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КУРС ЛЕКЦИЙ ПО АКУШЕРСТВУ

(для студентов с английским языком обучения)

Учебное пособие

COURSE OF LECTURES ON OBSTETRICS

(for students with English language of instruction)

Manual

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Пособие является конспектом лекций для самоподготовки к практическим занятиям студентов с английским языком обучения. В учебное пособие включены основные лекции по акушерству, входящие в обязательную программу для студентов 4 курсов медицинских вузов, клинических ординаторов и интернов с английским языком обучения.

Представлены наиболее актуальные проблемы акушерства и перинатологии. Изложение лекционного материала способствует самоконтролю учащихся в процессе обучения и более глубокому усвоению материала, особенно иностранными студентами, изучающими дисциплину «Акушерство» на английском языке.

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LESSON PLAN

Structure and functions of the reproductive system.

Fertilization. The structure and functions of the placenta.

Physiological changes during pregnancy.

Normal Labor Assessment of progress of labor and partography recording (first, second, third stages of the labor).

Multiple Pregnancy.

Hypertensive Disorders in Pregnancy.

Extragenital pathology and pregnancy.

Preterm labor.

Postterm pregnancy.

Premature Rupture of Membranes.

Abnormal Uterine Action.

Face and brow presentation.

Hemorrhage in Early Pregnancy.

Antepartum Hemorrhage.

Injuries to the Birth Canal.

Puerperal Sepsis.

Cesarean section.

STRUCTURE AND FUNCTIONS OF THE REPRODUCTIVE SYSTEM

Functions of the reproductive system:

- hormonal;
- menstrual;
- reproductive, prolongation of the genus.

The menstrual cycle. This is a regular natural change that occurs in the female reproductive system (especially in the uterus and ovaries), which makes pregnancy possible. The cycle is necessary for the production of oocytes and for the preparation of the uterus for pregnancy. The menstrual cycle occurs due to changes in the level of hormones. This cycle leads to a thickening of the functional layer of the uterine mucosa and the growth of the egg (which is necessary for pregnancy). The egg leaves the ovary on the fourteenth day of the cycle; the functional layer of the uterine mucosa provides nutrition to the embryo after implantation. If pregnancy does not occur, the functional layer of the endometrium is rejected, which is called ***menstruation.***

The characteristics of a normal menstrual cycle are:

- the presence of two-phase;
- the duration of menstruation is from 2–7 days;
- the cycle has a constant duration from 21 to 35 days;
- blood loss during menstruation is not more than 100 ml;
- absence of pain syndrome and violation of the general state of the body.

Pituitary hormones

1) anterior lobe

- **FSH**
- **LH**
- **Prolactin**
- **TSH**
- **ACTH**
- **STH**

2) posterior lobe

- **Oxytocin**
- **Vasopressin**

FSH:

- anterior lobe of the pituitary;
- increase in the first half of the menstrual cycle;
- increase estrogen levels (negative feedback);
- the growth of follicles in ovary;
- the proliferation of cells granuleza in follicles;
- synthesis of the aromatase-of the enzyme, turning the androgens in the estrogens in the ovaries;
- synthesis of estradiol in the ovary;
- synthesis receptors LH in cells preovulatory of the follicle.

LH:

- at mid-cycle initiating ovulation if the follicle is already primed by estrogen;
- ovulation takes place 36 hours after the LH surge;
- Synthesis of androgens in theca cells of follicles;
- luteinization granuleza cells;
- synthesis of progesterone in the corpus luteum.

Prolactin:

- is necessary for the lactation process;
- it may affect the formation of progesterone and the maintenance of the corpus luteum;
- in the uterus increases the number of progesterone receptors, promotes the implantation process;
- increases the sensitivity of the receptors to LH and FSH;
- stimulates the synthesis of insulin;
- stimulates lipogenesis;
- it affects appetite, emotional behavioral reactions, increases aggressiveness, lowers libido.

TSH:

- synthesis of $T_{4,}$ $T_{3;}$

- stimulates the growth and development of the body, the growth and differentiation of tissues;
- increases the need of tissues for oxygen;
- ↑ systemic BP;
- ↑ frequency and strength of heart contractions;
- ↑ the motor activity;
- increases body temperature, ↑ level of basal metabolism;
- prepares the ovaries for ovulation;
- controls the growth of uterine mucosa (endometrium), ready to accept the embryo.

ACTH (corticotropin):

- controls the synthesis and secretion of hormones of the adrenal cortex;
- synthesis and secretion of glucocorticoids – cortisol, cortisone, corticosterone;
- synthesis of progesterone, androgens and estrogens by the adrenal glands.

STH:

- the growth of long tubular bones of the limbs;
- anabolic and anti – catabolic effect;
- ↑ synthesis protein and inhibits its breakdown;
- helps to reduce the deposition of subcutaneous fat;
- increase the ratio of muscle mass to fa.t

Functions of the Ovaries

- Production of a mature oocyte, capable of fertilization and embryonic development.
- Production of ovarian steroids (estradiol, progesterone, androgens).
- Production of gonadal peptides (inhibin, activin).

Ovarian cycle:	Uterine cycle:
✓ follicular phase	✓ menses (desquamation)
✓ ovulation	✓ proliferative phase
✓ luteal phase	✓ secretory phase

The structure of the female reproductive system is shown in Figure 1

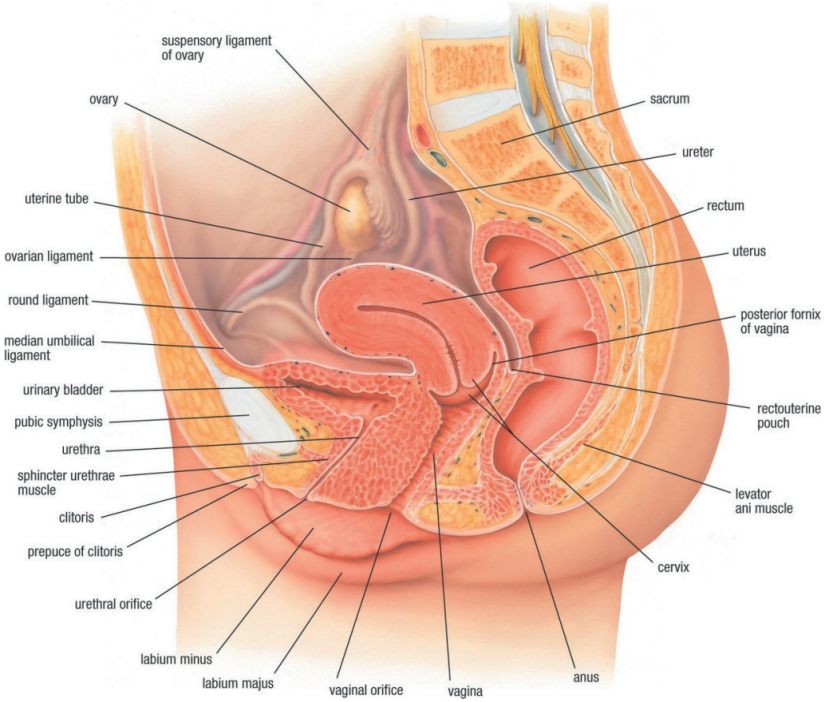


Fig. 1. Female urogenital system (midsaggital vew)

THE UTERUS

Uterine Wall Consists of 3 Layers:

- Myometrium – outer muscular layer.
- Endometrium – a thin, inner, glandular mucosa.
- Perimetrium – an incomplete serosa continuous with the peritoneum.

Uterine endometrium has two layers:

- functional layer: built up and shed each cycle;
- basal layer.

Changes in the endometrium during the uterine cycle are shown in Figure 2

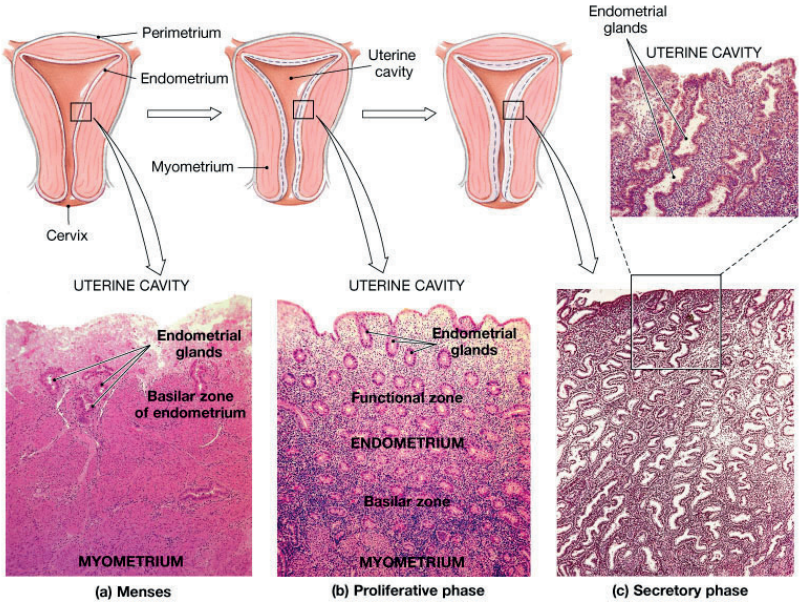


Fig. 2. The uterine cycle

Estrogens:

- stimulate growth of the vagina, uterus and oviducts in childhood;
- increase the thickness of the vaginal wall and distal one-third of the urethra by increased stratification of the epithelium;
- reduce vaginal pH by the action of the Doderlein's bacillus on the glycogen to form lactic acid;
- decrease viscosity of cervical mucus to facilitate sperm penetration;
- facilitate the development of primordial follicles;
- inhibit follicle stimulating hormone (FSH) secretion;
- stimulate proliferation of the endometrium;
- increase myometrial contractility;

- stimulate growth of breasts with duct proliferation;
- promote calcification of bone;
- promote female fat distribution;
- promote female hair distribution;
- metabolized by the liver and conjugated with glucuronic acid so that 65% is excreted in urine.

Progesterone

This hormone is produced by the corpus luteum in large amounts following ovulation and by the placenta in pregnancy.

Its functions are to:

- induce endometrial secretory changes;
- increase the growth of the myometrium in pregnancy;
- decrease myometrial activity in pregnancy;
- increase secretory activity in the uterine tubes;
- decrease motility of the uterine tubes;
- increase the glandular activity in the breasts;
- metabolized in the liver, 80% becomes pregnanediol.

The androgens – testosterone:

- the atrophy glandular tissue mammary glands;
- the atrophy of endometrium and myometrium;
- stimulation of atresia of follicles in the ovary;
- suppression of the secretion gonadotrop hormones of the pituitary;
- strengthening synthesis of osteoblasts, the growth of epifizar zones of bones;
- anabolic action (protein synthesis, increase muscle mass;)
- stimulation of growth rod hair in the androgen dependent and independent zones;
- strengthening of secretion sebaceous glands;
- delay fluid and electrolytes;
- the regulation of sexual behavior (libido).

