. In neonates, SCJ is located on the exocervix. At menarche, the production of estrogen causes the vaginal epithelium to fill with glycogen. Lactobacilli act on the glycogen and lower the pH, stimulating the subcolumnar reserve cells to undergo metaplasia.
- Metaplasia advances from the original SCJ inward, toward the internal os and over the columnar villi. This process establishes an area called the transformation zone (TZ). The TZ extends from the original SCJ to the physiologically active SCJ.

Pathogenesis of CIN and Invasive Carcinoma

The initial event in cervical dysplasia and carcinogenesis is likely to be infection with HPV. The mechanism by which HPV affects cellular growth and differentiation to interactions of **viral E6 and E7 proteins with p53 and Rb resulting in gene activation.**

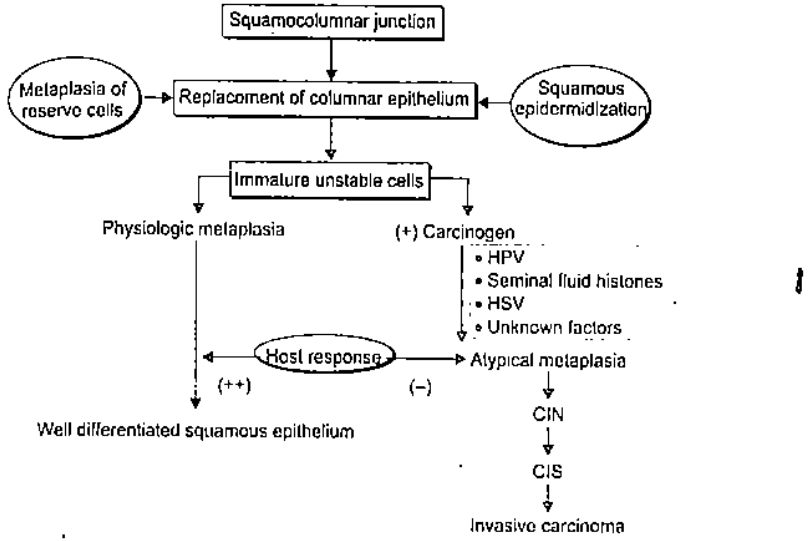


Fig. 8.1: Pathogenesis of CIN and invasive carcinoma

Diagnosis

- Early cervical cancer (stage 1a and b)
- Late/advanced carcinoma (stage 2 onwards).

Early

The concept of early CA cervix is not well defined, but it would include stages 1A, 1B which have got a better prognosis and maximal 5 year survival rate.

The presentation could be:

- Preclinical
- Clinical.

Preclinical

No symptoms or physical signs. The diagnosis is made during routine screening procedures or incidental on histopathology

- Exfoliative cytology (Pap smear) is the gold standard for screening.
  - The Papanicolaou test is a method of cervical screening used to detect potentially pre-cancerous and cancerous processes in the transformation zone. The test was invented by and named after the prominent Greek doctor Georgios Papanikolaou
  - The cells are examined under a microscope to look for abnormalities
  - **Abnormal smears should be subjected to colposcopic guided cervical biopsy**
  - In the absence of colposcopy, **Schiller's test** directed biopsy can be done.

- ***Liquid based cytology:*** Superior to Pap smear and has a better sensitivity and specificity
  - The sample is collected in a similar way to the Pap smear and the head of the spatula, where the cells are lodged, is broken off into a small glass vial containing preservative fluid, or rinsed directly into the preservative fluid
  - The sample is then spun and treated to remove any obscuring material like blood, mucus or pus
  - A thin layer of the cells is deposited onto a slide.
- *HPV DNA test:* The HPV test is a screening test for cervical cancer. The test detects the presence of HPV, the virus that causes cervical cancer. Certain types of HPV—including types 16 and 18—increase cervical cancer risk.
  The sample is taken at the same time as Pap smear.
- ***Colposcopy and cervical biopsy:*** An abnormal cytology report warrants further evaluation with colposcopy and cervical biopsy.
  - Three percent acetic acid is applied on the cervix and biopsy is to be taken from acetowhite areas (due to coagulation of nucleic acid)
  - The abnormal findings include:
    - Leukoplakia/acetowhite areas
    - Punctuation (dilated capillaries seen end on)
    - Mosaic pattern
    - Neovascularization/atypical blood vessels and branching.

If colposcopy is not available then Schiller's test directed biopsy can be taken.

Confirmation of Diagnosis is by Biopsy

Depending on the depth of invasion the diagnosis of stage 0 (carcinoma in situ) or stage 1A (up to 5 mm invasion below the basement membrane) or 1B (> 5 mm invasion) is made.

- Diagnostic cone biopsy should be done in following cases:
  - If there is a mismatch between cytology and histology
    (If Pap smear is abnormal but cervical biopsy is normal)
  - If entire TZ is not visualized on colposcopy (unsatisfactory colposcopy)
  - If endocervical curettage is positive.

Early Stage Clinical and Advanced/Late Carcinoma

Cervix maybe abnormal in appearance, with red granular gross erosion, ulcer, or nodular growth. These abnormalities can extend to the vagina.

Punch biopsy (and histopathology) taken from the growth or ulcer on cervix is mandatory and confirmatory.

Once the diagnosis is established:

- Complete blood count (CBC)
- RFT and
- LFT (to look for abnormalities from possible metastatic disease)
- **Cystoscopy and proctoscopy** should be performed in patients with a bulky primary tumor to help rule out local invasion of the bladder and the colon
- Barium enema studies can be used to evaluate extrinsic rectal compression from the cervical mass

- A routine chest radiograph is obtained to help rule out pulmonary metastasis
- A CT scan of the abdomen and pelvis is performed to look for metastasis in the liver, lymph nodes, or other organs and to help rule out hydronephrosis or hydroureter
- MRI or positron-emission tomography (PET) scanning is an alternative to CT scanning
- However, CT, MRI and PET Scans are **not allowed by FIGO for staging purpose.**

Staging Procedure

Physical examination	• Palpate lymph nodes • Examine vagine • Bimanual rectovaginal examination (under anesthesia recommended)
Radiologic studies (Allowed by FIGO)	• Intravenous pyelogram • Barium enema • Chest X-ray • Skeletal X-ray • Biopsy • Conization • Hysteroscopy • Colposcopy • Endocervical curettage • Cystoscopy • Proctoscopy
Optional studies (not allowed by FIGO)	• Computerized axial tomography • Lymphangiography • Ultrasonography • Magnetic resonance imaging • Radionuclide scanning • Laparoscopy

Clinical Features

Symptoms

- *Patient profile:* Patients are usually multiparous and in premenopausal age group (30–45 years) or even postmenopausal age
- In early stages there maybe no symptoms
- Clinically, the first symptom of cervical cancer is **abnormal vaginal bleeding**, intermittent or continuous
- Usually its **postcoital bleeding**
- Postmenopausal bleeding
- Vaginal discomfort
- Malodorous vaginal discharge
- Pelvic pain
- **Dysuria, frequency, hematuria** due to bladder involvement
- It can invade the rectum directly, leading to constipation, fistula
- Ureteral obstruction, with or without **hydroureter or hydronephrosis leading to uremia**
- The triad of leg edema, pain, and hydronephrosis suggests pelvic wall involvement
- Cachexia, anemia, and uremia.

On Examination

- PS: Growth could be **ulcerative or fungating** which **bleeds on touch**
- Bimanual examination, the indurated growth is felt and extent to vagina and sides can be felt
- Induration of the bladder base maybe felt through anterior fornix
- Rectal examination is important to note the involvement of parametrium and lateral pelvic wall. Induration is nodular.

Management of Cancer of Cervix Stagewise

Stage IA1

- Young patient/family not complete (to retain uterus) = **therapeutic conization**
- Old patient/family complete = **simple extrafascial hysterectomy.**

Stage IA2, IB, and IIA

Radical/Wertheim's hysterectomy	Concurrent chemoradiation (CTRT)
Only for stages: IA2, IB, IIA	I–IV IIb–IV

RT includes combined external beam radiation with brachytherapy

Cisplatin is given before RT as a radiosensitizer hence the preferred terminology is concurrent chemoradiation

- **All stages (I-IV) are radiosensitive**
- Stages of Ca cervix that are operable (radical/Wertheim's hysterectomy) are IA2, IB, and IIA
- **Stages IIB-IV are not operable and have to be treated with CTRT only**
- IA2, IB, IIA are radiosensitive and surgically operable, but **surgery is preferred over CTRT** for these stages for the following reasons:
  - Preservation of ovarian function
  - Preservation of vagina for coital function
  - Psychological benefit to the patient
- **Ca cervix almost never spreads to ovary and so when radical hysterectomy is done, oophorectomy is not required.**

Indications of Postoperative Radiotherapy

A randomized trial showed that patients with **parametrial involvement, positive pelvic nodes, or positive surgical margins** benefit from a postoperative combination of cisplatin-containing chemotherapy and pelvic irradiation.

Postoperative radiation therapy is also recommended in patients who have at least two intermediate risk factors (including tumor size greater than 2 cm, deep stromal invasion, or lymphovascular space invasion).

Comparison between the Two Modalities of Treatment for Ca Cervix

	Surgery	Radiation
Survival	85%	85%
Serious complications	Urologic fistulas 1–2%	Intestinal and urinary strictures and fistulas 1.4–5.3%
Vagina	Initially shortened but may lengthen with regular intercourse	Fibrosis and possible stenosis, particularly in postmenopausal patients
Ovaries	Can be conserved	Destroyed
Chronic effects	Bladder atony in 3%	Radiation fibrosis of bowel and bladder in 6–8%
Surgical mortality	1%	1% (from pulmonary embolism during intracavitatory therapy)

- Point A and Point B are in relation to radiotherapy for Ca Cervix

	Point A	Point B
Location	2 cm above and 2 cm lateral to external os	2 cm above and 5 cm lateral to external os
Structure present	Paracervical/Parametrial lymph node	Obturator lymph node
Dose of radiation	7000–8000 cGy	6000 cGy

RECENT ADVANCES

Radical trachelectomy: This involves removal of cervix, parametrium, vaginal cuff and pelvic lymphadenectomy. The uterus is preserved for further fertility.

The eligibility criteria include:

- Desire to preserve fertility/young patients
- FIGO stage 1A2 and 1B1
- Lesion size of 2 cm or smaller
- No lymph node metastasis.

However, it is not yet considered the standard of care, Wertheim's hysterectomy is the standard care for stages 1A2 and 1B1.

Q. FIGO staging of cervical cancer.

Clinical Staging of Cancer Cervix (FIGO)

	Preinvasive carcinoma
Stage 0	Carcinoma in situ, intraepithelial carcinoma (cases of stage 0 should not be included in any therapeutic statistics)
	Invasive carcinoma
Stage I	Carcinoma strictly confined to the cervix (extensions to the corpus should be disregarded)
Stage Ia	Preclinical carcinomas of the cervix, i.e. those diagnosed only by microscopy Stage 1a1: Lesion with <3 mm invasion Stage 1a2: Lesions detected microscopically and can be measured The upper limit of the measurement should show a depth of invasion of >3–5 mm taken from the base of the epithelium, either surface or glandular, from which it originates, and a second distinction, the horizontal spread, must not exceed 7 mm. Larger lesions should be staged as Ib

Contd...

Contd...

Invasive carcinoma	
Stage 1b	Lesion invasive >5 mm Stage 1b1: Lesions less than or equal to 4 cm Stage 1b2: Lesions larger than 4 cm
Stage II	The carcinoma extends beyond the cervix but has not extended onto the wall The carcinoma involves the vagina, but not the lower one-third Stage IIa: No obvious parametrial involvement Stage IIb: Obvious parametrial involvement
Stage III	The carcinoma has extended onto the pelvic wall. On rectal examination, there is no CA free space between the tumor and the pelvic wall. The tumor involves the lower one-third of the vagina. All cases with hydronephrosis or nonfunctioning kidney Stage IIIa: No extension to the pelvic wall Stage IIIb: Extension onto the pelvic wall and/or hydronephrosis or nonfunctioning kidney
Stage IV	The carcinoma has extended beyond the true pelvis or has clinically involved the mucosa of the bladder or rectum. A bullous edema, as such, does not permit a case to be allotted to stage IV Stage IVa: Spread of the growth to adjacent organs Stage IVb: Spread to distant organs

Recent advances	
FIGO (2009) Staging for Ca Cervix	
IA1	Confined to the cervix, diagnosed only by microscopy with invasion of <3 mm in depth and lateral spread 7 mm
IA2	Confined to cervix, diagnosed with microscopy with invasion of >3 mm and <5 mm with lateral spread of 7 mm
IB1	Clinically visible lesion or greater than A2, <4 cm in greatest dimension
IB2	Clinically visible lesion, >4 cm in greatest dimension
IIA1	Involvement of the upper two-thirds of the vagina, without parametrial invasion, <4 cm in greatest dimension
IIA2	>4 cm in greatest dimension
IIB	With parametrial involvement
IIIA/B	Unchanged
IVA/B	Unchanged

Q. Causes of postmenopausal bleeding.

Postmenopausal bleeding (PMB) is vaginal bleeding that happens at least 12 months after the periods have stopped due to menopause.

It should never be neglected and the patient should always be evaluated further.

Causes

- **Uterus/Endometrium:**
  - Atrophic endometrium/senile endometritis
  - Use of HRT
  - Endometrial hyperplasia; simple, complex, and atypical
  - Endometrial cancer. The probability of a woman presenting with PMB having endometrial cancer is 10%. However, 75–90% of women with endometrial cancer present with PMB
  - Endometrial polyps
  - Uterine sarcoma
  - Fibroids (very rare, suspect sarcomatous degeneration).
- **Cervix:**
  - Cancer
  - Erosion
  - Cervicitis
  - Polyps.
- **Vagina:**
  - Vaginal atrophy
  - Cancer of vagina.
- **Ovary:**
  - Ovarian cancer, especially estrogen-secreting (granulosa and theca cell) ovarian tumors.
- **Vulva:**
  - Vulval cancer.
- **Fallopian tube**

  Cancer (very rare)
  - Nongynecological causes including trauma or a bleeding disorder
  - Bleeding from the urinary tract or rectum maybe confused with postmenopausal bleeding.

Q. Risk factors, clinical features and management of endometrial CA.

INTRODUCTION

It the most common genital malignancy in **developed countries.** However in India and in developing countries CA cervix is the most common.

Adenocarcimoma is the most common variety of CA endometrium.

RISK FACTORS/ETIOLOGY

Estrogen-dependent cancer.

- **Persistent unopposed stimulation of endometrium with estrogen is the single most important factor** for the development of CA endometrium.
  - PCOS (anovulation, hyperestrogenism)
  - Granulosa cell tumor of ovary (secretes estrogen)
  - Unopposed estrogen therapy in HRT (in HRT both estrogen and progesterone to be given in patients with intact uterus)
  - Early menarche and late menopause (more exposure to estrogen. Risk is increased if menopause has not occurred by 52 years).
- Age: 75% patients are postmenopausal with **median age 60 years**
- 10% of patients with postmenopausal bleeding have CA endometrium
- Nulliparity

- **Obesity, hypertension, and diabetes mellitus** associated with CA endometrium = **corpus CA syndrome.** Obesity leads to high level of free estradiol
- Tamoxifen therapy: It is used for breast cancer treatment, it has weak estrogenic action on endometrium
- Lynch 2 syndrome: Hereditary nonpolyposis colon cancer with increased risk of endometrial, breast, and ovarian cancer
- Atypical endometrial hyperplasia can progress to carcinoma in about 29% cases.

Type of hyperplasia	Progression to CA (%)
Simple	1
Complex	3
Simple with atypia	8
Complex with atypia	29

COC, POP, DMPA, MIRENA and PREGNANCY all are protective for CA endometrium

COCs lower the risk of endometrial cancer by about 50%; the effect lasts for up to 15 years.

Pathology

The uterus maybe enlarged due to myohyperplasia, pyometra.

Localized

- Sessile or pedunculated
- Usual site fundus
- Myometrial involvement late.

Diffuse

Spreads through endometrium to invade myometrium may reach serosa.

Microscopic

- Adenocarcinoma is the MC variety of CA endometrium (80%)
- Papillary serous
- Clear cell
- Adenosquamous
- Mucinous
- Adenoacanthoma
- Squamous cell
- Mixed
- **Papillary serous variety and clear cell variety have worst prognosis**
- Among the two, **clear cell variety has poorer prognosis.**

Spread

- Direct: Myometrium, serosa, peritoneal cavity and cervix, tubes, and ovaries
- Lymphatic: Pelvic, paraaortic and rarely inguinal femoral nodes and ovary and tubes and vagina

- Pelvic lymph node involved in 4% cases in stage 1, grade 1 and 2 whereas it is 35–40% in stage 2
- Hematogenous: Lungs, liver, bones and brain.

Clinical Features

- **Postmenopausal bleeding:**
  Approximately 75% of women with endometrial cancer are postmenopausal. The most common symptom is postmenopausal bleeding. Bleeding may vary from slight to heavy and maybe continuous or irregular.
- Perimenopausal/premenopausal polymenorrhagia:
  - 25% of endometrial cancers are in patients who are perimenopausal or premenopausal
  - Heavy frequent menstrual periods or intermenstrual bleeding.
- Offensive watery discharge (pyometra)
- Pain: **Simpson's pain** = colicky pain in patients of CA endometrium.

On Examination

Pallor maybe present.

PS: Cervix is healthy looking, blood or purulent discharge maybe seen coming from os.

PV: The uterus maybe atrophic or normal or enlarged, mobile unless in late stages where it becomes fixed.

Regional lymph nodes and breast to be examined.

Investigations and Diagnosis

The diagnosis of CA endometrium has to be **by histopathological examination of endometrium. (Fractional curettage, D/C, endometrial biopsy).**

- **Fractional curettage is the investigation of choice for patients with postmenopausal bleeding.**
  - To be gently to prevent perforation
  - If pyometra, give antibiotics and delay the procedure by one week to avoid perforation and systemic infection
  - Endocervical curettage
  - UCL with sound
  - Dilatation of os
  - Uterine curettage
  - Specimens of cervical curettage and endometrium to be send separately for histopathology.
- D/C
- Endometrial biopsy using a curette or pipelle can be done as an outpatient procedure
- Hysteroscopically directed biopsy
- Pap smear is not reliable test but can also detect 50–60% of endometrial carcinomas.

Endometrial cancerous cells present in the posterior vaginal fornix can be detected by Pap smear.

- TVS: Findings suggestive are:
  - Thickened endometrium. **(even >5 mm in postmenopausal lady is significant)**
  - Hyperechoic endometrium with irregular outline
  - Increased vascularity on color Doppler
  - Intrauterine fluid.
- Special studies, such as CT scans of the abdomen and pelvis or MRIs are not routinely performed
- Preoperative evaluation includes:
  - CBC
  - FBS
  - PLBS
  - RFT
  - LFT
  - HIV, HBsAg, HCV
  - Chest X-ray
  - ECG.

Treatment

- Main stay of treatment is surgery and postoperative radiotherapy.
- CA endometrium is **surgically staged** and hence all patients **will first require staging laparotomy**.

Stage 1

- Surgery (total abdominal hysterectomy with bilateral salpingo-oophorectomy with lymph node sampling), followed by radiotherapy.

Postoperative Radiotherapy

- Vaginal cuff radiation 6000–7000 cGy for stage 1B
- Pelvis external beam radiation 4500–5000 cGy plus vaginal cuff boost for stage 1C.

Stage 2

- Modified radical hysterectomy, bilateral salpingo-oophorectomy with lymph node dissection, followed by radiotherapy
- Pelvis external beam radiation 4500–5000 cGy plus vaginal cuff boost for stage 2.

Stages 3 and 4

- Debulking surgery followed by radiotherapy
  - Pelvis external beam radiation 4500–5000 cGy plus vaginal cuff boost for stage 3 and whole abdominal radiation for stage 4.

Only patients with stage 1 A, grade 1 and 2 do not require postoperative radiotherapy.

A 2012 review found that for early stage primary endometrioid adenocarcinoma of the endometrium, laparoscopy and laparotomy are associated with similar rates of disease-free and overall survival and that laparoscopy is associated with reduced operative morbidity and shorter hospital stays.

- Chemotherapy and hormonal therapy is used in advanced or recurrent cases or in metastatic lesion
- Cytotoxic agents which can be used singly or in combination are:
  - Cisplatin
  - Carboplatin
  - Cyclophosphamide
  - Paclitaxel
  - Adriamycin.

Hormone treatment for endometrial cancer can include:

- Progestins—which are the main hormone treatment used
- Tamoxifen
- GnRH analogs
- Aromatase inhibitors.

If a tumor is well-differentiated and known to have progesterone and estrogen receptors, progestins maybe used in treatment. About 25% of metastatic endometrioid cancers show a response to progestins.

The main hormone treatment for endometrial cancer uses progesterone. The two most commonly used progestins are medroxyprogesterone acetate which can be given as an injection or as a pill and megestrol acetate. These drugs work by slowing the growth of endometrial cancer cells.

Q. FIGO staging CA endometrium.

FIGO Grading of Endometrial Carcinoma

Histopathologic degree of differentiation:

- G1: ≤ 5% nonsquamous or nonmorular growth pattern
- G2: 6–50% nonsquamous or nonmorular growth pattern
- G3: >50% nonsquamous or nonmorular growth pattern.

Surgical Staging for Endometrial Cancer

Stage	Finding
Ia G1 2 3	No myometrial invasion
Ib G1 2 3	<½ Myometrial invasion
Ic G1 2 3	>½ Myometrial invasion
IIa G 1 2 3	Extension to endocervical glands
IIb G1 2 3	Cervical stromal invasion
IIIa G 1 2 3	Positive uterine serosa, adnexa, and/or peritoneal cytology
IIIb G 1 2 3	Vaginal metastasis
IIIc G 1 2 3	Metastasis to pelvic and/or paraaortic lymph nodes
IVa G 1 2 3	Tumor invasion of bladder and/or bowel mucosa
IVb	Distant metastasis including intra-abdominal and/or inguinal lymph nodes.

Recent Advances

FIGO (2009)	Staging for Ca Endometrium
IA	Tumor confined to the uterus, no or <½ myometrial invasion
IB	Tumor confined to the uterus, >½ myometrial invasion
II	Cervical stromal invasion, but not beyond uterus
IIIA	Tumor invades serosa or adnexa
IIIB	Vaginal and/or parametrial involvement
IIIC1	Pelvic node involvement
IIIC2	Paraaortic involvement
IVA	Unchanged
IVB	Unchanged.

Q. Clinical features of epithelial ovarian CA.

Q. How will manage a case of epithelial ovarian malignancy.

INTRODUCTION

- Ovarian malignancy constitutes about 15–20% of all genital malignancy
- 20% of all ovarian neoplasms are malignant
- **85–90% of all primary ovarian malignancies are epithelial in origin**
- Germ cell tumors constitute 6–10% of all ovarian malignancies and sex cord stromal tumors accounts for 5–7% of all primary ovarian malignancies.

Clinical Features of Epithelial Ovarian Malignancy

Patient Profile

- Epithelian cancer mainly occurs in postmenopausal age group
- The peak incidence is at **55–60 years of age**
- Increase association with nulliparity and family history.

Symptoms

Most women with epithelial ovarian cancer have **no symptoms** for very long periods of time. When symptoms develop they are very **vague and nonspecific.**

- Bloating; abdominal distention, discomfort
- Dyspepsia, flatulence
- Early satiety, loss of appetite
- Indigestion and acid reflux
- Pressure effects on the bladder and rectum (increase frequency to urinate, constipation)
- Abdominal swelling, dull pain and discomfort in lower abdomen
- Shortness of breath, respiratory distress (in advanced cases, due to ascities or pleural effusion)
- Tiredness
- Sudden weight loss
- Dyspareunia
- Irregular menses or postmenopausal bleeding (very rarely).

Signs

General examination:

- Pallor
- Cachexia
- Jaundice
- Left supraclavicular lymph gland (Virchow's node) maybe enlarged
- Lower limb and vulval edema.

Per Abdomen

- Hepatomegaly: Firm and nodular
- Mass in hypogastrium, maybe bilateral with the following characteristics:
  - Solid or heterogenous
  - Restricted mobility
  - Irregular surface
  - Lower pole not reached
  - Dull on percussion
- Ascites.

PV

- The uterus is felt separately from the mass
- **Solid, irregular, fixed, pelvic mass is highly suggestive of ovarian malignancy**
- **Nodules maybe felt through the posterior fornix.**

Investigations

- Serum CA 125: Its not diagnostic and is for prognosis. However, it is useful to differentiate benign and malignant pelvic mass. For postmenopausal patient with adenexal mass a high CA125 levels (>95 IU/ml), there is a 96% positive predictive value for malignancy
- **The diagnosis of ovarian cancer requires exploratory laparotomy**
- **Preoperative evaluation includes:**
  - CBC
  - FBS
  - PLBS
  - RFT
  - LFT
  - HIV, HBsAg, HCV
  - ECG
- Chest X-ray
- IVP
- USG Pelvis and abdomen, limited value, but would detect ascites, involvement of omentum and the other ovary
- CT Scan and MRI: Limited role but will give an idea of extent of disease, lymph nodes, ascites and liver involvement
- **To rule out primary gastric and colon malignancy with ovarian metastasis:**
  - Upper and lower endoscopy

- Barium enema
- Upper GI series
  - Mammography if any breast mass
  - Cervical cytology (Pap smear)
  - Endometrial biopsy, endocervical curettage to exclude uterine or endocervical cancer
  - Fine-needle aspiration (FNA) or percutaneous biopsy of an adnexal mass is **not routinely recommended.** In most cases, this approach may only serve to delay diagnosis and treatment of ovarian cancer. Instead, if a clinical suggestion of ovarian cancer is present, the patient should undergo a laparotomy for diagnosis and staging.

Treatment

- **Surgery is the initial treatment of choice** for ovarian cancer, provided patients are medically fit. Patients who are not fit for surgery maybe given chemotherapy and considered for surgery later. The aim of surgery is to confirm the diagnosis, define the extent of disease, and resect all visible tumor. The role of cytoreduction was demonstrated by Griffiths in 1975.
- **Cytoreductive surgery:** This should be performed by a gynecologic oncologist at the time of initial laparotomy. **The volume of residual disease at the completion of surgery represents one of the most powerful prognostic factors.**
- **Residual disease of less than 1 cm is evidence of optimal cytoreduction,** although the greatest possible effort should be made to remove all obvious disease.
- ***For all stages 1-4: The main treatment consists of staging laparotomy, primary cytoreductive surgery followed by chemotherapy.***
- Basic steps involved in surgical staging: Midline or paramedian incision.
  - Send free fluid for cytology
  - If no free fluid, perform peritoneal washings and send it for cytology
  - Inspect and palpate all the intra-abdominal organs
  - Any suspicious area on peritoneal surfaces should be biopsied
  - Sample the diaphragm either by biopsy or scraping
  - Perform the infracolic omentectomy
  - Evaluate the pelvic and paraaortic lymph nodes. Enlarged nodes should be resected. If no metastasis are present pelvic lymphadenectomy should be performed.
- **Primary cytoreductive surgery:** The ovarian tumor should be removed intact (if possible) and a frozen histologic section should be obtained.
  - Total abdominal hysterectomy
  - Bilateral salpingo-oophorectomy
  - Infracolic omentectomy
  - Lymph node dissection and
  - Removal of all the metastatic deposits (as much as possible). All visible tumor should be removed. This may require extensive surgery, including bowel resection, excision of peritoneal implants, liver resection and splenectomy.

Conservative Surgery (Unilateral salpingo-oophorectomy)

- **Not recommended routinely except in young patients,** desirous of reproduction provided

- The tumor is stage 1A, well differentiated and opposite biopsy normal on frozen section
- The patient to br regularly followed up with TVS pelvis and CA 125 and definitive surgery done in future after family is complete.

Chemotherapy

- Postsurgery chemotherapy improves survival

Given in all cases except: Stage 1A, grade 1: No adjuvant chemotherapy required

- Given for 6 cycles at 3-4 weeks interval
- Platinum compounds (cisplatin, carboplatin) are most effective
- Taxane derivatives (paclitaxel, docetaxel) also very effective
- **Currently chemotherapy with carboplatin and paclitaxel is preferred in most patients as it is found to have better survival rate and better tolerated.**

Immunotherapy

Cytokines, interferon alpha and gamma, and interleukin 2 are being used as second line therapy along with chemotherapy. Use of monoclonal antibodies is under trial.

Interval Cytoreduction (after NACT)

In patients with advance stages (stage 3 and 4) and not fit for surgery (severe ascites and pleural effusion) or inadequate primary cytoreductive surgery.

Patients receive three cycles of neoadjuvant chemotherapy (NACT). Carboplatin and paclitaxel followed by interval cytoreductive surgery which is followed by three more cycles of NACT.

Prevention

Factors Reducing the Risk of Ovarian Cancer

- Use of OC pills/DMPA (since they cause anovulation)
- Breastfeeding
- Multiparity
- Pregnancy

Management Options in High-risk Women: BRCA1/2 Carriers

- Six monthly TVS
- OCPs (when not interested in fertility)
- Prophylactic oophorectomy (as soon as family is completed)
- Annual mammography.

9 Contraception

Q. Lactational amenorrhea as birth control.

INTRODUCTION

The period of lactational amenorrhea can act as method of contraception, though **not a very reliable method** as patient can conceive in this period.

Mechanism of Action (MOA)

Excessive secretion of prolactin, which controls lactation, **inhibits the ovarian function.**

Prolactin inhibits LH but has no effect on FSH. However, it partially inhibits ovarian response to both of these gonadotropins. As a result, while the prolactin level remains high, the ovary produces little estrogen and no progesterone. Hence, it **suppresses ovulation** and hence, ovulation and menstruation are affected.

Failure Rate

It is effective **maximum upto 6 months postpartum** and not reliable beyond that if there is exclusive breastfeeding, the failure rate of lactational amenorrhea method (LAM) (for 6 months only) is less than 2% when correctly and consistently used, but it is more otherwise. If any time during these 6 months the menses resumes, then it is not effective as contraception.

Advantages

The breastfeeding practices required by LAM have other health benefits for mother and baby:

- It provides the healthiest food for the baby.
- It protects the baby from life-threatening diarrhea.
- It protects the baby from diseases such as measles and pneumonia by passing on the mother's immunities to the baby.
- It helps to develop a close relationship between mother and baby.
- It protects the mother from diseases such as subinvolution, fibroadenosis, and fibroadenoma of the uterus.
- Breastfeeding reduces risks of breast cancer and epithelial ovarian cancer.

Q. Condoms.

INTRODUCTION

Male Condoms are contraceptive sheaths meant to cover the penis during coitus to prevent pregnancy. They are also known as French letters.

The condom is the **oldest and most widely used birth control device in the world.** Its invention is attributed to a physician named **Dr Condom,** who recommended it to Charles II.

Characteristics

Condoms are mostly made of **fine latex rubber** and are available in various shapes and colors. They are circular cylinders, 15–20 cm in length, 3–3.5 cm in diameter and 0.003–0.007 cm in thickness; they are closed at one end and open at the other with an integral rim.

Nonlatex forms of male condoms are now commercially made of **polyurethane.** Polyurethane condoms have a longer shelf life and can be used with oil-based lubricants, which can damage latex condoms.

It is most harmless method of contraception.

MOA: Barrier method, they prevent the union of egg and the sperm.

Failure rate: They have got a **very high failure rate.**

Total condom failure rates (breakage and slippage rate combined) range from **4% to 13%.**

Advantages and Noncontraceptive Benefits

- Cheap, easily available.
- No side effects.
- No medical supervision required (as in cases of IUCD and OC Pills).
- When used properly, the condoms give very good **protection against STDs.**
- These include not only traditional syphilis and gonorrhea but also trichomoniasis, moniliasis, nongonococcal urethritis, and infection with chlamydia and herpes virus. The condom seems to give best protection against sexually transmitted AIDS. Condoms also give protection against sexually transmitted hepatitis B virus. Protection against STD benefits male and female partners.
- **When used for more than 5 years, condom, reduce the chance of developing severe cervical dysplasia and cervical cancer as compared to the use of oral pills or to nonuse of contraceptives.**
- Condom catheter in males.
- To cover the TVS probe.
- After vaginoplasty, molds are used to prevent fibrosis, which are covered with condoms.
- **Shivkar's pack (condom tamponade)** for atonic PPH.
- In cases of antisperm antibodies present in cervical mucus.

Disadvantages

- **High failure rates**
- **Coitus dependent and male dependent**
- May slightly decrease the sexual pleasure
- Storage and disposal problems affect village people and reduce use of condoms.

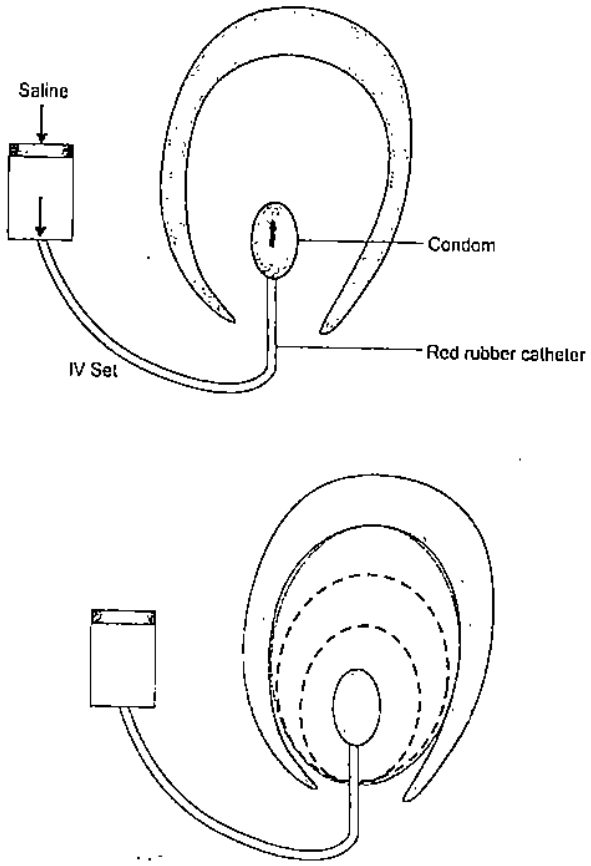


Fig. 9.1: Shivkar's pack

Disposal of Condoms

They should be wrapped in a piece of paper and thrown in dustbins or buried underneath the soil but should never be left in commodes or flushing-type latrine pans.

Female Condom

- A female condom, by the trade names of 'Femidom' or 'Reality,' is a new disposable barrier contraceptive for women. It consists of soft, loose-fitting polyurethane sac about 15 cm long and 7 cm in diameter.
- Sexual intercourse takes place within the cavity of the device.
- It is a women-controlled method and can even be used without the partner's cooperation. It prevents STDs including HIV/AIDS.

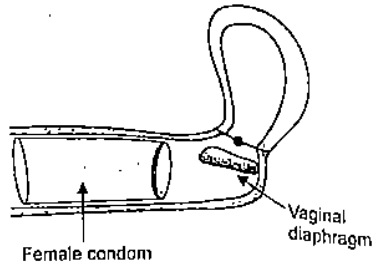


Fig. 9.2: Female condom and diaphragm

Disadvantages

1. Intercourse is noisy, and slippage occurs in about one in 5–10 uses; however, female condom rarely breaks.
2. Occasionally the penis is introduced, by mistake, outside the female condom, which may lead to pregnancy and STDs including HIV.
3. It is an expensive method.

Typical failure rate, as commonly used, is 21%.

Q. Vaginal sponge TODAY. Add a note on spermicidal agents.

Today is a soft, disposable foam sponge made of **polyurethane**. It is round shaped, with a depression at the center of the upper surface designed to fit over the cervix, and is saturated with **nonoxynol-9, the most powerful spermicide**. It has an attached nylon loop that helps in its removal.

MOA

It is **not a barrier method**. It **release spermicidal agent** which kills the sperms. It is moistened with water, squeezed gently to remove excess water and inserted high up in the vagina to cover the cervix. It **acts for 24 hours**, and intercourse may be repeated as often as desired during this period. Like the cervical cap, it can be introduced long before the sex act.

Failure Rate

The failure rate varies **between 9 and 27 per 100 users** in the first year.

It must be removed and thrown away after 8–24 hours **but not before 6 hours** of the las act. The real danger of the sponge is **development of TSS**, although it happens very rarely.

Spermicides

Spermicides are contraceptive chemical agents. They comprise a chemical capable c destroying sperms incorporated into an inert base. The commonly used spermicidal agent contain **nonionic surfactants** that alter sperm surface membrane permeability, causin osmotic changes resulting in the **killing of sperms**. Most spermicides contain nonoxynol-9 which is best for the purpose.

Their main role is to improve the contraceptive effect of other barrier methods. **They are mostly used along with diaphragms, cervical caps, and condoms.**

Spermicidal agents nowadays contain nonoxynol-9. A few products contain **octoxynol-9 and menfegol.**

There is no evidence that spermicides including nonoxynol-9 offer any protection against HIV and other STIs. Furthermore, there is some evidence that frequent use of nonoxynol-9 (twice a day or more) increases, rather than reduces, the chance of HIV transmission, perhaps by irritating the vaginal and cervical mucosa.

Typical average failure rate, as commonly used, is 21%.

Q. Generations of IUCD. Mechanism of action of IUCD and failure rates

INTRODUCTION

The intrauterine device (IUD) is the **second most commonly** used family planning method, after voluntary female sterilization (in India)

The IUD is one of the best methods of contraception during lactation

Generations of IUD:

- **First:** Inert devices, e.g. Lippes loop
- **Second:** All the copper-containing devices: For example, Multiload Cu 250, 375, Cu T 200, 220C, Cu T 380A, etc
- **Third:** Hormonal devices, e.g. Progestasert and Mirena

Mechanism of Action of IUCD

The precise mechanism of action of the IUD is still unknown.

- New studies prove that the IUDs act mostly by preventing sperms from fertilizing ova. The primary mechanisms of action of **copper-releasing IUD are by impeding sperm transport and inhibiting their capacity to fertilize ova.**
- All unmedicated and copper devices **produce an inflammatory or foreign body reaction, which in turn causes cellular and biochemical changes in the endometrium.** Prostaglandin level increase and the fibrinolytic mechanism needed for hemostasis are affected. Numerous polymorphs, giant cells, mononuclear cells, plasma cell, and macrophages appear in the endometrium as well as in the uterine and tubal fluids. These cells engulf or consume sperms and ova by the process of phagocytosis and thus prevent fertilization. Besides, normal cyclical changes in the endometrium may be delayed or deranged by the inflammatory reaction and liberation of prostaglandins, **making it inhospitable for implantation of the blastocyst.**
- When inserted postcoitally, IUDs **can prevent implantation of the fertilized ovum.**
- Copper causes more intense inflammatory reaction and interferes with enzymes in the uterus, the amount of DNA in endometrial cells, glycogen metabolism, and estrogen uptake by the uterine mucosa.
- Sperm motility, capacitation, and survival are also affected by the biochemical changes in the cervical mucus produced by copper.
- IUDs containing progesterone prevent sperm passing through the cervical mucus and maintain high progesterone level, and in consequence, relatively low estrogen levels locally. They, thereby, **keep the endometrium in a state in which implantation is hindered.**

Pearl Index of IUD

IUDs can be divided into three groups according to the pregnancy rate, indicating their contraceptive efficacy:

1. Group I (pregnancy rates greater than 2.0 per 100 women-year): Lippes loop, Cu 7 T 200
2. Group II (pregnancy rates less than 2.0 but more than 1 per 100 women-year): Nova T, ML Cu 250, and Cu T 220C
3. Group III (pregnancy rates less than 1 (mostly less than 0.5) per 100 women-year): Cu T 380A, Cu T 380S, ML Cu 375, and LNG 20

Q. Mirena/LNG IUD/LNG 20/Levonova/LNG IUS.

INTRODUCTION

Mirena is a hormone containing third generation IUCD.

Mirena contains a total of **52 mg levonorgestrel (LNG).** LNG is released into the uterine cavity at a rate of approximately **20 µg/day.**

These devices act mainly by local progestogenic effects and act for up to **5 years.**

Failure Rate

Pearl index after 5 years is **0.09/100 women-years (most effective reversible contraception available today).**

MOA

- Mirena **releases progesterone** in the uterine cavity and also has a **foreign body reaction in the uterus, it alters the endometrium and thus, makes it unsuitable for implantation**
- It also makes the **cervical mucus thick** and hence prevents the sperms from entering the uterus.
- It may also affect the sperm capacitation.
- Mirena eventually leads to **progesterone induced amenorrhea.**

Advantages and Noncontraceptive Benefits

Health benefits of mirena include:

- **Reduction of blood loss**, which benefits patients with anemia and dysfunctional uterine bleeding
- Reduction of pain and dysmenorrhea in **endometriosis and adenomyosis**
- Beneficial effect on fibroids
- The advantage that IUDs introduced 6 weeks after delivery do not influence lactation or affect infant growth and development
- Can be used in **prevention and treatment of endometrial hyperplasia**
- Decreases the risk of endometrial cancer
- Decreases the risk of PID and hence **protects against ectopic pregnancy.**

Drawbacks

- Irregular bleeding and oligomenorrhea, which happen quite commonly in the first 3–4 months

- Amenorrhea, which affects up to 20–50% cases by 1 year. But this is not at all harmful as it is a progesterone-induced amenorrhea.
- Difficulty of introduction, needing local anesthesia in many cases.

Q. Contraindications for use of IUD. Complications of IUCD.

INTRODUCTION

The intrauterine device (IUD) is the **second most commonly** used family planning method, after voluntary female sterilization (in India).
Contraindications for IUD use are absolute and relative.

Absolute contraindications include:

- Immediate postseptic abortion
- Pregnancy
- Pelvic tuberculosis
- Vaginal bleeding suspicious/unexplained
- Current pelvic inflammatory disease (PID)
- Puerperal sepsis
- Malignant trophoblast disease
- Current STDs
- Cervical cancer
- Uterine fibroids with distortion of uterine cavity
- Endometrial cancer
- Uterine anomalies like bicornuate, septate uterus.

Nulliparity, heart disease, fibroids with no cavity distortion and past history of PID are ***relative contraindications.***

Complications of IUD

1. **Menorrhagia and dysmenorrhea:**
   Increased bleeding is the greatest disadvantage of IUDs and, along with **pain**, accounts for their removal in 2–10 per 100 users in the first year.
2. **Misplaced IUD:** If the device is detected inside the peritoneal cavity, it should be removed as early as possible. Copper devices produce irritative reactions, inflammations, and a lot of adhesions. Copper devices in the peritoneal cavity usually need laparotomy for their removal, as they produce a good amount of adhesions, although it is possible to remove them by laparoscopy. Perforation occurs rarely, not more than **1.2 per 1000 insertions.**
   - The device may migrate into the peritoneal cavity or become embedded in the uterine musculature. Most perforations occur at the time when faulty insertion technique is followed.
   - The copper T devices are known to produce omental masses and adhesions, and progesterone devices can cause intraperitoneal bleeding and should **always be removed** urgently with laparoscopy (preferred) or laparotomy.
3. **Infections:** Doxycycline 200 mg or, better still, azithromycin 500 mg, administered orally 1 hour before insertion, reduces chance of infection.
   - **The presence of actinomyces has been found to increase with duration of use, especially after use of inert-tailed devices.**

- The infection in IUD users can be prevented by (a) proper selection of patients, excluding those cases who have active infection or are likely to have infection from the husband or other partners, (b) prophylactic antibiotic course, and (c) proper disinfection and the practice of aseptic techniques.

4. **Pregnancy:** As soon as pregnancy is confirmed, the IUD should be removed, if it can be done easily, to **reduce the risk of pelvic infection and miscarriage—the most frequent complication of pregnancy with an IUD in place.**
   If the IUD cannot be removed easily, it can be left in situ.
   There is **no risk at all of any congenital malformations** if IUD is left in situ.
5. **Ectopic pregnancy:** Several studies, including a WHO multicenter study, have found that **IUD users are 50% less likely to have ectopic pregnancy than women using no contraception.** The chance of ectopic pregnancy in IUD users is rare and varies form 0.25 to 1.5 per 1000 women-year. However, when pregnancy occurs, the chance of ectopic pregnancy is higher (about 30%) than in general population (about 0.5–0.8%) of all pregnancies.
6. Spontaneous Expulsion (5%): Usually in the first few months, more commonly during periods. More likely to occur in postabortal and puerperal insertions.
   Immediate (postinsertion) complications include:
   1. Cramp like pain: Transient. Can last for few hours. Relieved by analgesics and antispasmodics
   2. Syncopal attack (rare)
   3. Partial or complete perforation: Due to faulty technique and more likely in lactational period (soft uterus).

Q. Mechanism of action of oral contraceptive pills. What are the noncontraceptive benefits of OC pills?

INTRODUCTION

OC pills contain both estrogen **(ethinyl estradiol)** and progesterone **(e.g.** levonorgestrel/desogestrel/drospirenone/cyproterone).
They are highly effective method of birth control.

- **MOA:**
  Inhibition of ovulation: The combined pills inhibit ovulation by suppressing hypothalamic-releasing factors, which in turn leads to inappropriate secretion of FSH and LH. As a result, no LH surge occurs and **ovulation is suppressed.**
- **Alteration of endometrium:** OCs alter maturation of the endometrium, rendering it unsuitable for implantation of the fertilized ovum.
- **Changes in cervical mucus:** Cervical mucus becomes scanty, viscous, and cellular with low spinnbarkeit and no fernıng; these changes impair sperm transport and penetration.
- **Pearl Index:** Combined pills are very effective. The failure rate when correctly and consistently used is only **0.1%** or 1 per 1000 in the first year of use, but the typical failure rate, as is commonly used is 1.8%.
- The failures are mostly due to missed pills, delay in starting the next course, and stoppage of the drug due to side effect or fear complex without taking other contraceptive measures

Noncontraceptive benefits of OC pills.

- **Cure of menstrual disorders:** OCs cure dysmenorrhea and ovulation pain. Menorrhagia and metrorrhagia can always be controlled by the use of COCs. They also make the cycle regular.

- **Protection against cancer:** It has been conclusively proved that OCs directly prevent two common types of genital cancer: Endometrial cancer and ovarian cancer; it also indirectly prevents choriocarcinoma by preventing pregnancy.

  COCs decrease the ovarian cancer by about 40% and the effect persists for at least 10 years. COCs also lower the risk of endometrial cancer by about 50%; the effect lasts for up to 15 years.

  They also decrease the risk of colon cancer.
- Protection against benign tumors and related diseases:
  - ***Benign breast diseases (BBDs):*** It is well documented that BBDs, such as fibrocystic and fibroadenomatosis diseases, are reduced by 50–70% in pill users.
  - ***Ovarian functional cysts:*** Various studies have shown that low-dose OCs lower the risk of developing functional ovarian cysts. The risk of follicular cysts goes down by 50% and that of corpus luteum cysts by about 80%.
  - ***Fibromyoma of the uterus:*** The risk of uterine fibroid is reduced by about 30% in women who have used OCs for 10 years. Low-dose OCs help reduce fibroids and lessen menstrual flow.
- **Protection against:**
- **Ectopic pregnancy:** Chance of ectopic pregnancy with its grave consequences is lowered by 50% in low-dose OC users.
- **Pelvic inflammatory diseases:** Several studies have shown that regular pill users are protected from PIDs to the extent of 50%. OCs reduce PIDs by hindering the ascent of STD bacteria (including chlamydia) from the vagina upward by thickening the cervical mucus and lessening uterine motility, as well as by obviating illegal abortions and delivery of unwanted children.

  However, barrier contraceptives protect women better against STDs and HIV/AIDS than OCs do.
- **Anemia and malnutrition:** Pills reduce iron deficiency anemia by reducing menstrual flow in 60–80% of pill users; they improve nutrition of women by preventing repeated and frequent pregnancies.
- **Endometriosis:** Combined high-dose pills control endometriosis to a good extent when used continuously with increasing doses to produce pseudopregnancy.
- **Acne and hirsutism:** OCs are effective in treating acne and hirsutism by increasing sex-hormone-binding globulin and significantly decreasing free testosterone levels. Formulations with desogestrel, DRSP and cyproterone are specially effective in this respect.
- **Premenstrual syndrome**

  OCs and pills containing DRSP reduce premenstrual syndrome.
- **PCOS:** OC Pills are highly effective as they regularize the cycles and suppress acne and hirsutism. (OC pills containing cyproterone preferred).

Q. Side effects/risks of OC pills.

INTRODUCTION: AS IN PREVIOUS ANSWER

Important Side Effects/Risks of OC Pills

Breakthrough Bleeding

- This is slightly more common with the lowerdose pills. The women should have two pills a day for $\overline{2}$ or 3 days, which usually controls BTB; if not, EE 0.02 mg may be taken for 7 days along with the pills

- Hypomenorrhea happens sometimes with low-dose pills. The women should be reassured that is not harmful but rather good for health. But if they are not convinced, EE 0.02 mg may be added in the last 7 days for a few cycles
- Amenorrhea is usually temporary and not harmful
- Change to triphasic pills or supplementation with EE for two to three cycles usually cures amenorrhea.

Stroke and Myocardial Infarction

- Women who do not smoke, have their blood pressure checked, and do not have hypertension or diabetes are at **no increased risk** of myocardial infarction if they use low-dose COCs, irrespective of their age and duration of OC use
- The risk of hemorrhagic stroke does not increase in women below 35 years of age who do not smoke and are not hypertensive
- Current users of low-dose COCs have a low absolute risk of VTE mainly because incidence of VTE is very low in nonpregnant women
- Nevertheless, this risk is three to six times more than nonusers. The absolute risk of VTE attributable to OC use rises with increasing age, recent surgery, and some forms of thrombophilia
- Progestogens are associated with the increase of low-density lipoprotein cholesterol and a decrease of high-density cholesterol, which enhance the risk of atherosclerosis, coronary heart disease and cerebral thrombosis; but estrogens have the opposite effect, and these actions seem relatively balanced in low-dose COCs.

Breast and Cervical Cancer

- There is a small increase in risk of current users of the pill **(relative risk 1.24)**, and the risk reduces gradually over the 10 years after discontinuing use. Breast cancer in current or past OC users is largely localized in the breast—a condition that usually has a better prognosis
- The risk of breast cancer is due to the progestogen component of the pills, as the risk is same among users of progestogen-only methods
- Studies in developed and developing countries have shown a modest increase in the risk of cervical cancer (1.3–1.8-fold) among women who have used COCs for more than 5 years. However, it is not clear whether the increased risk is due to direct effect of the pill or some characteristics of the pills' users such as age at first intercourse, number of sexual partners, parity, and smoking status.

Liver Tumor

- OCs increase the incidence of a rare benign liver tumor, namely, primary hepatocellular adenoma

  Minor side effects like nausea, vomiting may happen initially.

  Other minor side effects include: mastalgia,weight gain and chlosma.

Q. Contraindications for combined OC pills.

INTRODUCTION: AS IN PREVIOUS ANSWER

Absolute contraindications include:

- Active liver disease (hepatitis/tumor)
- Postpartum: Breastfeeding

- Thrombophilias
- Ischemic heart disease
- Complicated migraine
- Pregnancy
- Complicated valvular heart disease
- Breast cancer (current or past history)
- Severe hypertension (systolic > 160 or diastolic >100)
- DM with vascular complications
- Current history of thromboembolism/stroke/deep vein thrombosis.

Smoking, age more than 35 years, mild hypertension and uncomplicated DM are **relative contraindications.**

Q. Injectable contraceptives.

Progestogen—only injectable contraceptives include:

- Depot-medroxyprogesterone acetate (DMPA)
- Norethisterone enanthate (NET EN or Noristerat).

One injection of Depo-Provera remains effective for **3 months**. It is administered in the form of a **150 mg injection** once every 3 months plus or minus 14 days.

One **200 mg NET EN** injection is to be taken **every 2 months**.

Both DMPA and NET EN are highly effective methods of contraception especially in lactating mothers where estrogen is containdicated.

Pearl Index

Typical failure rate of progestogen—only injectables, as commonly used, is **0.1–0.4%**.

Mechanism of Action

The injectable contraceptives act by **inhibiting ovulation** in most women. They also work by making **cervical mucus thick** and scanty, thus creating a barrier to sperm penetration, and making the **endometrium less suitable for implantation**.

Noncontraceptive Benefits

- It cures menstrual troubles like **menorrhagia and dysmenorrhea**
- Medical management of **endometriosis** (pseudopregnancy regimen)
- Prevention and treatment of endometrial hyperplasia
- **DMPA prevents sickling** and the development of abnormal-shaped red blood cells, and lessens episodic bone pain in women suffering from sickle cell diseases; it is thought to be the **best contraceptive for patients of sickle cell anemia**
- DMPA reduces the risk of pelvic inflammatory disease and ectopic pregnancy
- DMPA use **protects against the risk of endometrial and ovarian cancer**
- Injectables are suitable in cases with myoma and endometriosis, as contraception is provided without estrogen effect.

Side Effects

- Irregular menstrual bleeding and spotting, as well as temporary amenorrhea, are the most common side effects in DMPA and NET EN users
- **Weight gain:** The average weight gain is 1–3 kg in most cases
- There is a delay of few months in becoming pregnant following discontinuation of the injection
- **Bone density changes:** There is a risk of bone loss among long-term DMPA users leading to osteoporosis; however, this bone loss is reversible on cessation of the contraception.

Combined (estrogen + progesterone) monthly injectable contraceptives (not yet available in India):

- DMPA 25 mg plus estradiol cypionate 5 mg marketed as Cyclofem
- NET EN 50 mg plus estradiol valerate 5 mg marketed as Mesigna.

Q. Emergency contraception (Interceptives).

INTRODUCTION

Agents that do not interfere with fertilization but act on the endometrium to **prevent implantation are called interceptive agents.**

Indications

- Unplanned, unprotected intercourse
- After rape
- Rupture or tear in the condom at the time of intercourse.

Methods

Two methods of emergency contraception are available now: (1) hormonal and (2) mechanical (IUD).

There are two types of **hormonal** emergency contraception (emergency window = 72 hours)

- **LNG—only pills** (most commonly used)
- One tablet of 0.75 mg LNG pill should be taken as soon as possible after unprotected intercourse, followed by a same dose taken 12 hours later; both doses must be taken within 72 hours of intercourse
- Single 1.5 mg dose of LNG is as effective for emergency contraception as two 0.75 mg doses of LNG taken 12 hours apart.
  Failure rate (pregnancy rate) = **0–1%.**
- **Combined estrogen and progestogen pills (also known as the Yuzpe Regimen)**
- High-dose pills contain 50 µg of EE and 250 µg LNG (or 500 µg norgestrel)
- Two pills should be taken as soon as possible, but not later than 72 hours of unprotected coitus; this must be followed by two other pills 12 hours later
- When only low-dose pills containing 30 µg of EE and 150 µg of LNG (300 µg of norgestrel) are available, four pills should be taken as the first dose within 72-hours of unprotected intercourse, followed by four more pills after 12 hours.
  Main side effect is nausea and vomiting.
  Failure rate = **0–2%.**

Mechanical

IUDs introduced postcoitally can prevent pregnancy very successfully. (Failure rate = **0.1%**).

IUDs can be used postcoitally **up to 5 days following sexual exposure.**

Thus, this method can be used even after 48 hours more delay than the hormonal methods allow.

Mechanism of Action of EC

They may act through

- Inhibition or delay of ovulation
- Prevention of implantation in the altered endometrium (interception = main action), and
- Prevention of fertilization due to quick transport of sperms or ova
- **They cannot interrupt already established pregnancy (cannot cause an abortion).**

Q. *Indications of MTP.* Add a note on medical method of abortion in first trimester. Complications of surgical evacuation.

As per India's abortion laws, only qualified doctors under stipulated conditions can perform abortion on a woman in an approved clinic or hospital. The Indian abortion laws fall under the Medical Termination of Pregnancy (MTP) Act, which was enacted by the Indian Parliament in the year 1971. The MTP Act came into effect from April 1, 1972 and was once amended in 1975. The Medical Termination of Pregnancy (MTP) Act of India clearly states the conditions under which a pregnancy can be ended or aborted, the persons who are qualified to conduct the abortion:

- Pregnancy would involve serious risk of life or grave injury to physical and/or mental health of the pregnant woman **(therapeutic grounds)**
  Examples: Certain heart diseases like pulmonary hypertension (Eisenmenger's syndrome), grade 3/4 lesions, malignant hypertension, psychiatric illness
- There is substantial risk of child being born with serious physical and mental abnormalities so as to be handicapped in life **(Eugenic grounds)**
  Examples: Chromosomal abnormalities like trisomy 21,18,13, etc. congenital anomalies not compatible with life like anencephaly, complex heart diseases, bilateral renal agenesis, etc
- When pregnancy is caused by rape
- Pregnancy caused as result of failure of contraception.

Only the consent of the woman is required. The consent of male partner is not needed.

MTP in unmarried girls under the age of eighteen requires the consent of a guardian.

MTP in lunatics requires the consent of a guardian.

The length of the pregnancy **must not exceed twenty weeks** in order to qualify for an abortion.

Methods of First Trimester MTP

First Trimester MTP (Upto 12 weeks gestation)

Medical | Surgical

Medical

- It is now officially allowed upto **9 weeks of gestation**
- **Drugs used: Mifepristone followed by misoprostol**
- Mifepristone (RU486) is a progesterone antagonist
- It acts preferentially on target cells of the endometrium and deciduas, counteracting the effect of progesterone, which is essential for establishment and maintenance of pregnancy.
  - It affects the pituitary gonadotropic cells, producing a remarkable decrease of LH secretion, leading to luteolysis
  - It causes softening and ripening of the cervix and produces increased contractibility of the myometrium
  - It causes a marked increase in sensitivity of the uterus to exogenous PGs.

Dose

- Earlier 600 mg orally was used but now recent studies have shown that 200 mg is as effective, so **200 mg is used**. Patient takes this orally on day 1
- This is followed by **misoprostol tablets (PG E1 analogue) 400 microgram orally or 800 microgram vaginally 48 hours later** (day-3)
  Success rate is 96–98%
- **Follow-up visit and USG is required after 14 days to rule out retained products of conception.**
- If there are retained products a check curettage is necessary.

Contraindications due to medical reasons:

- Smoking with age 35 years
- Anemia – hemoglobin < 8 gm %
- **Suspected /confirmed ectopic pregnancy**/undiagnosed adnexal mass
- **Coagulopathy** or women on anticoagulant therapy
- Chronic **adrenal failure** or current use of systemic corticosteroids
- Uncontrolled hypertension with BP >160/100 mmhg
- Cardiovascular diseases such as angina, valvular disease, arrhythmia
- **Severe renal, liver or respiratory diseases**
- Glaucoma
- Uncontrolled seizure disorder
- Allergy or intolerance to mifepristone/misoprostol or other prostaglandins
- **Lack of access to 24-hours emergency services.**

Surgical

- Suction evacuation or manual vacuum aspiration
- Can be done upto 12 weeks of gestation.

Complications of suction evacuation

- **Uterine hemorrhage:** It occurs in 1–4% cases
- **Pelvic infection:** It ranges from 0.1% to 1.5%. It is due to incomplete evacuation and improper aseptic technique. The incidence can be reduced to a great extent by prophylactic use of antibiotic

- **Cervical injury:** This complication occurs in 0.01–1% cases
- **Uterine perforation:** This is the most dangerous complication, but fortunately it happens very rarely in 0.1–0.28% cases
  When perforation occurs or is suspected, the patient should be kept under observation and antibiotic should be started. Usually she can be discharged in 24 hours time. If there is strong suspicion or actual diagnosis of injury to the intestines or omentum, or if hemorrhage occurs, laparotomy should be performed followed by necessary steps
- **Retained products:** Incomplete abortion happens in 24% cases
- **Continuation of pregnancy:** In about 1% cases
- Delayed complications:
  - Cerivcal incompetence
  - Uterine synechiae.

10 Miscellaneous

Q. Dermoid cyst.

DERMOID CYST

- It is the MC benign germ cell tumor of the ovary. It is also called mature cystic teratoma
- Teratomas are germ cell tumors commonly composed of multiple cell types derived from **one or more of the 3 germ layers**. The word is derived from the Greek teras, meaning monster, which Virchow coined. Additionally, teratomas may be monodermal and highly specialized
- Mature cystic teratomas account for 10–20% of all ovarian neoplasms. They are the most common ovarian germ cell tumor and also the most common ovarian neoplasm in patients younger than 20 years. They are bilateral in 15–20% of cases
- The cyst is moderate in size. The capsule is tense and smooth
- On cut section there is one area of solid projection called **Rokitansky protuberance**, which is covered with skin with sweat and sebaceous glands. It is here that teeth and bones are found
- The predominant content of cyst are hair and sebaceous material. There may be clear CSF also
- There may be an area of thyroid tissue-struma ovarii, which maybe a/w hyperthyroidism.

Clinical Features

- Uncomplicated ovarian dermoids tend to be **asymptomatic** and are often discovered incidentally
- They do however predispose to **ovarian torsion**, and may then present with acute pelvic pain
- Lump in the lower abdomen
- Dull aching pain/heaviness in lower abdomen
- They are generally moderate in size and not felt per abdomen.

On pelvic examination:

- The uterus is felt separate from the mass
- A groove is felt between the mass and the uterus
- The lower pole of the cyst is felt through the fornix
- The dermoid cysts because of its fat content may float and may be felt in the anterior fornix **(Krustner's sign)**.

Investigations

Radiographic features:

Plain Film X-Ray

May show calcific and tooth components with the pelvis.

Pelvic Ultrasound

Ultrasound is the preferred imaging modality. Typically an ovarian dermoid is seen as a cystic adnexal mass with some mural components. Most lesions are **unilocular.**

The spectrum of sonographic features includes:
- **Rokitansky nodule** - Dermoid plug
- Diffusely or partially echogenic mass with posterior sound attenuation owing to sebaceous material and hair within the cyst cavity (echogenic interface at edge of mass that obscures deep structures): **The tip of the iceberg sign**
- Echogenic, shadowing calcific or dental components
- Presence of fluid-Fluid levels
- Multiple thin, echogenic bands caused by hair in the cyst cavity: **the dot-dash pattern.**

CT

CT has high sensitivity in the diagnosis of cystic teratomas though is not routinely recommended for this purpose.

Typically CT images demonstrate fat (areas with very low Hounsfield values), fat fluid level, calcification (sometimes tooth), Rokitansky protuberance and tufts of hair. The presence of most of the above tissues is diagnostic of ovarian cystic teratomas in 98% of cases. Whenever the size exceeds 10cms or soft tissue plugs and cauliflower appearance with irregular borders is seen, malignant transformation should be suspected.

Pelvic MRI

MR evaluation usually tends tend to be reserved for difficult cases, but is exquisitely sensitive to fat components. Enhancement is also able identify solid invasive components, and as such can be used to accurately locally stage malignant variants.

Complications

- Torsion is the most common (15-20%) cases
- Rupture is rare (1%)
- Rarely, within some mature teratomas certain elements (most commonly squamous components) undergo malignant transformation (1-2%).

Treatment and Prognosis

They are slow growing (1-2 mm a year) and therefore some advocate non surgical management.

Larger lesions are often surgically removed. Many recommend initial serial follow for lesions under 7 cm to monitor growth, beyond which a resection is advised.

Ovarian cystectomy leaving behind the healthy ovarian tissue is the operation of choice.

If the tumor is very big then oophrectomy may be required.

Laparotomy or laparoscopy can be done.

Laparoscopy is preferred nowadays.

Q. Mention complications of benign ovarian cysts and clinical features and management of torsion of ovarian tumor.

Q. DD and Management of ovarian torsion.

Complications of Benign Ovarian Cysts

1. Torsion
2. Infection
3. Rupture
4. Intracystic hemorrhage
5. Pseudomyxoma perotonei
6. Malignancy.

The torsion is more common in tumors with:
- Moderate size (too big and too small tumors will not undergo torsion)
- Moderate weight
- Free mobility
- Long pedicle.

Dermoid cyst has the maximum risk of torsion among all ovarian tumors.

PREDISPOSING FACTORS FOR TORSION

- Trauma
- Coitus
- Heavy physical exercises

Two groups of women show a particular tendency to be affected by ovarian torsion:
1. Women in their mid 20s and
2. Women who are postmenopausal.

Approximately 20% of cases of torsion occur during pregnancy.

Clinical Features

- Classically, patients present with the **sudden onset** (commonly during exercise or other agitating movement) of **severe, unilateral lower abdominal pain** that worsens intermittently over many hours
- A minority of patients, however, complain of mild pain that follows a more prolonged time course
- The pain usually is localized over the involved side, often radiating to the back, pelvis, or thigh. Approximately 25% of patients experience bilateral lower quadrant pain. It may be described as sharp and stabbing or, less frequently, crampy

- The patient may also complain of lump in the abdomen
- The lump may also be present before the pain
- Nausea and vomiting occur in approximately 70% of patients
- A history of previous episodes may be elicited, possibly attributable to partial, spontaneously resolving torsion. Fever may occur as a late finding as the ovary becomes necrotic.

Physical Examination

- General condition usually remains unaffected except the patient is in agony and pain
- The physical examination, like the history, is typically nonspecific and is highly variable
- A unilateral, tender, tense cystic mass with restricted mobility, in the hypogastrium and arising from pelvis
- However, the absence of such a finding does not exclude the diagnosis and the absence of tenderness cannot be used to rule out torsion
- Pelvic examination reveals the mass felt separate from the uterus
- Peritoneal findings are infrequent and indicate advanced disease if present.

Differential Diagnoses

1. Appendicitis
2. Diverticulitis
3. Ruptured ectopic pregnancy
4. Endometriosis
5. Torsion of subserous pedunculated fibroid
6. Bowel obstruction
7. Mesenteric ischemia
8. Nephrolithiasis
9. Pelvic inflammatory disease
10. Urinary tract infection.

Fate

- A partial torsion may untwist spontaneously
- In complete torsion there is obstruction of both venous and arterial system leading to venous congestion and extravasation of blood inside the cyst
- The cyst may rupture or become gangrenous and the omentum or intestine may get adhered to it.

Investigations

Ultrasonography and Color Doppler is the investigation of choice and should be the first examination performed.

- Typically, the affected ovary is enlarged
- It can show morphologic changes in the ovary and can help in determining whether blood flow is impaired
- Normal Doppler imaging must not, however, be used as a basis for excluding the diagnosis.
- Color Doppler sonography may be helpful in predicting viability of adnexal structures by depicting blood flow within the twisted vascular pedicle and presence of central venous flow.

Rarely, computed tomography (CT) or magnetic resonance imaging (MRI) is needed to make a definitive diagnosis.

CT or MRI can serve as a secondary modality when ultrasonographic findings are non-diagnostic.

Treatment

- Outpatient care has no role in the treatment of ovarian torsion. Patient should be admitted
- Pain medication may be given to a patient who presents with abdominal pain
- The use of nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids is acceptable
- Surgery: Laparotomy or laparoscopy. Laparoscopy is preferred
- Patients with either a suspected or confirmed diagnosis of ovarian torsion should be admitted. Laparoscopy can be used for both confirmation of the diagnosis and treatment
- The exact surgery depends on the viability of the tissue
- **Detorsion to be tried first.** If the ovaries and tubes look healthy then the cystectomy can be performed
- If there is necrosis then oophorectomy or salpingoohprectomy must be done.

Q. Cervical erosion (Ectopy).

DEFINITION

Normally, the endocervical canal is lined with columar epithelium and the ecto cervix with squamous epithelium. These connect at the squamo-columnar junction. **In cervical erosion, the columnar epithelium may extend further down and protrude on the ecto cervix.** It may also undergo squamous metaplasia and transform to stratified squamous epithelium.

Causes of Cervical Erosion

Excess Estrogen level: Cervical erosion is believed to be a response to high levels of circulating estrogen in the body.

1. *In pregnancy:* Cervical Erosion is a very common finding during pregnancy
   - It can cause mild bleeding during pregnancy, usually during sexual intercourse when the penis touches the cervix. The erosion disappears spontaneously 3-6 months after childbirth
2. *In women on birth control pills:* All birth control pills contain the hormones, estrogen and progesterone in different strengths
3. *At birth and puberty:* Cervical erosion is found in at least 1/3rd of all female babies. It is a response to the maternal estrogen that the babies are subjected to while still in the uterus. The erosion disappears as the influence decreases in few days but can reappear at puberty
4. *In women on hormone replacement therapy (HRT):* HRT in menopause mainly consists of replacement of estrogen in the body through pills, patches, creams, etc. This estrogen can cause cervical erosion.

Infections

The theory that infection is the cause of cervical erosion is discarded. Infection does not cause cervical erosion but it is rather the other way around—the changed cells of cervical erosion are more susceptible to various bacteria and fungi and tends to get infected very easily.

Miscellaneous Causes

It is believed by many that chronic infection of the vagina, vaginal douching and chemical contraceptions like anti-sperm gels can change the normal level of acidity of the vagina and cause cervical erosion. But these theories are yet to be proved.

Pathogenesis

The columnar epithelium of endo cervix replaces the squamous epithelium.

Usually single layer (flat type).

Hyperplastic and folds inwards → follicular erosion.

Folds inwards and outwards → papillary erosion.

During healing the SCJ moves towards the external os:
- The squamous epithelium grows beneath the columnar epithelium which gradually disintegrates
- Squamous metaplasia of columnar cells.

Symptoms of Cervical Erosion

- Asymptomatic.
- *Increased vaginal discharge:* The vaginal discharge is usually copious, clear or cloudy and slippery to the touch. If infections occur, there may be pus cells making the discharge mucopurulent. Infections can also cause the vaginal discharge to have a foul smell.
- *Bleeding:* Contact with the columnar cells of cervical erosion can cause the fragile tissue to break causing bleeding. This is usually seen after sexual intercourse or even after passing hard stool.
- *Other symptoms of cervical erosion:* Many symptoms like backache, chronic ill health and even infertility have been said to be due to cervical erosion. But it is more likely that these are the symptoms of chronic pelvic infection which may be the result or cause of cervical erosion.

Signs

On examination:
- The area of cervical erosion is seen as a **bright, red surface** surrounding and beyond the external os. It extends inside the cervix
- The margin is well defined and the whole area may be smeared with cervical discharge
- It feels soft but a little granular to the touch of the examining finger
- It can bleed a little during examination. It can look like and be mistaken for cervical cancer.

DD

- Ectopion
- Early cancer
- Primary sore of syphilis
- Tubercular ulcer.

Management

- PAP smear (cytology) to exclude dysplasia and malignancy
- In doubtful cases,a cervical biopsy should be done.

Treatment of Cervical Erosion

- During pregnancy the treatment should be withheld till 12 weeks post partum
- If the cervical erosion has no symptoms but have been discovered on routine examination, treatment is not necessary
- If symptoms are present however, active treatment becomes necessary. The aim of the treatment of cervical erosion is to destroy the columnar cells so that normal squamous cells can grow in their place:
  - *Electrocautery:* The cells are burned off by using heat generated by electric current
  - *Diathermy:* High temperatures are applied to the area of cervical erosion so that the cells are damaged
  - *Cryocautery:* Extreme cold generated by the application of nitrous oxide gas is used to destroy the cells
  - Laser vaporization.

Infections

Infections should also be controlled by antibiotics.

Post-operative advice: The area of cervical erosion takes 6-8 weeks to heal. So the patient is asked to avoid sex or use tampons during this period to avoid any injury.

Q. Define menopause. What are the various symptoms? Add a note on HRT.

Menopause

Menopause is defined as the permanent cessation of menses and is physiologically correlated with the decline in estrogen secretion resulting from the permanent loss of follicular/ovarian function.

The clinical diagnosis is confirmed following **stoppage of menses for 1 year** without any other pathology. So the woman is declared to have attained menopause only retrospectively.
- The time of menopause is determined genetically and occurs at the median age of 51 years in West and 47 years in India, range being 45-55 years
- **Premature ovarian failure is defined as menopause occurring spontaneously before 40 years of age.**

Delayed menopause is when the menopause occurs after the age of 55 years.

Menopausal Symptoms

1. Hot flushes/vasomotor symptoms
   - The classic symptom associated with *estrogen deficiency is the hot flash, also known as hot flush*

- This symptom is described as 'recurrent, transient periods of flushing, sweating and a sensation of heat, often accompanied by palpitation, feeling of anxiety and sometimes followed by chills'
- The entire episode usually lasts no more than 1-3 minutes and may recur as many as 30 times per day, although 5-10 times per day is probably more common
- Hot flashes are experienced by at least half of all women during natural menopause and even more women after surgical menopause
- In severe cases, hot flashes may be accompanied by fatigue, nervousness, anxiety, irritability, depression, and memory loss. These sensations, if occurring at night, are called as 'night sweats' and can lead to interruption of sleep patterns
- Physiologically, *hot flashes correspond to marked, episodic increase in the frequency and intensity of gonadotropin-releasing hormone (GnRH) pulses* from the hypothalamus and not due to increased GnRH secretion

2. *Urogenital atrophy:* Vaginal dryness, dyspareunia, recurrent vulvovaginal infections, and urinary tract infections and SUI
3. Mood swings, irritability, and depression, insomnia
4. Decreased libido
5. Memory loss/cognitive decline
6. *Osteoporosis:* Defined as the reduction in the quantity of bone, leading to enhanced susceptibility to fractures. Bones associated with postmenopausal fractures:
   - Spinal vertebra
   - Radius
   - Neck of femur.

HORMONE REPLACEMENT THERAPY

Based on the results of **Women's Health Initiative (WHI) trial**, the following are now the accepted indications for HRT:

- Menopausal symptoms such as hot flushes, vaginal dryness, mood swings, irritability, etc
- Prevention and treatment of osteoporosis
- Decreased libido.

HRT is not given for primary prevention of heart disease.

The current recommendation is to use HRT at the **lowest effective dose for a short period of time.**

The different hormones used are:

- **Estrogen (E) and progesterone (P) combination:**

The principle hormone used/needed is estrogen

- As unopposed estrogen is a risk factor for endometrial hyperplasia and cancer; in women with ***intact uterus both E + P should be given.***

In hysterectomized women, only E can be given.

- The most commonly prescribed oral estrogen is conjugated equine estrogen (CEE). The dose used generally 0.625-1.25 mg/day. Low dose 0.3 mg can also be used.

The most common progestin is medroxyprogesterone acetate (MPA) 2.5-5 mg/day. Micronized progesterone 100-200 mg/day or dydrogesterone 5-10 mg/day can also be used.

LNG-IUS (MIRENA) can also be inserted. It has no systemic side effects of progesterone

- Testosterone:
  - The most common indication for androgens is loss of libido.

Testosterone by peripheral conversion to estrogen will also relieve the hot flushes.

- Tibolone: (1.25-2.5 mg/day)
  - It is considered as designer HRT. It is a selective tissue estrogen activity regulator **(STEAR)**
  - It has estrogenic, progestogenic, and androgenic properties.
- Selective estrogen receptor modulators:
  - Raloxifene is a selective estrogen receptor modulator (SERM), which binds with higher affinity to estrogen alpha receptor than the beta receptors
  - Clinically raloxifene produces an effect similar to estrogen on skeletal and cardiovascular system, while behaving as an estrogen antagonist in the uterus and breast
  - Raloxifene maintains a favorable lipid profile and does not exert a proliferative effect on the endometrium
  - Effects on bone remodeling are similar to those of estrogen; there is a decrease in the incidence of fractures. Raloxifene is useful in decreasing the risk of osteoporosis
  - Unfortunately, raloxifene does not relieve hot flushes and can even worsen them
  - There is increased incidence of venous thromboembolism.

TYPES of HRT

- Estrogen-only HRT: usually recommended ONLY for women who have undergone hysterectomy. There is no need to take progestogen because there is no risk of endometrial cancer.
- E+P:
  - **Cyclical HRT:** Also known as sequential HRT, is often recommended for women who have menopausal symptoms but still have their periods.

There are two types of cyclical HRT:

1. Monthly HRT - Estrogen every day and progestogen for the last 12-14 days
2. 3 monthly HRT - Estrogen every day and progestogen for 12-14 days, every 13 weeks

Continuous combined HRT: It is usually recommended for women who are postmenopausal.

It involves taking estrogen and progestogen every day without a break.

Various ways HRT can be taken:

- *Tablets*
- ***Transdermal patch:*** *Patches are available containing 17 beta estradiol which release 40-80 mcg of estrogen/day. The patch to be applied below the waist line and to be changed twice a week*
- ***Subdermal implants*** - Under local anesthetic, small pellets of estrogen are inserted subcutaneously over the anterior abdominal wall or buttock or thigh. Can be kept for 6 months

- **Percutaneous Estrogen gel** – Delivers **1 mg of estradiol/day when applied to the skin**
- **Local estrogen vaginal cream:** (for urogenital atrophy) for vaginal dryness, atrophic vaginitis. It also reduces frequency, urgency and recurrent infections. Conjugated equine vaginal estrogen cream 1.25 mg daily is very effective.

CONTRAINDICATIONS OF HRT

- Active liver disease (hepatitis/tumor)
- Undiagnosed vaginal bleeding
- Thrombophilias
- IHD/CAD
- Complicated migraine
- Complicated valvular heart disease
- Breast and OVARIAN cancer (current or past history)
- Severe hypertension (systolic >160 or diastolic >100)
- DM with vascular complications
- History of thromboembolism/stroke/DVT.

Side Effects

- Fluid Retention
- Bloating
- Breast Tenderness
- Nausea
- Leg Cramps
- Headaches
- Acne.

Risks of HRT

- Breast cancer (Marginal increase in risk of breast cancer)
  - Using HRT for five years would only increase the average risk from 1% to 1.6%
  - Risk appears to return to normal within five years of stopping taking HRT.
- Ovarian cancer:
  - HRT slightly increases the risk of developing ovarian cancer
  - The longer HRT is taken, the more the risk increases
  - When HRT is stopped, risk returns to normal over the course of a few years.
- Increased risk of coronary heart disease (RR 1.29) and VTE.
- The risk of stroke is increased in women who smoke and are overweight.

Women starting HRT and aged below 60 are not at an increased risk of stroke.

Combined HRT **does not** increase the risk of endometrial cancer.

Q. Features of Turner's syndrome.

This condition occurs in about 1 in 2,500 newborn girls worldwide.

Features of Turner Syndrome (45 XO)

- Short stature
- Broad chest, widely spaced nipples (shield chest)
- Congenital lymphedema
- Cubitus valgus
- Webbed posterior neck
- High arched palate
- Ovarian dysgenesis and infertility (90%)
- Aortic coarctation or bicuspid aortic valves
- Normal intelligence
- Hypoplastic uterus (due to lack of estrogen).

Investigations

- Sex chromatin study negative
- Karyotype is 45 XO
- Sr E2 is very low
- Very high FSH and LH.

The primary treatments for nearly all girls and women include hormone therapies:

- **Growth hormone.** Growth hormone therapy is recommended for most girls with Turne syndrome. The goal is to increase height as much as possible
- Growth hormone treatment is usually given several times a week as injections of somatropi
- **Estrogen therapy.** Most girls with Turner syndrome need to start estrogen and relate hormone therapy in order to begin puberty and achieve adult sexual development
- Estrogen replacement therapy usually continues throughout life, until a woman reache the average age of menopause. Cyclical estrogen and progesterone therapy can also lea to regular menstruation in these patients.

Pregnancy and Fertility Treatment

Some women with Turner syndrome can become pregnant with IVF and donor oocyte.

This requires a specially designed hormone therapy to prepare the uterus for pregnancy.

PART 2

Obstetrics

11 Placental Functions and Physiological Changes

Q. Functions of placenta.

PLACENTAL FUNCTIONS

1. Transfer of nutrients and waste products from the mother to the fetus by the following mechanisms:

- Simple diffusion
- Facilitated diffusion
- Active transport
- Endocytosis
- Exocytosis
- Leakage.

This is responsible for the following functions:

- **Respiratory function:** By intake of oxygen and output of carbon dioxide by simple diffusion across the fetal membrane. The O_2 supply to the fetus is at the rate of 5 mL/kg/min and this is achieved with **cord blood flow of 165–330 mL/min.**
- **Excretory function:** By simple diffusion of waste products (eg: urea, uric acid, creatinine) from fetal to maternal circulation.
- **Nutritive function:**
  - Glucose: **Facilitated diffusion with help of GLUT 1.**
  - Lipids: Direct transfer.
  - Amino acids: **Active transport (ATPase).**
  - Water and electrolytes—Na, K, Cl by simple diffusion; Ca, Fe, phosphorus by active transport. Water soluble vitamins are actively transferred while fat soluble vitamins are slowly transferred.
  - Hormones: Insulin, steroids from the adrenals, thyroid, chorionic gonadotropin or placental lactogen cross the placenta at a very slow rate. Parathormone and calcitonin do not cross the placenta.

2. Endocrine Function

- Placenta produces **protein hormones** (hCG, hPL, PS BETA 1 G, PAPP-A) and **steroidal hormones** (estrogen and progesterone).

3. Enzymatic Function

- Placenta secretes many enzymes such as diamine oxidase, oxytocinase, phospholipase A2.

4. Barrier Function

- To protect the fetus from toxic effects of substances in the maternal blood. Most substances with high molecular weights (> 500 daltons) are held up. Antibodies and antigens cross the placenta in both directions. Maternal infections such as viral (rubella, chickenpox, measles, mumps, poliomyelitis), bacteria (tubercle bacillus, Treponema Pallidum), protozoa (Toxoplasma gondii, malarial parasites) can cross the placental barrier and affect the fetus. Most drugs can cross the placenta and affect the fetus.

5. Immunological Function

- The fetus and the placenta contain paternally determined antigens which can lead to immunological rejection. The placenta has some role in preventing such a rejection. Placental hormones have got some immunosuppressive effect. There is production of **blocking antibodies** by mother in response to **TLX (trophoblast lymphocyte cross reactive antigen)** which protect the fetus from rejection.

Q. Amniotic fluid

Amniotic fluid surrounds the fetus everywhere except at its attachment with the body stalk. The fluid is completely replaced in **every 3 hours.**

Source

1. Transudation of maternal serum across placenta.
2. Transudation of fetal circulation across the umbilical cord or placental membranes.
3. Secretion from amniotic epithelium.
4. Transudation of fetal plasma through nonkeratinized fetal skin before 20 weeks.
5. Fetal urine: **400–1200 ml/day** at term.
6. Fetal lung fluid.

Removal

1. Fetus swallows 400–700 ml of fluid per day.
2. Intramembranous absorption of water and solutes (200–500 ml/day) from the amniotic compartment to fetal circulation through the fetal surface of the placenta.

Volume

This is related to gestational age.

Gestational Age (weeks)	Volume (ml)
12	50
20	400
36–38	1000
40	800
43	200

Physical Features

- Faintly alkaline
- Low specific gravity 1.010
- An osmolarity of 250 mOsmol/Litre is suggestive of fetal maturity
- **Pale straw colour** due to exfoliated lanugo and epidermal cells from the fetal skin.

Color of amniotic fluid	Clinical Importance
Colorless	Preterm
Straw colored	Term
Meconium stained	Fetal distress
Golden	Rh incompatibility
Amber/saffron	Postdatism
Blood stained	Abruptio placenta
Tobacco juice	IUFD
Purulent	Chorioamnionitis

Composition

- **98–99% water**
- 1–2% solids (proteins, glucose, lipids, NPN, urea, uric acid, Na, K, Cl).

Functions

Main function = **protect the fetus**

- Shock absorber
- Maintains even temperature
- Allows free movements of fetus and prevents adhesions
- During labor, bag of water helps in cervical dilatation
- Flushes the birth canal and by its bactericidal action protects fetus and prevents ascending infection to the uterus.

Clinical Importance

- Study of amniotic fluid provides useful information about fetal well-being and maturity
- AFI (amniotic fluid index) is used for fetal well-being and to diagnose poly and oligohydroamnios
- ARM and drainage of liquor is a method for induction of labor.

Q. Tabulate the important physiological changes during pregnancy.

Q. CVS changes during pregnancy.

During pregnancy there are progressive anatomical, physiological, and biochemical changes in all systems of the body. It is a phenomenon of **maternal adaptation** to increasing demands of growing fetus.

PHYSIOLOGICAL CHANGES IN PREGNANCY

1. Hematological changes

• Blood volume (mL)	Increased	+30–40%
• Plasma volume (mL)	Increased	+40–50%
• RBC volume (mL)	Increased	+20–30%
• Total Hb (g)	Increased	+20%
• Hb (g%) PCV (%)	Decreased	–20%

2. Plasma protein changes in pregnancy

• Total protein (g)	Increased	+20–30%
• Plasma protein concentration (g%)	Decreased	–10%
• Albumin (g%)	Decreased	–30%
• Globulin (g%)	Slight increase	+5%
• Albumin: Globulin ratio	Decreased	–

3. Blood coagulation factors

Increased	Decreased	Unaffected
• Fibrinogen (+50%)	Factor XI	Clotting time
• ESR (4 times)	Factor XIII	Bleeding time
• Factor IX	Platelet count	
• X		
• VIII		
• VII		
• II		

Platelet count slightly decreases during pregnancy; however, there is no decline in platelet function.

4. Respiratory system changes in pregnancy

Increased	Decreased	Unaffected
• Tidal volume	Functional residual capacity	Respiratory rate
• Minute ventilation	Expiratory reserve volume	Vital capacity
• Minute O_2 uptake	Residual volume	Inspiratory reserve volume
• Inspiratory capacity	Total lung capacity	

5. Renal changes in pregnancy

Increased	Decreased
• Renal blood flow (+50%)	S. Creatinine
• GFR (+50%)	S. BUN
• Creatinine clearance	S. Uric acid
• Glucosuria	Plasma osmolality
• Aminoaciduria	S. $Na^+/K^+/Cl^-$

- S. Aldosterone increases in pregnancy.
- S. ADH (antidiuretic hormone) remains unchanged in pregnancy.

CVS

Anatomical Changes

- Due to elevation of diaphragm because of gravid uterus, the heart is pushed upwards and outwards with slight rotation to the left
- The apex beat is shifted to the 4th intercostal space about 2.5 cm outside the midclavicular line
- Systolic murmur can be heard in pulmonary or apical area
- ***Mammary murmur:*** Continuous hissing murmur in tricuspid area in left 2nd and 3rd intercostal space (due to increase in blood flow through internal mammary vessels)
- S3 and rarely S4 maybe auscultated
- *2D echo:* Increase in left ventricular end diastolic diameter. Increase in left and right atrial diameters
- *ECG:* Left axis deviation.

Cardiac Output (CO)

- ***Cardiac Output Increases by 40% during Pregnancy***
  - The cardiac output begins to rise from 5th week of gestation and reaches its peak at **30–32 weeks** and then remains static till term
  - So the maximum risk of a heart disease patient to have cardiac failure **during pregnancy** is at **32 weeks**
  - CO **increases by 50% during each uterine contraction in labor** and
  - There is **80% increase in CO immediately postpartum** (as the uterus contracts, blood from uterus is pushed back into the maternal system, also known as **autotransfusion**)
  - Therefore the risk of cardiac failure is maximum in the immediate postpartum period (followed by intrapartum). *To avoid this, diuretics should be given after placental delivery to heart disease patients.*
  - CO returns to prelabor value by one hour following delivery and to prepregnant levels by another **four weeks time.**

Hemodynamic changes during pregnancy			
	Nonpregnant	Pregnancy near term	Change
Cardiac output (L/min)	4.5	6.26	+ 40%
Stroke volume (ml)	65	75	+ 27%
Heart rate (per minute)	70	85	+ 17%
Blood pressure	Unaffected or midpregnancy drop of diastolic pressure by 5–10 mm Hg		
Venous pressure	10 cm (femoral)	20–25 cm water	+ 100%
Colloid oncotic pressure (mm Hg)	20	18	– 14%
Systemic vascular resistance (SVR)			– 21%
Pulmonary vascular resistance (PVR)			– 34%

Blood Pressure

- Systemic vascular resistance (SVR) decreases by 21% due to smooth muscle relaxing effect of:
  - Progesterone
  - NO
  - PG
  - ANP
- BP (CO × SVR) is decreased
- There is decrease in diastolic BP and MAP by 5–10 mm Hg.

Venous Pressure

- Antecubital venous pressure remains unaffected
- Femoral venous pressure (normal = 8–10 cm of water) is markedly raised (pressure of gravid uterus on common iliac veins) to about 25 cm of water during pregnancy in lying down position and to about 80 to 100 cm water in standing position.

Central Hemodynamics

- No significant change in CVP, MAP, and PCWP.

Regional Distribution

- Uterine blood flow is about 50 ml/min in nonpregnant state
- Uteroplacental blood flow increases progressively during pregnancy, ranging from approximately **700 to 900 ml/min near term.**

The increase is due to vasodilatation effect of:

- Progesterone
- NO
- PG
- ANP
- Pulmonary blood flow (normal = 6000 ml/min) is increased by 2500 ml/minute
- Renal blood flow (normal = 800 ml/min) increases by 400 ml/minute

12 Antenatal Care and Tests for Fetal Well-Being

Q. Biophysical profile (BPP).

The biophysical profile score is a method used to **assess the well-being of a fetus** at increased risk for death or damage in utero.

- Evaluates the fetus for the presence of five parameters using ultrasound and an electronic fetal heart rate monitor
- A score of 2 points is given for each parameter that meets criteria
- The USG (to evaluate 4 parameters) is continued until all criteria are met or 30 minutes have elapsed
- The points are then added for a **maximum score of 10.**

INDICATIONS

- **Nonreactive NST**
- **High-risk pregnancy** (Preeclampsia, IUGR, postdatism, diabetes, etc).

Components and their Scores for the Biophysical Profile (Manning's score)

Component	Score 2	Score 0
Nonstress test	≥2 accelerations of ≥15 beats/minutes for ≥15 seconds in 20–40 minutes, i.e. reactive NST	0 or 1 acceleration in 20–40 minutes
Fetal breathing	≥1 episode of rhythmic breathing lasting >30 seconds within 30 minutes	<30 seconds of breathing in 30 minutes
Fetal movement	≥3 discrete body or limb movements within 30 minutes	<3 discrete movements
Fetal tone	≥1 episode of extension of a fetal extremity with return to flexion or opening or closing of hand within 30 minutes	No movements or no extension/flexion
Amniotic fluid volume	Single vertical pocket >2 cm	Largest single vertical pocket ≤2 cm

Modified BPP = NST and AFI

Biophysical Profile Score, Interpretation, and Pregnancy Management

Biophysical profile score	Interpretation	Recommended management
10	Normal, nonasphyxiated	No fetal indication for intervention; repeat test weekly except in diabetic patient and postterm pregnancy (twice weekly)
8, Normal fluid	Normal, nonasphyxiated fetus	No fetal indication for intervention; repeat testing per protocol
8, Oligohydramnios	Chronic fetal asphyxia suspected	Deliver if ≥ 37 weeks, otherwise repeat testing
6	Possible fetal asphyxia	If amniotic fluid volume abnormal, deliver If normal fluid at > 36 week with favorable cervix, deliver If repeat test ≤ 6, deliver If repeat test > 6, observe and repeat per protocol
4	Probable fetal asphyxia	Repeat testing same day; if biophysical profile score ≤ 6, deliver
0–2	Almost certain fetal asphyxia	Deliver

It is a highly reliable test for fetal well-being. The BPP has a false-negative mortality rate (number of fetal deaths that occur within 1 week of a **normal** test result) of only **0.6–0.77** deaths per 1000 tests.

Q. What are the various tests for fetal well-being/fetal surveillance. Add a note on NST/ Cardiotocography.

Methods for Assessment of Fetal Well-being

Antepartum	Intrapartum	Postpartum
• Nonstress test (NST) • Biophysical profile (BPP) • AFI • Vibroacoustic stimulation test (VSAT) • Contraction stress test/oxytocin challenge test (CST/OCT) • Fetal kick count • Color Doppler USG	• CTG (cardiotocography) • Fetal heart rate (Doppler) • Fetal scalp electrode monitoring • Fetal pulse oximetry • Fetal scalp pH monitoring	Apgar score Umbilical cord pH

NST is a test for fetal well-being done in the antenatal period. The patient is **NOT in labor**, so there are **no contractions** and hence the name, nonstress test (as labor and contractions are considered as a stress for the fetus).

In NST, a continuous electronic monitoring of fetal heart rate is done along with fetal movements. With **fetal movements there should be acceleration in fetal heart rate**, which if present, indicates a healthy fetus. The accelerations in FHR with movements is **reflex mediated**.

Important indications for an NST are:

High risk pregnancies like:
- Diabetes
- Preeclampsia
- IUGR
- Decreased fetal movements
- Postdatism.

Testing should be started **after 30 weeks of gestation.** Reactive NST is valid **for 7 days** and hence frequency of testing should be weekly except in cases of **postdatism and maternal diabetes where a biweekly test is recommended.**

INTERPRETATION

- **Bradycardia:** The baseline fetal heart rate lesser than 110 beats/minute.
- **Tachycardia:** The baseline fetal heart rate greater than 160 beats/minute.

Beat-to-Beat Variability

- Normal beat-to-beat variability should be **6–25 beats/minute.**
- Diminished beat-to-beat variability can be an ominous sign and may indicate a seriously compromized fetus.
- Loss of beat-to-beat variability along with decelerations is associated with fetal academia.

Reactive (Reassuring) NST

Two or more accelerations of ≥ 15 beats/minute above the baseline, lasting for ≥ 15 seconds are present in 20–40 minutes observation period.

Nonreactive (Nonreassuring) NST

Presence of less than two fetal heart rate accelerations within a 20–40 minute testing period.

The false positive rate (healthy fetus but nonreactive NST) **of NST is 50%** whereas the **false negative rate** (number of fetal deaths that occur within 1 week of a normal test result) is **1.4–1.8 per 1000 tests.**

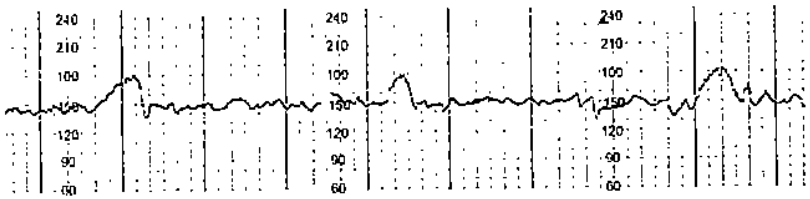


Fig. 12.1: Reactive NST

Note

The difference between NST and CTG is that in NST there are no contractions whereas **CTG is done during labor so the fetal heartbeat (cardio) and the uterine contractions (-toco-) both are recorded. So CTG is an intrapartum fetal monitoring test.**

The interpretations are same as NST.
There are three types of decelerations.

1. Early decelerations are due to **head compression** (stimulation of vagus nerve).
2. Late decelerations are due to **uteroplacental insufficiency (fetal distress/hypoxia)**.
3. Variable decelerations are due to **cord compression** (oligohydramnios in labor).

Features of Early Fetal Heart Rate Deceleration

Characteristics include gradual decrease in the heart rate with both onset and recovery coincident with the onset and recovery of the contraction (Fig. 12.2).

Features of Late Fetal Heart Rate Deceleration

Characteristics include gradual decrease in the heart rate with the nadir and recovery occurring after the end of the contraction. The nadir of the deceleration occurs 30 seconds or more after the onset of the deceleration (Fig. 12.3).

Features of Variable Fetal Heart Rate Decelerations

Characteristics include abrupt decrease in the heart rate with onset commonly varying with successive contractions. The decelerations measure ≥ 15 beats/minutes for 15 seconds or longer with an onset to nadir phase of less than 30 seconds. Total duration is less than 2 minutes (Fig. 12.4).

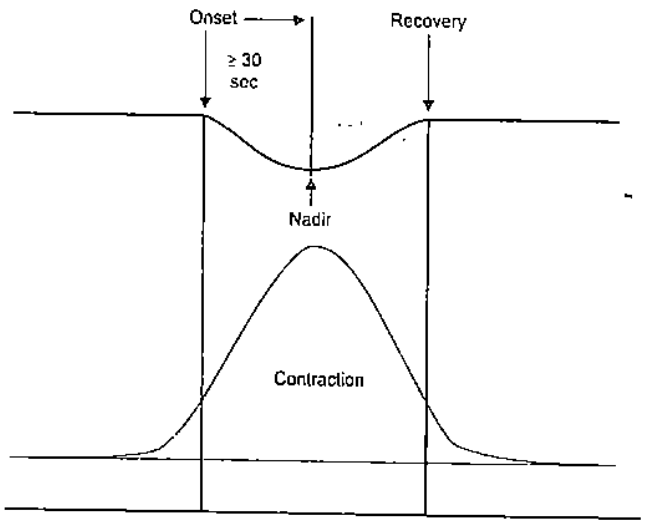


Fig. 12.2: Early deceleration

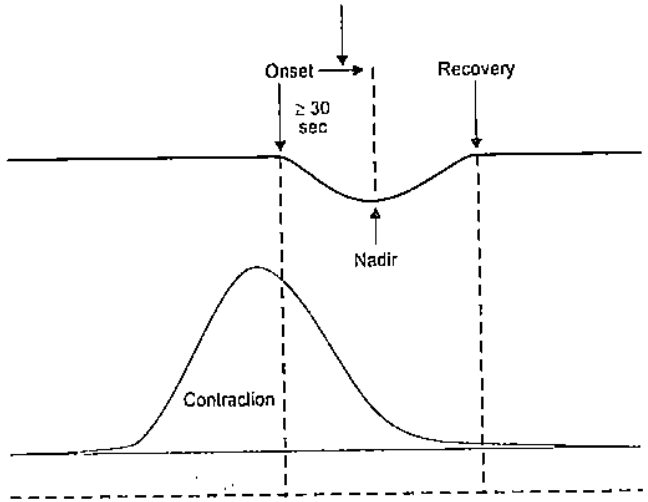


Fig. 12.3: Late deceleration

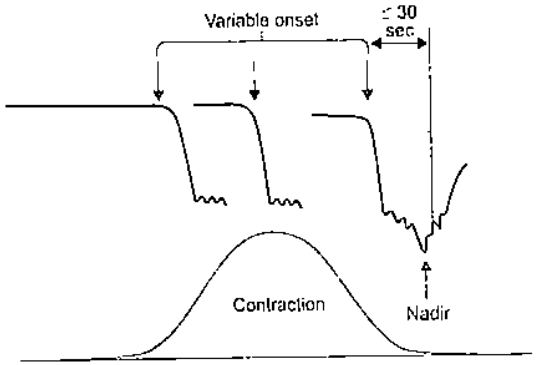


Fig. 12.4: Variable deceleration

Q. Components of antenatal care.

Q. Define antenatal care, components at first visit and schedule for subsequent visit in ideal setup for uncomplicated pregnancy.

DEFINITION

Antenatal care (ANC) is the **systematic supervision** (care, examination and advise) of the woman during pregnancy.

ANC Comprises of

- Careful **history taking and examination**
- Advice to pregnant women.

Aims and Objectives

1. *The primary aim of ANC is:* To promote and protect the health of women and the unborn fetus during pregnancy so as to achieve at the end, **a normal pregnancy and healthy mother and a healthy baby.**
2. To **reduce maternal and perinatal mortality and morbidity.**
3. Screen high risk cases.
4. To prevent or to detect and treat at the earliest any **complications.**
5. To **educate** the pregnant women about pregnancy and labor and the complications of pregnancies.
6. To discuss with couple the best approach (time and place) of safe delivery and the best way of bringing up their babies.
7. To ensure that the pregnant woman and her unborn child are in the best possible health prior to delivery. This is achieved by:
   - Nutritional advise
   - Iron, folate, and calcium supplementation
   - Providing treatment for conditions that affect mother such as malaria, tuberculosis, HIV, etc.
   - Tetanus toxoid immunization
   - Providing voluntary HIV testing and counseling
8. Providing information about advantages of breastfeeding and motivate for family planning and use of contraceptives.

At the First Visit

The first visit should not be deferred beyond the second missed period, ideally must be earlier.

- To assess the health of the mother and educate her the **importance of regular follow-up**
- To assess the gestational age and baseline investigations
- To start with iron, folic acid, and calcium supplements, if not already on it.

History Taking

Ask about

- Full name
- Husband's name
- Age (extremes of age is obstetric risk factor)
- Address
- Age of marriage
- Gravida and parity
- Religion
- Occupation of the patient and the husband (gives idea about her **physical activity, stress, occupational hazard** and socioeconomic condition. To anticipate complications of **anemia, prematurity, IUGR, etc. a/w low social status**)

- *Period of gestation:* Ask about the first day of the last **normal** menstrual period (LNMP) and assign the EDD and also inquire about the past menstrual cycle
- *Habits:* Smoking/chewing tobacco, alcohol, etc. and other addictions (frequency and quantity)
- *Complaints:* Bleeding or spotting since becoming aware of being pregnant, pain in abdomen, nausea, vomiting, sleep, appetite, bowel habit, etc.
- *History of present pregnancy:* Trimester wise complaints or complications noted. Medications, supplements, immunization, number of previous antenatal visits (booking status) noted
- *Obstetric history:* To know about any **antenatal, intrapartum and postpartum complications** of previous pregnancies:
  - Number and type of previous pregnancies (miscarriage, tubal pregnancy, preterm delivery)
  - Previous deliveries and any complication or procedure related to the previous deliveries (cesarian section and its indication, if known; forceps or vacuum extraction, manual removal of the placenta)
  - Date (month, year) and outcome of each event (live birth, still birth, abortion, ectopic, twins, hydatidiform mole, child with any abnormality, neonatal and infant death)
  - Birth weight if known
  - Sex of children
  - Special maternal complications, events, and interventions in previous pregnancies.

Past Medical and Surgical History

Ask about history of specific diseases and conditions, including: Tuberculosis, cardiovascular diseases, hypertension, chronic renal disease, epilepsy, diabetes mellitus, RTIs/STIs/HIV-AIDS, malaria, hepatitis and other liver diseases, any allergies, other chronic diseases, surgeries, blood transfusion, current use of medicines.

Family History

Family history of tuberculosis, hypertension, diabetes mellitus and any hereditary disease to be enquired.

Physical Exam

Perform routine physical examination and particularly pay attention to the followings:

- *Build:* Obese/average/thin
- *Nutrition:* Good/average/poor
- Height
- Weight (kilograms) for setting a baseline for further monitoring of appropriate weight gain
- Pallor/signs of anemia (pale complexion, fingernails, conjunctiva, oral mucosa, tip of tongue, and shortness of breath)
- Jaundice
- Glossitis, stomatitis
- *Neck:* Lymph nodes, neck veins, thyroid enlargement
- *Edema feet:* Examine both legs, medial malleolus and lower 1/3rd of tibia
- Pulse and blood pressure for detecting hypertension

- Chest and heart auscultation for detecting underlying cardiovascular and respiratory diseases
- Obstetric examination
  - Inspection
  - **Symphysis-fundal height**
  - *Palpation:* Various grips to determine lie and growth
  - Auscultation of FHS from 20 weeks onwards
- Breast exam for inverted nipple, which can impact breastfeeding
- External genitalia for vaginal discharge
- PAP smear maybe taken.

INVESTIGATIONS

Perform the following tests:

- *Routine urine analysis:* Protein, sugar, pus cells
- *Blood:* Blood group typing (ABO and Rh), Hemoglobin ,VDRL, blood sugars, TSH and HIV (after consent and counselling) and HBsAg
- PAP smear is routinely done at many clinics.

Special Investigations

- **Dual marker test at 11–13 weeks or triple marker test at 16–18 weeks** as a screening tests for trisomy
- USG:
  - USG in first trimester helps to detect:
    - Viable intrauterine pregnancy
    - Rule out ectopic pregnancy
    - Accurate dating
    - Number of fetuses
    - Any uterine and adnexal pathology
  - **NT scan at 11–13 weeks of gestation**
  - **Anomaly/malformation scan at 18–20 weeks of gestation.**

Repetition of Investigations

- Hemoglobin should be repeated at 28 weeks and 36 weeks.
- Blood sugars fasting and postprandial at 26–28 weeks.
- Urine checked for sugars and proteins at every antenatal visit.