FERTILIZATION. THE STRUCTURE AND FUNCTIONS OF THE PLACENTA

The results of fertilization:

- Stimulates the secondary oocyte to complete meiosis.
- Restores the normal diploid number of chromosomes (46).
- Results in variation of human species as maternal and paternal chromosomes intermingle.
- The embryo contains only maternal mitochondria because the sperm mitochondria are dispersed into the egg cytoplasm and discarded.
- Determines the sex of the embryo.
- The sex chromosome (Y or X) carried by the successful sperm determines embryonic sex.

Placenta (The placenta is characterized in Figure 3).

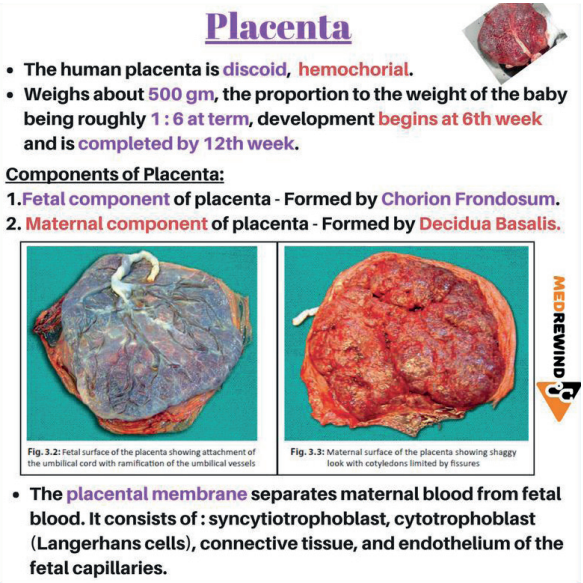


Fig. 3. Placenta

Placental function

1. Transfer of nutrients and waste products between the mother and fetus. In this respect it attributes to the following functions:

- Respiratory.
- Excretory.
- Nutritive.

2. Endocrine function: Placenta is an endocrine gland. It produces both steroid and peptide hormones to maintain pregnancy
3. Barrier function.
4. Immunological function.

PHYSIOLOGICAL CHANGES DURING PREGNANCY

GENITAL ORGANS

VULVA: Vulva becomes edematous and more vascular; superficial varicosities may appear specially in multiparae. Labia minora are pigmented and hypertrophied.

VAGINA: Vaginal walls becomes hypertrophied, edematous and more vascular.

UTERUS: There is enormous growth of the uterus during pregnancy. The uterus which in nonpregnant state weighs about 60 gm, with a cavity of 5–10 mL and measures about 7.5 cm in length, at term, weighs 900–1000 g and measures 35 cm in length.

ISTHMUS: During the first trimester, isthmus hypertrophies and elongates to about 3 times its original length. It becomes softer. With advancing pregnancy beyond 12 weeks, it progressively unfolds from above, downwards until it is incorporated into the uterine cavity.

CERVIX: There is hypertrophy and hyperplasia of the elastic and connective tissues.

FALLOPIAN TUBE: As the uterine end rises up and the fimbrial end is held up by the infundibulo-pelvic ligament, it is placed almost vertical by the side of the uterus.

OVARY: The growth and function of the corpus luteum reaches its maximum at 8th week when it measures about 2.5 cm and becomes cystic. It looks bright orange, later on becomes yellow and finally pale. Regression occurs following decline in the secretion of human chorionic gonadotropin (hCG) from the placenta.

BREASTS: The nipples become larger, erectile and deeply pigmented. Variable number of sebaceous glands (5–15) which remain invisible in the nonpregnant state in the areola, become hypertrophied and are called Montgomery's tubercles.

CUTANEOUS CHANGES:

Face (chloasma gravidarum or pregnancy mask): It is an extreme form of pigmentation around the cheek, forehead and around the eyes. It may be patchy or diffuse; disappears spontaneously after delivery.

Abdomen Linea nigra: It is a brownish black pigmented area in the midline stretching from the xiphisternum to the symphysis pubis. Striae gravidarum: These stretch marks represent the scar tissues in the deeper layer of the cutis.

WEIGHT GAIN

Reproductive weight gain: 6 kg

- Fetus – 3.3 kg, placenta – 0.6 kg and liquor – 0.8 kg
- Uterus – 0.9 kg and breasts – 0.4 kg
- Accumulation of fat (mainly) and protein – 3.5 kg

Net maternal weight gain: 6 kg

- Increase in blood volume – 1.3 kg
- Increase in extracellular fluid – 1.2 kg

HEMATOLOGICAL CHANGES

- *Blood volume* is markedly raised during pregnancy maximum 40–50% above the nonpregnant level at 30–32 weeks.
- *Plazma volume*: the maximum is reached to the extent of 50%. Total plasma volume increases to the extent of 1.25 liters.
- *The RBC mass* is increased to the extent of 20–30%. The disproportionate increase in plasma and RBC volume produces a state of hemodilution (fall in hematocrit) during pregnancy.
- *Neutrophilic leucocytosis* occurs to the extent of 8000/mm3 and even to 20,000/cu.mm in labor.
- *Total plasma:* protein increases from the normal 180 g (non-pregnant) to 230 g at term.
- *Blood coagulation factors*: Pregnancy is a hypercoagulable state. Fibrinogen level is raised by 50% from 200–400 mg/dL in nonpregnant to 300–600 mg/dL in pregnancy.

CARDIOVASCULAR SYSTEM

- the heart is pushed upwards and outwards with slight rotation to left;
- the apex beat is shifted to the 4th intercostal space about 2.5 cm outside the midclavicular line. Pulse rate is slightly raised, often with extrasystoles;
- a systolic murmur may be audible in the apical or pulmonary area;
- a continuous hissing murmur may be audible over the tricuspid area in the left second and third intercostal spaces called the "mammary murmur";
- blood pressure: there is overall decrease in diastolic blood pressure (BP) and mean arterial pressure (MAP) by 5–10 mm Hg;
- venous pressure: it is due to pressure exerted by the gravid uterus on the common iliac veins, more on the right side due to dextrorotation of the uterus.

GENERAL METABOLIC CHANGES: Total metabolism is increased due to the needs of the growing fetus and the uterus. Basal metabolic rate is increased to the extent of 30% higher than that of the average for the nonpregnant women.

- *Protein metabolism:* there is a positive nitrogenous balance throughout pregnancy. As the breakdown of amino acid to urea is suppressed, the blood urea level falls to 15–20 mg %.
- *Carbohydrate metabolism:* insulin secretion is increased, sensitivity of insulin receptors is decreased (44%), plasma insulin level is increased. This mechanism ensures continuous supply of glucose to the fetus.
- *Fat metabolism:* an average of 3–4 kg of fat is stored during pregnancy mostly in the abdominal wall, breasts, hips and thighs. Plasma lipids and lipoproteins increase appreciably during the later half of pregnancy due to increased estrogen, progesterone, hPL and leptin levels.
- *Lipid metabolism:* HDL level increases by 15%. LDL is utilized for placental steroid synthesis. This hyperlipidemia of normal pregnancy is not atherogenic.

- *Iron metabolism:* Total iron requirement during pregnancy is estimated approximately 1000 mg. This iron need is not squarely distributed throughout the pregnancy but mostly limited to the third trimester. Thus, in the second half, the daily requirement, actually becomes very much increased to the extent of about 6–7 mg. In the absence of iron supplementation, there is drop in hemoglobin, serum iron and serum ferritin concentration at term pregnancy. Thus pregnancy is an inevitable iron deficiency state.

SYSTEMIC CHANGES

Respiratory system: a state of hyperventilation occurs during pregnancy leading to increase in tidal volume and therefore respiratory minute volume by 40%. It is probably due to progesterone acting on the respiratory center and also to increase in sensitivity of the center to carbon dioxide. The woman feels shortness of breath. Pregnancy is in a state of respiratory alkalosis.

Urinary system:

- Kidney – There is dilatation of the ureter, renal pelvis and the calyces. The kidneys enlarge in length by 1 cm. Renal plasma flow is increased by 50–75%, maximum by the 16 weeks and is maintained until 34 weeks.
- Ureters become atonic; the stasis is marked between 20–24 weeks.
- Bladder: due to resetting of osmoregulation causing increased water intake and polyuria. In late pregnancy, frequency of micturition once more reappears.

Alimentary system:

- *Muscle tone and motility* of the entire gastrointestinal tract are diminished due to high progesterone level. Cardiac sphincter is relaxed. Risk of peptic ulcer disease is reduced.
- *Liver and gallbladder:* With the exception of alkaline phosphatase, other liver function tests (serum levels of bilirubin, AST, ALT, CPK, LDH) are unchanged.

Nervous system: postpartum blues, depression or "psychosis may develop in a susceptible individual.

Calcium metabolism and locomotor system: Daily requirement of calcium during pregnancy and lactation averages 1–1.5 g. Maternal total calcium levels fall but serum ionized calcium level is unchanged.

Endocrine system:

- *THE THYROID:* the serum protein bound iodine is increased in pregnancy, Thyroxin binding globulin (TBG) increases, thyroid hormones (T4) and TSH cross very minimally. Since the increase in TBG is dependent on estrogens, a failure of the PBI to rise may also indicate fetal compromise.
- *THE ADRENAL CORTEX*: there is significant increase in the serum levels of aldosterone, deoxycorticosterone (DOC), corticosteroid binding globulin (CBG), cortisol and free cortisol.
- *THE PARATHYROID GLAND:* PTH does not cross the placenta but the calcium ions do cross against a concentration gradient.
- *THE PANCREAS:* several anti-insulin factors and tissue insulin resistance modify the action of insulin during pregnancy.

NORMAL LABOR

Definition: Series of events that take place in the genital organs in an effort to expel the viable products of conception out of the womb through the vagina into the outer world is called Labor.

Delivery is the expulsion or extraction of a viable fetus out of the womb. It is not synonymous with labor; delivery can take place without labor as in elective cesarean section. Delivery may be vaginal, either spontaneous or aided or it may be abdominal.

Normal labor (eutocia): Labor is called normal if it fulfils the following criteria

- Spontaneous in onset and at term.
- With vertex presentation.
- Without undue prolongation.
- Natural termination with minimal aids.
- Without having any complications affecting the health of the mother and/or the baby.

Causes of onset of labor

Uterine distension: Stretching effect on the myometrium by the growing fetus and liquor amnii can explain the onset of labor at least in twins or polyhydramnios. Uterine stretch increases gap junction proteins, receptors for oxytocin and specific contraction associated proteins (CAPS).

- Fetoplacental contribution: Cascade of events activate *fetal hypothalamic pituitary adrenal axis* prior to onset of labor → increased CRH → increased release of ACTH → fetal adrenals → increased cortisol secretion → accelerated production of estrogen and prostaglandins from the placenta.
- Estrogen – the probable mechanisms are:

– *Increases* the release of oxytocin from maternal pituitary.

– *Promotes* the synthesis of myometrial receptors for oxytocin (by 100–200 folds), prostaglandins and increase in gap junctions in myometrial cells.