Supplements

- Folic acid 400 mcg is recommended (ideally to be started one month before conception) through out the pregnancy, especially in first trimester to prevent NTD and later megalobalstic anemia
- *Iron:* There is increase in iron requirement during pregnancy which cannot be met with dietary supplementation. Hence all pregnant mothers need iron therapy starting from 12–16 weeks onwards. One tablet containing 60 mg elemental iron to be taken every day, if hemoglobin is above 10 gm%. The dose is increased to 2–3 tablets in cases of anemia

- *Multivitamins:* Vitamin B complex, D supplementation should also ideally be recommended
- *Calcium:* As calcium requirement is doubled in pregnancy, patient should be advised to take at least one tablet of 500 mg calcium.

Immunization

Tetanus: Immunization protects both mother and neonate. In unprotected women 0.5 ml tetanus toxoid is given IM at 4–6 weeks interval for two such, the first dose between 16–24 weeks. If immunized in the past, a booster dose of 0.5 ml IM is given in last trimester.

Dietary Advice

Increase in calorie requirement is about **300 k.cal** over nonpregnant state during second half of pregnancy. There is also increase in requirement of protein, iron, and calcium. The patient is to be advised to eat balanced healthy diet. Women with normal BMI should eat to maintain the scheduled weight gain of 11–12 kgs in pregnancy.

Important Instructions

Patient to be motivated and persuaded to attend antenatal checkup on schedule date of visit.

To report early and immediately in cases of:

- Pain in abdomen
- Bleeding per vagina, even if slight
- Gush of watery fluid per vaginam (Suggestive of PROM)
- Decrease or absent fetal movements
- Untoward symptoms such as intense headache, epigastric pain, vomiting, and scanty urination.

Frequency of Visits

Generally checkup is done at interval of:

- 4 weeks upto 28 weeks, then
- 2 weekly upto 36 weeks and then
- Weekly till delivery.

In developing countries **WHO recommends at least four visits:**

- Second trimester 16–18 weeks
- 24–28 weeks
- 32 weeks
- 36 weeks.

13

Labor

Q. What are the stages of labor. Difference between true and false labor.

DEFINITION

Series of events that take place in genital organs in an effort to expel the viable products of conception out of womb through vagina into outer world is called as labor.

Labor has Four Stages

First Stage of Labor

- Begins with **regular uterine contractions and ends with complete cervical dilatation at 10 cm**
- Divided into a **latent phase** (<3 cm) and an **active phase** (3–10 cm)
- The latent phase begins with mild, irregular uterine contractions that soften and shorten the cervix
- Contractions become progressively more rhythmic and stronger
- The active phase usually begins at about 3–4 cm of cervical dilation and is characterized by rapid cervical dilation and descent of the presenting fetal part.

Second Stage of Labor

- Begins with complete cervical dilatation and ends with the **delivery of the fetus**
- In nulliparous women, the second stage should be considered prolonged if it exceeds 3 hours if regional anesthesia is administered or 2 hours in the absence of regional anesthesia
- In multiparous women, the second stage should be considered prolonged if it exceeds 2 hours with regional anesthesia or 1 hour without it.

Third Stage of Labor

- The period between the delivery of the fetus and the **delivery of the placenta** and fetal membranes
- Delivery of the placenta often takes less than 10 minutes, but the third stage may last as long as 30 minutes.

Fourth Stage

At least 1 hour observation after the expulsion of placenta and membranes. General condition of the mother and behavior of uterus are carefully to be monitored.

Difference Between True and False Labor

True labor	False labor
Contractions occur at regular intervals	Contractions occur at irregular intervals
Intervals gradually shorten	Intervals remain long
Intensity gradually increases	Intensity remains unchanged
Discomfort is in the back and abdomen	Discomfort is chiefly in the lower abdomen
Progressive effacement and dilatation of cervix	**Cervix does not dilate**
Discomfort is not stopped by sedation	Discomfort usually is relieved by sedation
Associated with show and formation of bag of forewaters	Not associated
A/w Descent of the presentating part	No descent

Q. Partogram/partograph.

Definition

Partogram is a composite graphical record of key data (maternal and fetal) during labor entered against time on a single sheet of paper.

It provides an accurate record of the progress of labor and any delay or deviation from normal may be detected quickly and treated accordingly. It was first devised by **Freidman in 1954**.

Components

- **Patient identification:** Name, gravida, parity
- **Time:** It is recorded at an interval of one hour. Zero time for spontaneous labor is time of admission in the labor ward and for induced labor is time of induction
- **Fetal heart rate:** It is recorded at an interval of **thirty minutes**
- State of membranes and color of liquor: 'I' designates intact membranes, 'C' designates clear and 'M' designates meconium stained liquor
- **Cervical dilatation** and descent of head
- *Uterine contractions:* Squares in vertical columns are shaded according to duration and intensity
- Drugs and fluids given to patient in labor
- *Blood pressure:* It is recorded in vertical lines at an interval of 2 hours
- *Pulse rate:* It is also recorded in vertical lines at an interval of 30 minutes
- *Oxytocin:* If it is used then concentration (U/L) is noted down in upper box; while dose (drops/minute) is noted in lower box
- Urine analysis
- Temperature record.

The concept of **'alert line' and 'action line' was introduced by Philpott and Castle in 1972.** The action line can be placed at 2–4 hours interval, to the right and parallel to alert line. In partograms recommended by **'WHO'** the distance between the alert and action lines is **4 hours.**

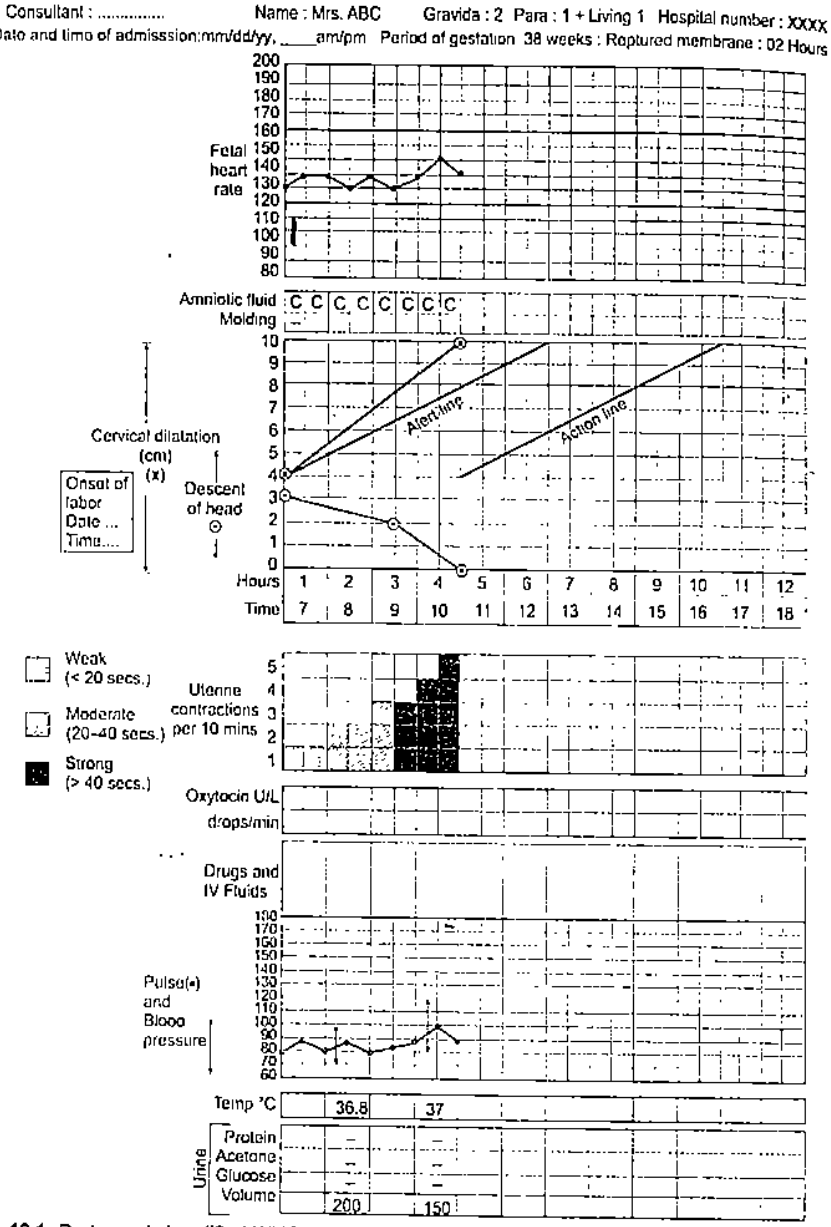


Fig. 13.1: Partograph (modified WHO) representing graphically the important observations in labor. The cervical dilatation and descent of head are shown in relation to alert and action lines. Intensity and duration of uterine contraction are shown with shades

Advantages

- Provides all important information and details on single sheet of paper
- No need to record labor events repeatedly
- **Early prediction** of any deviation from normal progress of labor ,so appropriate steps can be taken
- Facilitates handover procedure
- Partograph has **reduced the incidence of prolonged labor and LSCS rates**
- Improvement in **maternal and perinatal morbidity and mortality** (Fig. 13.1).

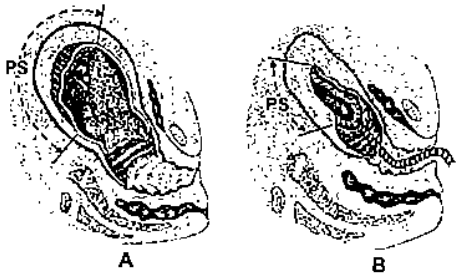
Q. Physiology and management of third stage of labor.

Third stage of labor is the period starting after the delivery of the fetus and till the delivery of the placenta and fetal membranes.

Physiology of 3rd Stage

It comprises of placental separation, its descent and finally expulsion with the membranes.

- After the baby is born, the muscles of the uterus contract, helping the placenta to separate from the uterine wall
- The amount of blood lost depends on how quickly this happens, since the uterus can contract more effectively after the placenta is expelled
- If the uterus does not contract normally (such as in uterine atony), the blood vessels at the placental site stay open and hemorrhage results. Because the estimated blood flow to the **uterus is 500 to 800 mL/minute at term,** most of which passes through the placenta, severe postpartum hemorrhage can happen within just a few minutes
- The muscle fibers of the uterus are in a **crisscross pattern** surrounding maternal blood vessels **(living ligature).** After the birth of the baby, these muscle fibers begin to **contract and retract**
- During the third stage, uterine contractions continue causing the placenta to separate from the uterine wall. Placental separation happens by contraction and retraction of the uterine muscles, **reducing the surface area at the placental site to about its half.** This reduction in size of the uterus is caused by **retraction** of the uterine muscle, a unique characteristic that helps maintain its shortened length after each contraction. As the placental area becomes smaller, the placenta begins to separate from the uterine wall because, unlike the uterus, it is not elastic and cannot contract and retract
- A shearing force is instituted between the placenta and the placental site which brings about the separation
- The plane of separation runs through **deep spongy layer of decidua basalis**
- At the area where the placenta separates from the uterus a clot forms. This clot—known as a retroplacental clot—collects between the uterine wall and the placenta and further promotes separation.



Figs 13.2 (A and B): Diagram showing area of placental site—(A) Before the delivery of the baby. (B) After the delivery of the baby. Note the reduction of the surface area of the placental site resulting in buckling of the placenta. PS = Placental surface

Methods of Placental Separation

There are 2 ways of placental separation:

- **Schultz (more common)**
- **Duncan Mathews.**

Schultz Mechanism

By far the most common mechanism of placental expulsion.

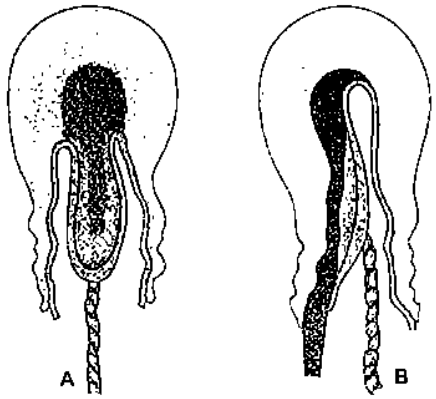
Delivery of the placenta with the fetal side presenting. Results when separation begins **centrally** with corresponding formation of a central retroplacental clot, which weighs the placenta so the central portion descends first.

This then inverts the placenta and amniotic sac and causes the membranes to peel-off the remainder of the decidua and trail behind the placenta. Bleeding associated with Schultz mechanism is not visible until the placenta and membranes are delivered, since the inverted membranes hold and catch the blood.

Duncan Mechanism

Delivery of the placenta with the maternal side presenting. Results when separation first takes place at the **margin or periphery of the placenta.** The placenta descends sideways and the amniotic sac, therefore, is not inverted but trails behind the placenta for delivery. Blood escapes between the membranes and uterine wall and is visible externally.

(Memory aid for remembering Schultz vs Duncan: Based on the appearance of the two different sides of the placenta. Fetal side is shiny and glistening because it is covered by membranes, therefore **'shiny Schultz'**. Maternal side is rough and red-looking, thus **'dirty Duncan.'** Remember: **S.S.C = Shiny Schultze Central**).



Figs 13.3 (A and B): Types of separation of the placenta—(A) Schultze method, (B) Mathews-Duncan method

Expulsion and Control of Bleeding

- After complete separation, the placenta is pushed into the flabby lower segment or upper vagina by effective contraction and retraction of uterus
- Thereafter, its expelled out by voluntary contraction of abdominal muscles or manual procedure
- The placental site is rapidly covered by a fibrin net and clots form
- The muscle fibers of the uterus (interlacing intermediate layer of myometrium) compress the blood vessels where the placenta was attached, helping to control bleeding at the placental site **(living ligature principal of hemostatsis)**
- Also, thrombosis occurs to occlude the torn sinuses
- **Apposition of walls of uterus (myotamponade)** also contribute.

Length of 3rd Stage

- Fifty percent of placental deliveries occur within 5 minutes, and 90 percent are delivered within 15 minutes
- A third stage of labor lasting longer that 18 minutes is associated with a significant risk of PPH
- When the third stage of labor lasts longer than 30 minutes, PPH occurs six times more often than it does among women whose third stage lasted less than 30 minutes.

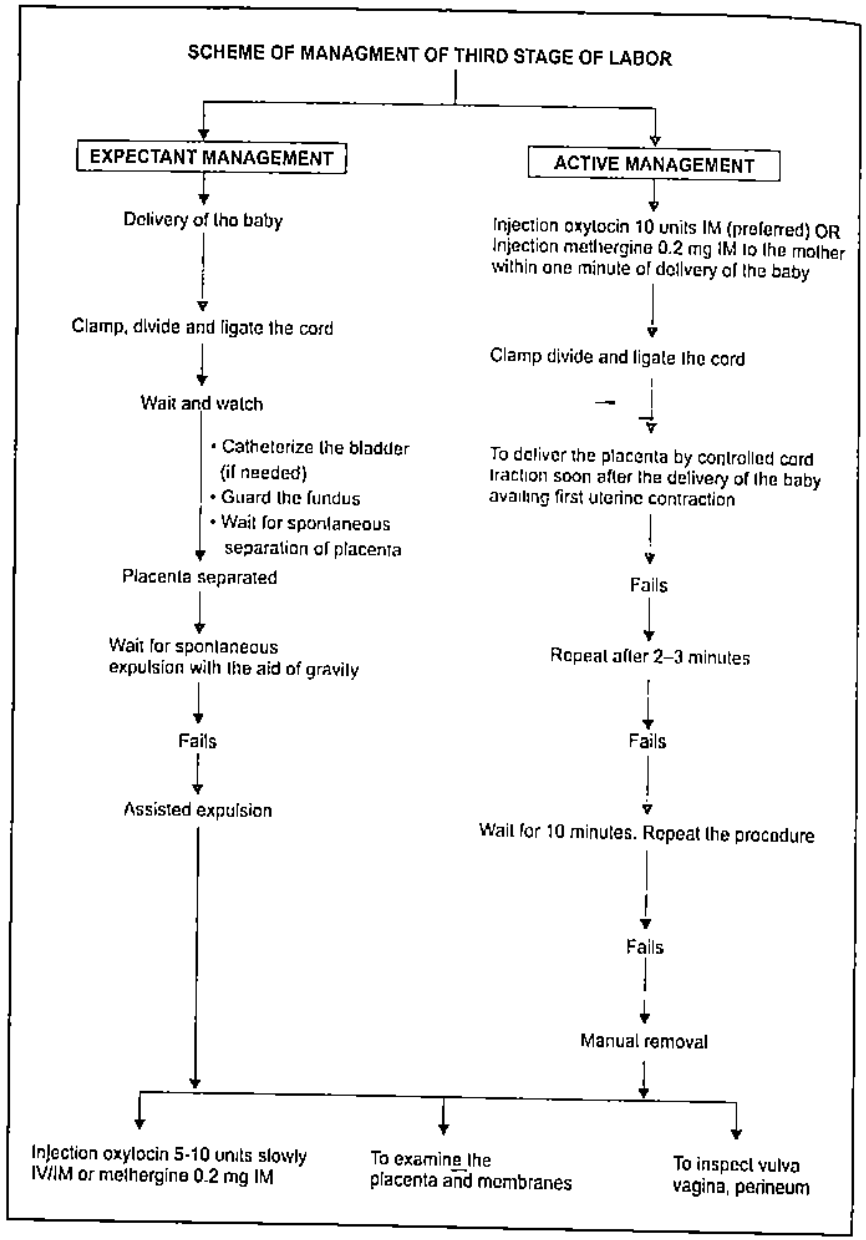
Management of 3rd Stage

Third stage is the most crucial stage of labor. Its most important and MOST dangerous complication is PPH.

Two methods of management are:

1. Expectant management
2. Active management (preferred) (Flow-Chart 13.1).

Flow Chart 13.1: Scheme of management of third stage of labor



	Physiologic (expectant) management	Active management*
Uterotonic	Uterotonic is not given before the placenta delivered	Uterotonic is given within one minute of the baby's birth (after ruling out the presence of a second baby)
Signs of placental separation	**Wait for signs of separation:** • Gush of blood • Lengthening of cord • Uterus becomes rounder and smaller as the placenta descends	**Do not wait for signs of placental separation. Instead:** • Palpate the uterus for a contraction • Wait for the uterus to contract • Apply CCT with countertraction
Delivery of the placenta	Placenta delivered by gravity assisted by maternal effort	Placenta delivered by CCT while supporting and stabilizing the uterus by applying countertraction
Uterine massage	**Massage the uterus after the placenta is delivered**	Massage the uterus *after* the placenta is delivered
Advantages	• Does not interfere with normal labor process • Does not require special drugs/supplies • May be appropriate when immediate care is needed for the baby (such as resuscitation) and no trained assistant is available • May not require a birth attendant with injection skills	• Decreases length of third stage • Decrease likelihood of prolonged third stage • Decreases average blood loss • Decreases the number of PPH cases • Decreases need for blood transfusion
Disadvantages	• Length of third stage is longer compared to AMTSL • Blood loss is greater compared to AMTSL • Increased risks of PPH	• Requires uterotonic and items needed for injection/injection safety • Requires a birth attendant with experience and skills giving injections and using CCT

Q. Active management of the third stage of labor (AMTSL).

Third stage of labor is the period starting after the delivery of the fetus and till the delivery of the placenta and fetal membranes.

Definition

Active management of the third stage of labor (AMTSL): A combination of actions performed during the third stage of labor to **prevent PPH.** AMTSL speeds delivery of the placenta by increasing uterine contractions and prevents PPH by minimizing uterine atony.

The components of AMTSL are:

- Administration of a **uterotonic drug** at the time of delivery of the **anterior shoulder or afterwards within one minute after the baby is born (oxytocin is the uterotonic of choice)**
- **Controlled cord traction** (CCT)
- **Uterine massage** immediately after delivery of the placenta.

Steps

1. WHO recommends oxytocin as the drug of choice for AMTSL
2. Administer a uterotonic drug (oxytocin or methergine) within one minute of the baby's birth
3. Before performing AMTSL, gently palpate the woman's abdomen **to rule out the presence of another baby**. At this point, do not massage the uterus
4. If there is not another baby, begin the procedure by giving the woman **10 IU of oxytocin IM** in the upper thigh. This should be done within one minute of childbirth. **In case of twins do it after the delivery of second baby**
5. Clamp and cut the cord following strict hygienic techniques after cord pulsations have ceased or approximately 2-3 minutes after birth of the baby, whichever comes first
6. Perform controlled cord traction.

Controlled Cord Traction (CCT): Brandt Andrew's Maneuver

Traction on the cord **during a contraction** (in downward and backward direction holding the clamp) combined with countertraction upward on the uterus towards umbilicus with the provider's hand placed immediately above the symphysis pubis approximately at the junction of upper and lower segment. CCT facilitates expulsion of the placenta once it has separated from the uterine wall.

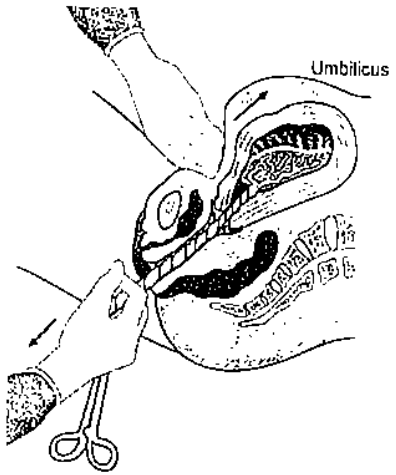


Fig. 13.4: Expression of the placenta by controlled cord traction

7. **Massage the uterus** immediately after delivery of the placenta and membranes until it is firm. Massaging the uterus stimulates uterine contractions and helps to prevent PPH
8. Examine the fetal and maternal sides of the placenta and membranes to ensure they are complete. A small amount of placental tissue or membranes remaining in the woman can prevent uterine contractions and cause PPH
9. Gently separate the labia and inspect the lower vagina and perineum for lacerations that may need to be repaired to prevent further blood loss. Suture the episiotomy
10. Perform a comprehensive examination of the woman and newborn one and six hours after childbirth. During the first two hours after the delivery of the placenta, monitor the woman at least every 15 minutes (more often if needed) to:
    - Palpate the uterus to check for firmness
    - Massage the uterus until firm. (Ask the woman to call for help if bleeding increases or her uterus gets soft.)
    - Check for excessive vaginal bleeding.

Advantages

AMTSL decreases:

- **Incidence of PPH by up to 60 percent**
- Length of third stage of labor
- Percentage of third stages of labor lasting longer than 30 minutes
- Need for blood transfusion
- Need for uterotonic drugs to manage PPH.

Disadvantage

The only disadvantage is **slightly increased incidence (1-2%) of retained placenta** and consequent manual removal of placenta. This is more likely to happen when methergin is used for AMTSL.

Q. Management of first stage of normal labor.

First stage of labor starts from onset of true labor pains (regular uterine contractions) and ends with complete cervical dilatation at 10 cm.

General Considerations

- Labor events have got great psychological, emotional and social impact to the woman
- Throughout labor, the patient is given **encouragement and emotional support**
- **Privacy** must be maintained
- She is explained about the events from time to time
- Comfortable environment, skill and confidence of the caregiver and the emotional support are all essential.

Management of normal labor aims at *maximal observation with minimal active intervention.* The idea is to maintain the normalcy and to detect any deviation from the normal at the earliest possible moment.

Antiseptics and Asepsis

Strict asepsis has to be maintained. Antiseptic and aseptic precautions are to be taken during vaginal examination and during conduction of delivery.

Patient Care

Shaving or hair clipping of the vulva is done.

The vulva and the perineum are washed liberally with soap and water and then with 10% Dettol solution or Hibitane (chlorhexidine) 1 in 2000.

Vaginal Examination in Labor

First vaginal examination should be done by a **senior doctor** to be more reliable and informative and it is to be done taking all aseptic precautions:

Complete examination should be done before fingers are withdrawn.

The following information are to be noted and recorded carefully:

- Cervical **dilatation** in centimetres
- Degree of **effacement** of cervix
- Status of **membranes** and if ruptured – color of the liquor: clear/meconium stained/blood stained
- Presenting part and its position
- Caput or moulding of the head
- **Station** of the head in relation to ischial spines
- Assessment of the **pelvis** specially in primigravida is to be done and presence of vulval varicosity, if any, are to be noted.

Indications of Vaginal Examinations

Vaginal examinations should be restricted to a minimum.

- At the onset of labor - To confirm the onset of labor
- *The progress of labor* can be judged on periodic examinations at an interval of 3–4 hours
- *Following rupture of the membrane* to exclude cord prolapse especially where the head is not yet engaged
- *Whenever any interference is contemplated*
- To confirm the actual coincidence of bearing down efforts with complete dilatation of the cervix and to *diagnose precisely the beginning of second stage.*

First Stage Management

Principles

- Noninterference with watchful expectancy so as to prepare the patient for natural birth
- To monitor carefully the progress of labor, maternal conditions and fetal behavior so as to detect any intrapartum complication early.

Preliminaries

- Enquiry is to be made about the onset of labor pains or leakage of liquor, if any
- Thorough general and obstetrical examinations including vaginal examinations are to be carried out and recorded
- Records of antenatal visits, investigation reports and any specific treatment given, if available, are to be reviewed.

Actual Management

- ***General:*** Encouragement, emotional support and assurance are given. *Constant supervision* is ensured.
- ***Bowel:*** An enema with soap and water or glycerine suppository is traditionally given in early stage.
- ***Rest and ambulation:*** If the membrane are intact, the patient is allowed to walk about. This attitude prevents vena cava compression and encourages descent of the head. Ambulation can reduce the duration of labor, need of analgesia.

  If, however, labor is monitored electronically or analgesic drug (epidural analgesia) is given, she should be in bed.
- ***Diet:*** There is delayed emptying of the stomach and low pH of the gastric contents in labor. *So food is withheld during active labor* **(to prevent Mendelson's syndrome).**

  Fluids in the form of plain water or fruit juice may be given in early labor.

  Intravenous fluid with ringer solution is started where any intervention is anticipated or the patient is under regional anesthesia.
- ***Bladder care:*** Patient is encouraged to pass urine by herself (bed pan can be given) as full bladder often inhibits uterine contraction and may lead to infection.

  If the patient fails to pass urine especially in late first stage, catheterization is to be done with strict aseptic precautions.
- ***Relief of pain:*** Different options include: Pethidine injections IM, Entonox inhalation (a mix of nitrous oxide 50% and oxygen 50%) and **epidural analgesia (considered the best and safe and should be offered if facilities are available)**
- Assessment of progress of labor and **partograph** recording:

Pulse is recorded every 30 minutes.

Blood pressure is recorded at every four hours.

Temperature is recorded every two hours. **Urine output** is recorded for volume, protein or acetone. Any drug (oxytocin or other) when given is recorded in the partograph.

Abdominal palpation – (a) Uterine cotractions: As regard the frequency, intensity and duration are assessed. *The number of contractions in 10 minutes and duration of each contraction in seconds are recorded in the partograph.* Partograph is charted every half an hour.

- *Pelvic grip:* Gradual disappearance of poles of the head which were felt previously, (usually occur in labor). Abdominal palpation for descent of the fetal head in terms of fifths felt above the brim is to be used
- Shifting of the maximal intensity of the fetal heart beat downwards and medially.

To Note the Fetal Well-Being

- Fetal heart rate (FHR) noted every half hour in the first stage and every 15 minutes in second stage or following rupture of the membranes. *To be of value, the observation should be made immediately following uterine contraction. The count should be made for 60 seconds.*

Ordinary stethoscope is quite suitable. Doppler, however, is helpful in the case of obesity and polyhydramnios.

Normal fetal heart rate ranges from 110–160 per minute.

Continuous electronic fetal monitoring/CTG: Recording of fetal heart rate and uterine contraction. It is commonly used for high risk pregnancies.

Vaginal examination: (To get information as mentioned above).

To Watch for Maternal and Fetal Distress

Evidences of Fetal Distress

Loss of beat to beat variability, decelerations, tachycardia, bradycardia.

Evidence of maternal distress are:
- Tachycardia (>100/minute)
- Anxious look with sunken eyes
- Dehydration, dry tongue, acetone smell in breath
- Hot, dry vagina often with offensive discharge
- Scanty high colored urine with presence of acetone.

14 Malpresentations and Malposition

Q. Etiology of breech and types of breech.

INTRODUCTION

Breech is the **most common malpresentation.** The lie is longitudinal and the podalic pole presents at the pelvic brim.

Etiology

Prematurity is the most common cause.

Fetal
- Multiple pregnancy
- Hydrocephalus (big head can fit well in the fundus)
- Polyhydramnios/oligohydramnios
- Trisomies, anencephaly and myotonic dystrophy.

Maternal
- Congenital malformation of the uterus
- Multiparity
- CPD
- Uterine fibroid/pelvic tumors
- Past history.

Placental
- Placenta previa
- Cornufundal attachment of placenta
- Short cord.

Prevalence of Breech Presentation by Gestational Age

Gestational age (weeks)	Breech (%)
28	24
30	17
32	11
34	6
36	5
37–40	4

Types of Breech

- ***Complete:*** Full flexed attitude, thighs are flexed at the hips and legs are flexed at the knees, so the presenting part comprises of the buttocks, external genitalia and two feet. It is usually seen in multipara.
- ***Incomplete:***
  - **Frank breech:** Thighs are flexed at the hips and legs are extended at the knees. The presenting part consists of external genitalia and buttocks. It is **commonly seen in primigravidas** (70%) due to tight abdominal wall, good uterine tone and early engagement
  - **Footling presentation:** Both the thighs and the legs are partially extended bringing the legs at the pelvic brim
  - **Knee presentation:** Thighs are extended but the knees are flexed, bringing the knees to the pelvic brim.

Q. Complications in breech presentation.

INTRODUCTION

Breech is the most common malpresentation. The lie is longitudinal and the podalic pole presents at the pelvic brim.

Complications: Maternal and Fetal

- Maternal:
  - Increased frequency of LSCS and operative vaginal delivery
  - Genital tract trauma
  - Anesthesia complications

- **Fetal:**

The corrected **perinatal mortality** (excluding congenital abnormalities) is **5–35/1000 births**. The fetal mortality is **least in frank breech and maximum in footling presentation** due to higher occurrence of cord prolapse.

- **Intrapartum fetal death**
- **Intracranial hemorrhage:** Compression followed by decompression of unmolded head leads to tear of tentorium cerebelli. This is more in preterm babies
- **Birth asphyxia** due to:
  - Cord compression after buttocks are delivered and when the head enters the pelvis
  - Retraction of the placental site
  - Premature attempt at respiration when the head is still inside
  - Delayed delivery of the head and
  - **Cord prolapse.**
- **Birth injuries** usually inflicted during manipulative deliveries:
  - Hematoma: Sternomastoid/thighs
  - **Fractures:** Femur/humerus/clavicle/odontoid process. There may be dislocation of the hip joint, mandible, C5 and C6 vertebrae
  - **Visceral injuries:** Rupture of liver, kidneys, adrenals, lungs and testicular hemorrhage

  - Nerve: Medullary coning, spinal cord injury, **brachial plexus** stretching leading to Erb's/Klumpkes paralysis
  - Long term neurological damage.

Q. ECV.

Q. Mention complications of ECV and what are the indications of LSCS for breech presentation.

External cephalic version (ECV) is a procedure used to turn a fetus from a breech position or transverse position into a **vertex position before labor begins**. It is done to bring the favorable cephalic pole in the lower pole of the uterus to **avoid complications of vaginal breech delivery and also to avoid LSCS.**

The **ACOG recommends** that efforts should be made to reduce breech presentation by external cephalic version (ECV) whenever possible.

The success rate for external cephalic version ranges from **35 to 85%, with an average of about 60%.**

When to perform:

ECV should be performed at **36 weeks of gestation** for the following reasons:

- If version results in fetal distress and need for immediate LSCS, iatrogenic prematurity is avoided
- The likelihood of spontaneous version is low
- An additional consideration in timing the version is that, although earlier attempts are more likely to be successful, they also are more likely to be associated with spontaneous reversion to breech.

Procedure

- It should be attempted at 36 weeks in labor-delivery complex
- Tocolytic drug should be given
- USG should be done to confirm the diagnosis and AFI
- **A reactive NST pre-procedure** should be ensured
- After emptying her bladder, the patient should be made to lie supine with her shoulders slightly raised and thighs slightly flexed
- Forward roll movement: The breech is mobilized with both hands to one iliac fossa towards which the fetal back lies. The podalic pole is grasped by the right hand like that of Pawliks grip and the left hand grasps the head. A firm intermittent unforceful pressure is exerted to the head and breech in opposite directions to keep the trunk well-flexed which facilitates the version and pushes the head towards the pelvis and breech towards the fundus till the lie becomes transverse. **The FHS should be checked.** The hand should be changed one after the other so as to hold the fetal poles without crossing over of hands. The intermittent pressure is exerted till the head is brought to the lower pole of the uterus
- **Postprocedure, a reactive NST should be obtained.** There may be transient bradycardia due to head compression which should settle within 10 minutes. If it persists, possibility of cord entanglement should be considered and reversion should be done

- The patient should be observed for 30 minutes for FHS monitoring and to assess for vaginal bleeding in case PROM has occurred
- Rh negative nonimmunized women should be given anti-D gamma globulin.

Contraindications for ECV

- Multiple pregnancy
- Previous LSCS (RISK OF SCAR RUPTURE)
- Severe preeclampsia (risk of abruption)
- Oligo/polyhydramnios
- Placenta previa/contracted pelvis **(version should not be attempted if there is a contraindication to vaginal delivery)**
- BOH
- IUGR, IUFD, Large fetus, major congenital anomalies
- PROM.

Complications of ECV

- Fetal distress
- IUFD
- Preterm labor, PROM
- Abruption
- Cord entanglement
- Amniotic fluid embolism
- Fetomaternal hemorrhage.

Successful Version is usually noted in:

- Complete breech
- Nonengaged breech
- Sacroanterior position
- Adequate liquor
- Nonobese patient.

Causes of Failed ECV

- Frank breech: Early engagement and difficulty in flexion due to splinting action of the fetal limbs
- Scanty liquor or big baby
- Mechanical: Obesity, irritable uterus, increased tone of abdominal muscles
- Short cord
- Uterine malformations: Septate/bicornuate.

Indications for Cesarean Section in Breech Presentation

Because of complications of vaginal breech delivery, LSCS in breech presentation is done **mainly for a good fetal outcome.**

Compared with vaginal birth, **planned cesarean section reduces perinatal or neonatal death** or serious neonatal morbidity. Hence in following cases LSCS is done:

- **Primi with breech: In modern day obstetrics primigravida with breech presentation should be delivered by LSCS**

- **Footling breech (very high risk of cord prolapse)**
- **Twins with first baby in breech** (risk of interlocking of twins and risk of cord prolapse)
- Previous LSCS with breech (risk of scar rupture)
- **Preterm breech** (risk of intraventricular hemorrhage increases with vaginal delivery)
- **Stargazing/flying fetus:** In perhaps 5% of term breech presentations, the fetal head may be in extreme hyperextension. This presentation is referred to as the stargazer fetus or the flying fetus. With such hyperextension, vaginal delivery may result in injury to the cervical spinal cord
- Big baby (fetal weight > 3.5 kgs) or contracted pelvis
- Precious pregnancy (like BOH, previous stillbirths, previous complications during vaginal breech delivery, IVF conception, etc.).

Q. Etiology of transverse lie.

INTRODUCTION

Definition

When the long axis of fetus lies perpendicular to the maternal spine or centralized uterine axis, it is called transverse lie. In transverse lie **shoulder is the presenting part.**

The dorsoanterior position is most common (60%).

- In dorsoposterior, the chance of fetal extension is common with increased risk of arm prolapse and cord prolapse.

Etiology

- Multiparity: Lax abdomen and imperfect uterine tone
- Prematurity
- Multiple pregnancy: More common for the second baby
- Polyhydramnios
- Uterine anomalies
- Placenta previa
- Pelvic tumors (fibroids/ovarian cysts)
- CPD/contracted pelvis.

There is no mechanism of labor in transverse lie. Delivery is by LSCS.

If the fetus is small (usually < 800 g) and the pelvis is large, spontaneous delivery is possible in transverse lie. The fetus is compressed with the head forced against the abdomen. A portion of the thoracic wall below the shoulder thus becomes the most dependent part, appearing at the vulva. The head and thorax then pass through the pelvic cavity at the same time, and the fetus, which is doubled upon itself, is expelled—this is referred to as **conduplicato corpore.**

Q. Clinical diagnosis and mechanism of labor in OP.

Q. Management of OP.

Q. Various outcome in OP.

Occipitoposterior Position

In a vertex presentation when the occiput is placed directly over the sacrum or sacroiliac joint, it is called an occipitoposterior position.

Incidence

- 10% of all vertex positions
- **Right OP is 5 times more common** than left OP since dextrorotation of the uterus and presence of the sigmoid colon on the left disfavor LOP.

Causes

- Pelvic Inlet: Usually associated with anthropoid or android pelvis
- Fetal head deflexion due to:
  - High pelvic inclination
  - Anterior placentation
  - Primary brachycephaly
- Abnormal uterine contraction.

Diagnosis

Abdominal Examination:

Umbilical Grip

- Fetal limbs are felt more easily on either side of the midline
- Fetal back is felt in the flank away from the midline
- Anterior shoulder away from the midline.

Pelvic Grip

- Head is not engaged
- The cephalic prominence (sinciput) is not as prominent as it is in OA.

Auscultation

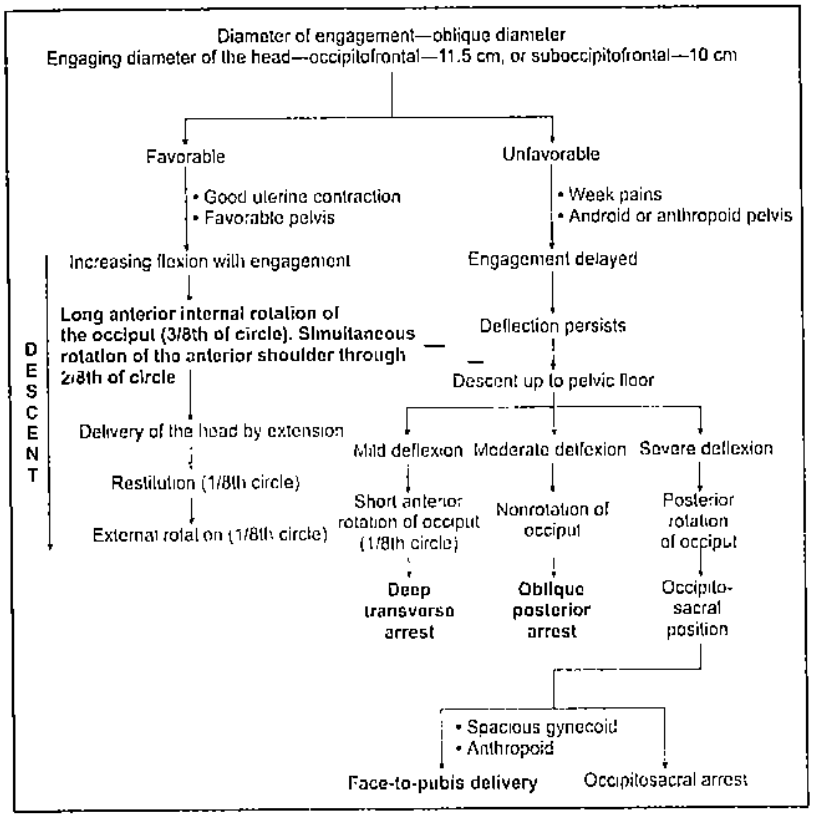
Maximum intensity of **FHS is on the flank** and often difficult to locate in LOP. **In direct OP, the FHS is noted directly in the midline.**

P/V

- Elongated bag of membranes
- Sagittal suture occupies any oblique diameters of the pelvis
- Posterior fontanelle is felt near the sacroiliac joint
- **Anterior fontanelle is felt more easily** and at a lower level than the posterior one
- In late labor, caput obliterates the sutures and fontanelles. For diagnosis, **the unfolded pinna points towards the occiput.**

Mechanism of Labor

Flow Chart 14.1: Scheme of mechanism of labor in occipitoposterior position



Course of Labor

Both first and second stage of labor are longer.

First Stage

Increased duration due to:

- **Delayed engagement** due to persistent deflexion of the head thereby increasing the diameter of engagement (occipitofrontal = 11.5 cm). The driving force transmitted through the fetal axis is not in alignment with the axis of the inlet
- Membrane status: Deflexed head becomes ovoid and this cannot fit well the spherical lower segment → loss of ball valve action during uterine contraction → early rupture of membranes and drainage of liquor

- Uterine contractions may be abnormal with slow cervical dilatation due to ill fitting of the deflexed head to the lower uterine segment. The occiput presses on the rectum causing premature desire to bear down in the first stage.

Second Stage

It is often delayed due to **long internal rotation or malrotation, with arrest** of the head at times.

Third Stage

Increased incidence of PPH and trauma to the genital tract.

Mode of Delivery

1. Long anterior rotation of occiput (OP becomes OA): Spontaneous or assisted vaginal delivery happens (90%).
2. Short posterior rotation: Spontaneous or assisted vaginal delivery may happen as face to **pubis** leading to increased risk of perineal injuries or there may be occipitosacral arrest.

   Reason for perineal injury: BPD (9.5 cm) stretches the perineum and occipitofrontal diameter (11.5 cm) emerges out of the introitus
3. Nonrotation or short anterior rotation: Spontaneous vaginal delivery is very rare. LSCS is done, else it may lead to prolonged/obstructed labor.

Management of Labor

The principles are:

- Early diagnosis
- Strict vigilance and watchful expectancy
- Judicious and timely interference.

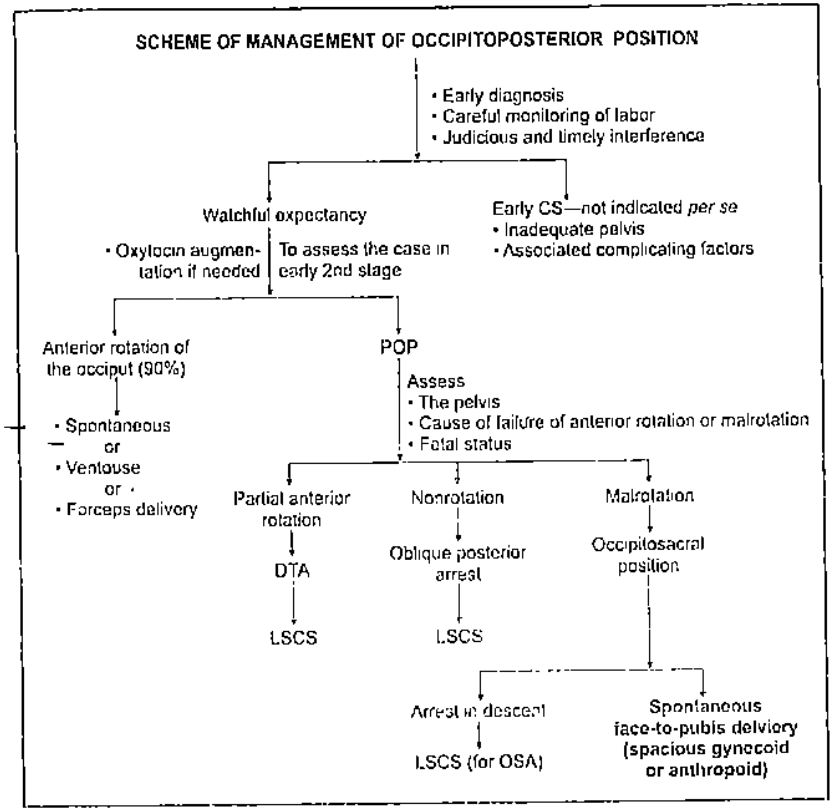
OP is per say not an indication for LSCS. In cases of pelvis inadequacy LSCS should be done.

- Labor is allowed to proceed like normal labor
- IV line, RL infusion started
- Blood to be crossed match and kept ready (LSCS may be needed and more risk of PPH).

Indications for LSCS in cases of OP:

- DTA
- Oblique posterior arrest
- Occipitosacral arrest (OSA) (Flow Chart 14.2).

Flow Chart 14.2: Scheme of management of occipitoposterior position



Q. Deep transverse arrest (DTA).

DEFINITION

The head is **deep into the cavity**, sagittal suture is in the **transverse bispinous diameter** and there is no progress in descent of the head even after 1 hour of full cervical dilatation.

This arrest may be due to nonrotation of primary occipitotransverse position or incomplete anterior rotation (1/8th of a circle) of oblique OP position.

Causes

- Pelvic architecture: Android pelvis
- Deflexed head
- Weak uterine contractions.

Diagnosis

- The head is engaged
- Sagittal suture is in the transverse bispinous diameter
- Anterior fontanelle is palpable.

Management

- If vaginal delivery is unsafe due to big baby or inadequate pelvis → LSCS
- If vaginal delivery is safe and the obstetrician is skilled, operative vaginal delivery can be attempted. Methods are:
  - Ventouse
  - Manual rotation and forceps application.
  - Forceps rotation and delivery with Kiellands forceps.

However in modern day obstetrics, manual rotation and Kiellands forceps are not done and LSCS is always to be done and preferred.

Complications

1. Prolonged labor
2. Obstructed labor with higher incidence of rupture uterus
3. Increased incidence of operative delivery
4. Increased incidence of trauma to the genital tract
5. Increased incidence of postpartum hemorrhage and puerperal infection
6. Increased incidence of perinatal morbidity and mortality.

15 Abortions/ Miscarriages

Q. Define spontaneous abortion. Discuss the etiologies of abortion in first and second trimester?

DEFINITIONS

Abortion is defined as expulsion/extraction from its mother of an embryo or fetus weighing **500 gms or less** when it is not capable of independent survival.

Abortion occurring without **medical or mechanical means** to empty the uterus is referred to as spontaneous. Spontaneous miscarriage is typically defined as a clinically recognized (i.e. by blood test, urine test, or ultrasonography) pregnancy loss before 20 weeks' gestation.

Approximately 5–15% of diagnosed pregnancies result in spontaneous miscarriage. **Seventy-five percent of abortion happen before 16 weeks and out of these around 75% happen before 8 weeks of gestation.**

ETIOLOGY

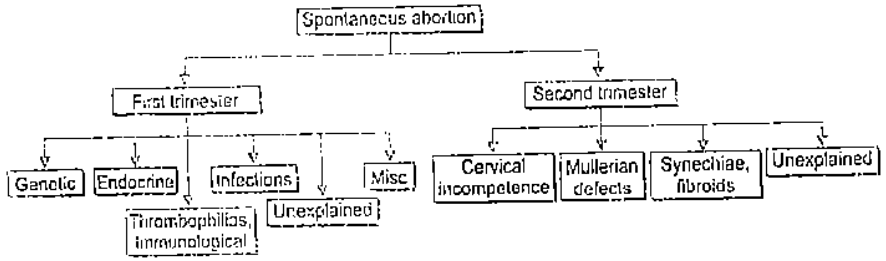
The causes of spontaneous abortions in first trimester are different from those in the second trimester.

Common Causes of Abortion

First Trimester

- **Genetic factors (50%):**
  - More than 80% of abortions occur in the first 12 weeks of pregnancy, and at least half result from chromosomal anomalies. After the first trimester, both the abortion rate and the incidence of chromosomal anomalies decrease. **Advance maternal age** is contributory factor.

Flow Chart 15.1: Main causes of abortion in first and second trimester



- **Trisomy 16 is the most common abnormal karyotype found in the abortus**
- **Monosomy X (45 X), the second most frequent** chromosomal abnormality (after trisomy), usually results in abortion and much less frequently in live born female infants (Turner syndrome)
- Advanced maternal and paternal ages do not increase the incidence of triploidy
- Euploid abortion: Euploid fetuses tend to abort later in gestation than aneuploid ones. Three-fourths of aneuploid abortions occur before 8 weeks; euploid abortions peak at about 13 weeks. The incidence of euploid abortions increase dramatically after maternal age exceeds 35 years.

- **Endocrine disorders (10–15%):**
  - **Luteal phase defect**/progesterone deficiency would always result in early first trimester abortions **(mainly before 8 weeks)**. After around 10 weeks the placenta completely secretes progesterone
  - **Thyroid disorders** both hypo and hyperthyroidism can lead to recurrent first trimester abortion
  - **Overt diabetes mellitus** would also lead to congenital anomalies and recurrent first trimester abortions.
- **Thrombophilias and Immunological disorders (autoimmune and alloimmune)-10%**
  - **Autoimmune factors:** (ANA and ANTI DNA antibodies and antiphospholipid antibodies)
  - Antiphospholipid antibodies are a family of autoantibodies that bind to negatively charged phospholipids, phospholipids-binding proteins, or a combination of the two. Two of these are **lupus anticoagulant and anticardiolipin antibody**, and have been implicated in spontaneous abortion
  - The mechanism of pregnancy loss in women with these antibodies involves **placental thrombosis and infarction**
  - In one postulated mechanism, antibodies may inhibit the release of prostacyclin, a potent vasodilator and inhibitor of platelet aggregation. In contrast, platelets produce thromboxane A2, a vasoconstrictor and platelet aggregator. They have also been shown to inhibit protein C activation,resulting in coagulation and fibrin formation
  - **Alloimmune disease:** Normally there are blocking antibodies which prevent the maternal immune cells from recognizing the fetus as foreign and prevent abortion
  - Lack of production of these antibodies can lead to abortion
  - **Inherited thrombophilias (protein C and S deficiency, factor-V Leiden mutation and hyperhomocysteinemia)** can lead to early and late miscarriages due to intravascular coagulation.
- **Infection:** For example, rubella, cytomegalovirus, and mycoplasmal, ureaplasmal, listerial, toxoplasmal infections.
- **Unexplained**
- Trauma/severe emotional shock.

Other factors that may contribute to miscarriage:

- **Exogenous factors**
  - Alcohol
  - Tobacco
  - Cocaine and other illicit drugs.

- **Chronic maternal health factors:** Severe hypertension, renal disease, systemic lupus erythematosus (SLE) can lead to both first and second trimester losses.

Second Trimester

- **Anatomic abnormalities:**
  - **Cervical incompetence** (congenital or acquired) (details in the next answer)
  - **Mullerian Fusion defects** (bicornuate uterus and unicornuate/septate uterus)
  - **Uterine synechiae:** Asherman's syndrome, characterized by uterine synechiae, usually results from destruction of large areas of endometrium by overzealous curettage.
    The risk is maximum if curettage is done in the postpartum period.
    If pregnancy follows, the amount of remaining endometrium may be insufficient to support the pregnancy, and abortion may ensue. A hysterosalpingogram that shows characteristic multiple filling defects may indicate Asherman syndrome, but hysteroscopy most accurately and directly identifies this condition.
- Uterine fibroid.
- **Maternal medical illness:** Chronic maternal health factors: Severe hypertension, renal disease, systemic lupus erythematosus (SLE) can lead to both first and second trimester losses.
- Unexplained.

Q. What are the causes and management of cervical incompetence?

Q. Cervical encerclage.

Q. OS tightening.

CERVICAL INCOMPETENCE

Definition

Mechanical or functional defect in the cervix which leads to inability of cervix to hold pregnancy.

Etiology

- Although the cause of cervical incompetence is obscure, previous **trauma** to the cervix—especially in the course of dilatation and curettage, **conization**, cauterization, or **amputation**—appears to be a factor in some cases
- In other instances, abnormal cervical development, including that following exposure to diethylstilbestrol in utero, may play a role
- It is also **a/w uterine anomalies** like bicornuate, unicornuate uterus
- Multiple pregnancy, past history.

Clinical Features

Classically, it is characterized by **painless** cervical dilatation in the **second trimester**, with prolapse and ballooning of membranes into the vagina, preterm premature rupture of membranes **(PPROM)**, followed by expulsion of an immature fetus. Unless effectively treated, this sequence may **repeat in future pregnancies**.

Internal examination in inter conceptional may reveal cervical tear (unilateral or bilateral) and gaping of cervix.

Investigations

The diagnosis is generally made on the **classical history.**

In the interconceptinol period:

1. Passage of no 6–8 Hegar's dilator without pain and absence of snap on withdrawal
2. Premenstrual hysterocervicography shows a **funnel shaped shadow** (the cervix is supposed to be closed due to progesterone).

During Pregnancy

1. **Cervical length less than 2.5 cm** is considered as short cervix
2. Funneling—ballooning of the membranes into a dilated internal OS >1 cm but with a closed external OS may also be seen
3. PS: Dilatation of cervix and herniation of membranes may be seen.

Treatment

- The treatment of classical cervical incompetence is **cerclage (OS tightening).**
- The operation is performed to surgically reinforce the weak cervix by some type of purse-string suturing.
- Cerclage procedure: Two types of vaginal operations are commonly used during pregnancy. One is **McDonald** and the other is **Shirodkar**. Success rates of these operations is **80–90%.**

Timing of Operation

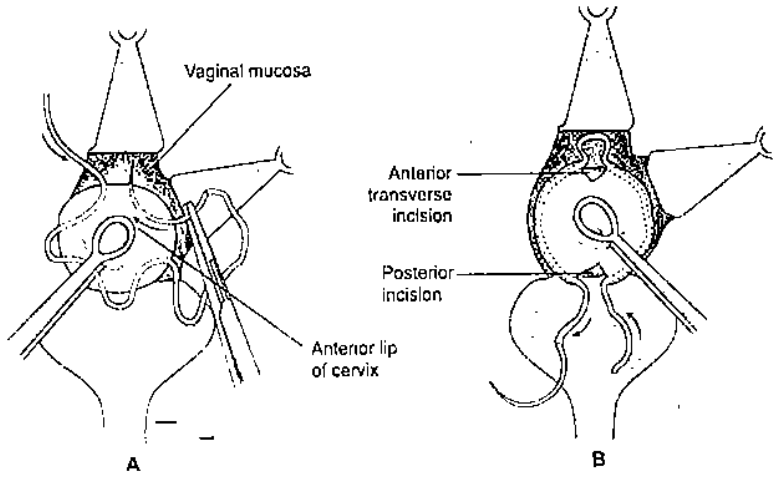
- Emergency cerclage is done when there is **cervical dilatation** and bulging membranes or detection of **short cervix (<2.5 cm) on USG**
- Prophylactic cercalge is done 2 weeks prior to the previous second trimester abortion.

Anesthesia

Regional (spinal or caudal block) or short general anesthesia (TIVA) (Figs 15.1A and B).

McDonald's Cerclage

- The McDonald's cerclage is performed using a permanent suture
- The bladder emptied, the cervix is exposed
- A purse string suture of **nonabsorbable material** like mersilene or Mersilk on a Mayo needle is inserted around the exocervix as high as possible to approximate to the level of the internal OS. This is at the junction of the rugose vagina and smooth cervix
- Five or six bites with the needle are made
- The stitch is pulled tight enough to close the internal OS, the knot being made in **front of the cervix.**



Figs 15.1 (A and B): Circlage operation—(A) McDonald technique; (B) Shirodkar's technique

Shirodkar's Cerclage

- The cervix is pulled down, a transverse incision is made above the cervix and the bladder is pushed well up above the internal OS
- A vertical incision is made posteriorly on cervicovaginal junction
- The **nonabsorbable suture** material (merseline) is passed **submucously** through the right and left corner of the anterior incision with an aneurysm needle so as to bring it out of the posterior incision
- The ends of the tape tied **posteriorly**
- The anterior and posterior incisions are closed with chromic catgut no. 0.

Postoperative

- **Bedrest** for 2–3 days
- **Uterine relaxants** (tocolytics like isoxsuprine, ritodrine, etc.) to be given (injectables followed by oral) for few days.

Advise on Discharge

- Avoid intercourse
- Routine antenatal care
- To report if bleeding, leaking PV or pain in abdomen.

Contraindications

- Vaginal bleeding
- Uterine contractions

- Ruptured membranes
- Intrauterine infections
- IUFD/severe congenital anomalies
- Dilation >4 cm.

Removal of Stitch

The knot is usually cut at **37 weeks** or any time **before, if the patient goes in labor.** If the knot is not cut, then during labor there can be cervical tears or rupture uterus.

Complications

While cerclage is generally a safe procedure, there are a number of potential complications that may arise during or after surgery. These include:

Immediate

- Risks associated with regional or general anesthesia
- Premature labor
- Premature rupture of membranes
- Chorioamnionitis
- Injury to the cervix or bladder
- Bleeding.

Delayed

- Cervical dystocia with failure to dilate requiring cesarean section
- Cervical tear/uterine rupture (may occur if the stitch is not removed before onset of labor)

Benson and Durfee is an **abdominal encerclage** operation reserved in cases when previously vaginal operations have failed (abortion has occurred in spite of vaginal cerclage).

Similar surgery can be done **laparoscopically during the nonpregnant state.**

16 Ectopic Pregnancy

Q. Discuss etiology of ectopic pregnancy.

Q. Signs and symptoms and management of acute/ruptured ectopic pregnancy.

Q. Signs and symptoms and management of unruptured ectopic pregnancy.

DEFINITION

The term ectopic is derived from the Greek word **ektopos, meaning out of place.**

In ectopic pregnancy the fertilized ovum is implanted and develops **outside the normal endometrial cavity,** the gestation grows and draws its blood supply from the site of abnormal implantation.

Incidence

- Increased due to increase in pelvic inflammatory disease, tuboplasty surgeries, IVF, and ovulation induction. The incidence is around **1 in 150–300 deliveries.**

Etiology

Pelvic Inflammatory Disease (PID)

- The most common cause of PID is an antecedent infection caused by Chlamydia trachomatis. Other organisms such as Neisseria gonorrhea, also increase the risk of ectopic pregnancy, and a history of salpingitis **increases the risk of ectopic pregnancy 4–10 fold**
- Genital TB also increases the risk of ectopic pregnancy
- Loss of cilia, narrowing of lumen and intra and peri tubal adhesions contribute.

Contraception Failure

- All contraceptive methods lead to an overall lower risk of pregnancy and therefore to an **overall lower risk** of ectopic pregnancy. However, **contraceptive failure, increases the risk of ectopic pregnancy.**
- IUCD: The modern copper IUD does not increase the risk of ectopic pregnancy. However, there is a **relative increase in tubal pregnancy** (7 times more) should pregnancy occur with IUCD in situ.
- Only progestasert has a rate of ectopic pregnancy higher than that for women not using any form of contraception.
- Sterilization: **There is 15–50% chance of ectopic pregnancy if pregnancy occurs (sterilization failure). The risk is highest with bipolar congulation.**

Tubal Surgery

- Previous tubal surgery has been demonstrated to increase the risk of developing ectopic pregnancy. Surgeries carrying higher risk of subsequent ectopic pregnancy include salpingostomy, neosalpingostomy, fimbrioplasty, tubal reanastomosis, and lysis of peri-tubal or periovarian adhesions.

History of Previous Ectopic Pregnancy

- After one ectopic pregnancy, a patient incurs a 7 to 13 fold increase in the likelihood of another ectopic pregnancy. Overall, a patient with a previous ectopic pregnancy has a **10–25% chance of a future tubal pregnancy.**

ART

- Ovulation induction with clomiphene citrate or gonadotropin therapy has been linked to a 4-fold increase in the risk of ectopic pregnancy.
  - The risk of ectopic pregnancy and heterotopic pregnancy dramatically increases when a patient has used assisted reproductive techniques—such as in vitro fertilization (IVF) or gamete intrafallopian transfer (GIFT)—to conceive. **The risk of ectopic is 5–7%**
  - Studies have demonstrated that up to 1% of pregnancies achieved through IVF or GIFT can result in a heterotopic gestation, compared with an incidence of 1 in 30,000 pregnancies for spontaneous conceptions.

Pelvic Adhesions

- Due to previous pelvic or abdominal surgery.

Increasing Age

The highest rate of ectopic pregnancy occurs in women aged 35–44 years. A 3- to 4-fold increase in the risk of developing an ectopic pregnancy exists compared with women aged 15–24 years.

Smoking

Cigarette smoking has been shown to be a risk factor for ectopic pregnancy development. Studies have demonstrated an elevated risk ranging from **1.6 to 3.5 times** that of nonsmokers.

Clinical Features

- **Acute (ruptured)**
- **Unruptured**
- **Subacute (chronic)**

ACUTE

Symptoms

- It is a/w tubal rupture or tubal abortion and massive intraperitoneal hemorrhage.
- Only 50% of patients with an ectopic pregnancy present with the **classic triad of abdominal pain, amenorrhea, and vaginal bleeding.**
- **Abdominal Pain:** It is the **most constant** feature (seen in almost 100% patients). It is acute, agonizing or colicky, in lower abdomen unilateral, bilateral or generalized. Shoulder tip pain (due to diaphragmatic irritation from hemoperitoneum) is seen in around 25% patients.
- **Amenorrhea:** There is generally short period (5–8 weeks) of amenorrhea (in around 75% patients) or it may even be absent.
- **Bleeding PV:** May be spotting or slight continuous bleeding (in around 70%). This is due to shedding of endometrium (due to decreased progesterone as there is insufficient HCG). There may also be expulsion of endometrial cast. Very rarely the bleeding maybe due to tubal abortion through uterine ostium in interstitial pregnancy.
- **Syncopal attack** and vomiting due to peritoneal irritation.

Signs

- Pallor present
- Tachycardia, feeble pulse
- Hypotension
- Cold clammy extremities
- Lower abdomen is **tense and tender, no mass felt** and shifting dullness is present
- Abdominal rigidity, involuntary guarding, and severe tenderness, as well as evidence of hypovolemic shock, such as orthostatic blood pressure changes and tachycardia, should alert the clinician to a surgical emergency; this may occur in up to 20% of cases.

PV

- Vaginal mucosa is blanched
- Uterus: Slightly bulky or normal size and **floats as if in water**
- Cervical motion tenderness
- Unilateral or bilateral tenderness on fornix palpation—Usually much worse on the affected side
- **No mass felt through the fornix usually.**

The presence of uterine contents in the vagina, which can be caused by shedding of endometrial lining stimulated by an ectopic pregnancy, may lead to a misdiagnosis of an incomplete or complete abortion and therefore a delayed or missed diagnosis of ectopic pregnancy.

UNRUPTURED TUBAL PREGNANCY

The physician should be **ectopic minded** and include ectopic pregnancy in D/D when a sexually active female has abnormal bleeding, more so in patients with risk factors.

Symptoms

- Presence of delayed periods/spotting with **features of early pregnancy**
- Flank pain, mild, colicky or continuous.

Signs: Bimanual Examination

The palpation should be **gentle to avoid iatrogenic rupture**

- Uterus is soft, normal or just bulky

- A well circumscribed, **pulsatile, tender mass felt through one of the fornices, separate from the uterus.**

CHRONIC/OLD ECTOPIC

Symptoms

- Short period of amenorrhea, 6–8 weeks
- *Lower abdominal pain:* Starts as acute and gradually becomes dull or colicky
- *Vaginal bleeding:* Scanty, dark and continuous. There could be expulsion of endometrial cast
- Bladder symptoms like dysuria, frequency or retention may be present
- Rectal tenesmus if infected hematocele.

Signs

- Patient looks ill with varying degree of pallor
- Tachycardia, even at rest
- **Features of shock are absent**
- Temperature: May be mildy elevated.

Abdominal Examination

Tenderness and guarding in lower abdomen on the affected side
- Irregular and tender mass may be felt in lower abdomen
- **Cullen's sign:** Dark bluish discoloration around umbilicus, suggestive of intraperitoneal hemorrhage
- *Bimanual examination:* Vaginal mucosa-pale
- Uterus normal size or bulky and may be pushed to one side
- **Cervical movement tenderness ++**
- **Ill defined, tender, boggy mass** felt through the posterolateral fornix, which may push the uterus to opposite side.

DD of Acute Ectopic

- Acute appendicitis
- Ruptured corpus luteum
- Ruptured chocolate cyst
- Ovarian torsion
- Perforated peptic ulcer.

Pregnancy test would be negative in all of the above.

DD of Chronic Ectopic

- Incomplete abortion
- Appendicitis
- Salpingitis
- Ruptured corpus luteum
- Ruptured chocolate cyst
- Ovarian torsion.

MANAGEMENT

Acute

Shock in early pregnancy should be thought to be due to ruptured ectopic unless proven otherwise.

Investigations

As it is an **emergency situation**, only hemoglobin and blood group and cross match should be done.

Treatment

- The principle is **resuscitation and laparotomy**
- Two wide bore IV lines
- IV fluids: RL (Crystalloid) and Colloids (hemaccel)
- Blood transfusion
- Exploratory laparotomy **(quick in, quick out)**
- **Linear salpingectomy** is the gold standard surgery. The tube is sent for histopathology
- The blood supply to the ovary is preserved, and oophorectomy is not to be done.

Chronic Ectopic Pregnancy

Investigations

- **Blood:**
  - CBC (Hemoglobin and total WBC count)
  - Blood grouping and cross match.
- **Serial beta hCG:**
  - Beta hCG is positive. A single value is not important as it diagnoses pregnancy but not its location. However a lower level than weeks of gestation would raise suspicion.
  - **Beta hCG would not double after 48 hours.**
  - As per the **Kadar's rule there would be < 66% increase** in beta hCG in cases of ectopic pregnancy.
- **USG:** TVS is the most important.
  - Uterus is empty. **Pseudo sac** maybe seen
  - Echogenic fluid in pouch of Douglas
  - Adenexal mass, separate from ovary
  - On **color Doppler, ring of fire appearance** around the mass.
- **Laparoscopy:** If the patient is hemodynamically stable, laparoscopy is preferred as it is not only diagnostic but also therapeutic. The blood in the POD is aspirated and salpingectomy can be done.
- **Sr progesterone:** > 25 ng/ml is suggestive of viable intrauterine pregnancy and < 5 ng/ml suggest an ectopic or abnormal intrauterine pregnancy.
- **Culdocentesis:** Very rarely done today. In absence of TVS or laparoscopy, it can be done through 18 G lumbar puncture needle, posterior fornix is punctured.

However a **negative culdocentesis does not rule out an ectopic, neither a positive result is specific.**

- **Dilatation and Curettage:** Very rarely needed. Chorionic villi float in normal saline is diagnostic of intrauterine pregnancy.

Management

If patient is unstable or if facilities for laparoscopy are not available:

- Exploratory laparotomy (quick in , quick out).
- Linear salpingectomy is the gold standard. The tube is sent for histopathology.
- The blood supply to the ovary is preserved, and oophorectomy is not to be done.
- If the patient is hemodynamically stable, **laparoscopy is preferred** as it is not only diagnostic but also therapeutic. The blood in the POD is aspirated and salpingectomy can be done.

Unruptured Ectopic

Investigations

- Serial beta hCG monitoring
- TVS
- Laparoscopy
  - Beta hCG is positive. A single value is not important as it diagnoses pregnancy but not its location. However, a lower level than weeks of gestation would raise suspicion.
  - Beta hCG would not double after 48 hours.
  - As per the **Kadar's rule there would be <66% increase** in beta hCG in cases of ectopic pregnancy.
  - USG (especially TVS) is probably the most important tool for diagnosing an extrauterine pregnancy, although it is more frequently used to confirm an intrauterine pregnancy.
    - **Presumed ectopic pregnancy:** An empty uterus on TVS in patients with a **serum β-hCG levels greater than** the discriminatory cut-off value is an ectopic pregnancy until proven otherwise. The endometrium may be thick and/or there could be presence of a pseudo sac.
    - **Definite ectopic pregnancy:** Presence of a thick, brightly echogenic, ring-like structure is located outside the uterus, with a gestational sac containing an obvious fetal pole, a yolk sac, or both. The endometrium could be thick or shows pseudo sac.
    - The presence of a tender adnexal mass on USG suggests an ectopic pregnancy.
  - Laparoscopy can be both **diagnostic as well as therapeutic.**

Management: Medical and Surgical

Medical management (Methotrexate) is the treatment of choice for an ectopic pregnancy whenever the required criteria are fulfilled.

The following criteria should be fulfilled for medical management of ectopic pregnancy:

- Patient should be **hemodynamically stable (unruptured tubal ectopic pregnancy)**

- Fetal cardiac activity absent. (Presence of cardiac activity is a relative contraindication)
- β-hCG levels < 5,000 μIU/mL (levels > 5,000 micro IU/mL is a relative contraindication)
- Gestational sac diameter **< 4 cm**
- Free fluid in POD < 100 mL
  - According to ACOG contraindications for methotrexate include: breastfeeding, alcoholism, immunodeficiency, liver or renal disease, blood dyscrasias, active pulmonary disease and peptic ulcer.

Candidates for methotrexate therapy must be hemodynamically stable. They are instructed that:

- Medical therapy fails in at least **5–10% of cases.**
- If tubal rupture occurs (a 5–10% chance), emergency surgery is necessary.
- If the woman is treated as an outpatient, rapid transportation must be reliably available.
- Signs and symptoms of tubal rupture such as vaginal bleeding, abdominal and pleuritic pain, weakness, dizziness, or syncope must be reported promptly.
- Until the ectopic pregnancy is resolved, sexual intercourse is prohibited, alcohol should be avoided, and folic acid supplements—including prenatal vitamins—should not be taken.

Methotrexate Therapy for Primary Treatment of Ectopic Pregnancy

Regimen	Follow-up
Single dose: Methotrexate, 50 mg/m²	Measure β-hCG at days 4 and 7 If difference is ≥ 15%, repeat weekly until undetectable If difference is <15%, repeat methotrexate dose and begin new day 1 If fetal cardiac activity present at day 7, repeat methotrexate dose, begin new day Surgical treatment if β-hCG levels not decreasing or fetal cardiac activity persists after three **doses of methotrexate**
Variable dose	
Methotrexate, 1 mg/kg IM, days 1, 3, 5, and 7 plus leukovorin, 0.1 mg/kg IM, days 2, 4, 6, and 8	Continue alternate day injection until β-hCG levels decreases to >15% in 48 h, or four does methotrexate given. Then, weekly β-hCG until undetectable

Surgical

Laparoscopy is preferred. If facilities are not available then laparotomy maybe done.
Salpingectomy is preferred.
Conservative surgeries: **Not routinely recommended because of high-risk of recurrence on the same side.**

Various conservative surgeries include:

- **Linear salpingostomy:** Linear incision on tube, remove the products, incision kept open
- **Linear salpingotomy:** Like above, but the incision is closed
- Segmental resection
- **Fimbrial expression:** In cases of distal pregnancy.

Anti D Injection

- In all Rh negative patients (and if Rh positive partner) injection **anti D** is to be given **50 mcg (<12 weeks gestation)** or **300 mcg (>12 weeks gestation)** in all cases of ectopic pregnancy (ruptured, chronic, unruptured)

In future pregnancy, TVS should be done early as there is a risk of recurrence.

17 Preeclampsia/ Eclampsia

Q. Classify hypertensive disorders of pregnancy.

Diagnosis of Hypertensive Disorders Complicating Pregnancy:

GESTATIONAL HYPERTENSION

- BP ≥ 140/90 mm Hg for first time during pregnancy
- **No proteinuria**
- BP returns to normal within 12 weeks postpartum
- Final diagnosis made only postpartum.

PREECLAMPSIA

Minimum Criteria

- BP ≥ 140/90 mm Hg after 20 weeks of gestation and
- **Proteinuria ≥ 300 mg per 24 h** or ≥ 1 + dipstick.

Increased Certainty of Preeclampsia

- BP ≥ 160/100 mm Hg
- Proteinuria 2.0 g per 24 h or ≥ 2 + dipstick
- Serum creatinine > 1.2 mg/dl unless known to be previously elevated
- Platelets < 100,000/mm^3
- Microangiopathic hemolysis (increased LDH)
- Elevated SGOT or SGPT
- Persistent headache or other cerebral or visual disturbances
- Persistent epigastric pain.

Hypertension is diagnosed when the resting blood pressure is 140/90 mm Hg or greater; Korotkoff phase V is used to define diastolic pressure. In the past, it had been recommended that an incremental increase of 30 mm Hg systolic or 15 mm Hg diastolic pressure be used as diagnostic criteria, even when absolute values were below 140/90 mm Hg. These criteria are **no longer recommended** because evidence shows that these women are not likely to suffer increased adverse pregnancy outcomes.

Edema has been abandoned as a diagnostic criterion because it occurs in too many normal pregnant women.

ECLAMPSIA

Seizures that cannot be attributed to other causes in a woman with preeclampsia.

SUPERIMPOSED PREECLAMPSIA (ON CHRONIC HYPERTENSION)

New-onset proteinuria ≥ 300 mg per 24 h in hypertensive women but no proteinuria before 20 weeks of gestation, Or

A sudden increase in proteinuria or blood pressure or platelet count < 100,000/mm³ in women with hypertension and proteinuria before 20 weeks of gestation.

CHRONIC HYPERTENSION

BP ≥ 140/90 mm Hg before pregnancy or diagnosed before 20 weeks of gestation, Or

Hypertension first diagnosed after 20 weeks of gestation and **persistent after 12 weeks postpartum.**

Q. Risk factors and etiopathology of preeclampsia.

DEFINITION

Preeclampsia is a **pregnancy specific syndrome** of reduced organ perfusion secondary to vasospasm and endothelial activation characterized by BP ≥ 140/90 mm Hg after 20 weeks of gestation and proteinuria ≥ 300 mg per 24 h after 20th week in a previously normotensive and nonproteinuric patient.

Risk Factors for Preeclampsia

- Patient younger than 20 or older than 35 years of age
- Young primigravida (exposed to chorionic villi for the first time)
- Vesicular mole, multiple pregnancy (exposed to a superabundance of chorionic villi)
- Maternal obesity, insulin resistance, pre-existing DM, and pre-existing hypertension/renal/vascular disease
- Past history: There is 25% chance of recurrence in subsequent pregnancy
- Family history of preeclampsia
- Thrombophilias (APLA syndrome, protein C, S deficiency, factor V leiden)
- New paternity
- Fetal hydrops
- Smoking is protective for preeclampsia
- Placenta previa has also been reported to reduce the risk of hypertensive disorders in pregnancy

According to Sibai, currently plausible potential causes include the following:

- Abnormal trophoblastic invasion of uterine vessels
- Immunological intolerance between maternal and fetoplacental tissues (decrease in Th1 and increase in Th2 helper T-cells)
- Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy (imbalance between vasoconstrictors and vasodilators, increase in TXA2, endothelin-1, and increase sensitivity to angiotensin II, whereas prostacyclin and NO decreases)
- Dietary deficiencies
- Genetic influences (HLA-DR4)
- Abnormal trophoblastic invasion: In normal implantation, the **uterine spiral arteries** undergo extensive remodeling as they are invaded by endovascular trophoblasts. In preeclampsia, however, there is **incomplete trophoblastic invasion.** In preeclampsia only the decidual vessels, but not myometrial vessels, become lined with endovascular trophoblasts.

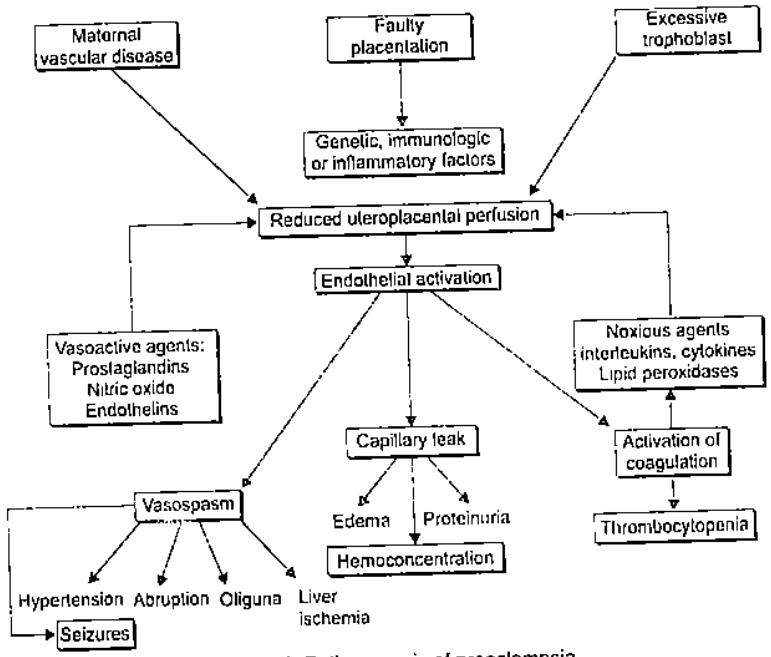


Fig. 17.1: Pathogenesis of preeclampsia

Q. Differences between mild and severe preeclampsia.

Abnormality	Mild	Severe
Diastolic blood pressure	<100 mm Hg	110 mm Hg or higher
Proteinuria	Trace to 1+	Persistent 2+ or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion	Absent	Present (eclampsia)
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Liver enzyme elevation	Minimal	Marked
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

Q. HELLP syndrome.

HELLP Syndrome

This is an acronym for hemolysis, elevated liver enzymes and low platletes.

- This is rare complication of preeclampsia (10–15% cases).

Criteria for the Diagnosis of HELLP Syndrome

Hemolysis (H)

- Schistocytes in the blood smear
- Bilirubin >1.2 mg/dl
- Absent plasma haptoglobin.

Elevated Liver Enzymes (EL)

- SGOT >72 IU/l
- LDH >600 IU/l.

Low Platelet Count (LP)

Platelets <100 × 103/mm^3

Complications

Maternal

- Eclampsia
- Abruption
- DIC
- Pulmonary edema, respiratory failure
- Pulmonary embolism, ARDS
- Stroke, cerebral edema
- Central venous thrombosis, seizures, retinal detachment
- Acute renal failure, chronic renal failure
- Hepatic (usually subcapsular) hematoma with possible rupture, ascites, nephrogenic diabetes insipidus
- Infection, sepsis
- Death
- Recurrence risk of HELLP syndrome = 3–19%.

Fetal/Neonatal

- Prematurity, RDS
- Intrauterine growth retardation
- Thrombocytopenia.

Investigations

Laboratory evaluation should include the following:

- Blood group and cross matching
- Complete blood cell (CBC) count with platelets: Anemia and thrombocytopenia

- **Coagulation studies:** BT, CT, PT, aPTT, Fibrinogen, D-dimer
- Peripheral smear: **Schistocytes, helmet cells, and burr cells** secondary to microangiopathic hemolytic anemia
- LFT: Bilirubin, SGOT, SGPT, LDH
- RFT: BUN, Creatinine
- Haptoglobin level: Decreased secondary to hemolysis.

Treatment

Multidisplinary team approach: Obstetrician, neonatologist, hematologist, intencivist and ICU facilities.

Delivery is the Definitive Treatment

- Although controversial, **corticosteroids** can be given as a treatment regimen for patients with HELLP
- Steroids are theorized to alter the degree of intravascular endothelial injury and prevent further hepatocyte death and platelet activation
- There is improved platelet counts, liver function, blood pressure, and urine output with the use of high-dose dexamethasone. **Intravenous glucocorticoids** appear superior to intramuscular steroids and are dose-dependent
- Steroids are also believed to improve fetal morbidity by reducing the incidence of respiratory distress syndrome and intraventricular hemorrhage, as well as maternal morbidity
- Dosing for high-risk patients with severe disease (platelet count < 20,000 or CNS dysfunction): 20 mg IV dexamethasone every 6 hours for up to 4 doses
- Dosing for all other patients with HELLP syndrome: 10 mg IV dexamethasone every 6 hours for 2 doses then 6 mg IV dexamethasone every 6 hours for 2 doses
- **Antihypertensives to control the high BP**
- **Prophylactic $MgSO_4$ should be started if impending eclampsia or actual eclampsia**
- Platelet transfusion and FFP maybe needed
- **Labor should be induced irrespective of weeks of gestation**
- LSCS for obstetric indication
- The route of delivery should be selected on obstetric indications including cervical status, obstetric history, the maternal and the fetal condition. If the cervix is unfavorable for induction of labor, cervical ripening should be the first step.

Q. Complications, investigations and management of preeclampsia.

INTRODUCTION

Definition

As above.

Complications

Maternal

Antenatal

- Eclampsia
- Abruption

- Preterm labor
- HELLP Syndrome
- DIC
- Blindness
- Cerebral hemorrhage
- ARDS
- Renal failure (oliguria, anuria)
- Increase risk of operative delivery.

Intrapartum

- Eclampsia

Postpartum

- PPH
- Eclampsia
- Shock
- Sepsis.

Remote

- Residual hypertension may be present in about 50% cases even after 6 months of delivery
- Recurrence risk: There is 25% chance of recurrence in subsequent pregnancy.

Fetal

- IUGR
- IUFD
- Prematurity
- Oligohydramnios
- Asphyxia.

Management

Investigations

- **URINE for proteins**/albumin. 24 hours urine protein
- **CBC:** There is hemoconcentration so HB values are false elevated. Low platlets indicate HELLP syndrome
- **Sr uric acid:** It is a **biochemical marker** of preeclampsia. Raised levels (>4.5 mg/dl) indicate renal involvement and also correlate with severity of preeclampsia, volume contraction and fetal jeopardy
- LFT: SGOT, SGPT, Bilirubin
- RFT: Sr creatinine

Coagulation profile maybe required in severe cases:

- BT
- CT
- PT, APTT

- Fibrinogen levels
- FDP
- **Fundoscopy:** Papilledema in severe cases, constriction of arterioles, alteration in a normal ratio of vein: Arteriole diameter from 3:2 to 3:1.

Tests for Fetal Well-being

- Fetal kick count
- NST weekly or biweekly
- BPP
- USG: For fetal growth and AFI
- Color Doppler.

Treatment

- Admit the patient
- Bedrest
- Diet: Diet to contain adequate protein about 100 mg/day. Salt and fluid restriction not needed
- BP charting 4 times/day
- Daily weight record
- Antihypertensives in pregnancy
  - Alpha methyldopa: 250–500 mg tds to qds
  - Nifedipine (always oral. Never sublingual): 5–10 mg bd to qds
  - Hydralazine 10–25 MG bds
  - Labetalol 100 mg bds to qds

ACE inhibitors are contraindicated.

Note: As per the latest guidelines DOC for hypertension in pregnancy = Labetalol followed by alpha methyldopa.

DOC for hypertensive crisis in pregnancy = Labetalol followed by hydralazine.

Diuretics: Not routinely recommended except in cases of cardiac failure or pulmonary edema or massive edema not relived by rest and causing discomfort to the patient.

Management

- On antihypertensives, if the BP is under control and there are no premonitory symptoms, then the pregnancy is allowed to continue **till 37 weeks** (keeping a close watch on maternal and fetal well-being)
- **Thereafter, the patient should be delivered even if the BP is under control,** as the risks of continuation of pregnancy far outweigh the benefits (as this is a pregnancy-induced condition and delivery is the ultimate or definitive treatment for pregnancy-induced hypertension)
- **It is not advisable to wait beyond 37 weeks** because the BP can rise and there can be complications (eclampsia, HELLP syndrome, IUFD, abruption, DIC, etc.) and there are no added benefits of continuing pregnancy beyond 37 weeks.

Impending Eclampsia

The dangerous symptoms **(premonitory symptoms)** that indicate impending eclampsia in case of preeclampsia are:

- Headache
- Epigastric pain
- Blurring of vision.
- Oliguria
- Nausea, vomiting

Whenever the above symptoms develop in a case of severe preeclampsia the patient is at a risk of eclampsia; the patient should be given anticonvulsant ($MgSO_4$) and antihypertensive medication, and the patient to be **delivered irrespective of the weeks of gestation.**

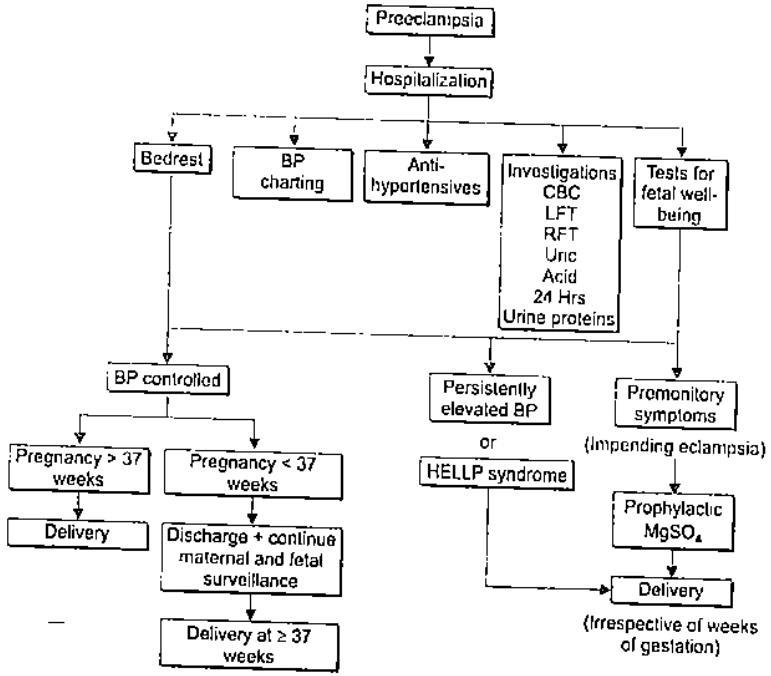
Magnesium sulfate is the drug of choice for eclampsia and also for **impending eclampsia.**

Prophylactic magnesium sulfate decreases the risk of convulsion, abruption, and maternal mortality in this scenario **(MAGPIE TRIAL).**

The indications for termination of pregnancy **irrespective of the weeks of gestation** in a case of preeclampsia are:

- Severe preeclampsia, with impending eclampsia
- Eclampsia (give $MgSO_4$ first, followed by induction of labor)
- HELLP syndrome.

Flow Chart 17.1: Management of preeclampsia



Methods of Delivery

- The route of delivery should be selected on obstetric indications including cervical status, obstetric history, the maternal and the fetal condition. If the cervix is unfavorable for induction of labor, cervical ripening with PGs gel or insets should be the first step
- If cervix favorable: oxytocin infusion, ARM
- LSCS for obstetric indications or in cases of severe preeclampsia and unripe, closed cervix where there would be prolonged induction-delivery interval

During Labor

- BP and urine output charting
- Strict monitoring of fetal heart sounds (continuous EFM preferred)
- **Avoid methergin** after baby delivery as it can raise the BP.

Prevention of Hypertension in Future Pregnancy

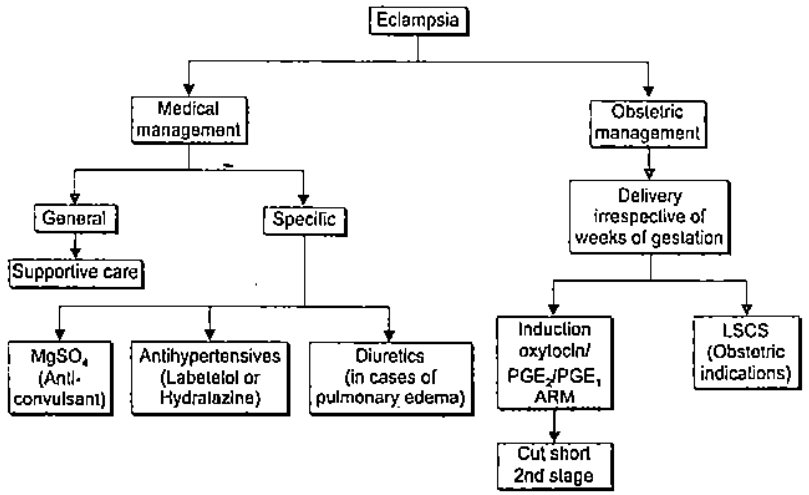
- **Low-dose aspirin** (75–150 mg/day till 34 weeks)
- Antioxidants (vitamin E, vitamin A, vitamin C, and **lycopene**)
- Calcium (2 g/day)
- Omega 3 fatty acids.

Q. Management of eclampsia.

Preeclampsia when complicated by convulsion and/or coma is called eclampsia.

MANAGEMENT

Flow Chart 17.2: Management of eclampsia



Medical

- Eclamptic convulsions are life-threatening emergencies and require the proper treatment to decrease maternal morbidity and mortality.
- ***Delivery is the only definitive treatment for eclampsia.***

Team Approach

The patient should be shifted urgently to a ***tertiary care center*** for further management.

An experienced obstetrician, a pediatrician or neonatologist, anesthetist, intensivist and ICU and NICU backup are essential.

General Management

Supportive Care

- To prevent maternal **injury and fall**
- To prevent aspiration
- **Maintain airway**
- Ensure oxygenation
- Place the patient in the left lateral position. This positioning decreases the risk of aspiration and will help to improve uterine blood flow
- Protect the patient against injury during the seizure by padding and raising guardrails, using a padded tongue blade between the teeth
- **Suctioning** the oral secretions
- Oxygenation by face mask (8–10 L/min). Monitor the oxygen saturation
- Sodium bicarbonate maybe given if acidosis
- Secure an intravenous (IV) line with a large-bore catheter for drawing specimens and administering fluids and medications
- **Catherization** to monitor the urine output and collect urine for proteins (proteinuria)
- Detailed history to be taken from relatives
- Examination: Quick but thorough general, abdominal and vaginal examination to be done.
- Monitor: Pulse, respiratory rate, BP half hourly. Also monitor the progress of labor and fetal heart rate
- Intensive monitoring of the patient is required and even ICU maybe needed
- Depending on the clinical course, regularly check the patient's neurologic status for signs of increased intracranial pressure or bleeding (e.g. funduscopic examination)
- Monitor fluid intake and urine output, maternal respiratory rate, and oxygenation, as indicated, and continuously monitor fetal status
- CVP and Pulmonary arterial pressure monitoring is rarely indicated but may be helpful in patients who have evidence of pulmonary edema or oliguria/anuria
- Dopamine infusion maybe needed in cases of anuria/oliguria
- IV fluids should be limited to isotonic solutions to replace urine output plus about 700–1000 mL/day to replace insensible losses
- IV antibiotics (generally cefotaxim or ceftriaxone) to prevent infection.

Specific Management

- Anticonvulsant therapy (magnesium sulfate)
- Blood pressure (BP) control
- Diuretics are used only in the setting of pulmonary edema.

Anticonvulsant

Magnesium sulfate is the DOC for eclampsia.

It can be given by various protocols:
- Pritchard
- Sibai
- Zuspan
- Sardesai.

Pritchard Protocol

Loading Dose

4 g (20 mL of 20%) IV over 4 min (only in severe preeclampsia-eclampsia) immediately followed by 10 g (20 mL of 50%) IM—5 g in each buttock.

If convulsions persist after 15 min: IV 2 g (10 mL of 20%) over 2 min (if the woman is large—4 g).

Maintenance

5 g (10 mL of 50%) IM every 4 h—alternate sides.

OR

Sibai Protocol

Loading Dose

6 g IV over 20 min

Maintenance

2–3 g/h IV

$MgSO_4$ is continued till 24 hours postdelivery or 24 hours after the last convulsion whicl ever is later.

Monitoring of $MgSO_4$ Therapy

- Patellar reflexes
- Respiratory rate (>14/min)
- Urine output (100 cc in 4 h or 30 cc/h).
  - **Therapeutic range of magnesium is 4–7 mEq/L.**
  - When plasma levels rise above 10 mEq/L, respiratory depression develops, and a 12 mEq/L or more, respiratory paralysis and arrest follow
  - Calcium gluconate, 1 g intravenously, is the drug of choice for $MgSO_4$ toxicity, alon with withholding further magnesium sulfate

Status Eclampticus

- For the rare patient who continues to have seizure activity while receiving adequate magnesium therapy, seizures maybe treated with sodium amobarbital, 250 mg IV over 3-5 minutes or thiopentone sodium 0.5 gm IV slowly. The procedure to be monitored by expert anesthetist
- If this fails complete anesthesia with muscle relaxant and assisted ventilation maybe required
- In unresponsive cases an emergency LSCS maybe lifesaving.

Antihypertensives

- Control of hypertension is essential to prevent further morbidity or possible mortality. The most commonly used antihypertensive agents are hydralazine, labetalol, and nifedipine
- As per the latest guidelines DOC for hypertensive crisis in pregnancy = Labetalol followed by hydralazine
- The goal is to maintain systolic BP between 140 and 160 mm Hg and diastolic BP between 90 and 110 mm Hg
- An IV bolus of hydralazine (5-10 mg every 20 min) or labetalol (20-40 mg every 15 min) is recommended. Other potent antihypertensive medications, such as sodium nitroprusside or nitroglycerin, can be used but are rarely required
- Care must be taken not to decrease the BP too drastically; an excessive decrease can cause inadequate uteroplacental perfusion and fetal compromise
- Diuretics are used *only in the setting of pulmonary edema.* Frusemide 40 mg IV followed by 20 g mannitol IV reduces pulmonary edema and also prevents ARDS.

Obstetric Management

Delivery is the only Definitive Treatment for Eclampsia

- Induction of labor must be initiated **(Irrespective of weeks of gestation)** after $MgSO_4$ and antihypertensives
- A dose of antenatal steroids maybe administered when gestational age is less than 34 weeks. Betamethasone (12 mg IM q24h × 2 doses) or dexamethasone (6 mg IM q12h × 4 doses) is recommended
- The mode of delivery should be based on obstetric indications but should be chosen with an awareness that *vaginal delivery is preferable from a maternal point of view*
- In the absence of fetal malpresentation or fetal distress, oxytocin or prostaglandins maybe initiated to induce labor
- Fetal heart rate and uterine contractions should be continuously monitored. Fetal bradycardia is common following the eclamptic seizure. Typically, emergency LSCS is not indicated for this post seizure transient bradycardia as it spontaneously resolves
- Prophylactic forceps or ventouse to be used to cut short second stage of labor
- LSCS is indicated in cases of:
  - Uncontrolled fits inspite of therapy
  - Unconscious patients and if prospects of vaginal delivery are remote
  - Worsening of maternal neurological, hepatic or renal condition and when anticipated delivery time is remote

  - Obstetric indications like fetal distress, abruption, malpresentations
  - It may also be considered in patients with an unfavorable cervix and a gestational age of 30 weeks or less (as induction under these circumstances may result in a prolonged intrapartum course and intrapartum complications)
- When emergent cesarean delivery is indicated, coagulopathy if present should be corrected before the procedure
- **Watch for PPH after delivery and methergin to be avoided.**

Anesthesia

- For nonemergency cesarean delivery, epidural or combined techniques of regional anesthesia are preferred
- Regional anesthesia is contraindicated in the presence of coagulopathy or severe thrombocytopenia (< 50,000 platelets/μl.)
- General anesthesia in women with eclampsia increases the risk of aspiration, and airway edema may make intubation difficult. It also can produce significant increases in systemic and cerebral pressures during intubation and extubation
- The use of spinal anesthesia requires caution because of the possibility of total sympathetic blockade, resulting in maternal hypotension and uteroplacental insufficiency.

18 Antepartum Hemorrhage (APH) and Postpartum Hemorrhage (PPH)

Q. Etiology, signs, symptoms and management of abruption.

Q. What are the types of abruption, management and complications?

ABRUPTIO PLACENTA/ABRUPTION

It is a form of APH where the bleeding occurs due to premature separation of **normally implanted** placenta. It is the **most common cause of APH**, followed by placenta previa.

Risk Factors for Abruptio Placenta

- Increased age and parity
- Preeclampsia and chronic hypertension. This is the **most important predisposing factor** The vasospasm in uteroplacental bed leads to anoxic endothelial damage and rupture of vessels or extravasation of blood
- Cigarette smoking and cocaine use (vasospasm and transient hypertension)
- Thrombophilia: Hereditary or acquired (APLA syndrome)
- Prior abruption (risk of recurrence is **17% for patients with one abruption and 25% for patients with more than one abruption)**
- Uterine leiomyoma: If the placenta is implanted over a fibroid
- Sudden decompression of uterus as in cases of multifetal gestation (after delivery of first baby) or in cases of PROM and polyhydramnios (sudden escape of liquor)
- External trauma or trauma during ECV
- Short cord
- Folic acid deficiency is also thought to play a role.

Signs and Symptoms of Abruptio Placenta

Sign or symptom	Frequency (%)
Vaginal bleeding	78
Uterine tenderness or back pain	66
Fetal distress	60
High-frequency contractions	17
Hypertonus	17
Idiopathic preterm labor	22
Dead fetus	15

Types/Varieties of Abruption

- *Revealed:* The blood insinuates between membranes and decidua and comes out of the external OS
- *Concealed:* The blood collects behind the placenta or the membrane and the decidua and does not come out of cervix
- *Mixed:* Combination of above 2 types.

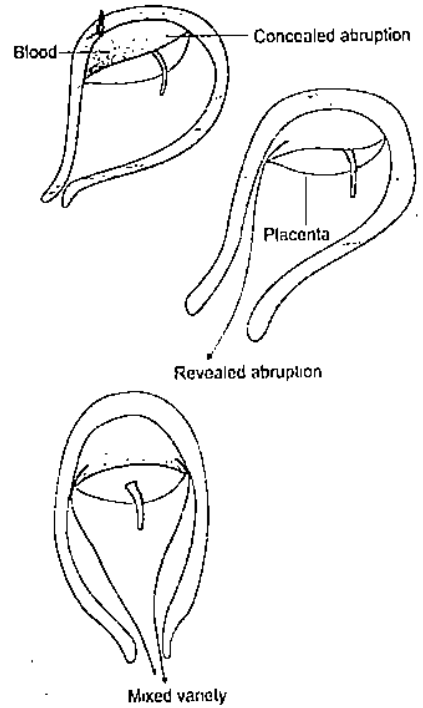


Fig. 18.1: Types of abruption

Clinical Features Depend on

- Degree of placental separation
- Speed at which the separation occurs
- Variety of abruption (concealed or revealed).

Revealed	Concealed
External bleeding (bleeding PV) present	Absent
Generally condition and pallor is proportional to the amount of blood loss	Shock and pallor out of proportion to the blood loss

Contd...

Contd...

Revealed	Concealed
Uterine height corresponds to period of gestation	More than period of gestation
Uterine feel normal with localized tenderness	Uterus tense, tender and tonically contracted
Fetal parts can be identified easily	Difficult to make out
Hb: Low, proportional to the amount of blood loss	Markedly low and out of proportion to the blood loss

Couvelaire Uterus

This is condition associated with **severe variety of concealed abruption.** This condition is diagnosed only laparotomy **(on operation table if the delivery is by LSCS.)**

There is widespread extravasation of blood into the uterine musculature and beneath the uterine serosa. This so-called uteroplacental apoplexy,was first described by Couvelaire in the early 1900s, is now frequently called Couvelaire uterus.

The uterus looks **dark port wine in color** which may be patchy or diffuse.

Such effusions of blood are also occasionally seen beneath the tubal serosa, in the connective tissue of the broad ligaments, and in the substance of the ovaries, as well as free in the peritoneal cavity.

These myometrial hemorrhages seldom interfere with uterine contractions to produce severe postpartum hemorrhage and are not an indication for hysterectomy.

Complications

Maternal

- Hemorrhage and hypovolemic shock
- DIC
- PPH (remember APH predisposes to PPH)
- Renal failure (hypovolemia and ATN), oliguria and anuria
- Sheehan's syndrome (postpartum pituitary necrosis)
- Increased risk of operative delivery (LSCS)
- Death.

Fetal

- Fetal distress/Hypoxia
- IUFD (with IUFD the placental detachment is usually greater than 50%)

- Prematurity
- Anemia in the new born.

MANAGEMENT

Emergency and Definitive

Emergency

- Two wide bore IV lines
- IV fluids (crystalloids and colloids)
- **Blood transfusion** (in cases of hemodynamic instability in the patient) and FFP and platelets transfusion in cases of DIC
- Close monitoring of maternal and fetal condition

Pritchard's rule for management of abruption: Keep **hematocrit (HCT) atleast 30% and maintain urine output of at least 30 ml/hour.**

Collect Blood for

- CBC and platelet count
- Grouping and cross matching
- DIC PROFILE
  BT, CT
  PT, aPTT
  Sr Fibrinogen
  FDP.

- ***Ultrasonography***

Helps in placental localization. In abruption its in the upper segment as compared to previa in which the placenta is in the lower segment.

Retroplacental collection can be seen.

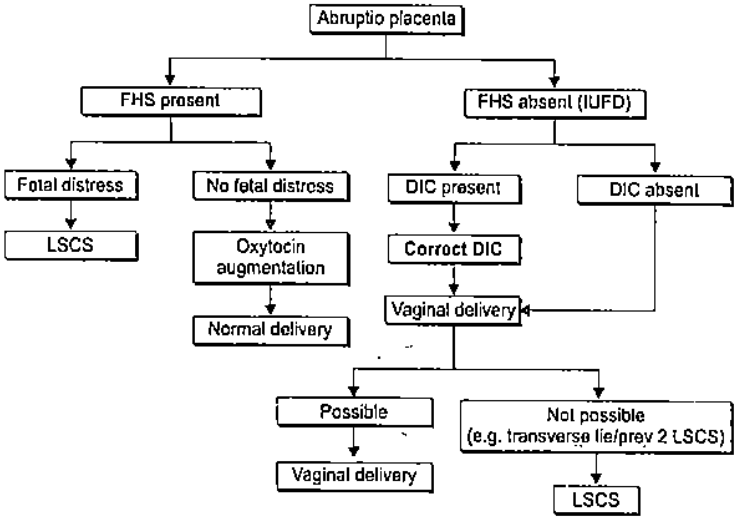
Early hemorrhage is hyperechoic or isoechoic.

Negative findings does not rule out abruption.

Definitive management = immediate delivery as abruption is progressive and the bleeding will only stop, once the placenta is delivered and the uterus contracts.

If the fetus is alive and there is **no fetal distress** (reassuring FHR tracing) and **there is a possibility that delivery can happen soon, then labor should be augmented** by ARM and oxytocin drip, keeping a close watch on FHR.

Flow Chart 18.1: Management of abruption



Q. What are the types of placenta previa?

Q. Clinical features and management of placenta previa.

PLACENTA PREVIA

Definition

Previa in latin means 'in front of', so the placenta is in front of presenting part. Placenta previa involves implantation of the placenta over the internal cervical OS. Variants include complete implantation over the OS (complete placenta previa), a placental edge partially covering the OS (partial placenta previa) or the placenta approaching the border of the OS (marginal placenta previa).

Four degrees of placenta previa have been recognized:

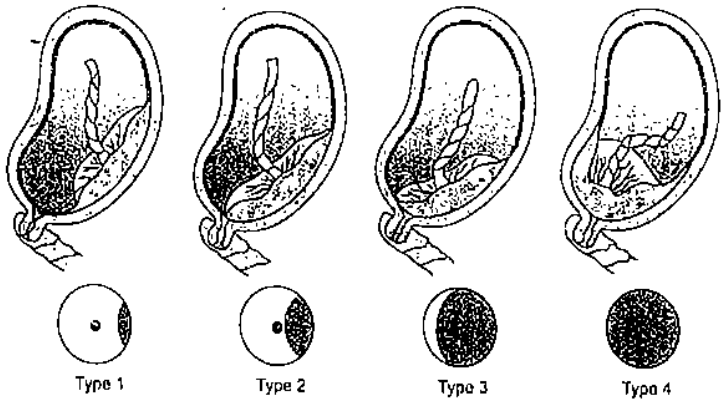


Fig. 18.2: Degree of placenta previa with findings on ultrasound examination

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

Risk Factors

- Increasing age and increasing parity
- **Past history** (12 times risk of another placenta previa)
- **Previous LSCS (probability of previa is four times greater than in patients without any uterine scar)**
- Multiple pregnancy
- Prematurity
- Smoking.

Clinical Features

- Remember 4 **P's:** Painless, profuse,periodic and purposeless (causeless)
- The classic presentation of placenta previa is **painless vaginal bleeding**
- Nearly two thirds of symptomatic patients present before 36 weeks' gestation, with half of these patients presenting before 30 weeks' gestation.

Signs

- General condition and anemia: Proportional to visible blood loss
- *Features of preeclampsia:* Unrelated
- *Height of uterus:* Proportionate to the period of gestation
- *Feel of uterus:* Soft and relaxed without any localized tenderness
- ***Malpresentation:*** Malpresentation is common (breech, transverse lie or unstable lie)
- The head is **high floating**
- ***Stallworthy's Sign:*** Slowing of fetal heart rate on pressing the head down in to the pelvis and prompt recovery on release of pressure is termed Stallworthy's sign. This sign is suggestive of posterior placenta previa.