– *Accelerates* lysosomal disintegration in the decidual and amnion cells resulting in increased prostaglandin (PGF2α) synthesis.

– Stimulates the synthesis of myometrial contractile protein–actomyosin through cAMP.

– *Increases* the excitability of the myometrial cell membranes.

- Progesterone: Increased fetal production of dehydroepiandrosterone sulfate (DHEA-S) and cortisol inhibits the conversion of fetal pregnenolone to progesterone. Progesterone levels therefore fall before labor. *It is the alteration* in the estrogen: progesterone ratio rather than the fall in the absolute concentration of progesterone which is linked with prostaglandin synthesis.
- Prostaglandins: Prostaglandins are the important factors which initiate and maintain labor. *The major sites of synthesis of prostaglandins are*–amnion, chorion, decidual cells and myometrium. *Synthesis is triggered by*–rise in estrogen level, glucocorticoids, mechanical stretching in late pregnancy, increase in cytokines (IL–1, 6, TNF), infection, vaginal examination, separation or rupture of the membranes. Prostaglandins enhance gap junction (intermembranous gap between two cells through which stimulus flows) formation.

Biochemical mechanisms involved in the synthesis of prostaglandins

Phospholipase A2 in the lysosomes of the fetal membranes near term → esterified arachidonic acid → formation of free arachidonic acid → synthesis of prostaglandins through prostaglandin synthetase. Prostaglandins (E2 and F2α) diffuse in the myometrium → act directly at the sarcoplasmic reticulum → inhibit intracellular cAMP generation → increase local free calcium ions → uterine contraction. Once the arachidonic acid cascade is initiated, prostaglandins themselves

will activate lysosomal enzyme systems. *The prostaglandin synthesis reaches a peak* during the birth of placenta probably contributing to its expulsion and to the control of postpartum hemorrhage.

Contractile system of the myometrium

The basic elements involved in the uterine contractile systems are –
(a) actin
(b) myosin
(c) adenosine triphosphate (ATP)
(d) the enzyme myosin light chain kinase (MLCK)
(e) Ca++

Structural unit of a myometrial cell is myofibril which contains the *proteins–actin and myosin.* The interaction of myosin and actin is essential for muscle contraction. The key process in actin–myosin interaction is myosin light chain phosphorylation. This reaction is controlled by *myosin light chain kinase (MLCK).* Oxytocin acts on myometrial receptors and activates phospholipase C → increases intracellular calcium level. Calcium is essential for the activation of MLCK and binds to the kinase as *calmodulin – calcium* complex.

Intracellular calcium levels are regulated by two general mechanisms: (a) Influx across the cell membrane and

(b) Release from intracellular storage sites. Calcium is stored within the cells in the sarcoplasmic reticulum and in mitochondria. Progesterone and cAMP promote calcium storage at these sites. PGF2α, E2 and oxytocin on the other hand stimulate its release.

- Intracellular Ca++ → Calmodulin Ca++ → MLCK → Phosphorylated Myosin + Actin → Myometrial contraction.
- Decease of intracellular Ca++ (or its shift to the storage sites) → dephosphorylation of myosin light chain → inactivation of myosin light chain kinase → Myometrial relaxation.

Uterine muscles have two types of adrenergic receptors – (a) α receptors, which on stimulation produce a decrease in cyclic AMP (adenosine monophosphate) and result in contraction of the uterus and (b) β receptors, which on stimulation produce rise in cyclic AMP and result in inhibition of uterine contraction.

PRELABOR *(Syn: premonitory stage)*: The premonitory stage may begin 2–3 weeks before the onset of true labor in primigravidae and a few days before in multiparae.

True labor pains are characterized by:

- Uterine contractions at regular intervals.
- Frequency of contractions increase gradually.
- Intensity and duration of contractions increase progressively.
- Associated with "show".
- Progressive effacement and dilatation of the cervix.
- Descent of the presenting part.
- Formation of the "bag of forewaters".
- Not relieved by enema or sedatives.

PHYSIOLOGY OF NORMAL LABOR

Tonus*: It is the intrauterine pressure in between contractions.* During pregnancy, as the uterus is quiescent (inactive), the tonus is of 2–3 mm Hg. During the first stage of labor, it varies from 8–10 mm Hg. It is inversely proportional to relaxation.*The factors which govern the tonus are:*

- Contractility of uterine muscles.
- Intra-abdominal pressure.
- Overdistension of uterus as in twins and hydramnios.

Intensity: *The intensity of uterine contraction describes the degree of uterine systole.* The intensity gradually increases with advancement of labor until it becomes maximum in the second stage during delivery of the baby. Intensity is initially influenced probably by hormones but subsequently depend on multiple origin of contractions. *Intrauterine pressure is raised to 40–50 mm Hg during first stage and about 100–120 mm Hg in second stage of labor during contractions. In spite of diminished pain in third stage, the intrauterine pressure is probably the same as that in the second stage.* The diminished pain is due to lack of stretching effect.

Duration: In the first stage, the contractions last for about 30 seconds initially but gradually increase in duration with the progress of labor.

Thus, in the second stage, the contractions last longer than in the first stage.

Frequency: In the early stage of labor, the contractions come at intervals of 10–15 minutes. The intervals gradually shorten with advancement of labor until in the second stage, when it comes every 2–3 minutes.

It is important to note that all the features of uterine contractions mentioned are very effective only when they are in combination.

LOWER SEGMENT OF UTERUS AND THE CLINICAL SIGNIFICANCE	
Anatomical features	*Clinical significance*
1. It is developed from the isthmus of the (nonpregnant) uterus which is bounded above anatomical and below by histological internal os. 2. In labor it is bounded above by the physiological retraction ring and below by the fibro muscular junction of cervix and uterus. 3. This segment is formed maximally during labor and the peritoneum is loosely attached anteriorly. 4. It measures 7.5–10 cm when fully formed and becomes cylindrical during the second stage of labor. 5. The wall becomes gradually thin due to: (I) Relaxation of the muscle fibers to allow elongation. (II) The muscle fibers are drawn up by the muscle fibers of the upper uterine segment by contraction and retraction during labor (III). Descent of the presenting part causes further stretching and thinning out of all. 6. This segment has got poor retractile property compared to the upper segment.	1. The phenomenon of receptive relaxation enables expulsion of the fetus by formation of complete birth canal along with the fully dilated cervix. 2. Implantation of placenta in lower segment is known as placenta previa. 3. It is through this segment that cesarean section is performed. 4. Poor decidual reaction in this segment facilitates morbid adherent placenta, once the placenta is implanted here. 5. In obstructed labor, the lower segment is very much stretched and thinned out and ultimately gives way (ruptures) specially in multiparae. 6. It is entirely the passive segment of the uterus. Because of poor retractile property, there is chance of postpartum hemorrhage if placenta is implanted over the area.

STAGES OF LABOR: ***Conventionally, events of labor are divided into three stages:***

- *First stage.*
- *Second stage.*
- *Third stage.*
- *Fourth stage*: It is the stage of observation for at least 1 hour after expulsion of the after-births. During this period, general condition of the patient and the behavior of the uterus are to be carefully monitored.

First stage of labor

First stage of labor starts from the onset of true labor pain and ends with full dilatation of the cervix. Its average duration is about 12 hours in primigravidae and 6 hours in multiparae. First stage consists of *latent phase* (upto 3 cm of cervical dilatation) and *active phase* (upto 10 cm). The stage is chiefly concerned with dilatation and effacement of the cervix. The stage is clinically manifested by progressive uterine contraction, dilatation and "effacement" of the cervix and ultimate rupture of the membranes. Maternal and fetal conditions remain unaffected except during uterine contraction.

Management consists of:

(1) non-interference with watchful expectancy

(2) women are given encouragement, emotional support and adequate pain relief during the entire course of labor

(3) to monitor carefully the progress of labor, maternal condition and fetal behavior so as to detect any deviation from the normal

(4) partograph is maintained.

ASSESSMENT OF PROGRESS OF LABOR AND PARTOGRAPH RECORDING

Pulse is recorded every 30 minutes and is marked with a dot (.) in the partograph. Blood pressure is recorded at every 1 hours and is marked with arrows (↔) Temperature is recorded every 2 hours.

Urine ouput is recorded for volume, protein or acetone. Any drug (oxytocin or other) when given is recorded in the partograph.

Abdominal palpation — (a) Uterine contractions: 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 as:

To note the fetal well being

Fetal heart rate (FHR) along with its rhythm and intensity should be 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. For routine clinical observation, ordinary stethoscope is quite suitable. Doppler ultrasonic cardiography (Dopplex), however, is helpful in the case of obesity and polyhydramnios. To avoid confusion of maternal and fetal heart rates, maternal pulse should be counted. Otherwise maternal tachycardia may be wrongly treated as fetal heart rate. Normal fetal heart rate ranges from 110–150 per minute.

Second stage of labor

The second stage of labor starts from full dilatation of the cervix and ends with expulsion of the fetus. Its average duration is two hours in primigravidae and 30 minutes in multiparae. The stage concerns with the descent and delivery of the fetus through the birth canal (see Fig. 4.). The stage is clinically manifested by increased frequency and

intensity of uterine contractions with appearance of "bearing down" efforts which result in expulsion of the fetus. The mother may show features of exhaustion.

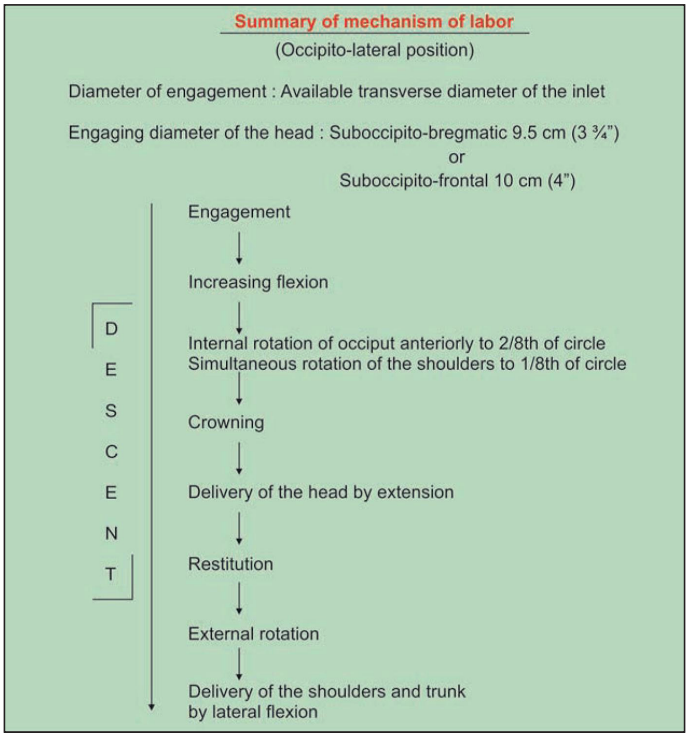


Fig. 4. Summary mechanism of labor

The principles in management are:

(1) To assist in the natural expulsion of the fetus slowly and steadily

(2) To prevent perineal injuries. During conduction of delivery, head is delivered slowly in between contractions. Flexion is maintained although so that smaller diameter of the head stretches the perineum. This, along with timely performed episiotomy (selective) prevents

perineal laceration. Shoulders are delivered slowly with next contraction.

Immediate care of the newborn includes clearing of the air passage and eyes, clamping and ligaturing of the umbilical cord and *Apgar* scoring.

Third stage of labor

The third stage begins after the expulsion of the fetus and ends with expulsion of the placenta and membranes. Its average duration is 15 minutes. The stage concerns with placental separation and its expulsion. The separation is achieved by marked reduction in the uterine surface area of the placental site following delivery due to retraction (see Fig. 5.).

The placenta being inelastic shears off its attachment through the deep spongy decidual layer. There are two ways of separation–central (Schultze) and marginal (Mathews-Duncan). The bleeding is controlled by effective myometrial contraction and retraction (living ligature) and by thrombosis. The expulsion may occur through "bearing down" efforts or more commonly, with assistance. The management is either by employing watchful expectancy or by active management (WHO) in cases where oxytocin 10 units IV (slowly) or IM/methergin 0.2 mg IV is administered within one minute following the delivery of the baby. The placenta and the membranes should be examined following their expulsion.

The third stage of labor comprises the phase of placental separation; its descent to the lower segment and finally its expulsion with the membranes.

- Central separation (Schultze): Detachment of placenta from its uterine attachment starts at the center resulting in opening up of few uterine sinuses and accumulation of blood behind the placenta (retroplacental hematoma). With increasing contraction, more and more detachment occurs facilitated

by weight of the placenta and retroplacental blood until whole of the placenta gets detached.

- Marginal separation (Mathews-Duncan):

Separation starts at the margin as it is mostly unsupported. With progressive uterine contraction, more and more areas of the placenta get separated. Marginal separation is found more frequently.

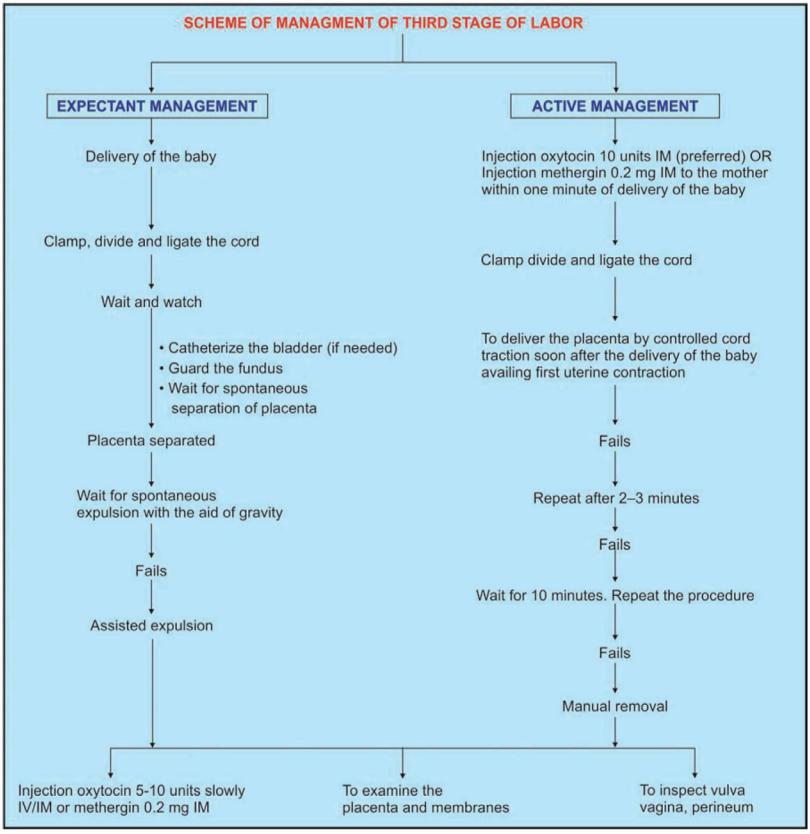


Fig. 5. Third stage of labor

MULTIPLE PREGNANCY

When more than one fetus simultaneously develops in the uterus, it is called multiple pregnancy.

Simultaneous development of two fetuses (twins) is the commonest; although rare, development of three fetuses (triplets), four fetuses (quadruplets), five fetuses (quintuplets) or six fetuses (sextuplets) may also occur.

Incidence. The incidence varies widely. It is highest in Nigeria being 1 in 20 and lowest in Far Eastern countries being 1 in 200 pregnancies. In India, the incidence is about 1 in 80. While the incidence of monozygotic twins remains fairly constant throughout the globe being 1 in 250, it is the dizygotic twins which are responsible for the wide variation of the incidence.

Etiology:

Prevalence of dizygotic twins is related to:

Race: The frequency is highest amongst Negroes, lowest amongst Mongols and intermediate amongst Caucasians.

Hereditary: There is hereditary predisposition likely to be more transmitted through the female (maternal side).

Advancing age of the mother: Increased incidence of twinning is observed with the advancing age of the mother, the maximum being between the age of 30–35 years. The incidence of twins is markedly reduced thereafter.

Influence of parity: The incidence is increased with increasing parity specially from 5th gravida onwards.

Iatrogenic: Drugs used for induction of ovulation may produce multiple fetuses to the extent of 20–40% following gonadotrophin therapy, although to a lesser extent (5–6%) following clomiphene citrate.

- Superfecundation is the fertilization of two different ova released in the same cycle, by separate acts of coitus within a short period of time.
- Superfetation is the fertilization of two ova released in different menstrual cycles. The nidation and development of one fetus over another fetus is theoretically possible until the decidual space is obliterated by 12 weeks of pregnancy.
- Fetus papyraceous or compressus is a state which occurs if one of the fetuses dies early. The dead fetus is flattened, mummified and compressed between the membranes of the living fetus and the uterine wall. It may occur in both varieties of twins, but is more common in monozygotic twins and is discovered at delivery or earlier by sonography.
- Fetus acardiacus occurs only in monozygotic twins. Part of one fetus remains amorphous and becomes parasitic without a heart.
- Hydatidiform mole (from one placenta) and a normal fetus and placenta (from the other conceptus) have been observed ultrasonographically.
- Vanishing twin: Serial ultrasound imaging in multiple pregnancy since early gestation has revealed occasional death of one fetus and continuation of pregnancy with the surviving one. The dead fetus (if within 14 weeks) simply 'vanishes' by resorption. The rate of disappearance could be to the extent of 40%.

Maternal physiological changes: Multiple pregnancy imposes physical changes on the mother in excess of those seen in singleton pregnancy.

- There is increase in weight gain and cardiac output.
- Plasma volume is increased by an addition of 500 mL. There is no corresponding increase in red cell volume resulting in exaggerated hemodilution and anemia.
- There is increased a fetoprotein level, tidal volume and glomerular filtration rate.

Lie and presentation: The mostcommon lie of the fetuses is longitudinal (90%) but malpresentations are quite common.

The combination of presentation of the fetuses are:

1. both vertex (50%);
2. first vertex and Fetus papyraceous or compressus second breech (30%);
3. first breech and second vertex (10%);
4. both breech (10%);
5. first vertex and second transverse and so on, but rarest one, being both transverse when the possibility of conjoined twins should be ruled out.

Diagnosis

History:

- History of ovulation inducing drugs specially gonadotrophins, for infertility or use of ART.
- Family history of twinning (more often present in the maternal side).

Symptoms: Minor ailments of normal pregnancy are often exaggerated. Some of the symptoms are related to the undue enlargement of the uterus:

- Increased nausea and vomiting in early months.
- Cardiorespiratory embarrassment which is evident in the later months – such as palpitation or shortness of breath.
- Tendency of swelling of the legs, varicose veins and hemorrhoids is greater.
- Unusual rate of abdominal enlargement and excessive fetal movements may be noticed by an experienced parous mother.

General examination:

- Prevalence of anemia is more than in singleton pregnancy
- Unusual weight gain, not explained by preeclampsia or obesity, is an important feature
- Evidence of preeclampsia (25%) is a common association.

Abdominal examination:

Inspection: The elongated shape of a normal pregnant uterus is changed to a more "barrel shape" and the abdomen is unduly enlarged.
Palpation:

- The height of the uterus is more than the period of amenorrhea. This discrepancy may only become evident from mid-pregnancy onwards.
- The girth of the abdomen at the level of umbilicus is more than the normal average at term (100 cm).
- Fetal bulk seems disproportionately larger in relation to the size of the fetal head.
- Palpation of too many fetal parts.
- Finding of two fetal heads or three fetal poles make the clinical diagnosis almost certain.
- Auscultation: Simultaneous hearing of two distinct fetal heart sounds located at separate spots with a silent area in between by two observers, gives a certain clue in the diagnosis of twins, provided the difference in heart rates is at least 10 beats per minute. The abdominal palpation and auscultation may not be carried out so easily, as described, because of the presence of hydramnios.

Sonography: In multifetal pregnancy it is done to obtain the following information:

1. Confirmation of diagnosis as early as 10th week of pregnancy.
2. Viability of fetuses, vanishing twin in the second trimester.
3. Chorionicity (lambda or twin peak sign)
4. Pregnancy dating.
5. Fetal anomalies
6. Fetal growth monitoring (at every 3–4 weeks interval) for *IUGR*.
7. Presentation and lie of the fetuses.
8. Twin transfusion (Doppler studies).
9. Placental localization.
10. Amniotic fluid volume.

Prognosis

Maternal mortality is increased in twins than in a singleton pregnancy. Death is mostly due to hemorrhage (before, during and after delivery), preeclampsia and anemia. Increased maternal morbidity is due to the prevalence of complications and increased operative interference.

Perinatal mortality is markedly increased mainly due to prematurity. It is 4–5 times higher than in a singleton pregnancy. It is extremely high in monoamniotic monozygotic twins due to cord entanglement. Onethird loss is due to stillbirth and two-third due to neonatal death.

During delivery the second baby is more at risk (50%) than the first one due to – (1) retraction of uterus leading to placental insufficiency (2) increased operative interference (3) increased incidence of cord prolapse

Complications of monochorionic twins

Twin-twin transfusion syndrome (TTTS) – It is a clinicopathological state, exclusively met with in monozygotic twins, where one twin appears to bleed into the other through some kind of placental vascular anastomosis. Clinical manifestations of twin transfusion syndrome occur when there is hemodynamic imbalance due to unidirectional deep arteriovenous anastomoses. As a result, the receptor twin becomes larger with hydramnios, polycythemic, hypertensive and hypervolemic, at the expense of the donor twin which becomes smaller with oligohydramnios, anemic, hypotensive and hypovolemic.

Management: Antenatal diagnosis is made by ultrasound with doppler blood flow study in the placental vascular bed.

(a) Repeated amniocentesis to control polyhydramnios in the recipient twin is done.

(b) Septostomy (making a hole in the dividing amniotic membrane).