Any pregnant patient beyond the first trimester who presents with vaginal bleeding requires a speculum examination followed by diagnostic ultrasound, unless previous documentation confirms no placenta previa. In placenta previa the blood is bright red in colour.

Because of the risk of provoking life-threatening hemorrhage, **a digital examination is absolutely contraindicated until placenta previa is excluded.** PV examination (if at all required) is only to be done in OT, with all facilities ready for cesarean section including anesthetist **(Double set up examination).**

Ultrasonography

The most useful and inexpensive study is transvaginal ultrasonography that provides 100% accuracy in identifying a placenta previa.

An alternative would be transabdominal ultrasonography that can be 95% accurate; however, the false-positive and false-negative rates can range from 2-25%. False positive result may be due to full bladder.

Transperineal sonography is another alternative.

Color Doppler: Prominent venous flow in hypoechoic areas near cervix is consistent with the diagnosis of placenta previa.

MRI

Very accurate, safe and without the risk of ionizing radiation. But expensive and not routinely done.

Management

Emergency

1. Two wide bore IV lines
2. IV fluids (crystalloids and colloids)
3. **Blood transfusion** (in cases of hemodynamic instability in the patient and FFP and platelets transfusion in cases of DIC)
4. Close monitoring of maternal and fetal condition
5. Abdominal examination to rule out tenderness and vulval inspection for presence of bleeding
6. Confirm the diagnosis with ultrasonography.

Collect blood for:

1. CBC and platelet count
2. Grouping and cross matching.

MacAfee and Johnson Regimen

(Conservative/Expectant Management in Placenta Previa)
This consists of complete bed rest, tocolysis, and close observation of patient.
Steroids are generally given to enhance lung maturity.

To undertake this regimen (to wait and watch), **all the three criteria should be fulfilled:**

- Mother should be hemodynamically stable.
- There should be no fetal distress.
- Pregnancy should be less than 36 weeks of gestation.

If any one of these criteria is not met, then the patient should be **delivered by LSCS.**

The expectant management is continued till 37 weeks. Then the patient is to be delivered by cesarean section, as now there is no point of further waiting.

Active management (Delivery).

In cases of placenta previa the delivery is by cesarean section.

Vaginal delivery is only possible in cases where the placental edge is **>3 cm away from internal os on USG (low lying placenta).**

Immediate Delivery (Irrespective of Weeks of Gestation) is to be done in following cases:

- Patient is in shock/hemodynamic instability (resuscitation followed by LSCS)
- Fetal Distress/IUFD/Severe congenital malformation in fetus
- Pregnancy > 37 Weeks.

Q. Differentiate between placenta previa and abruption.

Distinguishing Features of Placenta Previa and Abruptio Placenta

Clinical features	Placenta previa	Abruptio placenta
Nature of bleeding	a. Painless, profuse b. Bleeding is always revealed c. Periodic	a. Painful b. Revealed, concealed, or usually mixed c. Progressive
General condition and anemia	Proportional to visible blood loss	Out of proportion to the visible blood loss in concealed or mixed variety
Features of preeclampsia	Unrelated	Very likely to be present
Height of uterus	Proportionate height	May be disproportionately enlarged in concealed type
Feel of uterus	Soft and relaxed	Tonically conracted uterus
Malpresentation	Malpresentation is common (breech, transverse lie). The head is high floating	Unrelated the head may be engaged
Placentography	Placenta in lower segment	Placenta in upper segment
Tocolysis	Can be given	Never
Wait and watch	Can be done	Never
Delivery	LSCS	LSCS or vaginal delivery
DIC	Less common	More common

Q. Define PPH. What are the causes of PPH?

Q. Management of atonic PPH.

PPH

Definitions

- Blood loss of 500 ml or more (in cases of vaginal delivery) following birth of the baby.
- **Decrease in HCT by 10%** or more, after delivery **(ACOG).**
- Any amount of blood loss requiring 1 unit or more blood transfusion in the postpartum period.
- **Any amount of bleeding** from or into the genital tract following the birth of the baby upto the end of puerperium which causes **hemodynamic instability** in the patient.

Predisposing Factors and Causes of Immediate Postpartum Hemorrhage

Bleeding from placental implantation site

Hypotonic myometrium—uterine atony (MC) 80% cases

- **Hypertensive disorders:** Preeclampia, eclampsia
- **Antepartum hemorrhage:** Abruption (remember: APH predisposes to PPH)
- **Over distended uterus** (e.g. large fetus, twins, and hydramnios): Imperfect retraction
- Following prolonged/obstructed labor: Poor retraction and amnionitis
- Following precipitate labor
- Following oxytocin—induced or augmented labor
- **High parity:** Inadequate retraction, and increased risk of adherent placenta and anemia all contribute
- **Uterine atony in previous pregnancy**
- Chorioamnionitis
- Drugs—tocolytic agents, halothane
- Retained placental tissue, avulsed cotyledon and succenturiate lobe
- Abnormally adherent—accreta, increta, and percreta
- Fibroids: Mechanically cause imperfect retraction.

Trauma to the Genital Tract (20%)

- Large episiotomy, including extensions
- Lacerations of perineum, vagina, or cervix in cases of instrumental deliveries
- Ruptured uterus.

Coagulation Defects like DIC, HELLP Syndrome

Intensify all of the above.

MANAGEMENT

- Call for extra help
- Put two wide bore IV cannulas
- Send blood for grouping and crossmatching
- IV fluids (crystalloids and colloids) infuse normal saline 2 liters rapidly and plasma expanders like hemaccel
- Catheterize (Folleys' catheter) the patient and monitor the urine output
- To monitor the vitals like pulse, BP, and CVP if needed
- Give oxygen by mask
- Blood transfusion.

Specific

- Palpate the abdomen for the feel of the uterus
- If the uterus is flabby its atonic PPH
- If the uterus is well contracted and firm, the bleeding is due to trauma.

Atonic PPH

- Massage the uterus
- *Use of utero tonic drugs:*
  - Injection **oxytocin drip** (10-20 units in 500 ml of saline) @ 40-60 drops/minute. **As per the ACOG guidelines oxytocin is the first line drug of choice for atonic PPH**
  - Injection methergine 0.2 mg I V.
- The 15-methyl derivative of prostaglandin F2 (carboprost tromethamine). The initial recommended dose is 250 µg (0.25 mg) given intramuscularly, and this is repeated if necessary at 15-90 min intervals up to a maximum of eight doses.
- Misoprostol, a synthetic prostaglandin E1 analog, is also effective for the treatment of uterine atony. **WHO recommends that misoprostol (800 µg) be given per rectally.**

If the uterine atonicity persists:

Intrauterine packing.

- **Shivkars pack:** Condom inflated with saline acts as tamponade. Bakri balloon or Sengstaken-Blackemore tube can also be used. This can avoid a hysterectomy in around 80% patients.

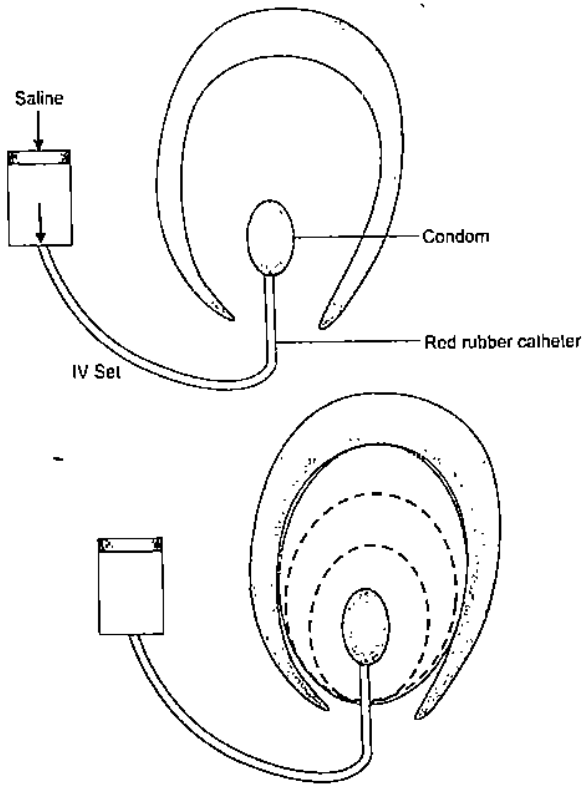


Fig. 18.3: Shivkar's pack

If all these medical methods fail to control the bleeding, then surgical methods are required.

Stepwise devascularization of the uterus:

- Uterine artery ligation

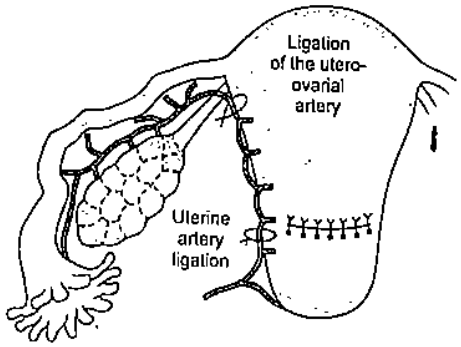


Fig. 18.4: Ligation of the utero-ovarian artery

- **Internal iliac artery ligation:**

Ligation of the internal iliac arteries **(anterior division)** at times reduces the hemorrhage appreciably.

The most important mechanism of action with internal iliac artery ligation is an 85-percent reduction in pulse pressure in those arteries distal to the ligation.

This converts an arterial pressure system into one with pressures approaching those in the venous circulation and more amenable to hemostasis via simple clot formation. Bilateral ligation of these arteries does not appear to interfere with subsequent reproduction.

- **Uterine compression sutures:**

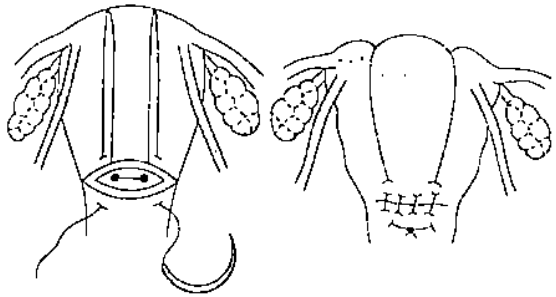


Fig. 18.5: B-Lynch brace suture for control of atonic PPH

1. In 1997, **B-Lynch** described a surgical technique for severe postpartum hemorrhage in which a pair of vertical brace chromic sutures were secured around the uterus, giving the appearance of suspenders, to compress together the anterior and posterior walls **(tamponade effect) success rate is about 80%.**

2. **Hayman sutures.**
3. **Cho square sutures.**
4. **Gunshella sutures.**

- **Uterine artery embolization** under fluoroscopic guidance can be tried.
- **Obstetric hysterectomy** is used as the last resort.

Hysterectomy performed at or following delivery may be lifesaving if there is severe obstetrical hemorrhage. It can be carried out in conjunction with cesarean delivery or following vaginal delivery.

19 Medical and Surgical Disorders

Q. Signs and symptoms suggestive of heart disease in pregnancy.

Metcalfe's Criteria for Heart Disease in Pregnancy (Finding suggestive of heart disease in pregnancy).

Symptoms

- Progressive dyspnea or orthopnea
- Nocturnal cough
- Hemoptysis
- Syncope
- Chest pain.

Clinical Findings

- Cyanosis
- Clubbing of fingers
- Persistent neck vein distention
- Systolic murmur grade 3/6 or greater
- Diastolic murmur
- Cardiomegaly
- Persistent split-second sound
- Criteria for pulmonary hypertension
- Persistent arrhythmias.

Q. Intrapartum management of cardiac patient.

INTRODUCTION

Mitral stenosis is the most common valvular disease in pregnancy.

The patient with heart disease belongs to a **high-risk category** and there is increased risk of mortality during pregnancy and especially during labor.

Team Approach

Obstetrician, cardiologist, anesthetist, neonatologist and ICU facilities.

General Measures for the Cardiac Patient in Labor

First Stage

- Labor and delivery in **lateral decubitus position/propped up position**
- Adequate pain relief **(epidural analgesia is the best).** Pain can cause tachycardia, which in turn can precipitate failure
- **Restrict IV fluids** to 75 ml/hour (except in aortic stenosis) to prevent CCF
- **Oxygen** by breathing mask @ 5 to 6 l/minute
- Careful monitoring of the vital parameters. Cardiac monitoring and pulse oximeter monitoring. CVP monitoring in selected cases. If pulse rate exceeds 110/min in between contractions, rapid digitalization is done by IV digoxin 0.5 mg
- Antibiotics (infective endocarditis prophylaxis = ampicillin and gentamicin). Ampicillin 2 gm and gentamicin 1.5 mg/kg at the onset of labor followed by repeat doses at 8 hourly interval.

Second Stage

- Avoid maternal pushing efforts and so to **cut short II stage of labor by use of prophylactic forceps or vacuum.**

Third Stage

- Prevention of postpartum pulmonary edema by giving **IV frusemide after placental delivery**
- **Methergine is absolutely contraindicated** and not to be given. To prevent excessive blood loss oxytocin infusion can be used.

Place for LSCS

- In heart disease patients, LSCS should be done for obstetric indications only
- Heart disease in which **elective LSCS should be done is Marfan syndrome with aortic root dilatation > 4 cm. Coarctation of aorta is a relative indication for elective LSCS.**

Q. Short note: Red degeneration.

INTRODUCTION

Fibroids are the most common benign tumors of the uterus. They are also the MC pelvic tumors in females.

Fibroids can undergo various types of degeneration. One of it is called red degeneration which occurs **exclusively during pregnancy.**

Red Degeneration
(Also Known as Carneous Degeneration)

- Occurs because fibroid overgrows its blood supply **(micronecrothrombosis)**
- Most commonly occurs in **second trimester of pregnancy** followed by in the **puerperium** period
- *Cut section:* Raw beefy appearance, fishy odor.

Clinical Features

- Patient presents with:
  - Acute abdomen
  - Vomiting
  - Fever.

There is presence of leukocytosis.

D/D

- Acute appendicitis
- Pyelonephritis
- Abruption.

Management

- Always conservative management (never surgery)
- Hospitalization
- Bed rest
- Analgesics
- IV fluids
- IV antibiotics (SOS).

Q. Complications of anemia in pregnancy.

Q. Investigations for case of anemia in pregnancy.

Q. Management of iron deficiency anemia in pregnancy.

ANEMIA

Introduction

Anemia is the MC hematological disorder to occur in pregnancy. **Dimorphic anemia** is the MC type of anemia.

It is the **MC indirect cause of maternal mortality.**

Complications

Maternal, fetal.

Maternal

Antenatal

- Infections: Anemia and malnutrition decreases resistance to infections
- **Cardiac failure** at 30 to 32 weeks
- Preterm labor
- Abruption (folic acid deficiency).

Intrapartum

Cardiac failure: Due to increased cardiac output.

Postpartum

- **PPH: Patients may not tolerate even minimal blood loss**
- **Maternal mortality**
- Puerperal sepsis
- Subinvolution
- Poor lactation
- Poor wound healing
- Puerperal venous and pulmonary embolism.

Fetal

- The baby does not have anemia at birth but the iron stores maybe less
- IUGR
- Increased in perinatal loss.

Criteria of Physiological Anemia

- Hb: 10 g%
- RBC: 3.2 million/mm^3
- PCV: 30%
- Peripheral smear: **Normocytic and normochromic**
- Mean corpuscular hemoglobin concentration remains unchanged in pregnancy.

Investigations

For the Degree of Anemia

- Hemoglobin
- Hematocrit (PCV).

Type of Anemia

- **Peripheral blood smear** (stained with **Leishman stain**) is the most important
- RBC morphology gives the idea about the type of anemia.

Iron Deficiency

- Hypochromic, microcytic
- Anisocytosis
- Poikilocytosis
- Increased in reticulocyte count.

Megaloblastic Anemia

Vitamin B_{12} and folic acid deficiency.

- Macrocytes
- Hypersegmented neutrophils

- Howell-Jolly bodies
- Giant polymorphs.

Hematological Indices

Indices	Iron deficiency anemia	Megaloblastic anemia
MCV	Decreased (less than 75 u^3)	Increased (>100 u^3)
MCH	Decreased (less than 25 pg)	Increased (>33 pg)
MCHC	Decreased (less than 30%)	Normal

Other Blood Investigations

In Iron Deficiency

- Serum iron below 30 µg/dl
- TIBC increased (> 400 µg/dl)
- Percentagae saturation decreased (10% or less)
- As per CDC, **Sr. ferritin less than 15 µg/L** confirms iron deficiency anemia.

In Megaloblastic Anemia

- Sr iron, normal or high
- Sr. folate <3 ng/ml
- Sr. B_{12} <90 pg/ml
- Associated leukopenia and thrombocytopenia.

To Find the Cause of Anemia

- Stool examination to detect helminthic infestation
- Clean catch mid stream, urine sample for pus cells, protein and sugars
- Other appropriate investigations as per history and clinical examination
- Hemoglobin electrophoresis to rule out thalassemia especially in cases of refractory anemia
- *In specific cases:* Chest X-ray to rule out TB, fractional test meal analysis of gastric juice for achlorhydria in pernicious anemia.

Treatment

Curative

General and specific.

General

- ***Diet:*** A balanced diet rich in iron and protein
  - The best source of iron is red meat, especially beef and liver. Chicken, turkey, pork, fish, and shellfish also are good sources of iron. Vegetarian sources include:
    - Iron-fortified breads and cereals
    - Peas; lentils; white, red, and baked beans; soybeans; and chickpeas
    - Tofu, jaggery
    - Dried fruits, such as prunes, raisins, and apricots
    - Spinach and other dark green leafy vegetables, prune juice
    - **Iron utensils should be used for cooking.**

Vitamin C

- Vitamin C helps the body absorb iron. Good sources of vitamin C are vegetables and fruits, especially citrus fruits. Citrus fruits include oranges, grapefruits.

Factors that inhibit iron absorption	Factors that enhance iron absorption
Foods rich in calcium	Heme iron
Tannins in tea	Ferrous iron (Fe^{2+})
Phytates in cereals	Ascorbic acid

- To cure the diseases contributing to anemia
- To eradicate any focus of infection
- **Antihelminthics** to eradicate worm infestation.

Specific Therapy

- Iron: Oral and parenteral
- Folic acid and vitamin B_{12} and C
- Once women become iron deficient in pregnancy it is not possible to ensure repletion through diet alone and oral supplementation is needed
- Oral iron is an effective, cheap and safe way to replace iron. Ferrous salts (sulphate, gluconate, fumarate, succinate) show only marginal differences between one another in efficiency of absorption of iron
- Ferric salts are much less well absorbed
- Generally a 200 mg tablet contains 60 mg elemental iron. One tablet is to be given three times a day (180 mg) to a max of six tablets. Higher doses should not be given, as absorption is saturated and side effects increased
- Oral iron supplementation should be taken on an **empty stomach**, as absorption is reduced or promoted by the same factors that affect absorption of dietary nonheme iron.

Side Effects

- Epigastric pain
- Nausea, vomiting
- Diarrhea or constipation
  - Patients need to be informed that **stools would black in color**
  - The side effects can be minimized by starting with a small dose or change of preparation
  - With oral treatment, the absorption of iron is an issue as it is effected by various factors
  - The serum iron maybe restored but there is difficulty in replenishing the stores.

Response is evident by:

- Sense of well-being
- Increased appetite
- Rise in hemoglobin and hematocrit which is preceeded by reticulocytosis.

Causes of Failure of Oral Treatment

- Improper typing of anemia: Wrong diagnosis, thalassemia
- Defective absorption
- Noncompliant patient
- Nontolerance
- Concurrent blood loss like piles or hookworm infestation.

Parenteral Iron

- Intravenous: Iron sucrose, iron dextran
- Intramuscular: Iron dextran and iron sorbitol citric acid complex.

Indications for Parenteral Iron

- Noncompliant patient
- Nontolerance to oral iron
- **Malabsorption syndrome**
  - **Parenteral iron is not given for rapid rise of Hb as the rise in Hb is the same with oral, IM and IV iron.**
  - It is about **0.7 to 1 g/dL per week**
  - **Fastest rise of Hb is with blood transfusion.**

The main advantages of parenteral iron are:
- Surety/certainty of administration
- Helps in replenishing the iron stores faster.

Different Formulae for Calculations of Dose of Parenteral Iron

- Formula 1 = (Normal Hb in g – patient's Hb in g) × Weight (in kg) × 2.21 + 1000 (for stores) = mg of iron needed
- Formula 2 = 250 mg of iron is required for each gram of Hb below normal
- Formula 3 = 0.3 × weight (in pounds) × (100 – Hb%) = mg of iron needed. Add 50% of this for stores.

For IV iron test dose is to be given by IV route and for IM iron test dose is to be given IM.

However for the newer preparation, **iron sucrose, test dose is not needed.**

Intramuscular injections are to be given by **Z technique** to prevent staining of the skin and to minimize the pain.

Side Effects

- Pain
- Abscess formation, skin discoloration
- *Reactions:* Fever, headache, lymphadenopathy, nausea, vomiting and rarely allergic reactions.

Blood Transfusion

Packed cells is preferred over whole blood (to avoid the risk of CCF).

To be given when rapid rise of hemoglobin is required:
- Severe anemia with advanced pregnancy (>36 weeks)
- Severe anemia in early labor
- Anytime if the patient has CCF due to severe anemia. (under the cover of diuretics).

Advantages of BT

- Increase in oxygen carrying capacity of blood
- Hb from lysed red cells maybe utilized for formation of new RBCs
- Stimulates erythropoiesis
- Improvement expected in 3 days.

Precautions

- Properly typed and cross matched blood to be used
- Patient needs to be admitted
- Blood to be given under the cover of diuretics (frusemide)
- To monitor vitals and watch for crepitations in the base of lungs
- Emergency resuscitation trolley ready to manage anaphylaxis reaction.

Management during Labor

- Blood to be collected for grouping and **cross matching and be kept ready.**

First Stage

- Patient should be in bed (lateral or propped up position preferred)
- Oxygen inhalation
- Strict asepsis
- To avoid fluid overload.

Second Stage

- Strict asepsis
- **Cut short second stage of labor** by use of prophylactic forceps or vacuum.

Third Stage

- **Active management of third stage of labor** (oxytocin or methergine following delivery of anterior shoulder) should be done to prevent PPH
- Blood loss to be replenished with BT if required
- To avoid fluid overload and watch for CCF.

Puerperium

- Prophylactic antibiotics to prevent infection
- Hematinics to be continued for at least 3 months postpartum

- **Contraceptive advice:** Patient to be counseled and explained the importance of birth spacing
- DMPA and POP are suitable. IUCDs preferably to be avoided
- Patient to be warned of danger of recurrence in subsequent pregnancies.

Q. Complications of diabetes in pregnancy.

INTRODUCTION

About 1 to 14% of all pregnancies are complicated by DM.

Diabetes in pregnancy could be:
- **Overt DM:** Preexisting diabetes which is detected before pregnancy or detected for first time during pregnancy.
- **Gestational diabetes (GDM):** It is defined as **carbohydrate intolerance** resulting in hyperglycemia of variable severity with onset or first recognition during pregnancy, whether or not insulin is used for treatment and whether or not the condition persists after pregnancy.

It generally develops at 26 to 28 weeks of gestation **as insulin resistance in pregnancy is maximum at 28 weeks of gestation.**

Pregnancy is a diabetogenic state because of:
- Insulin resistance
  - Production of HPL
  - Increased production of cortisol, estrogen, and progesterone
  - Increased destruction of insulin by kidneys and placenta
- Increased lipolysis
- Altered gluconeogenesis.

COMPLICATIONS

Effects of Pregnancy on Diabetes

- Increased insulin requirement
- Progression of diabetic retinopathy
- Worsening of diabetic nephropathy
- Worsening of diabetic cardiomyopathy.

Effects of Diabetes on Pregnancy

Complications: Maternal, Fetal, and Neonatal

Maternal

During pregnancy:
- *Abortion:* **Recurrent first trimester abortions** with uncontrolled diabetes
- Increased risk of preeclampsia (25%)
- *Polyhydramnios (25-50%):* Fetal hyperglycemia leading to polyuria, large baby and large placenta are responsible for polyhydramnios
- *Preterm labor (20%):* Due to overdistended uterus (polyhydramnios, macrosomy), infections and preeclampsia

- *Higher risk of infection:* UTI and vulvovaginitis
- *Maternal distress:* Due to polyhydramnios and macrosomy
- Ketoacidosis.

During labor

- Prolonged labor, obstructed labor
- *Shoulder dystocia:* Six times more risk compared to nondiabetics. With birthweight remaining same, the babies of diabetic mothers are more prone to develop shoulder dystocia compared to babies of nondiabetic mothers.
- *PPH:* Due to overdistented uterus (polyhydramnios, macrosomy) and preeclampsia
- *Pelvic floor trauma:* More risk of 3rd and 4th degree perineal tears
- *Operative delivery:* LSCS rates vary from 25 to 80%.

Puerperium

- Puerperal sepsis
- Lactational failure.

Late

- **Nearly 50% women with GDM will become overt DM over a period of 5 to 20 years.**

Fetal Effects

- **Congenital Anomalies** (3-10%)

It is related to severity of diabetes during organogenesis. It has been found that **higher the HbA1c higher is the risk of anomalies.**

Most common anomaly = **neural tube defects (anencephaly and spina bifida)** followed by cardiac anomalies.

Most specific anomaly = **Caudal regression syndrome/sacral agenesis.**

Central Nervous System

- Anencephaly and spina bifida
- Encephalocele
- Meningomyelocele and holoprosencephaly
- Microcephaly.

Cardiovascular

- Transposition of the great vessels
- Ventricular septal defect and atrial septal defect
- Hypoplastic left ventricle
- HOCM.

Note: VSD is the MC cardiac anomaly, TGV is the most specific cardiac anomaly in infants of diabetic mothers.

Skeletal

- Caudal regression syndrome (sacral agenesis).

Genitourinary

- Absent kidneys
- Polycystic kidneys
- Double ureter.

Gastrointestinal

- Tracheoesophageal fistula
- Bowel atresia
- Imperforate anus.

- ***Macrosomia (30–40%)***

ACOG definition: Birthweight > 4.5 kg

Pederson's Hypothesis

Maternal hyperglycemia causes fetal hyperglycemia, which in turn causes fetal hyperinsulinemia and also increase in IGF I and II which leads to fetal macrosomy.

Also elevation maternal free fatty acids leads to its increase transfer to the fetus.

- **Birth injuries:** Brachial plexus injury. Shoulder dystocia and prolonged labor are responsible
- **IUGR:** *Only in cases of long standing diabetes* **a/w maternal vasculopathy.**
- **Sudden IUFD at term:** Exact reason is not known but final event is hypoxia and acidosis.

There is increase in fetal oxygen demand and glycosylated HB binds more avidly to oxygen and releases less oxygen.

- *Gestational diabetes mostly develops at around 24–28 weeks, and hence, there is no risk of first trimester abortions and congenital anomalies in the fetus as sugars would be normal in the first trimester*
- **So remember that the 2 'A's: 'Anomalies' and 'abortions' are seen only in overt diabetes and not in GDM.**

Neonatal Effects

- Hyaline membrane disease/respiratory distress syndrome
- Hyperviscosity syndrome
- Genetic transmission (infants of mothers with type I diabetes have a 4 to 5% risk of acquiring diabetes; infants of mothers with type II diabetes have a 25 to 50% risk of diabetes)
- Hypoglycemia
- Hypocalcemia
- Hypomagnesemia
- Hyperbilirubinemia
- Polycythemia
- Cardiomyopathy
- Increase in PNM (2–3 times).

20 Preterm, Intrauterine Growth Restriction (IUGR) and Postdatism

Q. Etiology of preterm labor.

Q. Management of threatened preterm labor.

Q. Management of preterm labor.

DEFINITION

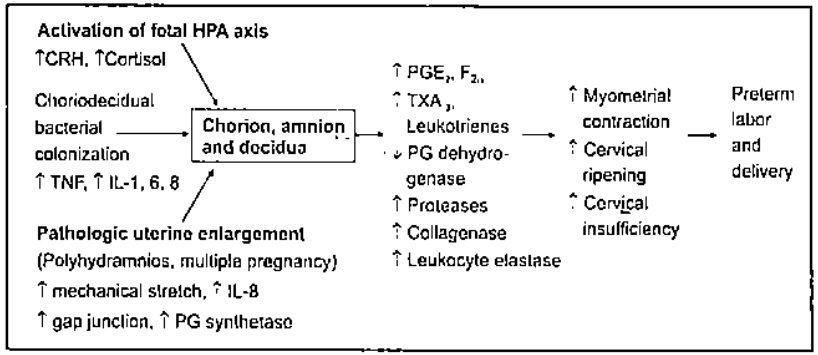
Onset of labor before 37th completed weeks of gestation is called preterm labor.

Incidence

Varies in the range of 5–10%.

Risk Factors/Etiology

Flow Chart 20.1: Etiopathogenesis of preterm labor



- **MC cause = Idiopathic.** In about 50% cases the cause is not known
- ***Infections:*** Urinary tract, dental caries, genital tract infections such as bacterial vaginosis, chlamydia, mycoplasma, etc.
- ***Over distended uterus:*** Multiple gestation, polyhydramnios, uterine anomalies like unicornuate, bicornuate uterus, macrosomy, etc.

- Prior preterm delivery: 16% risk of recurrence of birth before 34 weeks if prior one delivery is before 34 weeks and 41% risk of recurrence if prior two birth before 34 weeks.
- Pregnancy complications like preeclampsia, PROM, APH, IUFD
- *Medical and surgical illness:* Acute fever, APN, acute appendicitis, any abdominal surgeries
- Fibroids
- Smoking, illicit drug use (especially cocaine)
- Low socioeconomic status
- *Iatrogenic:* Indicated preterm delivery due to medical or obstetric complications like severe preeclampsia, eclampsia, uteroplacental insufficiency, etc.

Management

Investigations

- CBC
- Urine routine and culture and sensitivity
- High vaginal and cervical swab for culture
- USG for fetal biometry, weight, well-being, cervical length, dilatation of os, placental localization.

Treatment

- *Bedrest:* Preferably in left lateral position
- Adequate hydration
- Antibiotics if infection is present
- Two most important drugs used are **corticosteroids and tocolytic agents.**

Steroids

- Steroids (dexamethasone or betamethasone) are given to enhance **fetal lung maturity and they also decrease the incidence of intraventricular hemorrhage and NEC. This is beneficial if the delivery is delayed beyond 48 hours of the first dose.**
  - **Betamethasone 12 mg IM 2 doses 24 hours apart or dexamethasone 6 mg IM every 12 hours for 4 doses.**
  - **Betamethasone is preferred over dexamethasone, as it also prevents periventricular leukomalacia.**
- **Chorioamnionitis** and active infection in mother (e.g. open pulmonary koch) are the only contraindications for the use of steroids. They can be given to patients of hypertension and diabetes mellitus.
- Repeated doses of steroids (weekly) are **to be avoided** as they are a/w risk of necrotizing enterocolitis, intrauterine growth restriction (IUGR), pulmonary edema, and PIH.

Tocolytic Agents

- Beta 2 agonist (e.g. isoxsuprine, ritodrine, terbutaline, etc.)
- Calcium channel blocker (nifedipine): **Orally. Never sublingual**
- Indomethacin
- Magnesium sulfate
- Atosiban (oxytocin antagonist)
- Progesterone.

Tocolytics can be used for short-term use or long-term use. **Long-term use is generally avoided.**

The objectives of short-term use are:
- To delay the delivery for **at least 48 hours** for steroids to act (to prevent RDS and IVH)
- **In utero transfer** of the patient to higher center with NICU facilities.

Potential Complications of Tocolytic Agents

- **Beta-adrenergic agents**
  - Hyperglycemia
  - Hypokalemia
  - Hypotension
  - Pulmonary edema
  - Cardiac insufficiency
  - Arrhythmias
  - Myocardial ischemia
  - Maternal death.
- **Magnesium sulfate**
  - Pulmonary edema
  - Respiratory depression
  - Cardiac arrest
  - Maternal tetany
  - Muscular paralysis.
- **Indomethacin**
  - Oligohydramnios
  - Premature closure of DA
  - Renal failure
  - Gastrointestinal bleeding.
- **Nifedipine**
  - Transient hypotension
  - Headache

Contraindications to Tocolysis

Conditions where delivery is beneficial or needed are contraindications to tocolysis.
- Chorioamnionitis
- Severe preeclampsia/eclampsia
- Advanced labor
- Fetal distress
- Abruption
- IUFD
- Congenital anomalies not compatible with life
- Pregnancy >34 weeks.

Management of Preterm Labor

Multidisciplinary Team Approach (Obstetrician, anesthetist, neonatologist, and NICU FACILITIES)

- Patient is put to bed to prevent early ROM
- Adequate pain relief (epidural analgesia)

- Oxygen by breathing mask
- To watch for fetal distress during labor (**preferably continuous EFM**)
- **Birth should be gentle and slow to prevent sudden decompression**
- **Episiotomy preferred** to prevent head compression
- Tendency to delay is curtailed by low/outlet forceps
- **Early cord clamping** to prevent hypervolemia and hyperbilirubinemia in the baby
- Neonatologist to be present and immediately to shift to NICU

LSCS should be done for obstetric indications only.

Q. Complications of preterm neonate.

INTRODUCTION

- Baby born before 37 weeks of gestation is preterm. Preterm neonates can have various complications
- Developmental immaturity affects a wide range of organ systems
- The complex interplay of the mechanisms involved in preterm delivery, including inflammation and cytokine injury has been implicated in the pathogenesis of chronic lung disease, necrotizing enterocolitis, retinopathy of prematurity (ROP), and brain white matter injury in the preterm infant.

Lungs and Respiratory System

- **Respiratory Distress Syndrome**
  - RDS is associated with surfactant deficiency
  - The incidence of RDS increases with decreasing gestational age. Respiratory distress is less common in infants born at 33 to 36 weeks of gestation and is rare in full-term infants
  - Antenatal administration of glucocorticoids to women at risk for preterm delivery reduces the incidence and severity of RDS as well as the rate of mortality
  - Soon after birth, preterm infants with RDS develop rapid breathing, grunting, poor color, and crackling or diminished breath sounds breathing requires increased work. Respiratory failure because of fatigue, apnea, hypoxia, or an air leak (from alveolar injury) results from stiff lungs that need high pressures for ventilation.
- **Congenital pneumonia**
- **Bronchopulmonary Dysplasia and Chronic Lung Disease**
  - The chronic lung disease (CLD) that sometimes follows RDS in preterm infants is also called bronchopulmonary dysplasia (BPD). BPD/CLD is a chronic disorder that results from inflammation, injury, and scarring of the airways and the alveoli.
- **Apnea:** Another complication of preterm birth is apnea in which infants may stop breathing for 20 seconds or more, sometimes accompanied by bradycardia.

Gastrointestinal System

Preterm infants have difficulty with digesting nutrients because many specialized cells are not fully functional.

- **Necrotizing enterocolitis (NEC)**
  - It is an acute injury of the small or large intestines that causes inflammation and injury to the bowel lining and that primarily affects preterm infants.

  - NEC occurs in three percent of infants born before 33 weeks of gestation and in seven percent of infants with birth weights less than 1,500 grams.
  - It typically occurs within 2 weeks of birth and presents as feeding difficulties, abdominal swelling, hypotension, and other signs of sepsis. When NEC is suspected, infants are treated with antibiotics and bowel rest (i.e. no feedings).
  - The exact cause of NEC is unknown. The preterm infant's intestinal lining is fragile, and stresses (infections and insufficient oxygen or blood flow) can injure it. Inflammation is important in terms of both the etiology and the outcomes.
  - Injury to the GI tract lining can progress through the wall of the intestines, causing perforation and spilling of the intestinal contents into the abdomen, which causes peritonitis and sepsis.
- **Gastroesophageal reflux (GER)** is common in preterm infants, often presents as regurgitation, and may adversely affect growth and health. It may also be manifested by aspiration pneumonia, wheezing, or worsening of BPD/CLD because of an inability to protect the airway when refluxing.

Skin

- Skin plays important roles in fluid balance, temperature regulation, and the prevention of infection. The skin of infants born at the lower limit of viability is generally gelatinous, is easily injured when touched, allows tremendous loss of fluids, and does not provide an adequate barrier to infection.
- **Hypothermia** can develop as there is reduced subcutaneous and brown fat.

Infections and the Immune System

- Preterm infants have immature immune systems that are inefficient at fighting off the bacteria, viruses, and other organisms that can cause infections.
- The most serious manifestations of infections with these agents commonly seen in preterm infants include **pneumonia, sepsis, meningitis, and urinary tract infections.**
- As many as 65 percent of infants with birth weights of less than 1,000 grams have at least one infection during their initial hospitalization.
- Invasive fungal infections occur in six to seven percent of infants in an NICU, and the rates of such infections increase with decreasing gestational age and birth weight.

Cardiovascular System

- **PDA:** Which can lead to heart failure and reduced blood flow to vital body organs
- **Hypotension** is a frequent concern in preterm infants. The administration of boluses of normal saline and pressors is used to support blood pressure.

Hematologic System

- **Anemia:** Fetal blood loss, fetomaternal hemorrhage, and hemolysis can all result in congenital anemia, but the most common hematologic complication in preterm infants is anemia of prematurity. Anemia of prematurity is an exaggeration of the physiological anemia of infancy because of suppressed hematopoiesis for 6 to 12 weeks after birth and is earlier in onset and symptomatic.

Auditory System

- **Hearing disorder** is attributed to infections, immaturity, asphyxia, ototoxic medications, and hyperbilirubinemia.
- Ventilated infants are at increased risk for **otitis media**.

Ophthalmic System

- **ROP** is the most common eye abnormality in preterm infants. It is a neovascular retinal disorder, and its incidence increases with decreasing gestational age and decreasing birth weight. It is multifactorial in etiology with the primary determinant being immaturity with an avascular retina. Environmental factors, including hypoxia, hyperoxia, variations in blood pressure, sepsis, and acidosis may injure the endothelium of the immature retinal blood vessels.

Other ophthalmologic complications of prematurity include:
- Refractive disorders (especially myopia)
- Strabismus
- Amblyopia
- Optic nerve atrophy
- Cataracts
- Cortical visual impairment.

Late ophthalmologic problems include:
- Angle closure glaucoma
- Retinal detachment and
- Phthisis.

Central Nervous System

The most common signs of CNS injury in preterm infants are:
- IVH
- Intraparenchymal hemorrhage (Iph)
- White matter injury including periventricular leukomalacia
  - IVH generally begins with bleeding into the germinal matrix just below the lateral ventricles (i.e. a subependymal or germinal matrix hemorrhage).
  - The incidence and severity of IVH increase with decreasing gestational age and birth weight. Factors that contribute to IVH include hypotension, hypertension, fluctuating blood pressures, poor autoregulation of cerebral blood flow, disturbances in coagulation, hyperosmolarity, and injury to the vascular endothelium by oxygen free radicals.
  - Severe IVH can lead to ventricular dilation and posthemorrhagic hydrocephalus if there is an obstruction to the flow of cerebrospinal fluid with increased intracranial pressure.
- White matter injury and periventricular leukomalacia
- Injury to the periventricular white matter is a sign of CNS injury and is a complication of preterm birth. White matter injury includes a spectrum of CNS injuries, from focal cystic necrotic lesions (also called PVL) to ventricular dilation with irregular ventricular edges or cerebral atrophy.

Renal System

Oliguria, anuria as the immature kidneys are unable water, solute, and acid loads.

Q. Types of IUGR.

Q. Etiology of IUGR.

Q. Management of IUGR.

DEFINITION

Birthweight is below the tenth percentile of average for the gestational age.

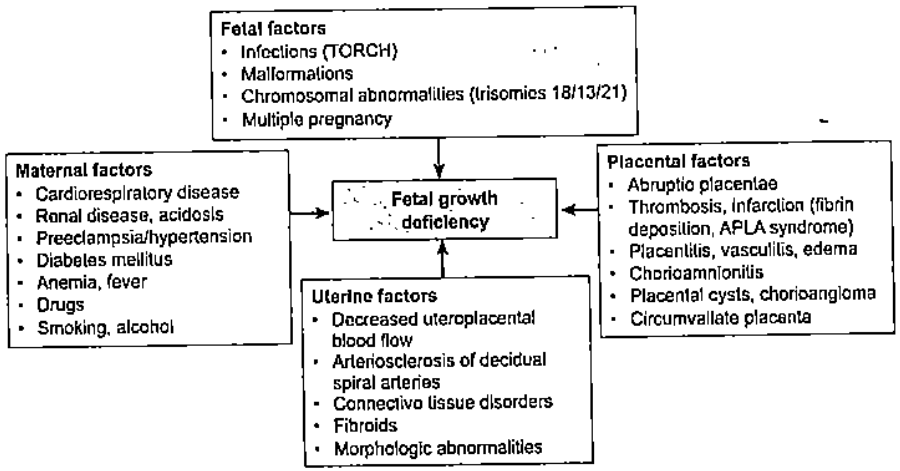
Comparison of symmetric and asymmetric IUGR fetuses

Symmetric (20%)	Asymmetric (80%)
Symmetrically small	Head larger than abdomen
Normal ponderal index	Low ponderal index
Head/abdomen and femur/abdomen ratios = normal	Elevated head/abdomen and femur/abdomen ratios
a/w genetic disease, infection	Placental vascular insufficiency
Total number of cells = less	Normal
Cell size = Normal	Smaller
Complicated neonatal course; Poor prognosis	Usually uncomplicated neonatal course and good prognosis
Insult is early in pregnancy	Fetus affected in later months
Brain sparing effect: Absent	Present

Incidence

About 5-15%

Causes of IUGR



The cause remains unknown in about 40% cases.

Diagnosis

Clinical

- Palpation of uterus for fundal height, approximate liquor volume and fetal weight
- **Symphysio-Fundal Height (SFH)** correlates with gestational age. It would be less than what it should be. A lag of 4 cm or more suggests growth restriction. Serial measurement is important
- Abdominal girth will be less than expected
- Maternal weight gain remains stationary or can even decrease.

USG

USG helps to diagnose IUGR and also its type (symmetric or asymmetric). Also it can detect associated oligohydramnios, the EBW and any congenital anomalies in the fetus.

USG Markers for Asymmetric IUGR

- **Abdominal circumference (on USG) is the best marker** for IUGR followed by ponderal index
- **Ponderal index** (PI) = fetal weight divided by third power of femur length. Normal = 8.3
- PI < 7 indicates IUGR
- FL/AC = 22% is normal, > 23.5% suggests IUGR
- **Normally after 34 weeks, HC/AC is less than one.** If it is more than one it suggests asymmetric IUGR
- Fetal glycogen stores from liver are depleted and there is redistribution of blood flow; therefore, AC is smaller than other parameters (BPD and femur length) on USG. FL is not affected by nutrition status
- **AFI:** In asymetric IUGR, the liquor is would be less because of brain sparing effect.

Color Doppler is the **best investigation** for the management of **asymmetrical** IUGR.

Serial color Doppler is very important.

Maternal uterine artery is only for prediction and not for management: Increased impedance of maternal uterine artery velocimetry **(presence of diastolic notch)** at 16-20 weeks is **predictive of preeclampsia and IUGR.**

Fetal vessels color Doppler is very important in management.

Fetal vessels which are examined are: Umbilical artery, middle cerebral artery (MCA) and ductus venosus

- Umbilical artery Doppler is considered abnormal if the S/D ratio is above the 95th percentile for gestational age **(rising S/D ratio is the earliest change in IUGR)**
- Absent diastolic flow in umbilical artery is an ominous sign, and IUFD can be expected within 7 days
- In extreme cases of growth restriction, end diastolic flow in **umbilical artery and ductus venosus may become reversed** and IUFD will occur within 48 hours

- As the S/D ratio begins to rise in fetus with asymmetric IUGR, **the blood flow in MCA** increases. There is redistribution of blood flow, and vital organs like brain continue to receive adequate blood at the expense of liver and kidney. This is called as **Brain-sparing effect.**
- **Absent and reversed diastolic flow in umbilical artery on color Doppler is an indication of immediate LSCS (as they indicate impending death).**

Management

In cases of symmetric IUGR, congenital anomalies, infections, and chromosomal abnormalities should be ruled out. There is no effective treatment.

For Asymmetric IUGR

- To monitor for fetal well-being very closely and
- Timely delivery to prevent IUFD.

General

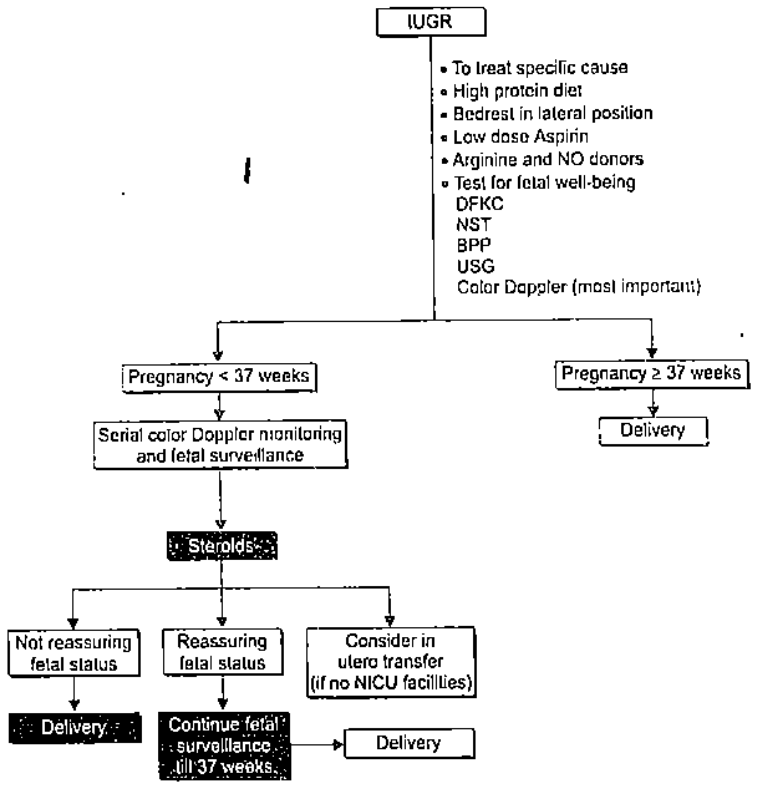
There is **no proven definitive treatment**, but the following may help:

- Adequate bed rest in left lateral position
- **High protein diet**, 300 extra calories/day, hyperalimentation by amino acids especially arginine
- **Corticosteroids** to be given prophylactically to enhance the lung maturity, as early delivery maybe necessary
- Avoiding smoking, nicotine, tobacco, and alcohol
- Control of hypertension, preeclampsia if present
- Maternal hyper oxygenation @2.5 litres/day
- Maternal volume expansion may help in improving placental perfusion
- **Low dose aspirin 75–150 mg** maybe helpful in selected cases of preeclampsia, thrombophilias
- DHA supplementation may also help
- **Arginine and nitric oxide donors** are known to increase the placental blood flow and help in utreoplacental insufficiency
- NTG patches applied on the abdomen are also known to have similar effect
- **Sildenafil 25 mg two to three times** a day can be tried to improve placental blood flow and help in utreoplacental insufficiency.

Test for Fetal Well-Being

- DFKC
- NST, weekly or biweekly
- BPP
- USG for AFI, EBW, placental maturity. Serial USG 3-4 weeks interval maybe needed
- Color Doppler is the most important and serial color Doppler helps in timing of the delivery as mentioned before (Flow Chart 20.2).

Flow Chart 20.2: Management of IUGR



Method of Delivery

- *In cases of elevated S/D ratio or increase in MCA flow:* Vaginal delivery is tried first keeping a close watch on fetal heart rate.
- PGs can be used if cervix is unfavorable or ARM and oxytocin in cases of favorable cervix. Liquor color will also guide futher management.
- **Absent and reversed diastolic flow in umbilical artery on color Doppler is an indication of immediate LSCS** (as they indicate impending death) as vaginal delivery is detrimental of the baby.

Care During Delivery

Multidisciplinary Team Approach

(Obstetrician, anesthetist, neonatologist and NICU facilities).

- Patient is put to bed to prevent early ROM
- Adequate pain relief (epidural analgesia)

- Oxygen by breathing mask
- To watch for fetal distress during labor **(preferably continuous EFM)**
- Birth should be gentle and slow to prevent sudden decompression
- Episiotomy preferred to prevent head compression
- Tendency to delay is curtailed by low/outlet forceps
- **Early cord clamping** to prevent hypervolemia and hyperbilirubinemia in the baby
- Neonatologist to be present and to shift baby to NICU if needed.

Q. Complications and management of postdatism.

DEFINITION

Postdatism is defined as pregnancy continuing beyond EDD or 40 weeks of gestation whereas Post-term is pregnancy continuing more than 42 weeks.

Etiology/Risk Factors

- Idiopathic
- Past history/Family history
- Anencephaly
- Fetal adrenal hypoplasia
- X-linked placental sulfatase deficiency
- Primiparity.

COMPLICATIONS

Fetal

Antenatal

Placental ageing leads to placental insufficiency leading to:

- **Oligohydramnios:** Liquor is 800 ml at 40 weeks and about 450 ml at 42 weeks
- MSAF (Meconium Stained Amniotic Fluid)
- Fetal hypoxia, distress and **sudden IUFD**.

Intrapartum

- Meconium aspiration
- Cord compression
- Shoulder dystocia, birth trauma
- Uterine dysfunction
- Increased risk of operative delivery
- Fetal hypoxia, distress.

After Birth

- Meconium aspiration syndrome
- Hypoglycemia
- Polycythemia.

Maternal

Increased risk of induction of labor, instrumental delivery and LSCS.

Clinical Features

In cases of post-term pregnancy the following features maybe seen:

- Stationary or falling weight
- Diminished abdominal girth due to decrease in liquor
- Uterus feels **'full of fetus' due to decrease in liquor**
- Hard skull bones on abdominal and vaginal examination.

Management

To confirm postdatism and fetal maturity:

Menstrual History

It is very useful to calculate the period of gestation if the patient is sure of her dates and has previous regular cycles.

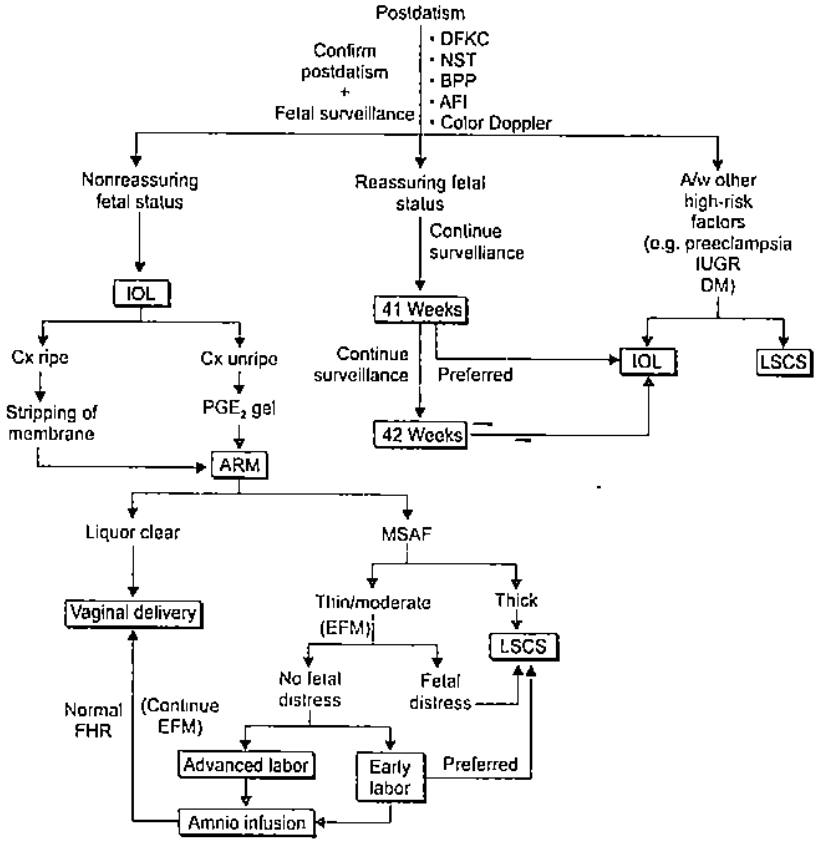
But in cases of mistaken/wrong dates or if the patient has conceived in lactational amenorrhea then the following maybe helpful:

- Clinical Criteria needed to confirm a term gestation are:
  - Fetal heart tones have been demonstrated for at least 20 completed weeks by stethoscope or at least 30 completed weeks by Doppler ultrasound
  - Appropriate uterine size was established by pelvic examination prior to 16 weeks.
- Ultrasound determinations needed to confirm a term gestation:
  - Gestational age based on the measurement of **crown-rump length obtained between 6–11 weeks of gestation (dating scan)**
  - **To confirm postdatism USG in first trimester (dating scan) is most useful.**
  - Very rarely amniocentesis and biochemical (L:S RATIO) and cytological evaluation of amniotic fluid may be required. However in presence of USG this is rarely required.

Assessment of fetal being/to detect placental insufficiency:

- **NST (Biweekly)**
- BPP and AFI
- Color Doppler
- Delivery should be done if there is evidence of fetal compromise or oligohydramnios.
- Prostaglandin can be used for cervical ripening and labor induction.

Flow Chart 20.3: Management of postdatism



IOL: Induction of labor.

21 Puerperal Sepsis

Q. Define puerperal pyrexia. Causes of puerperal pyrexia.

Q. Puerperal sepsis.

PUERPERAL PYREXIA

A rise in temperature reaching **100.4 °F (38 °C) or more** (measured orally) on **two separate occasions at 24 hours apart (excluding the first 24 hours) within the first 10 days** following delivery is called puerperal pyrexia.

Causes

- Puerperal sepsis
- Acute pyelonephritis,cystitis
- Breast engorgement, mastitis
- Wound infection (LSCS /episiotomy)
- Thrombophlebitis
- Atelectasis and pneumonia
- Unknown origin
- A recrudescence of malaria, pulmonary TB.

Puerperal Sepsis

An infection of the genital tract which occurs as a complication of delivery is called puerperal sepsis.

Postpartum uterine infection has been called variously endometritis, endomyometritis, and endoparametritis. Because infection actually involves not only the decidua but also the myometrium and parametrial tissues, the preferred term is metritis with pelvic cellulitis.

Route of Delivery

The **route of delivery** is the **single most significant risk factor** for the development of uterine infection.

Compared with cesarean delivery, metritis following vaginal delivery is relatively uncommon.

Most female pelvic infections are caused by bacteria indigenous to the female genital tract.

Predisposing Factors of Puerperal Sepsis

Antepartum

- Malnutrition and anemia
- Preeclampsia
- PROM
- Immunocompromised status (HIV)
- Diabetes mellitus
- Obesity.

Intrapartum

- Multiple cervical examinations
- Internal fetal monitoring
- Chorioamnionitis
- Retained placenta
- APH and PPH
- Prolonged labor, prolonged rupture of membranes (>18 hours)
- Operative delivery (LSCS)
- MSAF
- Traumatic operative delivery
- Placenta previa (placental site is close to vagina).

BACTERIA COMMONLY RESPONSIBLE FOR FEMALE GENITAL INFECTIONS

Aerobes

- Group A, B, D streptococci
- Enterococcus
- Gram-negative bacteria—*Escherichia coli, Klebsiella*, and *Proteus* species
- *Staphylococcus aureus*
- *Gardnerella vaginalis.*

Anaerobes

- Peptococcus species
- Peptostreptococcus species
- Bacteroides species
- Clostridium species
- Fusobacterium species
- Mobiluncus species.

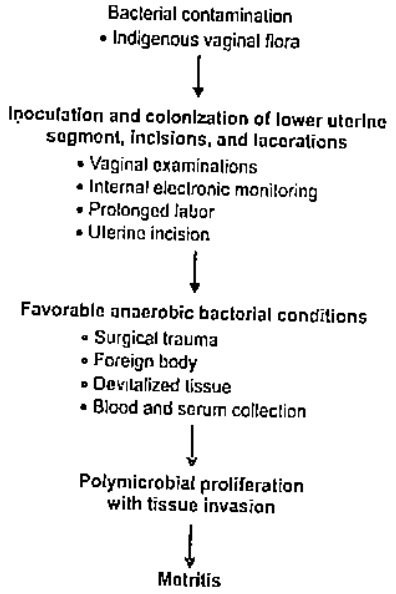
Other

- Mycoplasma species
- *Chlamydia trachomatis*
- *Neisseria gonorrhea.*

Most of the infections are polymicrobial

- **Fever is the most important criterion** for the diagnosis of postpartum metritis. Temperature commonly exceeds 38–39 °C. Chills may accompany fever and suggest bacteremia, which is documented in 10–20% of women with pelvic infection following cesarean delivery.
- Women have foul-smelling lochia without evidence for infection. Other infections, notably those due to **group A β-hemolytic streptococci, are frequently associated with scanty, odorless lochia.**
- Leukocytosis may range from 15,000 to 30,000 cells/μl.
- Complications of metritis that cause persistent fever despite appropriate therapy include a **parametrial phlegmon** or an area of intense cellulitis, a surgical incisional or pelvic abscess, and infected hematoma, and septic pelvic thrombophlebitis (Flow Chart 21.1).

Flow Chart 21.1: Pathogenesis of metritis following cesarean delivery



Clinical Features

Endometritis

- Rise in temperature with **chills and rigor**
- Tachycardia
- Lower abdominal tenderness on one or both sides of the abdomen
- Adnexal and parametrial tenderness
- **Foul-smelling lochia** without other evidence of infection
- Some infections, most notably caused by group A beta-hemolytic streptococci, are frequently associated with scanty, odorless lochia
- Uterus is tender and **subinvoluted.**

Wound Infections

Patients with wound infections, or episiotomy infections, have erythema, edema, tenderness out of proportion to expected postpartum pain, and discharge from the wound or episiotomy site. With severe infection there is fever with chills.

Parametritis

- The onset is usually about 7–10th day of puerperium
- Constant pelvic pain
- PV: **Unilateral tender indurated mass** pushing the uterus to the opposite side
- PR: Confirms the induration extending along uterosacral ligament
- Spiky temperature and fever with chills.

Pelvic Abscess

- Bulging, fluctuant mass in POD
- Swinging temperature
- Diarrhea.

Pelvic Peritonitis

- Pyrexia
- Tachycardia
- Lower abdominal pain and tenderness
- Forniceal and cervical movement tenderness.

Investigations

- Complete blood count: Differential count, platelets and Hb
- Electrolytes
- Thick blood film for malarial parasites
- Blood cultures, if sepsis is suspected
- Urinalysis, with cultures and sensitivity tests
- High vaginal and endocervical swabs for cultures (aerobic and anaerobic) and antibiotic sensitivity
- Wound cultures, if appropriate
- Coagulation studies, if pelvic thrombosis, deep vein thrombosis, pulmonary embolism, or invasive treatment (e.g., surgical procedure) is being considered
- Pelvic ultrasonography may be helpful in detecting retained products of conception, pelvic abscess, or infected hematoma. Color Doppler to detect venous thrombosis
- Contrast-enhanced CT or MRI are useful in establishing the diagnosis of septic pelvic thrombosis
- Chest X-ray to detect pulmonary pathology like collapse and atelectasis and also in cases of pulmonary Kochs.

Treatment

- Patient isolation especially if hemolytic streptococci is obtained on culture
- IV fluids

- To correct anemia with iron and blood transfusion in severe cases
- To maintain temperature, pulse, BP, urine output chart.

ANTIMICROBIAL REGIMENS FOR PELVIC INFECTION FOLLOWING CESAREAN DELIVERY

Regimen

- Clindamycin 900 mg + gentamicin 1.5 mg/kg, q8h intravenously
  Gold standard, 90–97% efficacy, once-daily gentamicin dosing acceptable
- Plus ampicillin: Added to regimen with sepsis syndrome or suspected enterococcal infection
- Clindamycin + aztreonam: Gentamicin substitute in patients with renal insufficiency
- Extended-spectrum penicillins
  Piperacillin, ampicillin/sulbactam can be used
- Imipenem + cilastatin: Reserved for special indications.

Surgical Treatment

Limited Role

- **Wound infection or episiotomy infection:** Drainage, debridement, and irrigation may be required. After the infection is controlled secondary suturing may be required.
- **Retained products:** Surgical evacuation under antibiotic cover
- **Pelvic abscess:** Drained by colpotomy under USG guidance
- **Unresponsive peritonitis:** Laparotomy and drainage of pus.
- Hysterectomy only in cases of **rupture or perforation having multiple abscesses, gangrenous uterus or gas gangrene infection.** Ruptured tubo ovarian abscess should be removed.

Q. Physiology of Lactation

500–800 ml of milk is produced by a healthy mother per day which requires 700 kcal/day

Physiological basis consists of 4 phases:

1. **Mammogenesis:** Growth of both ductal and lobuloalveolar systems occurs in pregnancy.
2. **Lactogenesis:** Colostrum secretion is noted during pregnancy and immediately post delivery. Milk secretion starts around the third postpartum day. Despite high prolactin levels in pregnancy, milk is not secreted as the high levels of estrogen and progesterone make the breast tissue unresponsive. **With withdrawal of estrogen and progesterone levels post delivery, prolactin acts on the breast tissue**. Growth hormone, thyroxine and insulin also increase secretory activity.
3. **Galactokinesis:** Milk is discharged due to the infant's suckling efforts and also a contractile mechanism which expresses milk from the alveoli into the ducts. **Oxytocin is a major galactokinetic hormone.**
4. **Galactopoeisis:** The most important hormone is **prolactin.** Suckling is essential for effective and continuous lactation. Periodic feeding is essential to relieve the pressure which then maintains the secretion.

Milk Ejection Reflex

Reflex by which milk is forced down into the ampulla of the lactiferous duct, where it can be sucked out by the baby. This reflex is inhibited by adverse psychic conditions, pain or breast engorgement (Fig. 21.1).

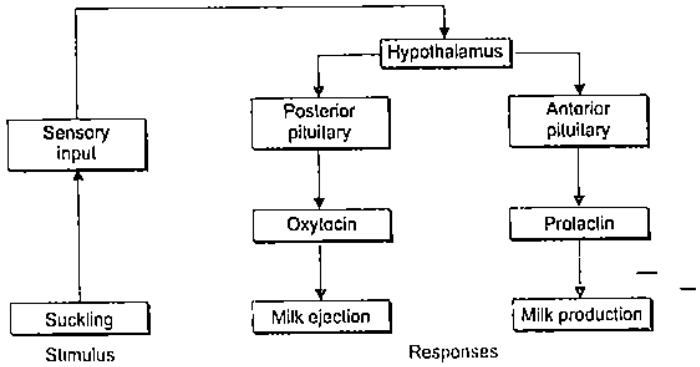


Fig. 21.1: Milk ejection reflex

Stimulation of Lactation

Early motivation for breastfeeding. Early (within half an hour of delivery) and exclusive breastfeeding in the correct position should be encouraged.

Important steps are:
- To put the baby to breast every 2 hours
- Plenty of oral fluids
- To avoid breast engorgement.

Drugs that improve milk production (galactogogues): Metoclopramide 10 mg thrice daily increases prolactin levels. Oxytocin contracts myoepithelial cells and causes milk let down.

Lactation Suppression

Indications

- IUFD, Neonatal death
- Breastfeeding not possible due to personal/medical reasons
- Breastfeeding is contraindicated.

Methods

- To stop breastfeeding
- To avoid breast handling
- Tight breast support, compression bandage
- Ice packs

- Analgesics
- Medical methods: **Cabergolin, bromocriptine**, estrogen, androgen, pyridoxine.

Q. Advantages of breastfeeding.

- Breast milk is the ideal and only natural food designed for baby.
  Carbohydrates: Mainly lactose, stimulates growth of intestinal flora and helps in vitamin B synthesis.

  Fat: Smaller fat globules, easily digested

  Proteins: Rich in lactalbumin and lactoglobulin

  Low osmotic load, **less burden on kidneys.**
- Protection against infections and deficiency states
  - **Vitamin D**, protects against rickets
  - Leukocytes, lactoperoxidase, lysozyme, lactoferrin, **interferons and immunoglobulins** Ig A,M and G all protect against infections
  - **Omega 3 fatty acids:** Neurological development.
- It is free. It is available whenever and wherever the baby needs a feed and its at the right temperature.
- It can build a strong physical and **emotional bond** between mother and baby.
- Breastfed babies have:
  - Less chance of diarrhea and vomiting ,fewer chest and ear infections
  - Less chance of being constipated
  - Less likelihood of becoming obese and therefore developing type 2 diabetes and other obesity-related illnesses later in life
  - Less chance of developing eczema
  - Decreased risk of developing chronic conditions, such as type I diabetes, celiac disease and Crohn's disease.
- Less risk of cancer: Breastfeeding can decrease baby's risk of some childhood cancers and **lowers mother's risk of premenopausal breast cancer and ovarian cancer.**
- Exclusive breastfeeding can also delay the return of periods and act as **natural contraception.**
- The oxytocin released when the baby nurses helps uterus contract, reducing postdelivery blood loss. Plus, breastfeeding will help in involution of the uterus.

Q. Baby friendly hospital.

The Baby Friendly Hospital Initiative (BFHI), also known as Baby Friendly Initiative (BFI), is a worldwide programme of the **World Health Organization and UNICEF, launched in 1991** following the adoption of the Innocenti Declaration on breastfeeding promotion in 1990.

The criteria for a hospital's Baby Friendly accreditation include:

1. Have a written breastfeeding policy that is routinely communicated to all health care staff.
2. Train all health care staff in skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding within one hour of birth.
5. Show mothers how to breastfeed and maintain lactation, even if they should be separated from their infants.

6. Give newborn infants no food or drink other than breastmilk, not even sips of water, unless medically indicated.
7. Practice rooming in—that is, allow mothers and infants to remain together 24 hours a day.
8. Encourage breastfeeding on demand.
9. Give no artificial teats or pacifiers (also called dummies or soothers) to breastfeeding infants.
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic.

22 Obstructed Labor and Rupture Uterus

Q. Causes, clinical features, and management of obstructed labor.

Q. Bandl's ring.

Q. Difference between Schroeder's ring and Bandl's ring.

INTRODUCTION

Obstructed labor is one of the common preventable causes of maternal and perinatal morbidity and mortality in developing countries in which there is no easy access to health facilities with the capability of carrying out operative deliveries.

Definition

Inspite of good/adequate uterine contractions, the progressive descent of presenting part is arrested due to mechanical obstruction (fault with passage and/or passenger).

The prevalence in 1–2% in developing countries.

Causes

There is no fault in power (remember the Power is always normal or even more than normal in obstructed labor).

Fault in Passage

- **CPD**
- **Contracted pelvis** (Undernutrition in childhood is common resulting in small pelves in women)
- Cervical dystocia (prolapse or scarring)
- Cervical or broad ligament fibroid
- Impacted ovarian tumor/nongravid horn of bicornaute uterus below presenting part.

Fault in Passenger

- **Transverse lie**
- Brow/mento posterior face presentation/compound presentation
- Occipito-posterior postion

- **Hydrocephalus/fetal ascites**
- **Macrosomy**
- Locked twins.

EFFECTS/COMPLICATIONS

Maternal

Immediate

- Exhaustion
- Dehydration, **ketoacidosis** (increased muscular activity without fluids and accumulation of lactic acid and ketones)
- Genital sepsis, chorioamnionitis
- **Rupture uterus** (spontaneous or traumatic following instrumental delivery)
- **PPH** and shock (atonic and traumatic)
- **Maternal morbidity and mortality** (death mainly due to rupture uterus, shock, and sepsis).

Late

- **Genitourinary fistula (VVF and RVF)**
- Vaginal atesia
- **Sheehans syndrome**
- Secondary amenorrhea (Following hysterectomy in cases of rupture uterus).

Fetal

- Asphyxia (tonic uterine contractions or cord prolapse in transverse lie)
- Acidosis (fetal hypoxia and maternal acidosis)
- Intracranial hemorrhage (supermoulding, tentorial tear or traumatic deliver)
- Infections
- IUFD
- Perinatal mortality.

Clinical Features

- Patient is in agony, discomfort, restless
- Exhaustion and ketoacidosis
- Other features as mentioned in table of Retraction/Bandl's ring below:

	Constriction ring (Schroeder's ring)	Retraction ring (Bandl's ring)
Nature	It is a manifestation of localized incoordinate uterine contraction	It is an end result of tonic uterine contraction and retraction
Cause	Undue irritability of the uterus	Following obstructed labor
Situation	Usually at the junction of upper and lower segment but may occur in other places. The position does not alter	Always situated at the junction of upper and lower segment. The position progressively moves upward

Contd...

Contd...

	Constriction ring (Schroeder's ring)	Retraction ring (Bandl's ring)
Uterus	Upper segment contracts and retracts with relaxation in between, lower segment remains thick and loose	Upper segment is tonically contracted with no relaxation. The wall becomes thicker; lower segment becomes distended and thinned out
Maternal condition	Almost unaffected unless the labor is prolonged	Maternal distress, exhaustion, sepsis appear early
Abdominal examination	• Uterus feels normal and not tender • Fetal parts are easily felt • Ring is not felt • Round ligament is not felt • FHS is usually present	• Uterus is tense and tender • Not easily felt • Ring is felt as a groove placed obliquely • Taut and tender round ligaments are felt • FHS is usually absent
Vaginal examination	• The lower segment is not pressed by the presenting part • Ring is felt usually above the head • Features of obstructed labor are absent	• Lower segment is very much pressed by the forcibly driven presenting part • Ring cannot be felt vaginally • Features of obstructed labor are present
End result	• Exhaustion to the mother is a late feature • Fetal anoxia due to prolonged uterine hypertonic state may appear late • Chance of uterine rupture is absent	• Exhaustion and sepsis appear early • Fetal death is usually early due to tonic contraction and exaggerated retraction • Ruptured uterus more in multigravidas and uterine exhaustion and rupture in primigravidas are the common mode to terminations
Principle of treatment	To relax the ring followed by delivery of the baby. LSCS and cut the ring if needed	**To relieve the obstruction by safe procedure (usually LSCS even in cases of IUFD) after excluding ruptured uterus**

TREATMENT

The principles are '**Never wait and Watch** and **Never use oxytocin**'

- To relieve the obstruction at the earliest by safe delivery
- To combat dehydration, ketoacidosis
- To control sepsis.

Preliminaries

- Two wide bore IV lines
- Send blood for cross matching and keep one pint ready
- IV fluids (RL) at least one liter in given in running drip. At least three liters of fluid required
- IV antibiotics cefotaxim or ceftriaxone 1 gm and metronidazole.

Definitive Treatment

- Rupture uterus must be ruled out first
- '**NEVER WAIT and WATCH and NEVER USE OXYTOCIN**' as it increases the risk of rupture uterus
- **There is NO PLACE for internal version and destructive operations in modern day obstetrics**
- LSCS gives the best results and would be required in majority cases (will have to be done **even in cases of IUFD**)
- If the head is low down (station +2 or +3) and vaginal delivery is not risky the forceps extraction can be done.

Prevention

- Antenatal detection of factors likely to cause obstructed labor (MACROSOMY, CPD, short stature, malpresentations, etc).
- Intranatal: Use of **partograph**, strict vigilance and timely intervention and referral if needed.

Q. Causes of rupture uterus.

Q. Etiology, clinical features, and management of rupture uterus.

Definition

Disruption in the continuity of all uterine layers: endometrium, myometrium and serosa anytime beyond 28 weeks of pregnancy is called rupture of uterus.

Incidence

1 in 2000 to 1 in 200 deliveries. Rupture uterus from obstructed labor is becoming less because of improved obstetric care, but prevalence of scar rupture is increased because of increase in LSCS rates.

Etiology

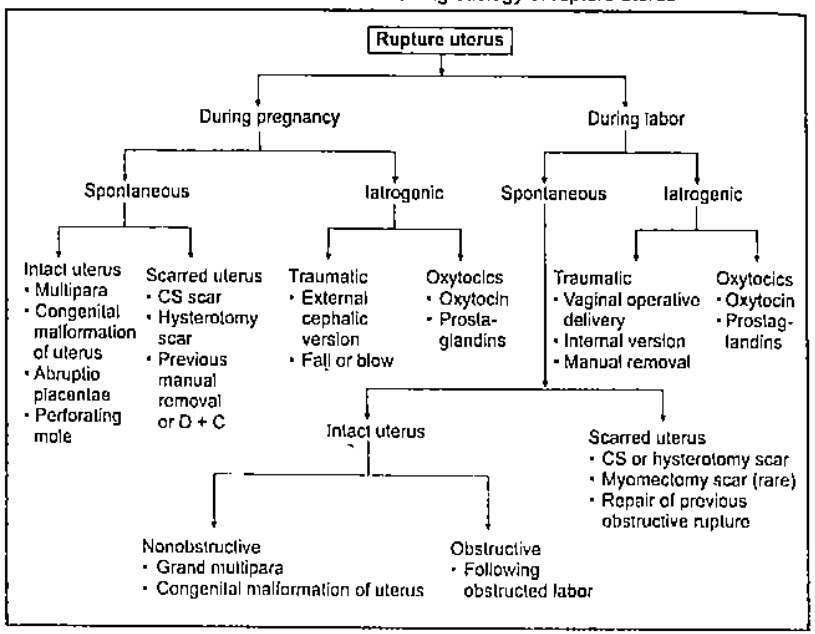
Rupture of previous LSCS scar during VBAC is one of the commonest cause of rupture uterus today.

- Spontaneous (intact or unscarred uterus)
- Scar rupture
- Iatrogenic.

The rupture can happen during pregnancy or during labor (Flow Chart 22.1).

- **Spontaneous rupture** during pregnancy is **very very rare** and is complete and involves the upper segment and occurs in later months of pregnancy
- Obstructive rupture (following obstructed labor) involves the lower segment and extends through one lateral wall to the upper segment
- Classical C. section scar or hysterotomy scar generally gives way in **third trimester** of pregnancy

Flow Chart 22.1: Scheme showing etiology of rupture uterus



- **LSCS scar generally ruptures in labor (mainly in second stage** or towards end of first stage) and unlikely to rupture during pregnancy
- Iatrogenic rupture is mainly due to injudicious use of **oxytocin or prostaglandins** (for induction or augmentation of labor) and **very very rarely due to Internal Podalic Version and destructive operations as they are not performed in modern day obstetrics.**

Clinical Features

- During pregnancy: Classical or hysterotomy scar:
  - Dull abdominal pain over scar area
  - Vaginal bleeding
  - Tenderness on palpation
  - Fetal distress/IUFD
  - Something giving way, acute pain and collapse when the rupture is complete

Following uterine rupture the most common electronic fetal monitoring finding tends to be sudden, severe heart rate decelerations that may evolve into late decelerations, bradycardia, and undetectable fetal heart action.

During Labor

Obstructive Rupture

- **Premonitory phase:**
  - Generally multipara in labor with features of obstruction

  - Strong frequent uterine contractions followed by continuous pain in suprapubic area
  - Dehydration, exhaustion, tachycardia, rise in temperature
  - Distended lower segment, Bandl's ring
  - Fetal distress/IUFD
  - Presenting part jammed in pelvis, dry, edematous vagina.
- **Phase of rupture:**
  - **Something giving way** at height of contraction
  - **Cessation of contractions**
  - Shock and exhaustion
  - **Superficial fetal parts**
  - Absence of FHS
  - A firm contracted uterus may at times be felt alongside the fetus
  - **PV: Loss of station** (recession of presenting part)
  - Bleeding PV.

With rupture and expulsion of the fetus into the peritoneal cavity, the chances for intact fetal survival are dismal and reported mortality rates range from 50 to 75%.

LSCS Scar Rupture

Impending scar rupture (Scar dehiscence)	Scar rupture
Unexplained tachycardia	Weak thready fast pulse
Hypotension	Shock
Fetal tachycardia	Persistent fetal bradycardia/IUFD
Uterine scar tenderness	Hematuria
Bleeding PV	Bleeding PV
Hematuria	Recession of presenting part

Rupture following Instrumental Or Manipulative Delivery

- Sudden deterioration of general condition of patient and vaginal bleeding following delivery
- Exploration of uterus to feel the rent confirms diagnosis
- Shock, broad ligament hematoma
- Shortening of cord immediately following a difficult vaginal delivery is pathognomic.

Treatment

Resuscitation

- Two wide bore IV lines
- Send blood for cross matching
- **IV fluids (RL)** in running drip
- IV antibiotics cefotaxim or ceftriaxone 1 gm and metronidazole
- **Start blood transfusion.**

Laparotomy

Hysterectomy

- **Unless there is sufficient reason to preserve the uterus: Quick subtotal hysterectomy** is needed in most of the cases, especially in multipara and obstructive rupture
- If condition permits and/or there is colporrhexis, total hysterectomy maybe done.

Repair

- Mostly in cases of scar rupture, where margins are clean or in obstructive rupture and desirous of child
- Excision of fibrous/necrosed tissue followed by suturing the defect
- **Sterilization (tubal ligation) to be offered.**

PREVENTION

- Antenatal detection of factors likely to cause obstructed labor (MACROSOMY, CPD, short stature, malpresentations, etc.) and rupture uterus (previous cesarean section, hysterotomy, myomectomy) and **mandatory hospital delivery**
- Avoid undue force in ECV
- Judicious selection of cases for VBAC and strict monitoring in labor
- Judicious use of oxytocin/PGs for induction/augmentation of labor and careful watch
- **Intranatal:** Use of partograph, strict vigilance and timely intervention and referral if needed
- IPV and destructive operations not to be done in modern day obstetrics.

23 Vesicular Mole and Liquor Disorders

Q. Mention the conditions in which uterus is more than weeks of gestation. Describe the clinical features of vesicular mole.

Q. Clinical features and complications of molar pregnancy.

Conditions in which uterus is **more than period of amenorrhea:**
- Wrong dates
- Twins, multiple pregnancy
- Polyhydramnios
- Macrosomy
- Ovarian tumors, fibroids
- Vesicular mole
- Concealed abruption.

DEFINITION

Gestational trophoblastic disease encompasses several disease processes that originate in the placenta. These include complete and partial moles, placental site trophoblastic tumors, choriocarcinomas, and invasive moles.

Vesicular mole is an abnormal condition of placenta where there is hydropic degeneration and proliferative changes in the young chorionic villi. It is a **benign condition with malignant potential.**

CLINICAL FEATURES OF VESICULAR MOLE

Incidence of molar pregnancy is highest in women aged **15 years or younger and those aged 45 years or older.** In the latter group, the relative frequency of the lesion is at least **10 times** greater than that at ages 20–40 years.

- **Uterine bleeding** is almost universal and may vary from spotting to profuse hemorrhage. It is the MC presenting feature. The discharge has **'white currant in red currant juice'** appearance.
- *Lower abdominal pain:* The pain can be due to: Overstretching of the uterus, uterine contractions, infection, concealed hemorrage and rarely perforation by invasive mole.
- *Constitutional symptoms:*
  - Excessive symptoms of pregnancy like nausea, vomiting and even hyperemesis (due to excessive hCG)
  - Thyrotoxic features like tachycardia, tremors

- Passage of grapes like vesicles is **pathognomonic**
- Fetal movements are not felt.

Signs

- Features suggestive of early pregnancy
- The patient looks ill
- Pallor is present and maybe out of proportion to the visible loss (due to concealed hemorrhage)
- Feature of preeclampsia like hypertension, proteinuria edema are present in about 50% cases.

Per Abdomen

- The size of the uterus is **more than period of amenorrhea**
- Uterus feels firm elastic (doughy)
- The fetal parts cannot be felt and external ballotment cannot be elicited
- Fetal heart sounds cannot be heard or detected.

PV

- Internal ballottment absent
- Bilateral or unilateral: Theca lutein cysts maybe palpable in 25–50% cases
- **Finding of vesicles in the discharge is pathognomic** and similarly if the os is open the vesicles maybe felt.

Investigations

- Blood group
- *Quantitative beta-hCG:* hCG levels greater than 100,000 mIU/mL indicate exuberant trophoblastic growth and raise suspicion for a molar pregnancy. Serum hCG value **>2 MOM** for the gestational age is of value. However, a molar pregnancy may have a normal hCG level. **Pre-evacuation levels are very important of follow-up**
- *Complete blood cell count with platelets:* Anemia could be present and coagulopathy could occur
- *Clotting function:* Test clotting function to exclude the development of a coagulopathy or to treat one if discovered
- Liver function tests
- Blood urea nitrogen (BUN) and serum creatinine
- *Thyroxine:* Although women with molar pregnancies are usually clinically euthyroid, plasma thyroxine is usually elevated above the reference range for pregnancy. Patient may present with signs and symptoms of hyperthyroidism
- *USG:* Classical finding is the **snow storm appearance.**
  Theca lutein cysts: These are ovarian cysts, maybe greater than 6 cm in diameter and accompanying ovarian enlargement. These are identified by ultrasonography. Patients may report pressure or pelvic pain. Because of the increased ovarian size, torsion is a risk. These cysts develop in response to high levels of beta-hCG. They spontaneously regress after the mole is evacuated, but it may take-up to 12 weeks for complete regression.

- *Chest X-ray:* Once a molar pregnancy is diagnosed, a baseline chest radiograph should be taken. The lungs are a primary site of metastasis for malignant trophoblastic tumors and also to rule out pulmonary embolism.
- Definitive diagnosis is made by **histological examination** of the products.

Complications

- **Perforation** of the uterus during suction and curettage sometimes occurs because the uterus is large and boggy. Rarely there maybe a perforating mole
- **Hemorrhage:** It could be due to separation of vesicles or rarely intraperitoneal hemorrhage due to perforating mole. Hemorrhage is a frequent complication during the evacuation of a molar pregnancy. For this reason, intravenous oxytocin should be started at the initiation of the suctioning. Blood for possible transfusion should be readily available
- **Malignant trophoblastic disease** develops in 20% of molar pregnancies. For this reason, quantitative hCG should be serially monitored
- **DIC:** Factors released by the molar tissue could trigger the coagulation cascade. Patients should be monitored for disseminated intravascular coagulopathy (DIC)
- **Acute pulmonary insufficiency:** Trophoblastic embolism could cause acute respiratory insufficiency. The greatest risk factor for this complication is a uterus larger than that expected for a gestational age of 16 weeks. The condition maybe fatal
- **Preeclampsia** and eclampsia very rarely.

Q. Follow-up of a case of vesicular mole.

INTRODUCTION

Gestational trophoblastic disease encompasses several disease processes that originate in the placenta. These include complete and partial moles, placental site trophoblastic tumors, choriocarcinomas, and invasive moles.

Vesicular mole is an abnormal condition of placenta where there is hydropic degeneration and proliferative changes in the young chorionic villi. It is a benign condition with malignant potential.

Aim of Follow-up

The prime objective is to diagnose persistent trophoblastic disease/choriocarcinoma.

Period of Follow-up

Routine follow-up is mandatory for all cases for at **least one year.** The reason is that the occurrence of choriocarcinoma is confined to this period.

Interval of Follow-up

- Serial quantitative serum beta-hCG levels should be determined
- Serum hCG levels are obtained *weekly* until the levels are negative or within reference range

- Levels should consistently drop and should never increase. **Normal levels are usually reached within 8–12 weeks after evacuation** of the hydatidiform mole. As long as the hCG levels are falling intervention is not needed
- Once levels have reached the reference range for 3–4 weeks, check them *monthly for 6 months*. Then the follow-up is discontinued and pregnancy allowed
- The patient should not become pregnant during the follow-up period
- If the serum hCG levels plateau or rise, the patient is considered to have malignant disease (i.e. gestational trophoblastic neoplasia) and metastatic disease needs to be excluded
- Effective contraception is recommended during the period of follow-up
- Estrogen-progestin contraceptives or depot medroxyprogesterone is usually used to prevent a subsequent pregnancy during the period of surveillance. IUCD is avoided because of risk of perforation.

Method Employed at Each Visit

History

Enquire about relevant symptoms like irregular vaginal bleeding, persistent cough, breathlessness or hemoptysis.

Abdominovaginal Examination to Note

- Uterine size and involution of the uterus
- Ovarian size for regression of the theca lutein cysts
- Malignant deposits **(bluish nodules)** in the anterior vaginal wall in the suburethral region.

Investigations

- Serum beta hCG levels as mentioned above
- Chest X-ray: If the pre-evacuation X-ray shows metastasis, it should be repeated at 4 weeks interval until remission is confirmed
  - It is then repeated at 3 months interval during the follow-up period
  - When the pre-evacuation X-ray is normal, it is repeated only when beta hCG plateaus or rises.

Q. Mention the conditions in which the uterus is more than weeks of gestation. Define polyhydramnios and, etiology, clinical features, complications, and management of polyhydramnios.

Conditions in which uterus is more than period of amenorrhea:
- Wrong dates (mistaken date of LMP)
- Twins, multiple pregnancy
- Polyhydramnios
- Macrosomy
- Ovarian tumors, fibroids
- Vesicular mole
- Concealed abruption.

INTRODUCTION

Amniotic fluid index (AFI) is an estimate of the amount of amniotic fluid and is an index for the fetal well-being. It is a part of the biophysical profile.

AFI is the score (expressed in cm) given to the amount of amniotic fluid seen on ultrasonography of a pregnant uterus. To determine the AFI, doctors may use a **four-quadrant technique,** when the deepest, unobstructed, vertical length of each pocket of fluid is measured in each quadrant and then added up to the others or the so-called **'Single Deepest Pocket'** technique.

Definitions

- More than two liter of amniotic fluid is termed as polyhydramnios, or
- AFI ≥ 25 cm, or
- Single largest vertical pocket of liquor > 8 cm (normal = 2–8 cm).

Classification

Single Largest Vertical Pocket (cm)

Mild	>8–11
Moderate	12–15
Severe	>15

Incidence

1–2% of cases.

Etiology

- **MC cause = Idiopathic**
- Fetal anomalies:
  - **Obstruction of fluid transit** through the gastrointestinal tract: Esophageal/duodenal atresia and diaphragmatic hernia
  - **Anencephaly:**
    - Transudation of fluid across the membranes
    - Absence of swallowing
    - Absent fetal pituitary (absence of ADH hormone implies that the baby passes more urine).
  - **Open spina bifida**
- Multiple pregnancy
- Hydrops fetalis (immune and nonimmune)
- Chromosomal abnormalities (e.g. trisomy 18)
- Twin-to-twin transfusion syndrome (the donor sac has oligohydramnios and recipient has polyhydramnios)
- Diabetes insipidus/Bartter's syndrome
- **Maternal: Diabetes mellitus** (leads to raised fetal blood sugars leads to fetal diuresis) and cardiac/renal disease (placental edema)
- **Placental:** Chorioangioma (a/w acute polyhydramnios).

Clinical Features

- In majority cases the liquor accumulation is gradual and patient maybe asymptomatic
- Respiratory: Dyspnea
- Palpitations
- Edema feet
- Varicocities in legs, vulva and hemorrhoids
- Polyhydramnios associated with fetal hydrops may cause the **MIRROR SYNDROME**, whereby the maternal condition mimics the fetus and mother develops edema, proteinuria, and PIH.

On Examination

- The abdomen is markedly enlarged with fullness in flanks
- The skin is **tense, shiny**
- Height of the uterus is more than period of amenorrhea
- Abdominal girth will be more than normal
- **Fluid thrill +**
- Fetal parts **cannot be well palpated.** External ballottment can be elicited more easily
- FHS are difficult to hear and Doppler maybe required
- PV: The cervix maybe dilated and effaced and tense bulging membranes maybe felt

Investigations

- USG
  - To estimate AFI
  - To rule out multiple pregnancies and various fetal anomalies
- ABO and Rh blood group (Rh isoimmunization can cause immune hydrops)
- FBS and PLBS and if needed GTT.

Complications

Maternal:

ANTENATAL
- *Preeclampsia*
- **Preterm labor**
- PROM
- Malpresentation.

INTRAPARTUM
- **Cord prolapse**
- Abruption (due to sudden decompression following sudden escape of large amount of liquor)
- Increased risk of operative delivery.

POSTPARTUM
- **PPH (Uterine Atony)**
- Subinvolution of uterus.

Fetal

Increase in perinatal morbidity and mortality due to prematurity, cord prolapse, abruption, and anomalies.

Treatment

Treatment is based on the underlying cause. Mild asymptomatic polyhydramnios is managed expectantly. For a woman with symptomatic polyhydramnios may need hospital admission. Antacids maybe prescribed to relieve heartburn and nausea.

No data to support dietary restriction of salt and fluid.

In severe cases:
- **Indomethacin (25 mg qds) and sulindac** are NSAIDs that decrease fetal urine production and are used in medical management of polyhydramnios in symptomatic patients.
- A major concern for the use of indomethacin/sulindac is the risk of premature closure of the fetal ductus arteriosus. Hence, these drugs **should not be used beyond 34 weeks** of gestation.

In Unresponsive Cases with Maternal Distress

- Pregnancy less than 37 weeks: **Amnioreduction (amniocentesis)** can be done. 1-1.5 liters of fluid should be removed slowly. The procedure may have to be repeated
- If pregnancy is more than 37 weeks: Induction of labor if no malpresentation.

During labor **controlled ARM** to be done **to prevent abruption and cord prolapse.**

Postpartum: To watch for PPH and **AMTSL should be done.**

Q. Mention conditions in which uterus is smaller than period of amenorrhea. Define oligohydramnios. Give etiology of oligohydramnios.

Q. Oligohydramnios.

Conditions in which uterus is smaller than period of amenorrhea:
- Wrong dates (mistaken date of LMP)
- IUGR
- IUFD
- Oligohydramnios.

INTRODUCTION

Amniotic fluid index (AFI) is an estimate of the amount of amniotic fluid and is an index for the fetal well-being. It is a part of the biophysical profile.

AFI is the score **(expressed in cm)** given to the amount of amniotic fluid seen on ultrasonography of a pregnant uterus. To determine the AFI, doctors may use a **four-quadrant technique**, when the deepest, unobstructed, vertical length of each pocket of fluid is measured in each quadrant and then added up to the others or the so-called **'Single Deepest Pocket'** technique.

Oligohydramnios

- AFI < 5 cm
  or
- Amniotic fluid less than 100 mL or
- Maximum vertical pocket < 2 cm.

Etiology

Fetal

- Chromosomal abnormalities
- Congenital anomalies (e.g. **renal agenesis and posterior urethral valves**)
- **IUGR**
- **Postdatism/post-term pregnancy**
- PROM
- Twin-to-twin transfusion
- Intrauterine infections.

Maternal

- **Uteroplacental insufficiency**
- Hypertension
- Preeclampsia
- NSAIDs, angiotensin: Converting enzyme inhibitors
- Dehydration
- Idiopathic.

Renal Agenesis

This defect has an incidence of about 1 in 4000 births. No kidneys are seen ultrasonographically at any point during gestation. The adrenal glands typically enlarge and occupy the renal fossae, which is termed the **lying down adrenal sign. Without kidneys**, no urine is produced and the resulting severe oligohydramnios leads to **pulmonary hypoplasia, limb contractures**, a distinctive **compressed face**, and death from cord compression or pulmonary hypoplasia. When this combination of abnormalities results from renal agenesis, it is called ***Potter syndrome after Dr Edith Potter*** who described it in 1946. When these abnormalities result from scanty amniotic fluid of some other etiology, it is called ***oligohydramnios sequence.***

- **Amnion nodosum** are tiny, light tan, creamy nodules in the amnion made up of vernix caseosa with hair, degenerated squames, and sebum. **They result from oligohydramnios** and are most commonly found in fetuses with renal agenesis and prolonged preterm ruptured membranes, or in the placenta of the donor fetus with twin-to-twin transfusion syndrome.
- Amniotic bands are caused when disruption of the amnion leads to formation of bands or strings that entrap the fetus and impair growth and development of the involved structure. Fetal conditions that appear to be the consequence of this phenomenon include intrauterine amputations.

Clinical Features

- Uterus size/height of the uterus is less than period of amenorrhea
- Abdominal girth will be less than normal

- **The uterus is 'full of fetus' due to scanty liquor**
- Malpresentation (breech) is common
- Evidence of IUGR.

Complications

- Tetrad of early-onset oligohydramnios:
  - Facial clefts (cleft lip/palate)
  - IUGR
  - Limb reduction defects
  - Pulmonary hypoplasia
- Cord compression in labor
- Malpresentations
- Increase operative delivery
- High perinatal morbidity and mortality.

Treatment

- Rule out congenital anomalies in the fetus
- **Treatment of hypertension and IUGR** if present (refer to IUGR answer)
- Hydration therapy (oral or IV) may help to increase the liquor volumes
- Antenatal amnioinfusion is not recommended
- However **amnioinfusion can be done in labor to prevent cord compression and improve neonatal outcome.**

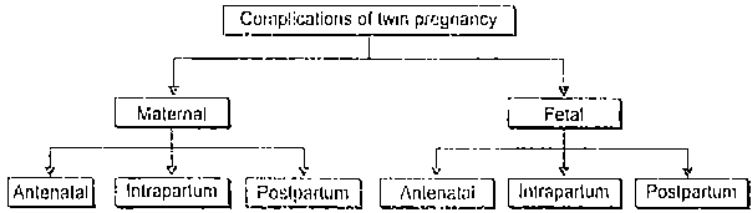
24

Twins

Q. Complications of twins.

Twins and higher order multiple pregnancies are at a risk of various complications as compared to singleton pregnancy.

Flow Chart 24.1: Complications of twin pregnancy



Maternal

Antenatal

There is increased risk of the following:

- **Anemia:** Due to increased requirements of iron, folic acid and vitamin B12 leading to dimorphic anemia
- **Hyperemesis,** *nausea:* Due to excessive hCG
- **Preeclampsia** *(25%):* Due to excessive chorionic villi. Multiple gestations are at increased risk for developing gestational hypertension and preeclampsia compared with singleton pregnancies. The incidence of preeclampsia is **2.6 times higher** in twin gestations than in singleton gestations
- **Polyhydramnios:** Due to increase in fetal renal perfusion. More common in monozygotic twins
- **Preterm labor** *(50%):* Over distention of the uterus, polyhydramnios and preeclampsia contribute
- **Gestational diabetes mellitus:** The incidence of gestational diabetes increases with each additional fetus in multiple gestations. Between 22–39% of triplet pregnancies and 3–6% of twin pregnancies are complicated by gestational diabetes. Each additional fetus increases the risk of gestational diabetes by an estimated factor of 1.8

- **APH (placenta previa + abruption):** Large placenta which encroaches on the lower segment contributes to placenta previa. Abruption could be due to:
  - Preeclampsia
  - Sudden decompression of the uterus after the delivery of the first baby
  - Sudden escape of liquor from hydro amniotic sac
  - Folic acid deficiency
- **Malpresentations:** More common compared to single pregnancy. It is **more common in the second baby.** In 70% cases the first baby is vertex and 50% cases both are vertex
- Mechanical distress such as palpitation, dyspnea, varicocities and hemorrhoids are increased compared to singleton pregnancy.

Intrapartum

- *Cord prolapse:* Around five times more common as compared to single term pregnancy and more common in second baby. Malpresentation and polyhydramnios contribute
- Cord entanglement and **interlocking of twins**
- *Operative delivery:* Increased risk of LSCS due to malpresentations, cord proplase, preeclampsia, prematurity, etc.

Postpartum

- **PPH:** Uterine atony due to overdistention of the uterus, polyhydramnios, preeclampsia, placenta previa and APH all contribute
- Subinvolution of the uterus due to overdistention
- Infection due to anemia and operative interference
- Lactation failure due to more demand.

Fetal

- **Congenital anomalies:** Congenital malformations are twice as common in twin pregnancies compared with singletons and four times more common in triplets. The rate of congenital anomalies in twins is estimated at approximately 4% compared with 2% in singletons. Monozygotic twins have twice the incidence of congenital abnormalities compared with dizygotic twins.
  The anomalies include NTD, hydrocephalus, cardiac anomalies and Down's syndrome.
- **Abortions:** Increased rate of both first and second trimester abortions. More in monozygotic twins.
- Prematurity is the most dangerous complication and contributes to morbidity and mortality
- IUGR: Intrauterine growth restriction (IUGR) and discordant growth contribute to adverse outcomes in multiple gestations. Growth restriction in multiple gestations is likely secondary to uteroplacental insufficiency but can also be secondary to structural anomalies, umbilical cord abnormalities, infections, genetic abnormalities or due to twin to twin transfusion syndrome. Approximately 14–25% of twin gestations are affected by growth restriction. One or both babies can be IUGR.
- **Asphyxia and still birth:** Due to prematurity, preeclampsia, gestational diabetes, cord prolapse, cord entanglement, abruption, malpresentation, and operative interference.
  The second baby is more at risk. Complications are more in monochorionic twins.

- **IUFD of one fetus.** More in monozygotic twin. If the loss happens in first trimester the affected fetus '**vanishes**' by resorption. If the death occurs in second trimester a fetus papyraceous or compressus may form. If the death occurs in late pregnancy the other fetus may develop acute hypotension, cerebral palsy or even death. Mother may develop DIC.
- Cord prolapse, cord entanglement and interlocking of twins.
- Increase perinatal mortality (PNM).

Best prognosis in seen in dichorionic, diamniotic variety (10–20% PNM).
Worst prognosis is with conjoint twins (70–90% PNM) followed by MC, MA (58–60% PNM).

Special Complications

- **Twin-to-twin transfusion syndrome** (occurs in monochorionic twins only):
  - In this syndrome, blood is transfused from a donor twin to its recipient sibling such that the donor becomes anemic and oligohydramniotic, and its growth maybe restricted, whereas the recipient has polyhydramnios and becomes polycythemic and may develop circulatory overload manifest as hydrops. Similarly, one portion of the placenta often appears pale compared with the rest of the placenta
  - This is due to deep arteriovenous anastomosis
  - Antenatal criteria recommended for defining the twin-to-twin transfusion syndrome include the following:
    - Same sex fetuses
    - Monochorionicity with placental vascular anastomoses
    - Weight difference between twins greater than 20%
    - Polyhydramnios in the larger twin
    - Oligohydramnios in donor twin, and
    - Hemoglobin difference greater than 5 g/dL
    - The donor twin has better prognosis.

Management

Diagnosis is made with USG and color Doppler.

- Repeated amniocentesis to control polyhydramnios in the recipient. This also improves circulation in donor twin
- Septostomy
- Laser photocoagulation of the anastomotic vessels
- Selective feticide/reduction of one twin done as last resort when survival of both the fetus is at risk.
- **Acardiac twin: Twin Reversed Arterial Perfusion (TRAP) sequence** is a rare (one in 35,000 births) but serious complication of monochorionic, monozygotic multiple gestation. In the TRAP sequence, there is usually a normally formed donor twin that has features of heart failure as well as a recipient twin that lacks a heart (acardius) and various other structures.
- **Discordant growth** (can occur in DZ and MZ twins): There is difference in weights of twins and is expressed as percentage of larger twin's weight:

Grade 1 = difference of 15–25%
Grade 2 = difference >25%

Q. Etiology of twins and types/varieties of twins.

When more than one fetus simultaneously develops in uterus it is called multiple pregnancy.

Incidence

- Incidence of monozygotic twin is constant throughout the world. It is one in 250
- The incidence of dizygotic twin varies from one in 20 in Nigeria to one in 80 in India
- With the advent of ART/IVF the incidence is on rise.

Etiology

Unknown for MZ Twins.

For DZ Twins

- *Increasing age and increasing parity:* The rate of natural twinning rises from zero at puberty, a time of minimal ovarian activity, to a peak at 37 years of age, when maximal hormonal stimulation increases the rate of double ovulation. This is in accordance with the first consistently observed sign of reproductive aging, an isolated rise in serum FSH. The fall in incidence after 37 years of age probably reflects depletion of the Graafian follicles
- Incidence increases from 5th gravida onwards
- Personal/family history of twinning. If the patient's mother or sister has twins, there is more chance of twins. Past history of twins also contributes
- *Ovulation induction agents:* **Three to eight percent risk** of twins with clomiphene citrate and **15–30%** with gonadotropins
- *IVF:* **20 to 45% risk of twins.** It depends on the patient's age and the number of embryos transferred
- Negroes have the highest risk and Mongols have the least risk.

Twins can be of two varieties: Dizygotic (DZ) and monozygotic (MZ).
DZ variety = 80% and MZ = 20% of all twins.
All dizygotic twins are Dichorionic and Diamniotic (DC, DA).

MZ twins are of following varieties depending upon the time of twinning:

- Within 72 hours of fertilization = DC, DA (around 30% of MZ twins and 6–7% of all twins)
- Between 4th and 8th day = MC, DA (66% of MZ twins and 13–14% of all twins)
- Between 8th and 12th day = MC, MA (1–3% of MZ twins and <1% of all twins)
- After 12 days = conjoint/Siamese twins (<1% of MZ twins and 0.002–0.008% of all twins).

Conjoined Twins (in Descending Order of Frequency): (Remember 'TOPIC')

- Thoracopagus (joined at thorax), MC variety
- Omphalopagus (abdomen)
- Pygopagus (buttocks)
- Ischiopagus (ischium)
- Craniopagus (head), least common variety.

Q. Clinical features and management of twin pregnancy.

Q. Intrapartum management of twins.

INTRODUCTION

Twin pregnancy belongs to a high-risk category as twins and higher order multiple pregnancies are at a risk of various complications as compared to singleton pregnancy.

Diagnosis

History

- Advanced maternal age and parity
- History of ovulation induction/ART
- Family/personal history of twins.

Symptoms

Exaggerated symptoms of normal pregnancy:

- Increase in nausea and vomiting and sometimes hyperemesis.

Symptoms due to over distended uterus:

- Cardiorespiratory embarassement—dyspnea, palpitation
- Swelling of legs, varicose veins and hemorrhoids
- Unusual rate of abdominal enlargement and excessive fetal movements.

ON EXAMINATION

General Examination

- Pallor (more risk of anemia) maybe present
- Excessive weight gain
- Edema feet
- High blood pressure (more risk of preeclampsia)
- Proteinuria.

Abdominal Examination

Inspection

Undue enlargement of the abdomen, 'barrel shape'.

Palpation

- Height of uterus and will be more than period of amenorrhea
- Abdominal girth more than normal average at term (100 cm)
- Presence of polyhydramnios
- Palpation of **too many fetal parts**
- **Finding of two fetal heads, or three fetal poles.**

Auscultation

Simultaneously hearing two distinct FHS with a silent area in between by two observers with a difference in heart rate of 10 beats/min.

Investigations

- Routine ANC investigations to be carried out
- **To keep in mind that there is more chance of anemia, preeclampsia and gestational diabetes**

- **USG:** Very useful for:
  - *Confirmation of diagnosis:* Count the number of gestational sacs and yolk sacs in first trimester
  - Dating the pregnancy
  - Viability of fetuses, vanishing twin in second trimester
  - Chorionicity establishment.

Signs for Chorionicity on USG

Dichorionicity	Monochorionicity
The 'twin-peak' sign/lambda sign (placenta intervenes between the membranes) Intervening membrane is >2 mm thick	'T' sign/inverted T sign (right angle relation between the placenta and fetal membranes) Intervening membrane is <2 mm

Twin Peak Sign/Lambda Sign Indicates Two Fused Placenta

- **Detailed NT scan at 11–13 weeks and anomaly scan at 18–20 weeks**
- Fetal growth at 3–4 weeks interval for IUGR/Discordant growth
- To detect malpresentations
- Color Doppler in cases of IUGR and TTTS
- Placental localization and AFI
- **Dual marker and triple marker test can also be carried out in twins as there is more risk of Down's syndrome.**

ANTENATAL MANAGEMENT

Early diagnosis is very important to detect chorionicity, amniocity and fetal anomalies.

Diet

Increase in dietary requirement. **Extra 300 Kcal/day** is needed over and above that needed for singleton pregnancy. Also increase in proteins needed.