(c) Laser photocoagulation to interrupt the anastomotic vessels on the chorionic plate can give some success.

(d) Selective reduction (feticide) of one twin is done when survival of both the fetuses is at risk.

Dead fetus syndrome – death of one twin (2–7%) is associated with poor outcome of the cotwin (25%) specially in monochorionic placenta.

Twin reversed arterial perfusion (TRAP) is characterized by an 'acardiac perfused twin' having blood supply from a normal cotwin via large arterio-arterial or vein to vein anastomosis.

Monoamniocity (2% of all twins) in monochorionic twins leads to high perinatal mortality due to cord problems (entanglement).

Conjoined twin is rare (1.3 per 100,000 births). Major cardiovascular connection leads to high mortality.

Antenatal management

Advice

- Diet: Increased dietary supplement is needed for increased energy supply to the extent of 300 K cal per day, over and above that needed in a singleton pregnancy. The increased protein demand is to be met with.
- Increased rest at home and early cessation of work is advised to prevent preterm labor and other complications.
- Supplement therapy:

1. Iron therapy is to be increased to the extent of 100–200 mg per day.

2. Additional vitamins, calcium and folic acid (5 mg) are to be given, over and above those prescribed for a singleton pregnancy.

- Interval of antenatal visit should be more frequent to detect at the earliest, the evidences of anemia, preterm labor or preeclampsia.
- Fetal surveillance is maintained by serial sonography at every 3–4 weeks interval.

Hospitalisation

- Routine hospital admission only for bed rest is not essential. However, bed rest even at home from 24 weeks onwards, not only ensures physical and mental rest but also improves uteroplacental circulation.

This results in:

1. increased birth weight of the babies;

2. decreased frequency of pre-eclampsia;

3. prolongation of the duration of pregnancy.

To prevent preterm delivery, routine use of betamimetics or circlage operation has got no significant benefit. Use of corticosteroids to accelerate fetal lung maturation is given (single dose) to women with preterm labor <34 weeks. Twins develop pulmonary maturity 3–4 weeks earlier than singletons. Scheme of management see Fig.6.

- Emergency: Development of complicating factors necessitates urgent admission irrespective of the period of gestation.

Indications of cesarean section:

The indications are broadly divided into:

- Obstetric causes.
- For twins.

Obstetric indication:

(1) Placenta previa

(2) Severe preeclampsia

(3) Previous cesarean section

(4) Cord prolapse of the first baby

(5) Abnormal uterine contractions

(6) Contracted pelvis.

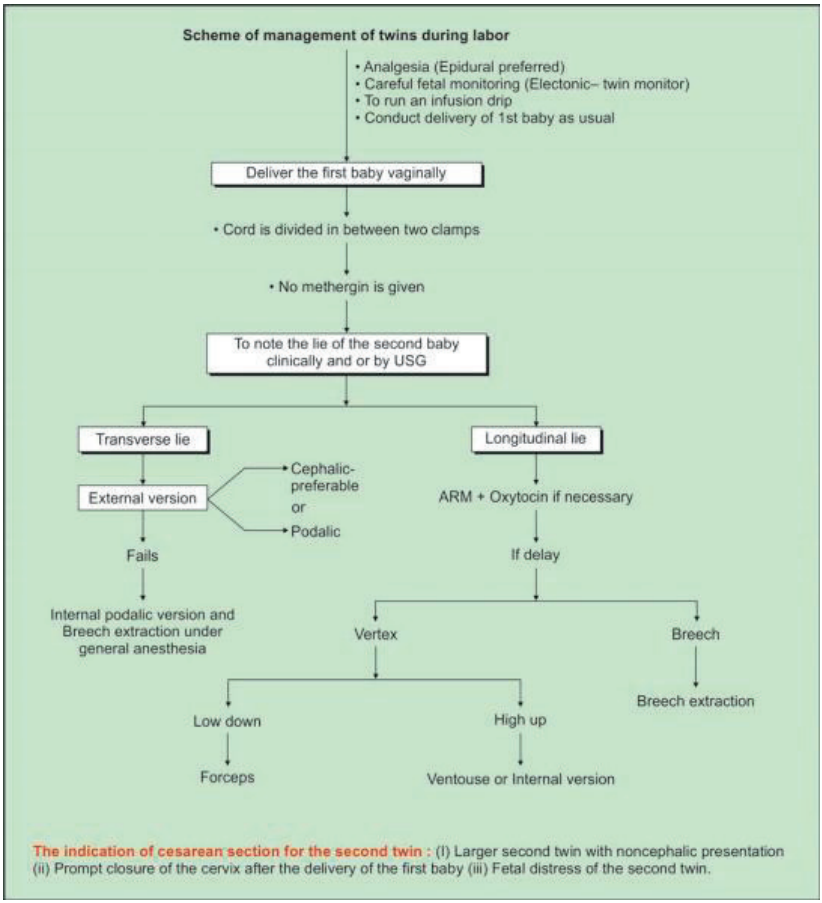


Fig. 6. Management of twins during labor

For twins:

1. Both the fetuses or even the first fetus with noncephalic (breech or transverse) presentation
2. Twins with complications: IUGR, Conjoint twins
3. Monoamniotic twins
4. Monochorionic twins with TTS
5. Collision of both the heads at brim preventing engagement of either head.

HYPERTENSIVE DISORDERS IN PREGNANCY

Hypertension is one of the common medical complications of pregnancy and contributes significantly to maternal and perinatal morbidity and mortality.

Classification in figure 7

Classification of Hypertension in Pregnancy (*National High Blood Pressure Education Program 2000*)

Disorder	Definition	Disorder	Definition
Hypertension	BP ≥ 140/90 mm Hg measured 2 times with at least a 6-hour interval	*Chronic hypertension with super imposed pre-eclampsia and eclampsia*	**The common causes of chronic hypertension:** (a) Essential hypertension (b) Chronic renal disease (reno vascular) (c) Coarctation of aorta (d) Endocrine disorders (diabetes mellitus, pheochromocytoma, thyrotoxicosis (e) Connective tissue diseases (Lupus erythematosus). ♦ **The criteria for diagnosis of super imposed pre-eclampsia:** (i) New onset of proteinuria >0.5 gm/24 hours specimen. (ii) Aggravation of hypertension. (iii) Thrombocytopenia or (iv) Raise of liver enzymes
Proteinuria	Urinary excretion of ≥ 0.3 gm protein/24 hours specimen or 0.1 gm/L		
Gestational hypertension	BP ≥ 140/90 mm Hg for the first time in pregnancy after 20 weeks, without proteinuria		
Pre-eclampsia	Gestational hypertension with proteinuria		
Eclampsia	Women with pre-eclampsia complicated with convulsions and/or coma		
Chronic hypertension	Known hypertension before pregnancy or hypertension diagnosed first time before 20 weeks of pregnancy		
Superimposed pre-eclampsia or eclampsia	Occurrence of new onset of proteinuria in women with chronic hypertension (see below)		

Fig. 7. Classification of Hypertension in pregnancy

Pre-eclampsia

Definition: Pre-eclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/90 mm Hg or more with proteinuria after the 20th week in a previously normotensive and nonproteinuric woman.

Diagnostic criteria of pre-eclampsia

- Hypertension: An absolute rise of blood pressure of at least 140/90 mm Hg.

Calculation based on mean arterial pressure (MAP) as advocated by Page:

Systolic pressure + (diastolic pressure × 2)/ 3 = MAP.

- Edema: Demonstration of pitting edema over the ankles after 12 hours bed rest or rapid gain in weight of more than 1 lb a week or more than 5 lb a month in the later months of pregnancy may be the earliest evidence of pre-eclampsia. However, some amount of edema is common (physiological) in a normal pregnancy.
- Proteinuria: Presence of total protein in 24 hours urine of more than 0.3 gm or >2+ (1.0 gm/L) on at least two random clean-catch urine samples tested > 4 hours apart in the absence of urinary tract infection is considered significant.

Incidence: The incidence of pre-eclampsia in hospital practice varies widely from 5 to 15%. The incidence in primigravidae is about 10% and in multigravidae 5%.

Etiopathogenesis of pre-eclampsia

1. There is an imbalance in different components of prostaglandins–relative or absolute deficiency of vasodilator prostaglandin (PGI2) from vascular endothelium and increased synthesis of thromboxane (TXA2), a potent vasoconstrictor in platelets.
2. There is increased vascular sensitivity to the pressor agent angiotensin-II. Angiotensinase activity is depressed, following proteinuria with elimination of α2 globulin (see scheme for pathophysiology).
3. Nitric oxide (NO): It is synthesized in the vascular endothelium and syncytiotrophoblast from L-arginine. It significantly relaxes vascular smooth muscle, inhibits platelet aggregation and prevents intervillous thrombosis. Deficiency of nitric oxide contributes to the development of hypertension.
4. Endothelin-1 is synthesized by endothelial cells, and it is a potent vasoconstrictor compared to angiotensin-II. Endothelin-1 also contributes to the cause of hypertension.

5. Inflammatory mediators: Cytokines [tumor necrosis factor (TNF-α), interleukins (IL-6) and others] derived from activated leukocytes cause endothelial injury
6. Abnormal lipid metabolism-results in more oxidative stress. Lipid peroxides, reactive oxygen species (ROS) and superoxide anion radicals – cause endothelial injury and dysfunction. Platelet and neutrophil activation, cytokines, superoxide radical production and endothelial damage are in a vicious cycle.
7. Others – mutation of factor V Leiden increases the risk.

Hence pre-eclampsia is characterized by endothelial dysfunction and vasospasm. Endothelial dysfunction is due to oxidative stress and the inflammatory mediators. Vasospasm results from the imbalance of vasodilators (PGI2, NO) and vasoconstrictors (Angiotensin-II, TXA2, Endothelin-1). Both are in a vicious cycle.

EDEMA: The cause of excessive accumulation of fluids in the extracellular tissue spaces is not clear. Probable explanations are: Increased oxidative stress → endothelial injury → increased capillary permeability. On this basis, the leaky capillaries and decreased blood osmotic pressure are the probable explanations.

PROTEINURIA: The probable chain of events is as follows. Spasm of the afferent glomerular arterioles → anoxic change to the endothelium of the glomerular tuft → glomerular endotheliosis → increased capillary permeability → increased leakage of proteins. Tubular reabsorption is simultaneously depressed. Albumin constitutes 50–60% and alpha globulin constitutes 10–15% of the total proteins excreted in the urine.

HELLP Syndrome: This is an acronym for Hemolysis (H), Elevated Liver enzymes (EL) and Low Platelet count (LP) (70 IU/L, LDH >600 IU/L) and bilirubin (>1.2 mg/dL). There may be subcapsular hematoma formation (which is diagnosed by CT scanning) and abnormal peripheral blood smear. Eventually liver may rupture to cause sudden hypotension, due to hemoperitoneum.

CLINICAL TYPES

- Mild: This includes cases of sustained rise of blood pressure of more than 140/90 mm Hg but less than 160 mm Hg systolic or 110 mm Hg diastolic without significant proteinuria.
- Severe:
  - A persistent systolic blood pressure of >160 mm Hg or diastolic pressure of >110 mm Hg.
  - Protein excretion of >5 gm/24 hr.
  - Oliguria (<400 ml/24 hr)
  - Platelet count < 100,000/mm3.
  - HELLP syndrome.
  - Cerebral or visual disturbances.
  - Persistent severe epigastric pain.
  - Retinal hemorrhages, exudates or papilledema.
  - Intrauterine growth restriction of the fetus.
  - Pulmonary edema.

CLINICAL FEATURES

SYMPTOMS: Pre-eclampsia is principally a syndrome of signs and when symptoms appear, it is usually late.

Mild symptoms: Slight swelling over the ankles which persists on rising from the bed in the morning or tightness of the ring on the finger is the early manifestation of pre-eclampsia edema. Gradually, the swelling may extend to the face, abdominal wall, vulva and even the whole body.

Alarming symptoms: The following are the ominous symptoms, which may be evident either singly or in combination. These are usually associated with acute onset of the syndrome.

(1) Headache – either located over the occipital or frontal region

(2) Disturbed sleep,

(3) Diminished urinary output – Urinary output of less than 400 ml in 24 hours is very ominous,

(4) Epigastric pain – acute pain in the epigastric region associated with vomiting, at times coffee color, is due to hemorrhagic gastritis or due to subcapsular hemorrhage in the liver,

(5) Eye symptoms – there may be blurring, scotomata, dimness of vision or at times complete blindness. Vision is usually regained within 4–6 weeks following delivery. The eye symptoms are due to spasm of retinal vessels (retinal infarction), occipital lobe damage (vasogenic edema) or retinal detachment. Reattachment of the retina occurs following subsidence of edema and normalization of blood pressure after delivery.

COMPLICATIONS OF PRE-ECLAMPSIA

Immediate:

1. Maternal

- During pregnancy: (a) Eclampsia (2%) — more in acute than in subacute cases, (b) Accidental hemorrhage, (c) Oliguria and anuria, (d) Dimness of vision and even blindness, (e) Preterm labor, (f) HELLP syndrome, (g) Cerebral hemorrhage, (h) Acute respiratory distress syndrome (ARDS)
- During labor:

(a) Eclampsia, (b) Postpartum hemorrhage – may be related with coagulation failure

- Puerperium: (a) Eclampsia — usually occurs within 48 hours, (b) Shock – puerperal vasomotor collapse is associated with reduced concentration of sodium and chloride due to sudden fall in corticosteroid level (c) Sepsis – due to increased incidence of induction, operative interference, and low vitality.

2. *The fetal* risk is related to the severity of pre-eclampsia, duration of the disease and degree of proteinuria. The following hazards may occur. (a) Intrauterine death—due to spasm of uteroplacental circulation leading to accidental hemorrhage or acute red infarction, (b) Intrauterine growth restriction – due to chronic placental insufficiency,

(c) Asphyxia, (d) Prematurity – either due to spontaneous preterm onset of labor or due to preterm induction.

Remote

- Residual hypertension: It may persist even after 6 months following delivery in about 50% cases. It is more related to familial diathesis and underlying thrombophilias (protein C, protein S deficiency, antiphospholipid syndrome). Microvascular dysfunction due to insulin resistance is also there.
- Recurrent pre-eclampsia: There is 25% chance of pre-eclampsia to recur in subsequent pregnancies. This too is related with familial diathesis, personal predisposition with underlying thrombophilias.
- Chronic renal disease: There is high incidence of glomerulonephritis in women with pre-eclampsia remote from term. This is more likely due to pre-existent underlying renal disease.
- Risk of placental abruption for those women with pre-eclampsia ranges from 5–20 percent and women with HELLP syndrome, the risk of pre-eclampsia in subsequent pregnancy is about 20 percent.

Prognosis: The prognosis of pre-eclampsia depends on the period of gestation, severity of disease and response to treatment.

Maternal mortality: Increased maternal deaths are mainly related to eclampsia, accidental hemorrhage, acute renal failure, pulmonary edema, disseminated intravascular coagulopathy and HELLP syndrome. Though mortality has been reduced significantly in the advanced countries, it still remains high in the developing world.

Perinatal mortality: Although the maternal mortality has been reduced significantly, the perinatal mortality still remains very high even in the developed countries (7–10%). In the developing countries, the perinatal mortality remains to the extent of about 20%, about 50% of which being stillborn.

Prophylactic measures for prevention of preeclampsia

- Regular antenatal check up for early detection of rapid gain in weight or a tendency of rising blood pressure specially the diastolic one.
- Antithrombotic agents: Low dose aspirin 60 mg daily beginning early in pregnancy in potentially high-risk patients is given. It selectively reduces platelet thromboxane production. Aspirin in low doses is known to inhibit cyclo-oxygenase in platelets thereby preventing the formation of thromboxane A2 without interfering with prostacyclin generation.
- Heparin or low molecular weight heparin is useful in women with thrombophilia and with high risk pregnancy.
- Calcium supplementation (2 gm per day) reduces the risk of gestational hypertension.
- Antioxidants, vitamins E and C and nutritional supplementation with magnesium, zinc, fish oil and low salt diet have been tried but are of limited benefit.
- Balanced diet rich in protein may reduce the risk.

Antihypertensives: Antihypertensive drugs have limited value in controlling blood pressure due to preeclampsia (see Fig. 8, Fig. 9.).

The indications are:

(1) Persistent rise of blood pressure specially where the diastolic pressure is over 110 mm Hg. The use is more urgent if associated with proteinuria.

(2) In severe pre-eclampsia to bring down the blood pressure during continued pregnancy and during the period of induction of labor, the common oral drugs used are:

Drug	Mode of action	Dose
• Methyl-dopa	Central and peripheral anti-adrenergic action	250–500 mg tid or qid
• Labetalol	Adrenoceptor antagonist (α and β blockers)	100 mg tid or qid
• Nifedipine	Calcium channel blocker	10–20 mg bid
• Hydralazine	Vascular smooth muscle relaxant	10–25 mg bid

Fig. 8. Antihypertensive drugs

Hypertensive crisis: Any of the following drugs can be used when the BP is >160/110 mm Hg or the mean arterial pressure (MAP) is >125 mm Hg:

Table 17.1

Drug	Onset of action	Dose schedule	Maximum dose	Maintenance dose
Labetalol *	5 min	10–20 mg IV every 10 min	300 mg IV	40 mg/hr
Hydralazine	10 min	5 mg IV every 30 min	30 mg IV	10 mg/hr
Nifedipine	10 min	10–20 mg oral, can be repeated in 30 min	240 mg/24 hr	4–6 hours interval
Nitroglycerin	0.5–5 min	5 µg/min IV	Short-term therapy only when the other drugs have failed (see p. 507)	
Sodium nitroprusside		0.25–5 µg/kg/min IV		

* To avoid labetalol in women having asthma or cardiac failure.

Fig. 9. Antihypertensive drugs

Complete remission of all signs and symptoms is uncommon until after delivery and the underlying disease pathology persists.

Group A: If the duration of pregnancy is remote from term, the patient may be discharged with advice to attend the antenatal clinic after one week. These women are not cured as majority (90%) develop recurrence. If the patient is near term, she should be kept for a few days till completion of 37th week. Thereafter, decision is to be taken either to deliver her or to wait for spontaneous onset of labor by the due date. It is not wise to allow the pregnancy to continue beyond the expected date.

Group B: If the pregnancy is beyond 37 completed weeks, delivery is to be considered without delay. If less than 37 weeks, expectant treatment may be extended judiciously at least up to 34 weeks. Careful maternal and fetal well-being are to be monitored during the period.

Group C: The couple is counseled. Termination of pregnancy (delivery) is considered irrespective of duration of gestation. Seizure prophylaxis (magnesium sulfate) should be started. Steroid therapy is considered if the duration of pregnancy is < 34 weeks. It prevents neonatal RDS, IVH and maternal thrombocytopenia.

Methods of delivery:

- Induction of labor
- Cesarean section

Indications: It is indeed difficult to lay down hard and fast rules for the indications for induction.

(1) Aggravation of the preeclamptic features in spite of medical treatment and/or appearance of newer symptoms such as epigastric pain. (2) Hypertension persists in spite of medical treatment with pregnancy reaching 37 weeks or more.

(3) Acute fulminating pre-eclampsia irrespective of the period of gestation.

(4) Tendency of pregnancy to overrun the expected date.

Methods: If the cervix is ripe, surgical induction by low rupture of the membranes is the method of choice. Oxytocin infusion may be added. If the cervix is unripe, prostaglandin (PGE2) gel 500 μg intracervical or 1–2 mg in the posterior fornix is inserted to make the cervix ripe when low rupture of the membranes can be performed. In severe pre-eclampsia, antihypertensive drugs should be used during induction.

Cesarean section Indications:

(1) When an urgent termination is indicated and the cervix is unfavorable (unripe and closed).

(2) Severe pre-eclampsia with a tendency to prolong the induction–delivery interval.

(3) Associated complicating factors, such as elderly primigravidae, contracted pelvis, malpresentation, etc. The operation should be done by an experienced surgeon with the help of an expert anesthetist. Epidural anesthesia is preferred, unless there is coagulopathy.

Eclampsia – pre-eclampsia when complicated with generalized tonic–clonic convulsions and/or coma is called eclampsia.

Incidence: the hospital incidence in India ranges from 1 in 500 to 1 in 30. It is more common in primigravidae (75%), five times more common in twins than in singleton pregnancies and occurs between the 36th week and term in more than 50%.

Cause of convulsion: The cause of cerebral irritation leading to convulsion is not clear. The irritation may be provoked by:

(1) Anoxia – spasm of the cerebral vessels → increased cerebral vascular resistance → fall in cerebral oxygen consumption → anoxia,

(2) Cerebral edema – may contribute to irritation,

(3) Cerebral dysrhythmia – increases following anoxia or edema. There is excessive release of excitatory neurotransmitters (glutamate).

Clinical features of eclampsia

The fits are epileptiform and consist of four stages.

– *Premonitory stage:* The patient becomes unconscious. There is twitching of the muscles of the face, tongue, and limbs. Eyeballs roll or are turned to one side and become fixed. This stage lasts for about 30 seconds.

– *Tonic stage:* The whole body goes into a tonic spasm – the trunk-opisthotonus, limbs are flexed and hands clenched. Respiration ceases and the tongue protrudes between the teeth. Cyanosis appears. Eyeballs become fixed. This stage lasts for about 30 seconds.

– *Clonic stage:* All the voluntary muscles undergo alternate contraction and relaxation. The twitchings start in the face then involve one side of the extremities and ultimately the whole body is involved in the convulsion. Biting of the tongue occurs. Breathing is stertorous and bloodstained frothy secretions fill the mouth; cyanosis gradually disappears. This stage lasts for 1–4 minutes.