Supplements

- Iron increased to 100–200 mg/day. Additional vitamins, calcium and folic acid (4 mg instead of 400 mcg) are also needed
- Adequate rest to the mother
- To watch for preterm labor. Corticosteroids can be given to accelerate lung maturity
- Fetal surveillance by USG for fetal growth at 3 to 4 weeks interval. **NST, BPP and color Doppler for fetal well-being**
- **More frequent antenatal visits** to detect anemia, preeclampsia, preterm labor and gestational diabetes.

Intrapartum Management

Multidisciplinary team approach (obstetrician, anesthetist, neonatologist and NICU facilities).

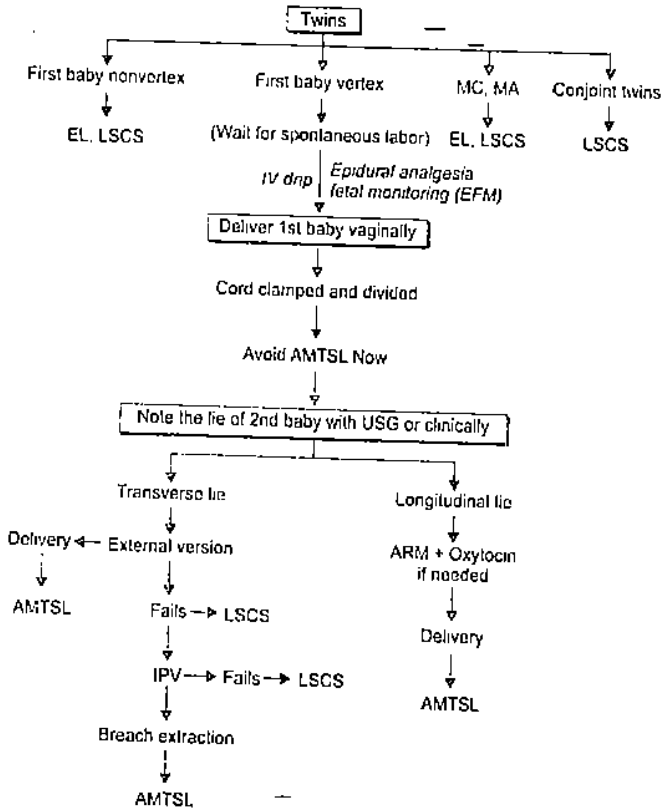
- **Skilled obstetrician should be present**
- **Presence of USG is very helpful and should be there**
- Secure IV line (as urgent IV therapy maybe needed), send blood for cross match and RL drip can be started.
  - Patient is put to bed to prevent early ROM

- Adequate pain relief (epidural analgesia preferred)
- Careful fetal monitoring (**preferably continuous EFM**)
- **PV examination after rupture of membranes to rule out cord prolapse**
- Neonatologist and a trained assistant to be present for delivery
- NICU backup.

Delivery

- Route of delivery is decided by the position of first baby
- Only if the first fetus is in vertex position, then normal vaginal delivery is possible
- **Twins with first fetus in nonvertex position** (breech, transverse, oblique, etc.) are to be **delivered by LSCS**
- **MC, MA variety twins are always to be delivered by LSCS** (even if the first fetus is in vertex position) because of very high risk of **cord prolapse and cord entanglement** (Flow Chart 24.2).

Flow Chart 24.2: Management of twins



EL = Elective; AMTSL = Active Management Third Stage Labor

The only indication of IPV in modern day obstetrics is transverse lie in second baby of twins.

Indications of LSCS for Second Baby

- Fetal distress
- Cord prolapse
- Abruption (due to sudden decompression) after delivery of the first baby
- Larger second twin with noncephalic presentation
- Prompt closure of cervix after delivery of first baby
- Failed ECV or IPV done for second baby in transverse lie.

Management of Third Stage

- **AMTSL to be done after the delivery of the second baby to prevent PPH**
- Advisable to **continue oxytocin drip** for at least one hour after delivery of second baby
- Closely monitor the patient in postpartum period.

Mother to be given encouragement and support to manage both babies.

Contraception

Contraceptive advice to be given to mother on discharge.

25 Induction of Labor and Operative Delivery

Q. What is induction of labor. What are the indications and methods for IOL?

Q. Contraindications for IOL.

INTRODUCTION

Induction of labor (IOL) needs to be considered when the risk-benefit analysis indicates that **delivering the baby is a safer option for the baby, the mother, or both, rather than continuing the pregnancy,** and when there are no clear indications for cesarean section and no contraindications for vaginal delivery.

DEFINITION

IOL means initiation of uterine contractions by medical, surgical or combined for the purpose of vaginal delivery.

INDICATIONS

Maternal and Fetal Conditions

Maternal

- Gestational hypertension, preeclampsia, eclampsia
- Premature rupture of membranes
- Postdatism, postterm
- Abruptio placentae
- Chorioamnionitis
- Maternal medical conditions (e.g. diabetes mellitus, renal disease, chronic pulmonary disease, chronic hypertension, cholestasis of pregnancy)
- Polyhydramnios
- Maternal request.

Fetal

- Fetal demise (IUFD)
- Fetal compromise (e.g. severe fetal growth restriction, isoimmunization, oligohydramnios)
- Major congenital anomaly.

Contraindications to labor induction (all cases where vaginal delivery is not possible or contraindicated).

- Contracted pelvis, severe CPD
- Transverse lie, oblique lie, footling breech
- Umbilical cord prolapse
- Active genital herpes infection
- Placenta previa
- Vasa previa
- Previous classical cesarean section, hysterotomy
- Previous myomectomy entering the endometrial cavity
- Cervical cancer
- Fetal distress, severe fetal compromise
- Previous 2 or more LSCS.

Bishop Scoring System Used for Assessment of Inducibility (asked as short note)

	Factor				
Score	Dilatation (cm)	Effacement (%)	Station	Cervical consistency	Cervical position
0	Closed	0–30	–3	Firm	Posterior
1	1–2	40–50	–2	Medium	Midposition
2	3–4	60–70	–1	Soft	Anterior
3	≥5	>80	+1 to +2	–	–

A score of **9 conveys a high likelihood for a successful induction.** Score of 4 or less identifies unfavorable cervix and needs for cervical ripening.

Methods of Induction of Labor

- Medical
- Surgical
- Combined.

MEDICAL

- Oxytocin
- Prostaglandins (PGE1 tablets, PGE2 gel, tablets, inserts)
- Mifepristone.

Oxytocin

- As pregnancy progresses, the number of oxytocin receptors in the uterus increases (by 100-fold at 32 weeks and by 300-fold at the onset of labor).
- Oxytocin activates the phospholipase C-inositol pathway and increases intracellular calcium levels, stimulating contractions in myometrial smooth muscle.
- **Oxytocin is the preferred pharmacologic agent for inducing labor when the cervix is favorable or ripe.**

- **2.5–5 units oxytocin** in 500 ml RL is used. The drip is generally started at 8–10 drops/minute and titrated as required.

Prostaglandins

- Alter the extracellular ground substance of the cervix, and **PGE2 increases the activity of collagenase in the cervix.**
- They cause an increase in elastase, glycosaminoglycan, dermatan sulfate, and hyaluronic acid levels in the cervix and facilitate the dilatation.
- Increase in intracellular calcium levels, causing contraction of myometrial muscle.

Dinoprostone

For the purpose of cervical ripening and induction, dinoprostone gel (**PGE2** = 0.5 mg), tablets and dinoprostone inserts (10 mg) are available.

The gel can be repeated **every 6 hourly for 2–3 doses as required.**

Misoprostol

Misoprostol is a synthetic **PGE1** analog that has been found to be a safe and inexpensive agent for cervical ripening and IOL. It can be used by **vaginal, oral, sublingual and buccal route.**

Generally 25 mcg intravaginally every four to six hours is used. Higher doses or shorter dosing intervals are associated with a higher incidence of side effects, especially hyperstimulation.

Finally, uterine rupture in women with previous cesarean section is also a possible complication, limiting its use to women who do not have a uterine scar.

Risks associated with the use of prostaglandins include:
- **Uterine hyperstimulation (tachysystole)** and subsequent fetal distress
- MSAF (meconium stained amniotic fluid)
- Very rarely rupture uterus and
- Maternal side effects such as nausea, vomiting, diarrhea, and fever.

PGF2alpha CANNOT be used for Induction of Labor

Mifepristone

Mifepristone (200MG orally or vaginally) is an antiprogesterone agent. Progesterone inhibits contractions of the uterus, while mifepristone counteracts this action.

SURGICAL METHODS

Stripping of the Membranes

- Stripping of the membranes causes an **increase in the activity of phospholipase A2 and prostaglandin as well as causing mechanical dilation of the cervix, which releases prostaglandins.**
- The membranes are stripped by inserting the examining finger through the internal cervical OS and moving it in a circular direction to detach the inferior pole of the membranes from the lower uterine segment.
- Risks of this technique include infection, bleeding, accidental rupture of the membranes, and patient discomfort.

Amniotomy/ARM (Artificial Rupture of Membrane) (Short Note)

Mechanism of Action

- It is hypothesized that amniotomy increases the production of, or causes a release of, prostaglandins locally
- It can be combined with oxytocin.

Advantages

- High success rate
- Chance to observe the **amniotic fluid,** blood stained or MSAF and hence helps in further management
- Access to use fetal scalp electrode, fetal scalp blood, and intrauterine pressure catheter.

Limitations

It cannot be done in an unfavorable cervix (long, firm and OS closed). The cervix should be at least one finger dilated.

Contraindications

- Maternal HIV infection
- Active genital herpes infection
- Floating/unengaged head (can lead to cord prolapse)
- Polyhydramnios (sudden decompression leads to abruption, so in these cases controlled ARM should be done).

Complications

Risks associated with this procedure include:
- Umbilical cord prolapse or compression
- Maternal or neonatal infection
- FHR deceleration
- Bleeding from low-lying placenta or vasa previa
- Possible fetal injury
- Abruption due to sudden decompression in cases of polyhydramnios
- AFE (very rare).

Mechanical Modalities

All mechanical modalities share a similar mechanism of action: Some form of local pressure that stimulates the release of prostaglandins.
- Hygroscopic dilators **(very very rarely used)** absorb endocervical and local tissue fluids, causing the device to expand within the endocervix and providing controlled mechanical pressure. The products available include natural osmotic dilators (e.g. Laminaria japonicum) and synthetic osmotic dilators (e.g. Lamicel). The main advantages of using hygroscopic dilators include outpatient placement and no FHR-monitoring requirements.
- Balloon devices provide mechanical pressure directly on the cervix as the balloon is filled. A Foley catheter (26 Fr) or specifically designed balloon devices can be used.

The risks associated with these methods include infection, bleeding, membrane rupture, and placental disruption.

Q. Prerequisites for Forceps Application.

The prerequisites for successful application of forceps:

- The head must be engaged
- The fetus must present as a vertex or by the face with the chin anterior
- The position of the fetal head must be precisely known
- **As per the ACOG guidelines station should be > = + 2**
- The cervix must be completely dilated (10 cm)
- The membranes must be ruptured
- There should be no suspected cephalopelvic disproportion.(pelvis deemed adequate)
- Bladder must be empty
- Informed consent (verbal or written)
- Experienced operator , a valid indication and neonatologist
- Backup plan in case of failure (LSCS facilities)
- Adequate maternal analgesia
- Willingness to abandon the procedure when difficulties faced.

Generally, the indications and prerequisites for the use of the vacuum extractor for delivery are the same as for forceps delivery.

Q. Indications for LSCS.

INDICATIONS

- Absolute
- Relative
- Maternal
- Fetal.

Absolute

- Central placenta previa
- Placenta accreta
- Contracted pelvis (severe CPD)
- Pelvic mass (cervical fibroid, broad ligament fibroid)
- Previous classical cesarean section
- Previous 2 or more LSCS
- Advanced carcinoma cervix
- Vasa previa
- Cord presentation.

Relative

Complications of labor and factors impeding vaginal delivery, such as:

- Prolonged labor, obstructed labor or a failure to progress (Dystocia)
- Fetal distress
- Cord prolapse

- Abnormal presentation (Breech, brow, transverse lie, oblique lie)
- Abruption
- Failed labor induction
- Failed instrumental delivery by forceps or ventouse (sometimes a trial of forceps/ventouse delivery is attempted, and if unsuccessful, it will be switched to a cesarean section.)
- Macrosomy
- Triplets and higher order pregnancy
- Severe IUGR
- Previous myomectomy scar
- Other complications of pregnancy, pre-existing conditions and concomitant disease, such as:
  - Preeclampsia, hypertension
  - Certain heart disease (Marfan's syndrome)
  - HIV infection of the mother
  - Sexually transmitted diseases, such as genital herpes (which can be passed on to the baby if the baby is born vaginally)
  - Precious pregnancy (BOH, IVF conception, elderly primigravida, long standing infertility conception, etc.)
  - Previous uterine rupture
  - Rare cases of posthumous birth after the death of the mother.

Q. Episiotomy

Definition

An episiotomy (perineotomy) is a **planned, surgical incision** on the perineum and the posterior vaginal wall during second stage of labor. The incision is actually a second degree perineal injury, and is sutured closed after delivery. It is the **most common obstetric procedure performed on women,** and although its routine use in childbirth has steadily declined in recent decades.

Objectives

- To prevent or **minimize overstretching and tearing of perineal muscles,** episiotomy is done as prophylaxis against soft-tissue trauma(tears can involve the perineal skin or extend to the muscles and the anal sphincter and anus).
- To enlarge the introitus so as to reduce the strain and stress on fetal head and facilitate easy and safe delivery of the fetus.

Indications

- When perineal muscles are excessively rigid/inelastic perineum
- Instrumental delivery: Forceps, ventouse
- There is a serious risk to the mother of second- or third-degree tearing in cases like:
  - The baby is very large
  - Face to pubis delivery
  - Face delivery
  - After coming head of breech

- When a woman has undergone FGM (female genital mutilation) or perineal reconstructive surgery
- Prolonged late decelerations or fetal bradycardia during active pushing
- Shoulder dystocia. Episiotomy does not directly resolve this problem, but it is indicated to allow the operator more room to perform maneuvers to free shoulders from the pelvis.

Timing

Bulging thin perineum during contraction just prior to or at the time of **crowning** (when 3–4 cm of the head is visible).

Anesthesia

1% lignocaine, local anesthesia.

Types

There are four main types of episiotomy:

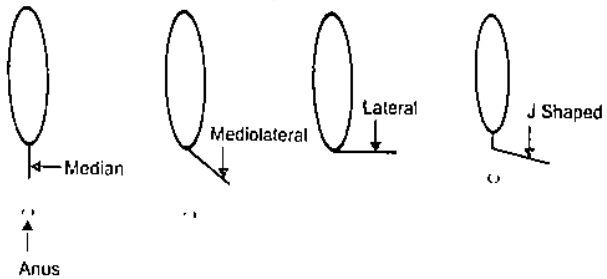


Fig. 25.1: Types of episiotomy

Mediolateral

The incision is made downward and outward from midpoint of Fourchette either to right or left. It is directed diagonally in straight line which runs about 2.5 cm away from the anus (midpoint between anus and ischial tuberosity). **Most common type.**

Median

The incision commences from centre of the Fourchette and extends on posterior side along midline for about 2.5 cm.

Lateral

The incision starts from about 1 cm away from the centre of fourchette and extends laterally. Drawback include chance of injury to Bartholin's duct; thus some practitioners have totally condemned it.

J-shaped

The incision begins in the centre of the Fourchette and is directed posteriorly along midline for about 1.5 cm and then directed downwards and outwards along 5 or 7 O'clock position to avoid the anal sphincter. **Very rarely done.**

Structures cut are:

- Posterior vaginal wall
- Superficial and deep transverse perineal muscles bulbospongiosus, and part of levator ani and fascia covering those muscles
- Transverse perineal branches of pudendal vessels and nerves
- Subcutaneous tissue and skin.

Repair

Perfect hemostasis, obliteration of dead space and tension free suture are the principles to be followed.

It is done 3 layers in following order with number 0 chromic catgut or ploy glactin (vicryl rapide) sutures

1. Vaginal mucosa: Sutured by continuous/continuous interlocking sutures.
2. Perineal muscles: Interrupted sutures.
3. Skin and subcutaneous tissue: Interrupted or vertical mattress.

Wound Care

- The wound is to be kept open, dry and clean. Antiseptic ointment/cream to be applied 2–3 times a day and each time following urination and defecation.
- Analgesics and anti-inflammatory tablets and oral antibiotics for 3–5 days.
- The stitches need not be removed as they are absorbable.

Complications

Immediate and Late

Immediate

- **Extension:** Extension to rectum. More likely in median episiotomy
- Forniceal tear
- Vulval hematoma
- Infection
- Wound dehiscence
- Injury to rectal spincter
- Rectovaginal fistula
- Necrotizing fasciitis (very rare).

Late

- **Dyspareunia:** This is due to faulty repair technique leading to narrow introitus and perineal scar
- **Scar endometriosis (rare).**

26 Previous Lower Segment Cesarean Section (LSCS)/ Vaginal Birth After Cesarean (VBAC)

Q. Clinical features of previous cesarean section scar rupture/dehiscence. Mention the rupture rates of various scars.

INTRODUCTION

Pregnancy with a previous cesarean delivery belongs to a high-risk category and is quite prevalent today due to liberal use of primary cesarean section.

The dictum has changed from 'once a cesarean always a cesarean' to **'mandatory hospital delivery and individualization of the case'**.

CLINICAL FEATURES OF SCAR RUPTURE/DEHISCENCE

The scar begins to rupture from inside-out. **In dehiscence the serosa is intact.** Rupture is complete thickness, involves all layers and peritoneal cavity of the mother communicates with amniotic cavity of the baby.

Impending scar rupture (scar dehiscence)	Ruptured uterus
Unexplained tachycardia	Weak thready fast pulse
Hypotension	Shock
Fetal tachycardia	Persistent fetal bradycardia/IUFD
Uterine scar tenderness / suprapubic pain especially in between contractions	Hematuria
Bleeding pv	Bleeding pv
Hematuria	**Cessation of uterine contractions**
	Recession of presenting part (Loss of station)

Change in fetal heart rate (tachycardia/loss of beat to beat variability/decelerations) is the **earliest and the most consistent sign** of impending scar dehiscence, followed by maternal tachycardia.

Estimated Risks for Uterine Rupture in Women with a Prior Cesarean Delivery

Prior uterine incision	Estimated rupture (%)
Classical	4–9
T shaped	4–9
Low vertical	1–7
Low transverse	0.2–1.5

Q. Criteria/guidelines for VBAC.

Q. How will manage a case of previous LSCS?

INTRODUCTION

Pregnancy with a previous cesarean delivery belongs to a **high risk category** and is quite prevalent today due to liberal use of primary cesarean section.

The dictum has changed from 'once a cesarean always a cesarean' to **'mandatory hospital delivery and individualization of the case'**.

Recommendations of the ACOG Useful for the selection of candidates for vaginal birth after cesarean delivery **(trial of scar)**

- No more than one prior low-transverse cesarean delivery **(ONLY 1 PREVIOUS LSCS)**
- Clinically adequate pelvis **(no CPD)**
- No other uterine scars or previous rupture
- Physician immediately available throughout active labor who is capable of monitoring labor and performing emergency cesarean delivery
- Availability of resources (anesthesia, OT, blood) for emergency cesarean delivery
- **Patient consent.**

Management of Case of Previous Cesarean Section

- **It is important to take note in the first visit itself whether the previous cesarean section was LSCS (lower segment transverse) or classical cesarean**
- Regular antenatal checkup and build up Hb
- To enquiry about **pain/tenderness over the scar area** or vaginal bleeding at every visit
- Patients with previous classical scar or hysterotomy scar: **Admit at 36 weeks** (as chance of rupture of such scar is more in last few weeks of pregnancy)
- **Formulate the route of delivery:** The most important decision is to whether go for repeat elective cesarean section or VBAC
- **Previous classical cesarean section or hysterotomy: Elective repeat ceserean section at 38 weeks of gestation or even earlier if required**
- Patients with previous LSCS, enquiry about:
  - Indication of prior cesarean section (recurrent or nonrecurrent)
  - Number of previous LSCS **(Patients with previous 2 or more LSCS → ELECTIVE REPEAT LSCS** at 38–39 weeks of gestation or even earlier if required)
  - Whether it was elective or emergency

  - Any intraoperative or postoperative complications and wound healing of the previous scar. Any infection or prior wound gape may weaken the scar.
  - **(Ideally go through the previous cesarean section notes or discharge summary if available).**
- Fetal weight estimation: Clinically and USG
- **Clinical pelvimetry:** For VBAC, the pelvis should be adequate , there should not be any CPD
- Patients with **previous LSCS and having CPD/contracted pelvis → ELECTIVE REPEAT LSCS** at 38–39 weeks of gestation or even earlier if required
- Patient to be explained about the risk and benefits of VBAC.

The following points are to be kept in mind:

- In women with uterine malformations who have undergone cesarean delivery, the risks for uterine rupture in a subsequent pregnancy may be as high as with a classical incision.
- Women who have previously sustained a uterine rupture are at increased risk of recurrence. Those with a rupture confined to the lower segment have been reported to have a 6% recurrence risk in subsequent labor, whereas those whose prior rupture included the upper uterus have a 32% recurrence risk.
- The rate of uterine rupture is increased nearly fivefold in women with two previous cesarean deliveries compared with that in those only with one—3.7% versus 0.8%.
- **Any previous vaginal delivery, either before or following a cesarean birth, significantly improves the prognosis for a subsequent successful VBAC.**
- The success rate for a trial of scar depends to some extent on the indication for the previous cesarean delivery. Generally, about **60–80%** of trials after prior cesarean birth result in vaginal delivery, with **success being maximum if previous cesarean section was because of breech presentation.**
- Women attempting VBAC who had no previous vaginal deliveries, the relative risk of uterine rupture is more than doubled when the birth weight is at least 4000 g.
- As maternal weight increases, the rate of VBAC success decreases.

MANAGEMENT OF LABOR

Team Approach

Senior obstetrician, pediatrician and anesthetist and OT backup.

- Spontaneous onset of labor is desired
- Induction with PG increases the risk of scar rupture and hence ACOG discourages the use of prostaglandin cervical ripening agents
- IV LINE with RL drip
- Collect blood for grouping and cross matching
- Closely monitor labor
- Vital parameters (pulse, BP)
- **To watch for scar tenderness** (in between contractions, if we palpate the lower uterine segment the patient winces with pain)
- Fetal heart rate monitoring **(electronically preferred)**
- **To watch for progress of labor, chart a partogram**
  - Any attempt to induce cervical ripening or to induce or augment labor increases the risk of uterine rupture in women undergoing a trial of scar.
  - ACOG have concluded that oxytocin may be used for both labor induction and augmentation with close patient monitoring, in women undergoing a trial of scar.

- **Epidural analgesia** is NOT contraindicated
- Prophylactic forceps or vacuum to **cut short second stage of labor**
- If the progress is unsatisfactory or evidence of scar tenderness/dehiscence → emergency LSCS
- Postpartum routine exploration of the scar is not advised. It should only be done in cases where there is excessive vaginal bleeding or maternal hypotension inspite of well contracted uterus
- During 3rd time LSCS patient is to be counselled and **tubal ligation should be offered.**

Q. Difference between LSCS scar and classical cesarean section scar.

Q. Merits/advantages of lower segment operation over classical cesarean section.

	Lower segment	Classical
Techniques	Operative field less bloody because of less vascularity The wall is thin, and as such apposition is perfect	More bloody because of increased vascularity The wall is thick, and coaptation of the margins is not perfect
Postoperative	Hemorrhage and shock—less peritonitis is less even in infected uterus because of perfect peritonization and, if occurs, localized to pelvis	More Chance of peritonitis is more in presence of uterine sepsis
	Peritoneal adhesions and intestinal obstruction are less Convalescence is better Mortality is much lower	More because of imperfect peritonization Relatively poor Mortality is high
Wound healing	The scar is better healed because: Perfect apposition of the thin margins Chance of blood collecting in the wound is less	The scar is weak because: Imperfect apposition because of thick margins Chance of blood collection in the wound is more, which hinders union
	The wound remains quiescent during healing process	The wound is in a state of tension and due to contraction and relaxation of the upper segment. As a result, the knots may slip or the sutures may become lax
	Chance of gutter formation is unlikely as placental implantation is usually fundal	Chance of gutter formation on the inner aspect is likely because of (a) inclusion of the deciduous or (b) inadequate coaptation of the friable inner part when the placenta is anteriorly situated
During future pregnancy	Scar rupture is less (mainly in labor): 0.2–1.5%	More risk of rupture (mainly in third trimester) 4–9%
Following rupture	Maternal death: Less Perinatal death 1 in 8	Maternal death: 5% Perinatal death 6 in 8

27

Miscellaneous

Q. Define maternal death and MMR. What are the causes of maternal mortality.

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

- MMR **(maternal mortality ratio) is maternal deaths per 100,000 live births**
- It varies from 4–40/100,000 live births in developed countries and 100–700 in developing countries. In India it **178 per 100,000 live births**
- Maternal mortality rate indicates maternal deaths divided by number of women of reproductive age (15–49) and expressed per 100,000 women of reproductive age per year. In India it is about 120
- Every day, approximately 800 women die from preventable causes related to pregnancy and childbirth
- 99% of all maternal deaths occur in developing countries
- Maternal mortality is higher in women living in rural areas and among poorer communities
- Young adolescents face a higher risk of complications and death as a result of pregnancy than older women.

CLASSIFICATION OF CAUSES

- **Direct (75–80%)**
- **Indirect (20–25%)**
- Nonobstetric.

Direct deaths are those resulting from complication of pregnancy/delivery or its management:

The most common causes are:

- **Hemorrhage (APH and PPH) (20–25%)**
- Unsafe abortion (10–13%)
- Hypertensive disorders of pregnancy (preeclampsia, eclampsia) (12%)
- Postpartum infections (puerperal sepsis) (10–15%)
- Obstructed labor (8%)
- Ectopic gestation

Indirect deaths include conditions present before or developed during pregnancy but aggrevated by physiological effects of pregnancy and strain of labor:

- **Anemia**
- HIV/AIDS
- **Cardiovascular disease**
- Diabetes
- Viral hepatitis.

All of which may complicate pregnancy or be aggravated by it.

- Nonobstetric = accidents.
- **Hemorrhage (mainly PPH) is the most common cause of maternal mortality in India**
- **Hypertensive disorders of pregnancy (preeclampsia, eclampsia) are the most common cause of maternal mortality in developed countries**
- **Anemia is the most common indirect cause of maternal mortality.**

CONCLUSION

- **Skilled care** before, during and after childbirth, **good family planning services** and **education** can save the lives of women and newborn babies.
- Between 1990 and 2013, maternal mortality worldwide dropped by almost 50%.

Q. Lochia.

It is the vaginal discharge for the first fortnight during puerperium. The discharge originates from the uterine body, cervix and vagina.

Odor and reaction: It has got a peculiar offensive fishy smell. Its reaction is alkaline tending to become acid towards the end.

Color: Depending upon the variation of the color of the discharge, it is named as:

- **Lochia rubra** (red): 1–4 days
- **Lochia Serosa** (5–9) days: The color is yellowish or pink or pale brownish
- **Lochia alba** (pale white): 10–15 days.

Composition: Lochia Rubra consists of blood, shreds of fetal membranes and decidua, vernix caseosa, lanugo and meconium.

Lochia serosa consists of less RBC but **more leukocytes**, wound exudate, mucus from the cervix and microorganisms (anaerobic streptococci and staphylococci). **The presence of bacteria is not pathognomonic unless associated with clinical signs of sepsis.**

Lochia alba contains plenty of decidual cells, leukocytes, mucus, cholesterin crystals, fatty and granular epithelial cells and microorganisms.

Amount: The average amount of discharge for the first 5–6 days, is estimated to be 250 ml.

Normal Duration: The normal duration may extend upto 3 weeks. The red lochia may persist for longer duration especially in women who get up from the bed for the first time late. The discharge may be scanty, especially following premature labor or may be excessive in twin delivery or hydramnios.

Clinical importance: The character of the lochial discharge gives useful information about the abnormal puerperal state. **The vulval pads are to be inspected daily to get information:**

- **Odor**: If malodorous, indicates infection. **Retained plug or cotton piece inside the vagina should be kept in mind**
- **Amount**: Scanty or absent—signifies infection or lochiometra. If excessive—indicates infection
- **Color**: Persistence of red color beyond the normal limit signifies subinvolution or retained bits of conceptus
- **Duration**: Duration of the lochia alba beyond 3 weeks suggests local genital lesion.

Q. Cause of IUFD.

IUFD includes all deaths of fetus weighing 500 gms or more, occurring during pregnancy (antepartum) or during labor (intrapartum). For all practical purpose, antepartum death occurring beyond the period of viability is termed as intrauterine fetal death.

Etiology

- Maternal (5–10%)
- Fetal (25–40%)
- Placental (25–35%)
- Unexplained (25–35%).

Maternal (5–10%)

- **Antiphospholipid antibodies** (Lupus anticoagulant and ACL-anticardiolipin antibodies
- **Diabetes**
- **Hypertensive disorders** (preeclampsia, eclampsia)
- Thrombophilias (factor V Leiden, protein C and S deficiency)
- Abnormal labor (prolonged, obstructed labor, uterine rupture)
- Sepsis
- Acidosis/hypoxia
- Postterm pregnancy
- Drugs.

Fetal (25–40%)

- **Chromosomal anomalies**
- Nonchromosomal birth defects
- Nonimmune hydrops
- Infections.

Placental (25–35%)

- **Abruption**
- **Cord accident**
- **Placental insufficiency**
- Intrapartum asphyxia
- Previa
- Twin-to-twin transfusion
- Chorioamnionitis.

Unexplained (25–35%)

Q. USG in obstetrics.

INTRODUCTION

Ultrasounds are sound waves with frequencies higher than the upper audible limit of human hearing (**>2MHz**). The clinical application of ultrasound in obstetrics was introduced and popularized by **Ian Donald**. USG is an essential tool in managing almost every pregnancy.

The commonly used frequency range in obstetrics is **3–5 MHz for abdominal transducers and 5–7 MHz for vaginal transducers**.

In clinical practice USG images are:

- B mode: 2 D images are obtained routinely
- M mode: To study motion, e.g. fetal heart rate
- Doppler ultrasound: To study the blood flow.

Indications/Uses of USG in Obstetrics

First Trimester

In the first trimester, a standard ultrasound examination typically includes:

- Diagnosis of pregnancy
- Gestational sac size, location, and number
- **Fetal cardiac activity (viability)**
- **Diagnosis of ectopic pregnancy**
- Measurement of **crown-rump length (dating of gestational age)**
- Fetal number, including number of amnionic sacs and **chorionicity for multiple gestations**
- Diagnosis of molar pregnancy
- Assessment of embryonic/fetal anatomy appropriate for the first trimester **(nasal bone, anencephaly)**
- CVS is always done USG guided
- Embryo reduction in cases of multiple pregnancy
- **NT (nuchal translucency) assessment**
- Evaluation of the maternal uterus, ovaries, pelvic mass.

Second Trimester

In the second trimester, a standard ultrasound exam typically includes:

- **Detailed fetal anatomical survey/anomaly scan**
- Cervical incompetence/cervical length assessment
- Placental localization
- Baseline record of biometry
- Amniocentesis and cordocentesis are done USG guided
- Amniocity and chorionicity for multiple gestations if not done in first trimester

Third Trimester

- Assessment of growth/monitoring IUGR
- Estimation of fetal weight

- Lie and presentation
- Placental localization/Bleeding in 3rd trimester
- Diagnosis of IUFD
- AFI (oligohydramnios/polyhydramnios)
- Fetal well being/**BPP**
- **Intrauterine transfusion (IUT)** is always done USG guided
- Before and after ECV.

Color Doppler

Indications

- **IUGR** (most important investigation for management) (details in IUGR answer)
- **Rh isoimmunization management.** Peak systolic velocity (PSV) in the middle cerebral artery is increased with fetal anemia because of increased cardiac output and decreased blood viscosity. PSV in MCA is now used in management of Rh isoimmunized fetuses and timing of IUT
- Prediction of PIH
- Diagnosis of **placenta accreta/percreta, vasa previa**
- In cases of ectopic pregnancy **(ring of fire appearance)** of ectopic sac.

Recent Advances

3D and 4D USG

- 3D USG creates a 3 dimensional image of the fetus. It is considered better than 2D USG for detecting **facial clefts**, CNS and CVS defects and also a **life like photo** improves antenatal parental bonding
- **4D allows a 3-dimensional picture in real time**, rather than delayed, due to the lag associated with the computer constructed image, as in classic 3-dimensional ultrasound.

Q. Shoulder dystocia.

DEFINITION

Shoulder dystocia is an uncommon obstetric complication of cephalic vaginal deliveries during which the **fetal shoulders do not deliver after the head has emerged** out of the introitus. A head-to-body delivery time **exceeding 60 sec** is used to define shoulder dystocia.

It occurs when one or both **shoulders becomes impacted against the bones** of the maternal pelvis.

Incidence: 0.2–1%

- Risk factors include: **D, O, P, E.**
  D = Diabetes mellitus
  O = Obesity
  P = Postdatism and previous history
  E = Excessive weight gain during pregnancy (mother or fetus).

Complications

Maternal

- **PPH:** Usually from uterine atony, but also from vaginal and cervical lacerations
- Increase in operative delivery.

Fetal

- Significant **fetal/perinatal morbidity and even mortality**
- Asphyxia
- Transient **Erb or Klumpke brachial plexus palsies** are the most common injury
- Clavicular fractures and humeral fractures and sternocleidomastoid hematoma.

Diagnosis

- Definite recoil of head back against perineum **(turtle neck sign)**
- **Fetal face becomes plethoric.**

Management of Shoulder Dystocia

- **Call for extra help**
- To involve anesthetist and pediatrician (for neonatal resuscitation)
- **Extend the episiotomy, remove the lithotomy position**
- **Never give fundal pressure (as it causes further impaction of shoulder)**
- **Moderate suprapubic pressure** should be applied by an assistant while downward traction is applied to the fetal head. This will reduce the bisacromial diameter
- Check if it is a unilateral shoulder dystocia (posterior shoulder is in hollow of sacrum, anterior is above pelvic brim) or a bilateral shoulder dystocia (both shoulders above pelvic brim)
- If it is **bilateral shoulder dystocia,** directly proceed to perform **LSCS** after doing the **Zavanelli** maneuver (cephalic replacement back into the vagina and then cesarean delivery)
- The rest of the maneuvers can be tried for unilateral shoulder dystocia, and if they fail, then proceed for **Zavanelli maneuver** and LSCS
- The **McRoberts maneuver:** The maneuver consists of removing the legs from the stirrups and sharply flexing them up onto the abdomen. This procedure causes straightening of the sacrum relative to the lumbar vertebrae, rotation of the symphysis pubis toward the maternal head, and a decrease in the angle of pelvic inclination
- Woods reported that, by progressively rotating the posterior shoulder 180° in a corkscrew fashion, the impacted anterior shoulder could be released. This is frequently referred to as the **Woods corkscrew maneuver**
- Delivery of the posterior shoulder
- **Rubin's maneuver: Posterior pressure on the anterior shoulder**
- **Gaskin maneuver:** Involves moving the mother to an **all fours position** with the back arched, widening the pelvic outlet
- Cleidotomy consists of cutting the clavicle and is usually used for a dead fetus. Symphysiotomy has also been applied successfully.

As per ACOG guidelines, planned cesarean delivery is to be considered for the nondiabetic woman carrying a fetus with an estimated fetal weight exceeding **5000 g** or the diabetic woman whose fetus is estimated to weigh more than **4500 g** to avoid the risk of shoulder dystocia.

Q. Uterine inversion.

It is an extremely rare and a life threatening complication in the 3rd stage in which the **uterus is turned inside out partially or completely.**

Incidence is about **1 in 20,000 deliveries.**

CAUSES AND RISK FACTORS

These include:

- It is well established that ***mismanagement of the third stage of labor*** (combination of premature traction on umbilical cord and fundal pressure before separation of placenta) is the **commonest cause of acute uterine inversion**
- Uterine atony
- Fundal implantation of a **morbidly adherent placenta**
- Manual removal of the placenta
- Precipitate labor
- Short umbilical cord
- Connective tissue disorders (Marfan syndrome, Ehlers-Danlos syndrome)
- Sudden emptying of a distended uterus
- Spontaneous inversion of unknown etiology: Localized atony on placental site over fundus and associated sharp rise of intrabdominal pressure.

However in up to 50% of cases, no risk factors are identified and there is no mismanagement of the third stage. This condition can, therefore, be unpredictable.

Classification according to Severity of Uterine Inversion

First degree (incomplete): The inverted fundus extends down but remains above the cervical ring (internal os).

Second degree (incomplete): The inverted fundus extends through the cervical ring but remains within the vagina.

Third degree (complete): The inverted fundus extends out of the introitus so the endometrium (with or without the placenta) is visible outside the vulva.

Fourth degree (total): The vagina is also inverted.

Classification According to Timing of the Event

- Acute occurs within 24 hours of birth
- Subacute occurs after 24 hours, within 4 weeks
- Chronic occurs after 4 weeks, rare.

Symptoms

Acute severe lower abdominal pain with bearing down sensation.

Signs

- **Shock (neurogenic)** thought to be due to the parasympathetic effect caused by tension on the nerves due to stretching of the ligaments supporting the uterus and ovaries
- Cupping or dimpling on fundal surface in mild degrees
- Uterine **fundus is not palpable** abdominally
- Bimanual examination can confirm the diagnosis and degree of inversion
- In complete variety a **pear shaped mass reddish purple in color, protrudes outside the vulva, broad end pointing downwards**
- PPH.

Treatment

Delay in treatment increases the mortality rate. It is necessary that a number of steps be taken immediately and simultaneously:

- Call for help, including an anesthesiologist immediately.
  - Administer oxygen
  - Insert 2 wide bore IV cannulas
  - Group and cross match 4 units of blood
  - Resuscitate with rapid infusion of crystalloids
  - Monitor vital signs.

Manual Replacement (Johnson Maneuver)

- Immediately push up on the fundus with the palm of the hand and fingers towards umbilicus in the direction of the long axis of the vagina to replace the freshly inverted uterus **(to replace that part first which has inverted last)**
  - Do not remove the placenta if it is adherent
  - Maintain hand in place until a sustained contraction
- Anesthesia (preferably halothane or enflurane) and tocolytics have been used successfully for uterine relaxation and repositioning
- As soon as the uterus is restored to its normal configuration, **oxytocin drip started to contract the uterus** while the operator maintains the fundus in normal position
  - Once uterine inversion corrected, perform manual removal of placenta in theatre under anesthesia if still attached
  - Give stat dose oxytocin 10 IU IV
  - Commence oxytocin infusion (oxytocin 40 IU in 500 mL sodium chloride 0.9%) at 125 mL/hr over 4 hours.

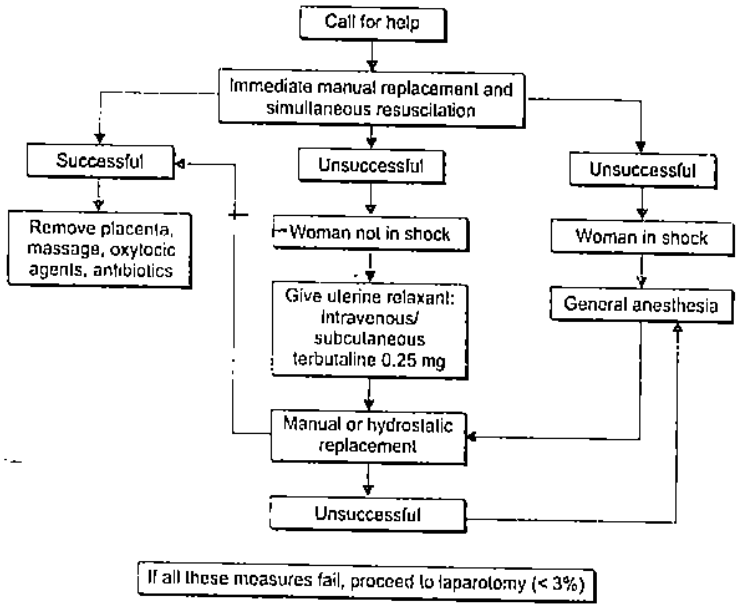
O'Sullivan's hydrostatic technique

- If initial uterine replacement unsuccessful:
  - O'Sullivans hydrostatic repositioning can be done in theatre or in labor ward +/- anesthesia
  - Exclude uterine rupture first
  - Position in lithotomy

- With 2 bags of warmed irrigation fluid and wide bore IV set +/- silastic ventouse cup to produce a better seal
- Run copious amounts of warmed fluid into the vagina (by gravity or pressure)

If unsuccessful repeat or consider surgical management (laparotomy or transvaginal cervical incision).

Flow Chart 27.1: Management of uterine inversion



Various Surgeries for Inversion of Uterus

Hydrostatic technique	Abdominal	Vaginal
O'Sullivan	Haultain	Kustner
Ogueh	Huntington	Spinelli
	Ocejo	

Complications

- Neurogenic shock
- PPH
- Pulmonary embolism
- Maternal mortality
- If left uncared for: Infection, sloughing and chronic inversion.

Q. Hyperemesis gravidarum.

DEFINITION

It is a severe type of vomiting of pregnancy which has got deleterious effect on the health of the mother and/or incapacitates her in day to day activities.

Incidence

There has been marked fall in the incidence during the last 30 years. **Less than 1 in 1000 pregnancies.**

Etiology

Obscure but the following are the known facts:

- It is mostly limited to the first trimester
- It is more common in **first pregnancy**, with a tendency to recur again in subsequent pregnancies It has got a **familial history**
- It is more prevalent in **hydatidiform mole and multiple pregnancy**
- It is more common in unplanned pregnancies.

Theories

- *Hormonal*
  - Excess of HCG or higher biological activity of hCG is associated: Frequency of vomiting at the peak level of hCG and also increased association with v. mole and twins
  - High serum level of estrogen
  - Progesterone excess leading to relaxation of the cardiac sphincter and impaired gastric motility.

  Other hormones involved are:
  Thyroxin, prolactin, leptin and adrenocortical hormones
- *Psychogenic:* Neurogenic element sometimes plays a role, as evidenced by its subsidence after shifting the patient from the home surroundings. Conversion disorder, somatization are the other theories
- *Dietetic deficiency*: Probably due to low carbohydrate reserve
- *Allergic* or *immunological basis.*

Metabolic, Biochemical and Circulatory Changes

The changes are due to the combined effect of **dehydration and starvation** consequent upon vomiting.

Metabolic

- Glycogen depletion
- The fat reserve is broken down
- There is incomplete oxidation of fat and accumulation of ketone bodies. The acetone is ultimately excreted through the kidneys and in the breath.

Biochemical

- Decrease in Na, K and Cl
- **Ketoacidosis**
- Increase in blood urea and uric acid
- Hypoglycemia; hypoproteinemia; hypovitaminosis and rarely hyper bilirubinemia.

Circulatory

- Hemoconcentration leading to rise in hematocrit values
- Slight increase in the WBC count with increase in eosinophils.

CLINICAL COURSE

- Early
- Late (moderate to severe).

The patient is usually a nullipara, in early pregnancy. The onset is insidious.

Early: Vomiting occurs throughout the day. Normal day to day activities are curtailed. **There is no evidence of dehydration or starvation.**

Late: Evidences of dehydration and starvation are present.

Symptoms

- Vomiting is increased in frequency with retching
- Urine quantity is diminished even to the stage of oliguria
- Epigastric pain, constipation may occur. Complications may appear if not treated.

Signs

Features of dehydration and ketoacidos.

Dry coated tongue, sunken eyes, acetone smell in breath, tachycardia, hypotension, rise in temperature may be noted, jaundice is a late feature.

Such late cases are rarely seen these days.

Investigations

- **Urinalysis:** (1) Quantity - small (2) Dark Color (3) High specific gravity with acid reaction (4) Presence of acetone, occasional presence of protein and rarely bile pigments (5) Diminished or even absence of chloride
- **Biochemical and circulatory changes:** Mentioned previously
- **Ophthalmoscopic examination** is required if the patient is seriously ill. Retinal hemorrhage and detachment of the retina are the most unfavourable signs
- **ECG** when there is abnormal serum potassium level.

Diagnosis

The pregnancy is to be confirmed first.

Ultrasonography is useful not only to confirm the pregnancy but also to exclude other obstetric (hydatidiform mole, multiple pregnancy), gynecological, surgical or medical causes of vomiting.

Complications

- Ketoacidosis
  The following complications may occur which are fortunately rare nowadays.
- Neurologic complications
  - Wernicke's encephalopathy due to thiamine deficiency
  - Pontine myelinolysis
  - Peripheral neuritis
  - Korsakoff's psychosis.
- Stress ulcer in stomach
- Esophageal tear (Mallory-Weiss syndrome)
- Jaundice
- Convulsions and coma and
- Renal failure.

Management

The principles in the management are:

- To control vomiting
- To correct the fluids and electrolytes imbalance
- To correct metabolic disturbances (acidosis and alkalosis)
- To prevent the serious complications of severe vomiting.

Hospitalization

Surprisingly the patient improves rapidly (with the same diet and drugs used at home)

NBM: Till at least 24 hours after the cessation of vomiting.

IV Fluids

- ***3 Litres /24 hours*** (half is 5% dextrose and half is Ringer's solution)
- Extra amount of 5% dextrose equal to the amount of vomitus and urine in 24 hours, is to be added
- Serum electrolyte to be corrected if there is any abnormality
- Enteral nutrition through nasogastric tube may also be given.

Drugs

- **Antiemetic drugs: Promethazine** 25 mg or **prochlorperazine** 5 mg may be administered twice or thrice daily intramuscularly.
  - Trifluoperazine 1 mg twice daily intramuscularly is a potent antiemetic therapy.
  - **Vitamin B_6 and Doxylamine** are also safe and effective.
  - **Metoclopramide** stimulates gastric and intestinal motility without stimulating the secretions. It is found useful.

- **Hydrocortisone** 100 mg IV in the drip is given in a case with hypotension or in intractable vomiting. Prednisolone is also used in severe cases.
- **Nutritional support** - with vitamin B_1, Vit. B_6, Vit. C and Vit. B_{12} are given.

Nursing care: **Sympathetic but firm handling** of the patient is essential. Social and psychological support should be extended.

Hyperemesis progress chart is helpful to assess the progress of patient while in hospital.

Daily record of pulse, temperature, blood pressure at least twice daily, intake-output, **urine** for acetone, protein, bile, blood biochemistry and ECG (when serum potassium is abnormal) are important.

Clinical features of improvement are evidenced by - (a) subsidence of vomiting (b) feeling of hunger (c) better look (d) disappearance of acetone from the breath and urine (e) normal pulse and blood pressure and (f) normal urine output.

Diet: Before the intravenous fluid is omitted, the foods are given orally. At first, dry carbohydrate foods like biscuits, bread and toast are given. Small but frequent feeds are recommended. Gradually full diet is restored.

Termination of pregnancy is rarely indicated. Intractable hyperemesis gravidarum in spite of therapy is rare these days.

Q. Management of Rh isoimmunized pregnancy. Add a note on prevention of isoimmunization (Anti D).

INTRODUCTION

Rh isoimmunized pregnancy means that the Rh negative mother has already been sensitized **(positive indirect Coombs' Test)** to Rh positive cells and **developed antibodies** against them.

Antibodies once formed remain life long.

These antibodies are of 2 types:
1. IgM: Cannot cross the placenta and are not harmful to the fetus
2. IgG: Cross the placenta and will cause fetal RBC hemolysis (of Rh positive fetus).

It can lead to development of:
- Hydrops fetalis (most serious form) and even IUFD
- Icterus gravis neonatorum (intermediate variety)
- Congenital anemia of newborn (least severe and mild variety).

Management

Rh isoimmunized patients should be managed in centres with special **'fetal medicine unit'** *and facilities for* **intrauterine blood transfusion.**

Also NICU, facilities for exchange transfusion and expert neonatologist would be needed to tackle the affected babies.

- In case of first affected pregnancy, Rh isoimmunized women should undergo determination of their anti-D antibody titers or quantitative estimation of antibody levels

- These are usually performed monthly until 24 weeks of gestation, after which time titers should be repeated every 2 weeks
- If titers remain below the critical titer, delivery can occur at term
- A critical titer is defined as the titer associated with a significant risk for fetal hydrops
- Usually, pregnancies in which antibody titers are **1:8** or lower can be managed by serial monitoring of maternal antibody titers
- But if the titer is **1:16 or higher**, fetal wellness assessment is mandatory by ultrasonography to evaluation of middle cerebral artery peak systolic velocity **(MCA-PSV)** or **serial amniocentesis for delta OD450** if the former is not available.

Management of Women with a History of a Previous Anemic Fetus or Infant

- When there is a history of a previous anemic fetus or newborn, the probability of subsequent affected Rh D-incompatible fetus is more than 80%
- In these cases, maternal antibody titers are not predictive for the severity of fetal anemia and fetal clinical surveillance, by assessment of middle cerebral artery peak systolic velocity (MCA-PSV) or serial amniocentesis for delta OD450 should be started at **18-20 weeks anyways or 10 weeks prior to the date of previous IUFD or hemolytic manifestation of the fetus.**

Amniocentesis and Amniotic Fluid Evaluation

- When fetal blood cells undergo hemolysis, breakdown pigments, mostly bilirubin,are present in the supernatant of amniotic fluid
- The amount of amniotic fluid bilirubin correlates roughly with the degree of hemolysis and thus indirectly predicts the severity of the fetal anemia
- The concentration is measured by a continuously recording spectrophotometer and is demonstrable as a change in absorbance **('deviation bulge') at 450 nm**, referred to as **ΔOD450**, and the value is plotted on **Liley's chart.**

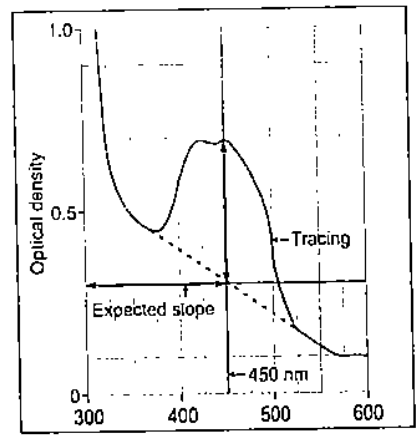


Fig. 27.1: Spectrophotometric analysis of amniotic fluid showing optical density difference at 450 nm wave length with 'deviation bulge' in Rh hemolytic disease

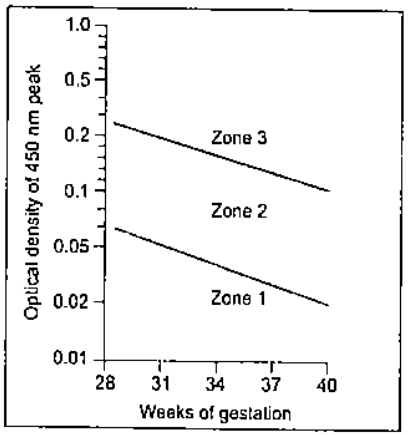
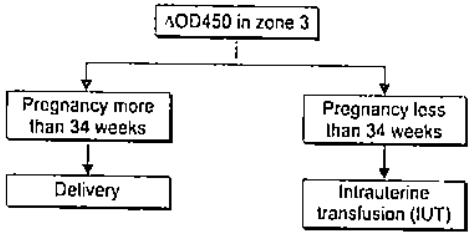


Fig. 27.2: Plotting of the 'deviation bulge' in Liley's prediction chart at different periods of gestation

- Optical density values in **zone 1** indicate a fetus that will have only **mild disease** or unlikely to be affected. Amniocentesis should be repeated in **3–4 weeks**
- In **zone 2,** the fetus is at moderate risk. In low zone 2, the expected fetal hemoglobin concentration is between 11.0 and 13.9 g/dl, whereas in upper zone 2, the anticipated hemoglobin level ranges from 8.0 to 10.9 g/dl. Amniocentesis should be repeated **after 1 week.**
- **Values in zone 3 indicate a severely affected fetus with a hemoglobin level of less than 8.0 g/dl,** and, without therapy, death is predicted within **7–10 days.** A value in zone 3 demands immediate **fetal red blood cell transfusion (intrauterine transfusion) or delivery depending on weeks of gestation**



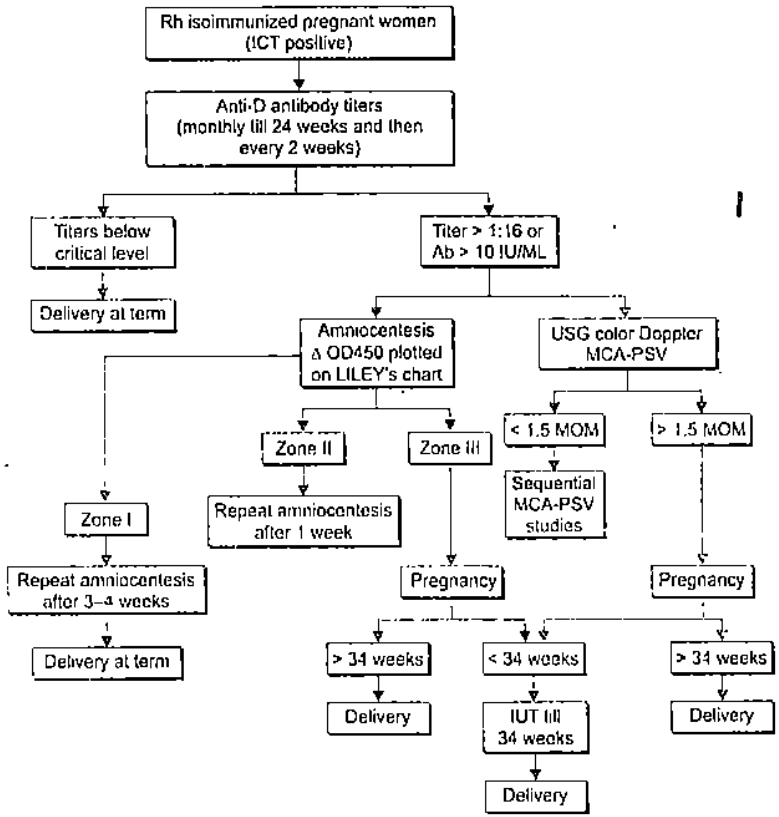
Recent Advances

Fetal anemia can be predicted noninvasively using middle cerebral artery (MCA) Color Doppler. The anemic fetus shunts blood preferentially to the brain to maintain adequate oxygenation. **Peak systolic velocity (PSV) in the MCA is increased with fetal anemia because of increased cardiac output and decreased blood viscosity.**

Nowadays this method is preferred over amniocentesis (which is an invasive procedure).