– *Stage of coma:* Following the fit, the patient passes on to the stage of coma. It may last for a brief period or in others deep coma persists till another convulsion. On occasion, the patient appears to be

in a confused state following the fit and fails to remember the happenings. Rarely, the coma occurs without prior convulsion.

Prognosis

Maternal: Immediate: Once the convulsion occurs, the prognosis becomes uncertain. Prognosis depends on many factors and the ominous features are:

(1) Long interval between the onset of fit and commencement of treatment (late referral).
(2) Antepartum eclampsia specially with long delivery interval.
(3) Number of fits more than 10.
(4) Coma in between fits.
(5) Temperature over 102°F with pulse rate above 120/minute.
(6) Blood pressure over 200 mm Hg systolic.
(7) Oliguria (< 400 mL/24 hours) with proteinuria > 5 gm/24 hours.
(8) Nonresponse to treatment.
(9) Jaundice.

Mortality: Maternal mortality in eclampsia is very high in India and varies from 2–30%, much more in rural based hospital than in the urban counterpart. However, if treated early and adequately, the mortality should be even less than 2%.

Causes of maternal deaths:

(1) Cardiac failure.
(2) Pulmonary edema.
(3) Aspiration and/or septic pneumonia.
(4) Cerebral hemorrhage.
(5) Acute renal failure.
(6) Cardiopulmonary arrest.
(7) Adult respiratory distress syndrome (ARDS).
(8) Pulmonary embolism.
(9) Postpartum shock.
(10) Puerperal sepsis. Maternal complications are higher in antepartum eclampsia.

Remote: If the patient recovers from acute illness, she is likely to recover rapidly within 2–3 weeks. Recurrence of eclampsia in subsequent pregnancies is uncommon, although chance of pre-eclampsia is about 30%.

Fetal: The perinatal mortality is very high to the extent of about 30–50%. The causes are:

(1) Prematurity – spontaneous or induced,
(2) Intrauterine asphyxia due to placental insufficiency arising out of infarction, retroplacental hemorrhage and spasm of uteroplacental vasculature,
(3) Effects of the drugs used to control convulsions,
(4) Trauma during operative delivery.

MANAGEMENT HOSPITAL – THE PRINCIPLES OF MANAGEMENT ARE:

- Maintain: airway, breathing & circulation
- Hemodynamic stabilization (control BP)
- Oxygen administration 8–10 L/min
- Organize investigations
- Arrest convulsions (see below)
- Deliver by 6-8 hours
- Ventilatory support (if needed)
- Prevention of complications
- Prevention of injury
- Postpartum care (intensive)

Magnesium sulfate is the drug of choice (see Fig.10.).

Regimens of $MgSO_4$ for the management of severe pre-eclampsia and eclampsia		
Regimen	**Loading dose**	**Maintenance dose**
Intramuscular (Pritchard)	4 gm IV over 3–5 min followed by 10 gm deep IM (5 gm in each buttock)	5 gm IM 4 hourly in alternate buttock
Intravenous (Zuspan or Sibai)	4–6 gm IV over 15–20 min	1–2 gm/hr IV infusion

Fig. 10. Magnesium sulfate doses

OBSTETRIC MANAGEMENT:

During pregnancy: In majority of cases with antepartum eclampsia, labor start soon after convulsions. But when labor fails to start, the management depends on – (i) whether the fits are controlled or not and (ii) the maturity of the fetus. The decision to deliver is made once the woman is stable.

- *Fits controlled:*

– Baby mature: Delivery should be done.

(a) If the cervix is favorable and there is no contraindication of vaginal delivery, surgical induction by low rupture of the membranes is done. Oxytocin drip may be added, if needed.

(b) When the cervix is unfavorable, cervical ripening with PGE2 gel or pessary could be achieved before ARM.

(c) If the cervix is unfavorable and/or there is obstetric contraindication of vaginal delivery, cesarean section is done.

– Baby premature (<37 weeks): Delivery is recommended in a set up with neonatal intensive care unit (NICU). The underlying disease process of pre-eclampsia eclampsia persists until the woman delivers. At times the disease process may flare up. Moreover, there lies the risk of recurrent convulsions and IUFD. Steroid therapy is given when pregnancy is less than 34 weeks. Conservative management at very early pregnancy may improve perinatal outcome but this must be carefully balanced with maternal well being (RCOG-2006).

– Baby dead: The preeclamptic process gradually subsides and eventually expulsion of the baby occurs. Otherwise medical method of induction is started.

- *Fits not controlled:*

If the fits are not controlled with anticonvulsant within a reasonable period (6–8 hours), termination of pregnancy should be done. If vaginal examination indicates a quick response to induction, low rupture of the membranes is done. Oxytocin infusion may be added. The uterus responds well to oxytocin in such cases. In presence of unfavorable factors, cesarean section gives a quick response.

During labor: In the absence of any contraindication to vaginal delivery, as soon as the labor is well established, low rupture of the membranes is to be done to accelerate the labor. The dose schedule of antihypertensive and anticonvulsant drugs may be increased to quieten the patient. Second stage should be curtailed by forceps, ventouse or craniotomy, if the baby is dead. Prophylactic intravenous ergometrine or syntometrine following the delivery of the anterior shoulder should not be given as it may produce further rise of blood pressure. Instead, 10 units of oxytocin IM or IV slowly should be given. One should remain vigilant about postpartum hemorrhage and shock.

Indications of cesarean section:

1. Uncontrolled fits in spite of therapy.
2. Unconscious patient and poor prospect of vaginal delivery.
3. Obstetric indications (malpresentation).

Follow up and prognosis: Patient should be followed up in the post-natal clinic by 6 weeks time. Persistence of hypertension, proteinuria and abnormal blood biochemistry necessitates further investigation and consultation with a physician. Further pregnancy should be deferred till they are controlled.

Recurrence risk varies between 2 and 25%. The risk of pre-eclampsia and eclampsia to the daughter of an eclampsia patient is about 25% and 3%, respectively.

Atypical eclampsia is defined when eclampsia occurs before 20th week of pregnancy or more than 48 hours postpartum. Patients are treated with parenteral magnesium sulfate.

GESTATIONAL HYPERTENSION

A sustained rise of blood pressure to 140/90 mm Hg or more on at least two occasions 4 or more hours apart beyond the 20th week of pregnancy or during the first 24 hours after delivery in a previously normotensive woman is called gestational hypertension. It is associated with a much higher incidence of essential hypertension in later life than pre-eclampsia. Both, thus appear to be two phases of the same

disorder. It should fulfill the following criteria: (1) Absence of any evidences for the underlying cause of hypertension (2) Unassociated with other evidences of pre-eclampsia (edema or proteinuria). (3) Majority of cases are > 37 weeks pregnancy. (4) Not associated with hemoconcentration, thrombocytopenia, raised serum uric acid level or hepatic dysfunction. (5) The blood pressure should come down to normal within 6 weeks following delivery.

Gestational edema is excessive accumulation of fluid with demonstrable pitting edema over the ankles greater than 1 + after 12 hours in bed or gain in weight of 2 kg or more in a week due to influence of pregnancy.

Gestational proteinuria is the presence of protein of more than 0.3 gm in the 24 hours urine during or under the influence of pregnancy in the absence of hypertension, edema or renal infection. It may be orthostatic proteinuria.

CHRONIC HYPERTENSION IN PREGNANCY

Chronic hypertensive disease (CHD) is defined as the presence of hypertension of any cause antedating or before the 20th week of pregnancy and its presence beyond the 12 weeks after delivery. The condition poses a difficult problem as regards the diagnosis and management when seen for the first time, beyond the 20th week of pregnancy. Overall incidence is 2–4% of which 90% are due to essential hypertension. See the differential diagnosis in Figure 11.

The high risk factors for CHD are: (1) Age (> 40 years), (2) Duration of hypertension (>15 years), (3) Level of BP (>160/110 mm of Hg), (4) Presence of any medical disorder (renovascular), and (5) Presence of thrombophilias. Majority of women with CHD are low risk and have satisfactory maternal and fetal outcome without any antihypertensive therapy.

Table 17.2: Differential features of Pre-eclampsia with gestational and essential hypertension			
	Pre-eclampsia	**Gestational hypertension**	**Essential hypertension**
Age	Mostly young	Young	Usually elderly
Parity	Primigravidae — common	Primigravidae	Multipara — common
Past history	Pre-eclampsia in previous pregnancy	May be present	Pre-pregnant hypertension present
Family history	May be present	Unrelated	Often present
Onset of hypertension	After 20th week of pregnancy	Usually in third trimester	Before 20th week of pregnancy
Follow up BP following delivery	Subsides completely	Subsides completely	Persists even after 3 months
Proteinuria	Present	Absent	Usually absent
Eye changes	Usually none. Extreme cases— retinal edema, constriction of arterioles, nicking of the veins	None	Silver wiring of the arterioles. Hypertensive retinopathy
Specific blood values	• Hemoconcentration + • Thrombocytopenia + • Serum uric acid > 5 mg/dl • Raised liver enzymes	Absent	Not significant

Fig. 11. Differential diagnosis of pre-eclampsia with gestational and essential hypertension

EXTRAGENITAL DISEASES

Heart disease in pregnancy

Factors responsible for cardiac failure:

- Advanced age
- Cardiac arrhythmias or left ventricular hypertrophy
- History of previous heart failure
- Appearance of 'risk factors' in pregnancy are: infection, anemia, hypertension, excessive weight gain and multiple pregnancy
- Inadequate supervision.

Maternal:

The prognosis depends on:

- Nature of lesion
- Functional capacity of the heart
- Quality of medical supervision provided during pregnancy, labor and puerperium
- Appearance of the risk factors mentioned earlier. Maternal mortality is lowest in rheumatic heart lesions and acyanotic group of heart diseasesless than 1%. With elevation of pulmonary vascular resistance specially with cyanotic heart lesions, the mortality may be raised to even 50% (Eisenmenger's syndrome). Most of the deaths occur due to cardiac failure and the maximum deaths occur following birth.

The other causes of death are:

- pulmonary edema
- pulmonary embolism
- active rheumatic carditis
- subacute bacterial endocarditis and
- rupture of cerebral aneurysm in coarctation of aorta. Fetal congenital cardiac disease is increased by 3–10% if either of the parents have congenital lesions.

DIAGNOSIS: (see Fig. 12.)