If the PSV is above the cutoff **(more than 1.5 MOM** for the corresponding gestational age) it indicates moderate to severe fetal anemia. Cordocentesis and IUT or delivery is indicated depending upon the weeks of gestation.

Flow Chart 27.2: Management of Rh isoimmunized pregnancy



Methods of Delivery

Vaginal: PG gel can be used for cervical ripening.

LSCS

- For obstetric indications
- In cases of preterm fetus with unfavourable cervix
- Cases where IUT has been done, LSCS is safer and preferred.

Care during Delivery

- **Early cord clamping** (as soon as possible) to prevent further antibody transfer
- **Cord to be kept long 15–20 cm** for exchange transfusion, if needed
- Avoid MROP at LSCS and minimize the spillage of blood in peritoneal cavity
- Cord blood investigations: Hb, ABO and Rh grouping, DCT and bilirubin.

As the mother is already sensitized there is no role of anti-D and hence it is not to be given after delivery.

IUT

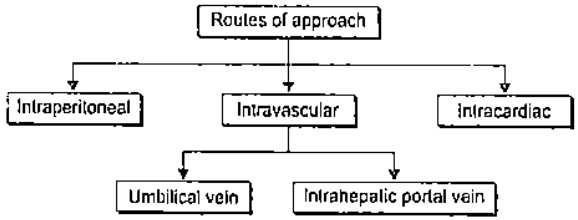
IUT continues to be the mainstay of therapy for severe Rh isoimmunization. It is indicated in cases where there is **severe affection of the fetus prior to 34 weeks of gestation.**

The purpose of intrauterine transfusion are to:
- **Treat hydrops fetalis and severe fetal anemia** (and thereby preventing IUFD) and maintain the fetal hemoglobin value at more than 9 g/dL
- Prolong /allow pregnancy **to complete 34 weeks** of gestation.

When to Perform

Nicolaides has recommend that transfusions be commenced when the hemoglobin is at least 2 g/dl below the mean for normal fetuses of corresponding gestational age. Other clinicians perform transfusions when the **fetal Hct is below 30%**, which is 2 standard deviations below the mean at all gestational ages. **Hydropic changes occur when Hct<15%.**

Serial intrauterine transfusions, if required, are usually performed until 34 weeks of gestation, because after this time the risk of the procedure is greater than benefits and so delivery should be done.



- Fresh **O-negative** double packed cells (with Hct 70–80%) crossed match with mother is to be transfused
- **The procedure is done in sterile room or OT under strict aseptic precautions with antibiotic cover under USG guidance**
- Tocolytics are given to prophylactically. Also steroids are given to enhance lung maturity
- **Temporary fetal paralysis is achieved by injection pancuronium/atracurium or vecuronium**
- It is given IM into fetal thigh, gluteal or deltoid
- Fetal paralysis last for 3–4 hours
- Fetal surveillance with USG and NST to be done in post transfusion period.

Intravascular Transfusion

- Cordocentesis is done and blood is transfused into umbilical vein under USG guidance
- The amount of blood required to be transfused is calculated by various formulas depending upon fetal Hct and donor Hct

- **Goal is to achieve a Hct of 50%**
- **Multiple transfusions may be needed 10–14 days as fetal fetal Hct drops at the rate of 1%/day.**

Intraperitoneal Transfusion

- It is done if vascular access is not available
- Blood is transfused in fetal peritoneal cavity which is taken up by subdiaphragmatic lymphatics
- The amount of blood transfused is calculated as number of weeks of gestation over 20, multiplied by 10 (e.g. at 30 weeks: 10 × 10 = 100 ml).

Complications

- Umbilical cord hematoma
- Fetal bradycardia
- Chorioamnionitis
- Preterm labor
- Abruption
- Fetal injury
- Volume overload
- IUFD (procedure related fetal loss is 4–5%).

Advantages

- Correction of fetal anemia and improved oxygenation
- Improved fetal hepatic function
- Helps to prolong pregnancy thereby avoiding birth of a extremely preterm fetus.

Overall neonatal survival is 90–100% for nonhydropic fetus and about 50–70% for hydropic fetus.

Other Theraples

For the rare patient with very early (≤18 weeks) severe isoimmunization, **plasma exchange (plasmapheresis) and administration of high dose intravenous immunoglobulin G(IVIG)** may maintain the fetal hematocrit above life-threatening levels long enough to achieve a gestational age when IUT is technically feasible.

PREVENTION OF ISOIMMUNIZATION

ANTI-D

- It is an IgG antibody that is given by i.m. route
- Monoclonal and polyclonal varieties are available.

Mechanism of Action

It binds to fetal RBCs and blocks the the Rh antigens of fetal cells, so that they cannot stimulate the maternal immune system and hence prevents the antibody formation.

To prevent maternal Rh sensitization, **women who are Rh negative** (and not yet senisitized/ ICT negative) should receive ANTI -D injections in the following situations:

- **At 28 weeks of gestation to all unsensitized Rh-negative mothers** (married to Rh positive partner). This reduces the risk of sensitization from 1.2% to 0.2 %
  - Postpartum within 72 hours (as early as possible) **if the baby's blood group is Rh positive**
- After abortion, MTP, ectopic pregnancy, molar pregnancy
- After amniocentesis, CVS, cordocentesis
- After ECV
- After manual removal of placenta (in any situation where fetomaternal hemorrhage is expected).

When the mother is already sensitized (positive indirect Coombs' test or positive Rh titer), there is no role of anti-D.

300 µg will protect the mother from fetal hemorrhage of up to **15 mL of D-positive red cells or 30 mL of fetal whole blood.**

The amount of fetal blood entering maternal circulation may be calculated by **Kleihauer-Betke (KB) test. It is not done routinely but to be done in cases when large FMH is suspected.**

Indications and Recommended Dose of Anti-D

Indications	Recommended dose (µg)
First trimester abortion/MTP	50
First trimester ectopic pregnancy	50
Second trimester abortion/MTP	300
Second trimester amniocentesis	300
Prophylaxis at 28 weeks	300
After delivery	300

Q. Anencephaly.

INTRODUCTION

The incidence of NTDs is **1-2 per 1000 live births**, and they are second only to cardiac anomalies, which are the most frequent structural fetal malformation.

- Anencephaly is a lethal NTD characterized by **absence of the brain and cranium above the base of the skull and orbits** but the facial portion is normal.
- It can be diagnosed as early as the **first trimester on USG.**
- 70% of fetuses are female.
- Face presentation is the most common presentation.
- Recurrence risk is 5% after one affected fetus and 13% after two affected fetuses.
- Frog eyes are seen.

Polyhydramnios is commonly seen due to the following reasons:

a. Transudation of fluid across the membranes
b. Absence of swallowing
c. Absent fetal pituitary (absence of ADH hormone implies that the baby passes more urine)
   - Postdatism is seen as fetal pituitary plays an important role in initiation of labor.
   - However preterm labor can also be there due to polyhydramnios.
   - **Pseudoshoulder dystocia** is seen as the soft head/face can slip through incompletely dilated cervix.

Management

- If diagnosed before 20 weeks of gestation MTP should be offered and done after counseling patient, as its a lethal anomaly.
- If diagnosed later legally MTP is not allowed. Labor may be induced. The uterus is often refractory to oxytocin and hence PGs may be required.

Prevention

- Some NTDs are associated with a specific mutation in the methylenetetrahydrofolate reductase gene, the adverse effects of which can be largely overcome by **periconceptional folic acid supplementation.**
- More than half of NTDs could be prevented with daily intake of **400 µg of folic acid throughout the periconceptional period** (1 month before pregnancy and the first trimester of pregnancy).
- However in patients with **past history of NTDs, 4 mg folic acid** is recommended throughout the periconceptional period.

PART 3

Pediatrics

– Short Notes

- APGAR Score
- Asphyxia Neonatorum
- Care of the Newborn at Birth
- Causes of Convulsion in Neonate
- Down Syndrome
- Kernicterus
- Neonatal Jaundice
- Neonatal Resuscitation

Pediatrics Short Notes

Q. APGAR Score.

INTRODUCTION

Apgar score, introduced by *Dr Virginia Apgar* (American Anesthesiologist at Columbia Presbyterian Medical Center, 1952) is a quantitative method for assessing the Infant's respiratory, circulatory and neurological status.

Apgar Score System

	0	1	2
Color of the body (Appearance)	Blue/pale	Body pink Extremities blue	Pink completely
Heart rate/min (Pulse)	Absent	<100	>100
Reflex stimulation (Grimace)	No response	Grimace	Cries, coughs or sneezes
Muscle tone (Activity)	Flaccid	Some flexion	Actively moving the extremities
Respiratory effort (Respiration)	None	Slow, irregular	Good crying

- 8–10 Normal
- 5–7 Moderately asphyxiated
- <4 Severe distress.

Note: The ideal score at 1 minute is **9** as the periphery is always blue (acrocyanosis) and **score is not 10 at 1 minute.**

Timing of the Score

- At first cry
- After regular respiration has been established
- Delayed to detect any neurological deficit.

To satisfy the above criteria the Apgar score is taken at 1 minute, 5 minutes and 10 to 20 minutes (Extended APGAR, if required).

Importance

- Apgar score is a **'monitoring score'** *to* determine the **efficacy of resuscitation**
- Gives an overall view of condition of newborn
- As the score declines it indicates drop in arterial pH, i.e. acidosis
- Prognostic value: If score is **< 4 at 20 minutes;** it indicates a very bad prognosis
- Five minutes score is more useful indicator to the status of newborn than the 1 min score
- If the score is low it may indicate any of the following:
  - Birth asphyxia
  - Drugs given to mother during labor
  - Congenital malformations
  - Intrauterine infections/septicemia.

Fallacies

- It is a subjective scoring (except heart rate)
- It ignores the time of first cry of newborn
- One minute score is not useful in deciding upon the intervention necessary for resuscitation as action must be initiated before that
- Apgar scoring cannot be used in:
  - Preterm baby
  - Neurologically floppy infant
  - Severely sedated infant
  - Infant with Erb's palsy
- Respiration cannot be judged if the newborn is on IPPR
- Score at 1 minute cannot be taken as record of situation at birth
- It does not give any idea of the duration of asphyxia, e.g. perinatal asphyxia, hypothermia, metabolic derangements in the mouth
- It does not give the severity of the asphyxia, 1 and 5 min score cannot tell the progression. Hence 10 and 20 minutes scores are done to tell about possible neurological deficit.

2. Asphyxia neonatorum.

INTRODUCTION

Asphyxia neonatorum is the consequence due to lack of oxygen and/or lack of perfusion to various organs resulting from nonestablishment of satisfactory pulmonary respiration at birth.

Etiology

- **Continuation of intrauterine hypoxia (90%)**
  - *Placenta:* Functional failure of placenta as respiratory organ
    - Anatomical changes in the placenta: Thin small placenta, circumvallate placenta
    - **Uteroplacental insufficiency**
    - Premature separation of placenta/abruption
    - Extensive infarcts with/without postdatism
    - Hypertensive disorders in pregnancy
    - Supine hypotensive syndrome

  - Cord compression/prolapse, true knot in the cord, vascular anomalies in the cord, etc.
  - *Maternal hypoxic states:* Anemia, eclampsia, cyanotic cardiovascular disorders, status asthmaticus, shock.

- ***Birth trauma to the neonate:***

Malpresentations/Prolonged head compression
(forceps/vacuum)/prolonged second stage of labor

↓

Raised intracranial tension
Cerebral edema and congestion

↓

Asphyxia

↓

Premature attempts of fetus to initiate respiration (*in utero*) leads to
Increased venous pressure
Aspiration of amniotic fluid

↓

IUFD or Delivery of asphyxiated baby

- ***Prenatal and intranatal medications to the mother:***

Morphine pethidine, anesthetic agents depress respiratory
centers of the newborn directly

↓

Continuing intrauterine asphyxia

↓

Premature respiratory efforts *in utero*

↓

Transient period of rhythmic respiratory effort

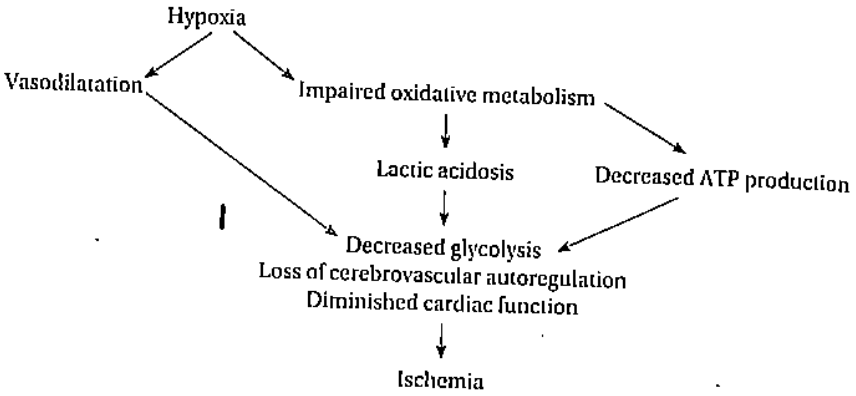
↓

Followed by primary apnea

↓

Secondary/terminal apnea due to
Respiratory depression
Acidosis
Hypoglycemia

Pathophysiology of Hypoxic Ischemic Encephalopathy



Diagnostic Criteria for Birth Asphyxia

Any four of the following:

1. Evidence of fetal distress
2. Acute metabolic acidosis
3. Apgar at 5 minutes < 5
4. Initiation of respiration > 5 minutes
5. Features of hypoxic ischemic encephalopathy
6. Multiorgan damage.

Clinical Features and Complications

Depend on: Etiology, intensity and duration of O_2 lack, plasma CO_2 excess and subsequent acidosis.

- CNS: Hypoxic ischemic damage in form of cortical necrosis, cortical infarcts, necrosis of thalamus/basal ganglia. Signs of raised ICT
- Renal: Features of acute tubular necrosis, syndrome of inappropriate secretion of ADH
- GIT: Necrotizing enterocolitis, liver damage
- Cardiac: Transient myocardial ischemia, mitral regurgitation, tricuspid regurgitation
- Respiratory: Pulmonary hemorrhage, pulmonary edema, increased pulmonary resistance, respiratory distress syndrome and meconium aspiration
- Hematological: Disseminated intravascular coagulation.

Management

Prophylactic

- High-risk cases segregated
- Intrapartum fetal monitoring particularly in high-risk cases to determine fetal distress and timely intervention
- Careful management of labor in high-risk conditions

- Judicious administration of anesthetic agents and depressant drugs during labor
- Proper care of newborn at the time of birth.

Definitive

- Maintain hydration IV fluids
- Maintain temperature
- Symptomatic treatment of hypoglycemia, hypocalcemia, convulsions, etc.
- Treatment of cerebral edema.

Poor Prognostic Factors

- Severe prolonged asphyxia
  - Seizures of early onset, specially difficult to treat
  - Raised intracranial pressure
- Abnormal neurological signs at the time of discharge
  - Sarnat stage 3
  - Hypodensities on CT brain
  - Persistent oliguria.

Q. Care of the newborn at birth.

PRINCIPLES

- Maintenance of temperature
- Initiation of respiration
- Maintenance of asepsis
- Diagnosis of severe congenital malformations.

STEPS OF NEONATAL CARE

Preparation

- Temperature of the labor room to be kept at about **30 °C to prevent hypothermia.** Exposure to cold may lead to metabolic acidosis, hypoglycemia and increased renal losses of solute and water
- Prewarm towels are kept ready
- **Radiant warmer** of receiving trolley are kept on.

Reception

- Keep under warmer (at a distance of 80 cm)
- Air passage suctioned after the baby's head comes out. **Air passages cleared by** suction of **oral cavity, oropharynx and nose (in the same order)**
- The baby should be received in a sterile prewarmed towel and then kept on warm surface in a head low position
- Wipe and **clean dry the baby**, special care should be taken to **dry the head**, which forms a large part of surface area in a newborn.

Apgar Scoring

- Apgar scoring to be done at 1 minute and 5 minutes.

Resuscitation

Resuscitation if required: Four Cardinal principles of neonatal resuscitation:

1. **Minimize immediate heat loss**
2. **Ensure open airway**
   - Proper position
   - Suction of mouth followed by nose (to prevent aspiration)
   - If thick meconium, endotracheal intubation and suction
3. **To initiate breathing**
   - Tactile stimulation (gentle rubbing on back/flicking soles of feet)
   - Face mask and AMBU bag positive pressure breathing
   - Endotracheal intubation and mechanical ventilation if required
4. **To maintain circulation**
   - Chest compression
   - Medication.

Physical Examination of the Baby

To be conducted
- Soon after birth
- 24 hours of age
- Before discharge from the nursery.

First examination is done to determine:
- Any congenital anomalies
- To categorize the baby in the birth weight, gestational age groups
- To detect other disorder which may affect neonatal course

- Umbilical cord	- Look for single umbilical artery
- Ears	- Low set ears, preauricular tags of sinus which are associated with congenital renal malformations
- Mouth	- Cleft palate/lip Tracheoesophageal fistula
- Nose	- Choanal atresia
- Neck	- Branchial/thyroglossal cysts, Neonatal goiter, Cystic hygroma Webbed neck in Turner's syndrome
- Chest	- Auscultate for murmur
- Arms	- Erb's palsy, Klumpke's palsy
- Genitalia	- Imperforate hymen with hydrocolpos Hypospadias/Epispadias Cryptorchidism Ambiguous genitalia
- Feet	- Talipes equinovarus
- Rectum	- Record initial temperature Exclude imperforate anus

Gestational Age Estimation

Plantar creases, descent of testes

Preterm	<37 weeks
Post-term	>42 weeks

Maintenance of Body Temperature

- Environmental temperature around 25 °C
- Preterm and sick-thermo-neutral environment
- Larger and older babies -32 °C with 40-60% relative humidity
- Warm, woollen clothes
- Radiant warmer/incubator/servocontroller.

Identification Tag

To prevent accidental exchange of babies. Also footprint of the baby is taken on the case paper to confirm identification.

Transfer to Nursery/Mother's Room and Care in the Nursery

- Baby bath: Not routinely favored to prevent cross infection vernix, blood and meconium wiped off using sterile moist swabs and skin is kept dry using soft towel. Baby bath may be given day prior to discharge. Use of bland soap, soft towel and separate bath tub for each baby.
- Weight: Baby should be weighed naked.
- Care of umbilical cord:
  - Inspect for slippage of ligature
  - Watch for foul smell, delayed separation, periumbilical redness (Suggestive of sepsis)
  - Cord stump falls by 5 to 10 days.
- Eye care: Clean with normal saline
- Medications:
  - Vit K 1 mg IM to minimize hemorrhagic tendency
  - Prophylactic antibiotic therapy required if:
    - Premature rupture of membranes
    - Instrumentation (laryngoscopic intubation)
- Close observation
  - Excessive mucus secretion
  - Bleeding from umbilical cord stump
  - Hourly temperature till it stabilizes above 36 °C
- Rooming in as early as possible even within half hour after delivery baby is given to the mother.

Advantages are easy establishment of breastfeeding and emotional bond development.

Q. Causes of convulsion in neonate.

Neonatal seizures or neonatal convulsions are epileptic fits occurring from birth to the end of the neonatal period. The neonatal period is the most vulnerable of all periods of life for developing seizures, particularly in the **first 1–2 days to the first week from birth.**

The prevalence is approximately 1.5% and overall incidence approximately 3 per 1000 live births.

	Frequency
Hypoxia-Ischemia/BIRTH ASPHYXIA • Prenatal (hypoxemia, fetal distress, abruptio placentae, cord compression) • Perinatal (iatrogenic, maternal hemorrhage, fetal distress) • Postnatal (cardiorespiratory causes such as hyaline membrane disease, congenital heart disease, pulmonary hypertension)	+++++++
Hemorrhage and intracerebral infarction • Intraventricular and periventricular (mainly preterm neonates) • Intracerebral (spontaneous, traumatic) • Subarachnoid • Subdural hematoma • Cerebral artery and vein infarction	++++
Trauma • Intracranial hemorrhage • Cortical vein thrombosis	++++
Infections • Encephalitis, meningitis, brain abscess • Intrauterine (rubella, toxoplasmosis, syphilis, viral – such as cytomegalovirus, herpes simplex virus, human immunodeficiency virus, coxsackie virus B) • Postnatal (beta-hemolytic streptococci, *Escherichia coli* infection, herpes simplex virus, *Mycoplasma*)	++++
Metabolic • Hypoglycemia (glucose levels <20 mg/d in preterm and, <30 mg/d in full-term babies indicating hypoglycemia; mainly associated with prenatal or perinatal insults) • Neonates of diabetic mothers • Pancreatic disease • Glucogen storage disease (idiopathic) • Hypocalcemia (early, in first 2–3 days, mainly in preterm neonates with prenatal or perinatal insults; late, at 5–14 days, is mainly nutritional; maternal hyperparathyroidism; DiGeorge's syndrome) • Hypomagnesemia (may accompany or occur independently of hypocalcemia) • Hyponatremia (mainly associated with prenatal or perinatal insults; inappropriate secretion of antidiuretic hormone) • Hypernatremia (mainly nutritional or iatrogenic) • Inborn errors of metabolism (amino acid and organic acid disorders, hyperammonemias; they usually manifest with peculiar odors, protein intolerance, acidosis, alkalosis, lethargy, or stupor) • Pyridoxine dependency • Kernicterus	++

Contd...

Contd...

	Frequency
Malformations of cerebral development • All disorders of neuronal induction, segmentation, migration, myelination and synaplogenesis such as polymicrogyria, neuronal heterotopias, lissencephaly, holoprosencephaly, and hydranencephaly	++
Neurocutaneous syndromes • Tuberous sclerosis, incontinentia pigmenti	++++
Drug withdrawal and toxic • Withdrawal from narcotic-analgesics, sedative-hypnotics, and alcohol; heroin- and methadone-addicted mothers; barbiturates	+++
Inadvertent injections of local anesthetics during delivery	++
Idiopathic benign neonatal seizures (familial and nonfamilial)	

Q. Down Syndrome.

INTRODUCTION

Down syndrome is the **most common autosomal trisomy.** In Down syndrome the chromosome number 21 is present in triplicate. Down syndrome is a common cause of mental retardation.

Genetic Basis

- ***Nondysjunction* (95% of patients)** Meiotic separation does not occur for number 21 chromosome. One of the gamete carries an extra chromosome. On fertilization of this gamete, it leads to trisomy 21.
- ***Translocation* (4% of cases)** when part of chromosome 21 becomes translocated onto another chromosome usually a D group (number 13, 14 or 15) or a G-group (number 21 or 22) chromosome. Affected people have two normal copies of chromosome 21 plus extra material from chromosome 21 attached to another chromosome, resulting in three copies of genetic material from chromosome 21.
- ***Mosaicism* (1% of cases)** Some cells having 46 chromosomes and two number 21 chromosomes and some cells having 47 and three 21chromosomes.

Incidence

Down syndrome occurs in 1 in 600 to 1 in 800 live births **(50% of cases abort in early pregnancy).**

Risk in Relation to Increasing Maternal Age

Age of mother	Approximate risk of Down syndrome
20 years	1 in 2000
30 years	1 in 1000
40 years	1 in 100
45 years	1 in 30

Clinical Features

Mosaic Down syndrome children have milder clinical presentation as compared to other 2 types:

Dysmorphic Features

1. **Dysmorphic facial features:**
   - Flat facial profile
   - Short, upslanting palpebral fissures
   - Brushfield spots on iris
   - Flat nasal bridge with *epicanthal folds*
   - Small, mouth with protruding tongue
   - *Short ears* with abnormal ear lobes
   - Cataracts and squint is common
   - High arched palate with small teeth
   - *'Scrotal'* (Furrowed) tongue.
2. **Other dysmorphic features:**
   - Microcephaly, Brachycephaly
   - Skin: Excess posterior neck skin
   - Short stature/short 'long' bones
   - Clinodactyly with hypoplastic middle phalanx.
   - Single palmar crease *(Simian crease)*
   - Sandle gap.

Functional and Structural Abnormalities

- ***Hypotonia:*** Frequent accompaniment. Most noticeable in the newborn (poor Moro's reflex and hyperflexibility of joints, hip dysplasia)
- *Mental retardation* and developmental delay. The mean IQ is 50.
- *Cardiac defects:*
  - Endocardial cushion defects
  - Septal defects (VSD/ASD)
  - Patent ductus arteriosus
- Abdomen and pelvis
  - Duodenal artesia and Hirschsprung disease
  - Omphalocele, congenital diaphragmatic hernia
  - Small penis
  - Cryptorchidism
  - Renal pylectasis
- *Hypothyroidism and leukemia* occur at higher frequency
- *Social aspects:* Behave as good babies, happy children and tend towards mimicry, are friendly and have a good sense of rhythm and enjoy music.

MANAGEMENT

Counseling

- To be given after confirmation of diagnosis
- Both the parents should be present
- Counseling given by a team of pediatrician, geneticist and psychiatrist
- Explain the parents about the disease; that the child is going to be mentally retarded, require special schooling
- Explain about congenital heart diseases, other abnormalities.

Counseling about the Recurrence Risk

Chromosome constitution			Risk of offspring	
Affected child	Father	Mother		
Trisomy 21 (Nondisjunction)	N	N	Mother <30 year in present pregnancy	2–3%
			Mother >30 year; had Down baby before 30 year of age	Risk at mothers age +1%
			Mother >30 year; had Down baby after 30 year age	Risk at mother's age
Translocations 14/21, 15/21, 13/21, 21/22	N	C		11.9%
	C	N		2–3%
Translocations 21/21	N	C		100%
	C	N		100%
Mosaic	N	N		2–3%

C = Carrier, N = Normal

In Future Pregnancy

The only 100% confirmatory test for Down syndrome is karyotyping, the sample for which can be obtained by chorionic villus sampling (CVS) or amniocentesis. Hence, in a patient who has a past history of fetus with Down syndrome, **fetal karyotyping should to be done in all future pregnancies.**

Prevention

Early Detection of Down's Syndrome and MTP

Antenatally NT SCAN and dual marker test (hCG + PAPP A) at 11–13 weeks of gestation or **triple marker test,** should routinely be offered to all patients as **a screening test.**

Triple Marker Test

This is a **screening** test done between **16 and 18 weeks of gestation,** mainly to identify a mother who is at a high-risk of having a fetus with trisomy 21. It involves estimation of 3 hormones: hCG, AFP, and unconjugated estriol (UE3).

Interpretation

In following cases **confirmatory test (karyotyping) should be done** and if abnormal MTP should be offered:

- Maternal age >35 years
- Previous autosomal trisomy birth
- Major fetal structural defect identified by ultrasound/increase NT
- High-risk detected by dual/triple marker test.

Q. Kernicterus.

DEFINITION

Unconjugated hyperbilirubinemia in the neonatal period can cause bilirubin encephalopathy with necrosis of neurons in the basal ganglia, hippocampus, and subthalamic nuclei. This *'nuclear staining'* with bilirubin is called **'Kernicterus'** (***Bilirubin brain damage/ Bilirubin encephalopathy***).

Pathophysiology

- Unconjugated bilirubin can be bound to albumin or exist as free bilirubin if albumin is exhausted
- 3 gm of albumin binds with 21–24 mg indirect bilirubin
- A newborn has about 3 gm% of albumin in blood. **If the indirect bilirubin goes above 24–25 mg%** then albumin is not available for binding and the excess indirect bilirubin accumulates
- The bilirubin bound to albumin cannot cross the blood brain barrier. However, the indirect bilirubin not bound to albumin can do so
- Once bound to the neurons, *the free indirect bilirubin* acts as a mitochondrial toxin. This leads to *mitochondrial damage*. Once the free indirect bilirubin binds to mitochondria, it is almost irreversible change but the bilirubin can still be dissociated and hence this effect can be considered partly reversible
- It is also said, that bilirubin reversibly *inhibits phosphorylation* of 'synapsin' which is believed to regulate neurotransmitter release. After initial entry into the neurons, the bilirubin departs leaving behind neuronal atrophy and gliosis
- Also, several other cytoplasmic proteins such as ligandin, fatty acid binding protein and lipoprotein show high affinity for bilirubin. As the bilirubin binding capacity of the tissue proteins is exhausted, the free bilirubin seeps into the neurones.

High-risk Factors

Prematurity and Preterm Infants

- Blood brain barrier is weak
- Capacity of albumin to bind to bilirubin is also low.

Neonates Suffering from

- Hypoglycemia
- Hypothermia
- Acidosis
- Serious metabolic illnesses.

All these lead to a fall in blood pH and uncouples bilirubin from bilirubin-albumin complex.

Conditions like

- Hypoxia
- Hemolysis
- Septicemia
- Ketoacidosis.

Can lead to increased levels of free bilirubin in the blood.

Drugs

- Salicylates
- Sulfonamides
- Nonesterified fatty acids.

These decrease bilirubin binding with albumin.

Also, following causes a decreased threshold for bilirubin brain damage:

- Hypocalcemia
- Hypomagnesemia
- Birth asphyxia
- Respiratory distress syndrome
- Hyaline-membrane-disease.

Parts of Brain Affected by Hyperbilirubinemia

- Eighth nerve nucleus
- Basal ganglia
- Cerebral cortex (if free indirect bilirubin levels are very high).

Clinical Features

- Appear between 3 to 7 days of life
- Lethargy, refusal of feeds, shrill cry
- Icterus, bulging fontanelle, setting sun sign, dampened reflexes, convulsions, rigidity, ophisthotonus, abnormal Moro's reflex
- In very mild type there may just be deafness and hearing loss (detected by brainstem evoked response potentials - BERA on follow-up).

Stages

- Stage 1 Hypotonia, poor Moro's reflex. lethargy, vomiting, high pitched cry
- Stage 2 Seizures, rigidity, opisthotonus, fever, oculogyric crisis, paralysis of upward gaze
- Stage 3 Decrease in spasticity (one week of age)
- Stage 4 Late sequale.

Sequale

- Deafness (VIII nerve nucleus affected)
- Spastic/Athetoid type of cerebral palsy (basal ganglia affected)
- Intellectual retardation and learning disabilities (cortical involvement)
- Paralysis of upward gaze
- Epilepsy
- Dental dysplasia
- Brownish staining to teeth.

Investigations

- Serum bilirubin: Direct, indirect
- Serum albumin
- Blood electrolytes (r/o acidosis) a Blood culture (r/o septicemia) o Blood sugar (r/o hypoglycemia)
- Hb, CBC, ESR (hemolytic process).

Management

Treating the indirect hyperbilirubinemia:
- Phototherapy
- Exchange transfusion
- Use of tin-metalloporphyrin.

(For details please refer to note on Neonatal Jaundice)
- Rehabilitation of the patient later on is equally important (in cases of cerebral palsy, MR, deafness, etc.). Proper referrals should be made
- The prime objective has to be preventing the rise of bilirubin to toxic levels which may cause kernicterus.

Q. Neonatal jaundice.

DEFINITION

Physiological Jaundice

Unconjugated hyperbilirubinemia with following characteristics:
- *In full term newborns:* Peak bilirubin level of 6-8 mg/dl by 3 days of life and maximum reached up to 12 mg/dl. Returns to normal by 10th day of life
- *In preterm newborns:* Peak bilirubin level by fifth day of life rising up to 15 mg/dl. Returns to normal level by 15th day.

Pathological Jaundice

Nonphysiological hyperbilirubinemia with any of following characteristics:
- Clinical jaundice prior to 36 hours of age
- Clinical jaundice persisting for more than 8 days in full term babies or 14 days in preterm newborns
- Serum bilirubin increasing to more than 5 mg/dl/day
- Total serum bilirubin more than 15 mg/dl in formula fed term baby or > 17 mg/dl in breast-fed term baby.

Incidence

- 25-60% of full term newborns develop clinical jaundice
- 70-85% of preterm infants develop clinical jaundice
- 3% of normal term infants show bilirubin levels more than 15 mg/dl.

Note

- 1 gm of hemoglobin produces 34 mg of bilirubin
- Normal newborn produces 6-10 mg of bilirubin/kg body weight/day as compared to 3-4 mg/kg/day in adult.

Possible Mechanisms involved in Physiologic Jaundice

- Increased bilirubin load on liver cells:
  - Increased RBC volume/kg as compared to adult
  - Decreased RBC survival 90 days
  - Increased enterohepatic circulation of bilirubin
  - Defective hepatic uptake of bilirubin from plasma
- *Defective bilirubin conjugation*: Decreased UDP glucoronyl transferase activity and decreased UDP glucose dehydrogenase activity
- Defective bilirubin excretion.

Factors Exaggerating Physiologic Jaundice

- Male sex
- Prematurity
- Maternal diabetes
- Hypoxia
- Cutaneous bruising and cephalhematoma.

Causes of Pathological Jaundice

Overproduction

- **Hemolytic disorders**
  - Extracorpuscular
    - Immunological: Rh incompatibility, ABO incompatibility
    - Nonimmunological: Sepsis, Vit. K induced hemolysis.

Intracorpuscular
- Hemoglobinopathies: Thalassemia
- Enzyme defects: G-6PD deficiency, Pyruvate kinase deficiency
- Membrane defects: Hereditary spherocytosis, elliptocytosis, poikilocytosis.

- **Extravascular blood**
  - Cephalhematoma and other hematomas
  - Pulmonary and cerebral hemorrhage.
- **Polycythemia**: *Chronic fetal hypoxia*
- **Exaggerated enterohepatic circulation**
  - Mechanical obstruction: Atresia and stenosis, Meconium ileus, Hirschprung's disease
  - Reduced peristalsis: Underfeeding.

Under Secretion

- **Obstructive disorders**
  - Biliary atresia
  - Choledochal cyst
  - Tumor or band
  - Dubin Johnson syndrome
  - Rotor's syndrome.
- **Metabolic and endocrinal disorders**
  - Galactosemia
  - Criggler: Najar syndrome
  - Gilberts syndrome
  - Hypothyroidism
  - Hypopituitarism
- **Prematurity.**

Mixed

- *Intrauterine infections:* Toxoplasmosis, Rubella, Herpes zoster, Syphilis
- Maternal diabetes
- *Respiratory distress syndrome*
- Asphyxia
- *Erythroblastosis fetalis.*

BREAST MILK JAUNDICE

In up to 1% breastfed child, the serum bilirubin levels remain high after the third day instead of the usual fall and may rise up to 20-30 mg% by two weeks of age. With continued breastfeeding the levels stay elevated and then gradually fall to return to normal by 1 to 3 months of age. If breastfeeding is stopped the bilirubin level falls rapidly in 48 hours. This is harmless condition and kernicterus does not occur.

Clinical Examination

- **Severity of jaundice (Krammer's rule)**
  - Icterus limited to head and neck — 4-8 mg%
  - Icterus involving upper trunk — 5-12 mg%
  - Icterus over lower trunk — 8-16 mg%
  - Icterus over arms, legs — 11-18 mg%
  - Icterus over palms and soles — >15 mg%
- **Color of jaundice**
  - Hemolytic : Lemon yellow
  - Hepatic : Bright yellow/orange yellow
  - Obstructive : Green yellow
- Gestational age: Prematurity
- General condition of newborn: Sepsis, fever, tachycardia, tachypnea, evidence of injuries
- Umbilical discharge: Umbilical sepsis
  - Pallor, hemorrhage, petechiae
- Hepato-/spleno-megaly
  - Neurological signs
  - Cry: Shrill cry in kernicterus
  - Tone
  - Reflexes: Moro's and sucking reflexes are weak or absent
- Features of endocrinal disorders
- Any congenital malformation.

Investigations

- Hb, CBC, Reticulocyte count
  - Peripheral smear for RBC morphology
- ABO and Rh grouping of mother and baby
- Coomb's test of mother and baby
- Serum bilirubin: Direct and indirect
- Blood culture
- Serum albumin
- Other liver function tests
- G-6-PD enzyme studies
- Ultrasonography of abdomen and liver scans
- T3, T4, TSH

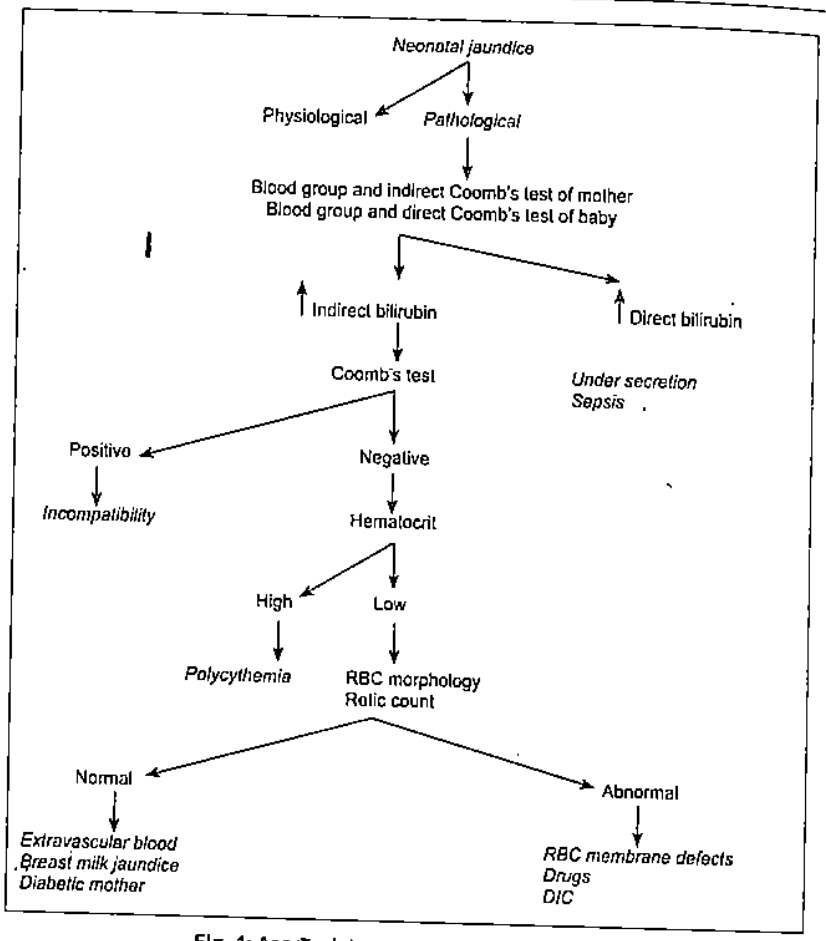


Fig. 1: Approach to a case of neonatal jaundice

Treatment

Physiological jaundice:

- No specific treatment required
  - Continue breastfeeding
  - Watch for any complicating illness or sudden rise in bilirubin levels
  - Treat/prevent any exaggerating factor.

Pathological Jaundice

Treatment Modalities

- Phototherapy

- Exchange transfusion
- Phenobarbital
- Agar-Agar
- Albumin infusion
- Tin protoporphyrin.

Phototherapy

Mode of Action

Phototherapy helps by lowering unconjugated hyperbilirubinemia.

- ***Structural isomerization:*** Phototherapy modify bilirubin deposited in the skin by photo-isomerization process forming bilirubin isomer, which is rapidly excreted in the bile with a half life of about 2 hours.
- ***Intramolecular cyclization:*** To lumirubin, which is rapidly excreted in bile and urine without conjugation
- ***Photo-oxidation:*** Bilirubin absorbs light and get oxidised to polar color less products which are excreted in urine.

Indications

- Serum bilirubin above 15 mg% for term infants and 10 mg% for preterm infants.
- Serum bilirubin 5 mg% or more in *the* first 24 hours.
- In hemolytic disease of newborn—immediately after birth.
- Adjunct to exchange transfusion in hemolytic disease of newborn.
- Prophylactically in:
  - Very low birth weight babies who are likely to develop dangerous levels of bilirubin.
  - Severely bruised premature infants.

Contraindications

- Obstructive jaundice—**ineffective in reducing direct hyperbilirubinemia**
- Light sensitive porphyria.

Technique

- Light source: Four blue/green light fluorescent lamps
  - Position of infant: Placed naked at a distance of 45 cm below light source
- Protection of infant: Eye patches to protect from retinal damage and diaper to protects external gonads
  - Duration of phototherapy: 24-48 hours exposure is generally long enough to bring down serum bilirubin level to safe limit.

Side Effects of Phototherapy

- Dehydration
- Diarrhea
- Skin burns/rash

- Tanning
- Bronze baby syndrome—reduced hepatic excretion of bilirubin photoproduct
- Decreased serum calcium levels
- Hyperthermia
- Hemolysis
- Retinal damage.

Exchange Transfusion

Aims and Objectives

- *Remove* excess bilirubin and other harmful substances and to replace the blood by healthy donor blood
- Correct anemia by replacing blood of low PCV with that of normal PCV avoiding circulatory overload also.

Indications

Uncomplicated hemolytic disorders:
- Severe anemia Hb < 8 gm%
- Serum bilirubin > 20 mg% at any time
- Serum bilirubin rising by > 1 mg% per day
- Cord bilirubin > 4.5 mg% and cord Hb < 11gm%
- Serum bilirubin rising > 0.5 mg% with hemoglobin level between 11 and 13 gm%.

Complicated hemolytic disorders:
- Hydrops fetalis
- Impending heart failure.

Donor Blood

- Should be fresh (less than 3 days old)
- Amount required.

Double volume exchange: 160 ml/kg (exchanges 87% of infants blood volume).

Single volume exchange: 80 ml/kg (exchanges 63% of infants blood volume).

Blood Group

In Rh incompatibility: O Rh negative blood, cross matched against mother's blood.

In ABO incompatibility: Same ABO and Rh group as of baby.

Tested for HIV, HBsAg and VDRL.

Technique

- Strict asepsis
- Cardiac monitor

- Umbilical vein cannulated with umbilical catheter
  - Exchange transfusion done by push-pull method.

Complications

- Vascular: Embolisation, thrombosis, vasospasm
- Cardiac: Arrhythmias, arrest circulatory overload
- Electrolyte and acid base: Hyperkalemia, hypocalcemia, acidosis
- Bleeding: Thrombocytopenia, deficient dolling factors
- Infections
- Others: Hypo/hyperthermia, perforation of vessels, transient maculopapular rash.

Phenobarbital

Mechanism of Action

Induces activity of the enzyme glucoronyl transferase. Increases bilirubin conjugation and excretion.

Dosage and Role in Neonatal Jaundice

Therapeutic: 5-8 mg/kg/day to the newborn: Indicated only in Crigler-Najjar syndrome type II and other conjugated hyperbilirubinemia.

Disadvantages/Side Effects

Take time (3-4 days) to become therapeutically active.

Child may become drowsy which is also an early feature of kernicterus. Slow feeding.

AGAR

Mechanism of action: Binds bilirubin in gut and diminishes enterohepatic circulation.

Dosage and role in neonatal jaundice: 125 mg every 3 hourly in mild to moderate hyperbilirubinemia as an adjunct.

ALBUMIN INFUSION

Mechanism of action: Raises bilirubin binding capacity.

Dosage and role in neonatal jaundice: 1 mg/kg of salt free albumin can be used as an alternative to exchange transfusion in very small infants.

Tin Protoporphyrin

Hemeoxygenase enzyme inhibitor.

Q. Neonatal resuscitation.

Globally, over 5 million neonatal deaths occur each year. However outcomes might be improved for more than 1 million infants per year through effective implementation of simple resuscitative techniques.

ANTICIPATORY PREPARATION FOR RESUSCITATION

High-risk babies are more likely to develop anoxia, e.g.

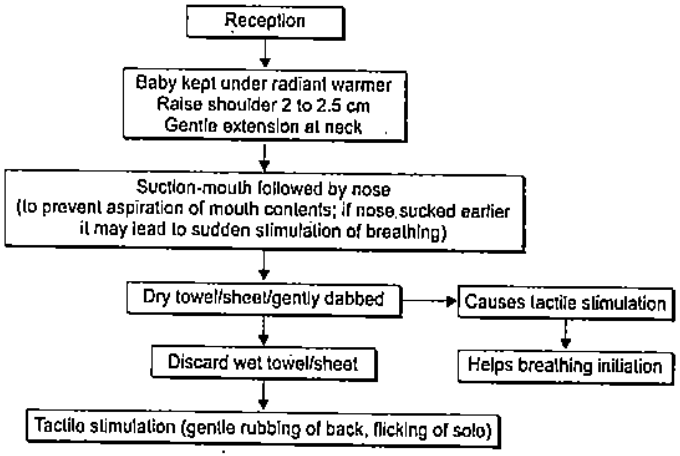
- *Maternal factors:* Complicated pregnancies like preeclampsia, APH, BOH, etc.
- *Fetal factors:* Preterm delivery, multiple births, IUGR
- *Complicated labor:* Malpresentations, LSCS/instrumental delivery, cord prolapse, fetal distress/passage of meconium.

Plan of Action

Depends on respiratory rate, heart rate, and color.

FOUR CARDINAL PRINCIPLES OF NEONATAL RESUSCITATION

1. Minimize immediate heat loss
2. Ensure open airway:
   - Proper position
   - Suction of mouth followed by nose (to prevent aspiration)
   - If thick meconium, endotracheal intubation and suction.
3. To initiate breathing:
   - Tactile stimulation (gentle rubbing on back/flicking soles of feet)
   - Face mask and AMBU bag positive pressure breathing
   - Endotracheal intubation and mechanical ventilation if required.
4. To maintain circulation:
   - Chest compression
   - Medication.



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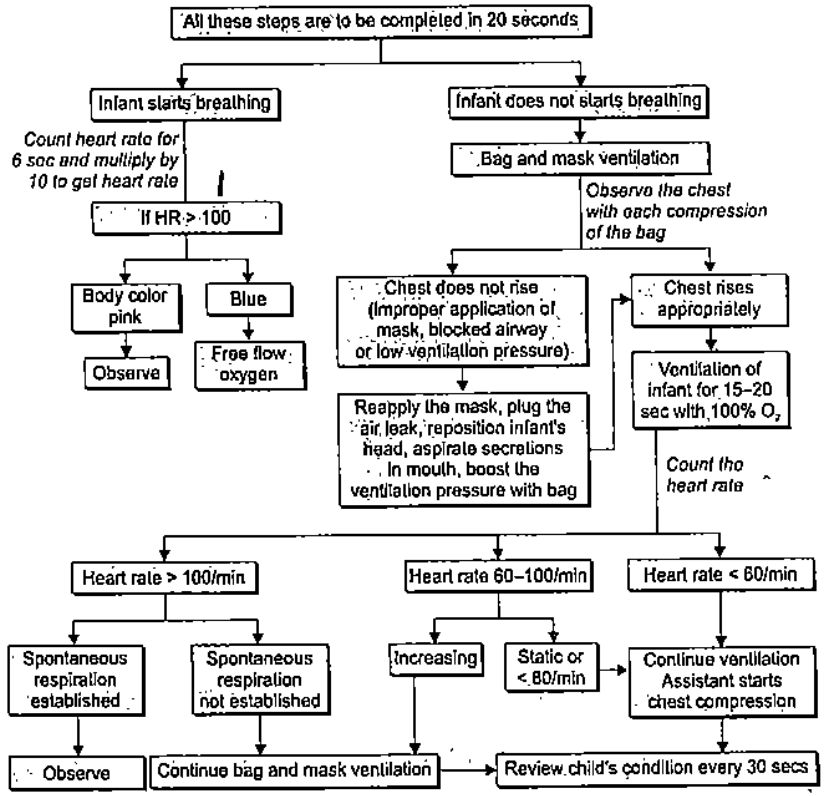

Fig. 2: Protocol for resuscitation

Note: Never slap the back/squeeze the rib cage/force the thighs on abdomen/dilate anal sphincter.

- If ventilation is required for >2 minutes put an orogastric tube to relieve abdominal distension
- If IPPR and chest compression fails, do endotracheal intubation with IPPR and give medications if required:

Chest Compression

- Firm support at back
- Done with 2 fingers (index and middle) or balls of two thumbs over the lower third of sternum area just below a line drawn between nipples
- Compression of 1to 1.5 cm
- Rate 120/min
- Condition reviewed every 30 secs.

Heart rate <80/min	Heart rate >80/min
↓	↓
Continue chest compression	Discontinue chest compression, continue bag and mask ventilation with 100% O_2

Endotracheal Intubation

Indications

1. Prolonged IPPR is required .
2. Bag and mask is ineffective
3. Tracheal suction is required: **Meconium aspiration**
4. **Diaphragmatic hernia (bag and mask ventilation is contraindicated here).**

- Laryngoscope inserted with left hand beyond base of tongue to rest in vallecula and blade is lifted straight up (not tilted up) to visualize epiglottis and glottis. Suction if required to visualize glottis
- Insert the endotracheal tube (ETT) to a length of 4 cm into the glottis
- Resuscitation bag attached to ETT. Baby ventilated with 100% O_2. Auscultate for breath sounds
- Confirm proper position
- If breath sounds only on one side, withdraw by 1 cm and check again
- If no breath sounds, tube is in esophagus, therefore reinsert the tube.

Medications

- Epinephrine hydrochloride: 1 in 10,000 solution, 0.1–0.3 ml/kg IV or through ETT if HR <80/min despite 30 sec ventilation with 100% O_2 and chest compression
  If HR = 0 give immediately
  Repeat after 5 mins if required
- Volume expanders: 10 ml/kg IV over 5–10 mins if acute blood loss
- Dopamine hydrochloride: If poor peripheral perfusion/thready pulse or shock state. Rate, 5 microgram/kg/min in an IV drip. (This increases BP and stabilizes the heart rate)
- Naloxone hydrochloride: If mother was administered narcotics (<4 hr) and baby has severe respiratory depression: 0.1 mg/ml/kg IV, IM or via ETT
- $NaHCO_3$: 4.2% or half diluted 7.5% or 8.4%. Rate 2 mEq./kg IV slowly over 2 mins only in cases of documented metabolic acidosis.

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