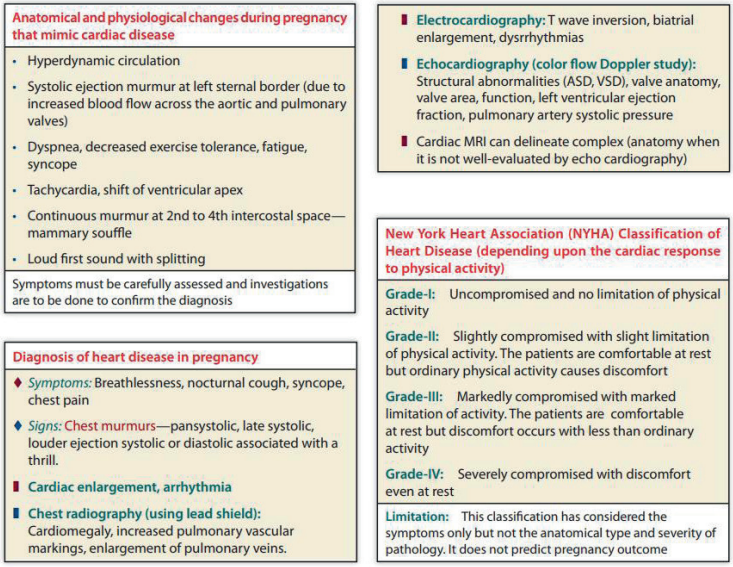


Fig. 12. Diagnostics tests

General management

Principles:

– Early diagnosis and evaluation of anatomical type and functional grade of the case – To detect the high-risk factors and to prevent cardiac failure

– Combined (obstetrician and cardiologist) care and mandatory hospital delivery.

Special care in each antenatal visit is to detect and to treat the risk factors that precipitate cardiac failure in pregnancy. Risk factors for cardiac failure are:

Infections – Urinary tract, dental and respiratory tract.

- Anemia/
- Obesity Hypertension Arrhythmias Hyperthyroidism Drugs – Betamimetics.
- Excess intake of caffeine, alcohol, high calorie diet.

ROLE OF ANTICOAGULANTS: Anticoagulants are necessary in cases of congenital heart disease who have pulmonary hypertension, artificial valve replacements or atrial fibrillation.

- heparin 5000 units twice daily subcutaneously up to 12th week; upto 7 days postpartum. Warfarin is then to be continued.

- warfarin tablet 3 mg. daily to be taken at the same time each day and continued up to 36 weeks.

ADMISSION:

Elective:

- Grade – I: At least two weeks prior to the expected date of delivery.
- Grade – II: At 28th week specially in case of unfavorable social surroundings.
- Grade – III and IV: As soon as pregnancy is diagnosed. The patient should be kept in the hospital throughout pregnancy.

Emergency:

- Deterioration of the functional grading.
- Appearance of dyspnea or cough or basal crepitations or tachyarrhythmias.
- Appearance of any pregnancy complication like anemia, pre-eclampsia.

MANAGEMENT DURING LABOR

Place of induction: Most patients with cardiac disease go into spontaneous labor and deliver without any difficulty. However, induction (vaginal PGE2) may be employed in very selected cases for obstetric indications. One should guard against infection and pulmonary edema due to fluid overload.

LABOUR:

First stage:

– Position: The patient should be in lateral recumbant position to minimize aortocaval compression – Oxygen is to be administered (5–6 l/min) if required – Analgesia in the majority, is best given by epidural.

– Fluids should not be infused more than 75 mL/hour to prevent pulmonary edema. – Careful watch of the pulse and respiration rate. If the pulse rate exceeds 110 per minute in between uterine contractions, rapid digitalization is done by intravenous digoxin 0.5 mg.

– Cardiac monitoring and pulse oxymetry can detect arrhythmias and hypoxemia early. – Central venous pressure monitoring may be needed in selected cases.

Prophylactic antibiotics is to prevent puerperal endocarditis: intravenous ampicillin 2 g and gentamicin 1.5 mg/kg (not to exceed 80 mg), 8 hours interval.

All high-risk patients with structural heart disease, complex cyanotic congenital heart disease, prosthetic valves need endocarditis prophylaxis.

Second stage: No maternal pushing and the tendency to delay in the second stage of labor is to be curtailed by forceps or ventouse under pudendal and/or perineal block anesthesia.

Third stage: oxytocin can be given by infusion.

PLACE OF CESAREAN SECTION: In general, there is no indication of cesarean section for heart disease.

CARDIAC INDICATIONS OF CESAREAN DELIVERY:

- Coarctation of aorta
- Aortic dissection or aneurysm Aortopathy with aortic root > 4 cm Warfarin treatment within 2 weeks.

Barrier method of contraceptives (condom) is the best.

MANAGEMENT OF CARDIAC FAILURE IN PREGNANCY:

Propped up position

- O2 administration Monitoring with ECG and pulse oximetry.
- Diuretic: Frusemide (Loop) (40–80 mg) IV.
- Mechanical ventilation Injection morphine 15 mg IM.
- Digoxin 0.5 mg IM followed by tab digoxin 0.25 mg P.O. (Digoxin crosses the placenta and is excreted in breast milk).
- Dysrhythmias – quinidine or electrical cardioversion.
- Tachyarrhythmias – Adenosine (3–12 mg) IV.

SPECIFIC HEART DISEASE DURING PREGNANCY AND THE MANAGEMENT

RHEUMATIC HEART DISEASE:

MITRAL STENOSIS is the commonest heart lesion met during pregnancy. Normal mitral valve area ranges between 4 and 6 cm^2. Symptoms usually appear when stenosis narrows this to less than 2.5 cm^2. In asymptomatic cases, the mortality is < 1% but once it is significantly symptomatic, mortality ranges between 5 and 15%. Diagnosis and management has been mentioned earlier. During labor continuous epidural analgesia is ideal and intravenous fluid overload is to be avoided.

PLACE OF VALVOTOMY: It is better to withheld elective cardiac surgery during pregnancy. Surgery should be considered in cases of unresponsive failure with pregnancy beyond 12 weeks. Best time of surgery is between 14–18 weeks. Valve replacement, commissurotomy, balloon-valvotomy can be carried out in early second trimester. Atrial fibrillation is a complication. Digoxin, β blockers and anticoagulation (heparin) should be used.

AORTIC STENOSIS: Most cases of aortic stenosis are congenital, some are rheumatic in origin. Normal aortic valve area is 3–4 cm^2. When it is reduced to < 1 cm^2, stenosis is significant. Maternal mortality of significant aortic stenosis is about 15–20% with perinatal

loss of about 30%. Epidural anesthesia is contraindicated. During labor, fluid therapy (125–150 mL/h) should not be restricted. Left ventricular after load is high and the pregnant patient is sensitive to haemorrhage.

Common symptoms are angina, syncope and left ventricular failure. Medical management is not helpful in a symptomatic patient. Valve replacement is the definitive treatment. Mechanical valves need anti-coagulation. Open heart surgery is preferably avoided in pregnancy. Aortic balloon valvoplasty may be done as a palliative procedure.

CONGENITAL HEART DISEASE:

Major maternal risks in pregnancy are:

1. Cyanosis.
2. Left ventricular dysfunction.
3. Pulmonary hypertension.

Acyanotic (L to R shunt)

Atrial Septal Defect (ASD): ASD (ostium secundum type) is the most common congenital heart lesion during pregnancy. Even uncorrected ASD tolerates pregnancy and labor well. Congestive cardiac failure unresponsive to medical therapy requires surgical correction. Shunt reversal is the major risk which may develop in hypovolemia. Such cases may occur in hemorrhagic conditions and following injudicious administration of epidural anesthesia. In the absence of arrhythmias, and pulmonary hypertension, ASD does not usually complicate pregnancy.

Patent Ductus Arteriosus (PDA): Presence of continuous murmur at the upper left sternal border is suggestive of diagnosis. Most patients with PDA tolerate pregnancy well. Pulmonary hypertension may cause maternal death. Surgical correction during pregnancy can be performed provided there is no pulmonary hypertension. Epidural analgesia is better avoided to minimize shunt reversal due to systemic hypotension. Fetal loss may be up to 7% and there is 4% chance that the child of this parent will suffer from the same abnormality. Endocarditic prophylaxis should be given.

Ventricular Septal Defect (VSD): In general, if the defect is less than 1.25 cm^2, pulmonary hypertension and heart failure do not develop. Pregnancy is well tolerated with small to moderate left to right shunt or with moderate pulmonary hypertension. The major risk is shunt reversal leading to circulatory collapse and cyanosis. Hypotension is to be avoided. Fetal loss may be up to 20%.

Mitral Valve Prolapse (MVP): Is the commonest congenital valvular lesion. Most of them are asymptomatic. Women tolerate pregnancy and labor well. Endocarditis prophylaxis is given.

B. Cyanotic (R to L shunt)

Fallot's tetralogy: It is the most common form of cyanotic heart lesion. It is a combination of ventricular septal defect, pulmonary valve stenosis, right ventricular hypertrophy and an overriding aorta. After surgical correction, patients tolerate pregnancy well. Surgically uncorrected patients are at increased risk. Complications like bacterial endocarditis, brain abscess and cerebral embolism are more common. Maternal mortality is 5–10% and the perinatal mortality is 30–40%. IUGR is common. Systemic hypotension is dangerous which may lead even to death. Epidural or spinal anesthesia is avoided. Pregnancy is discouraged in women with uncorrected tetralogy.

Eisenmenger's syndrome:_Patients with Eisenmenger's syndrome have pulmonary hypertension with shunt (right to left) through an open ductus, an atrial or ventricular septal defect. Maternal mortality is about 50% and so also the perinatal loss (50%). Termination of pregnancy should be seriously considered. Heparin should be used throughout pregnancy as there is risk of systemic and pulmonary thromboembolism. Epidural anesthesia is contraindicated. Inhaled nitric oxide or I.V. prostacyclin is used as a pulmonary vasodilator. Complications are: hemoptysis, arrhythmia, cerebrovascular accident and hypoxemia; hyperviscosity syndrome and sudden death

DIABETES MELLITUS AND PREGNANCY

Diabetes mellitus is a chronic metabolic disorder due to either insulin deficiency (relative or absolute) or due to peripheral tissue resistance (decreased sensitivity) to the action of insulin. The pathophysiology involved are: 1- decreased sensitivity of skeletal muscles and liver to insulin (insulin resistance) and -2 inadequate secretion of insulin (β cell dysfunction). The defect lies both in insulin secretion and action. The ultimate effect is the hyperglycemia. Two types are generally described.

Type–1 (IDDM) is characterized by young age onset (Juvenile) and absolute insulinopenia. They have genetic predisposition with presence of autoantibodies.

Type–2 (NIDDM) is characterized by late age onset, overweight woman and peripheral tissue (skeletal muscle, liver) insulin resistance (hyper insulinemia). Genetic predisposition is also observed.

Alternation in lipid metabolism in diabetes: Decrease in HDL cholesterol is observed specially with type-1 diabetes. HDL acts as a plasma antioxidant. Fall in HDL may be a cause for congenital malformations as oxidative stress is a potential factor. Increased free fatty acids have been associated with fetal over-growth.

About 1–14 percent of all pregnancies are complicated by diabetes mellitus and 90 percent of them are gestational diabetes mellitus (GDM). Nearly 50 percent of women with GDM will become overt diabetes (type-2) over a period of 5 to 20 years.

GESTATIONAL DIABETES MELLITUS (GDM)

NOMENCLATURE: GDM is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy.

The potential candidates for GDM are:

Positive family history of diabetes (parents or sibling). Family history should include uncles, aunts and grandparents

a) Having a previous birth of an overweight baby of 4 kg or more
b) Previous stillbirth with pancreatic islet hyperplasia revealed on autopsy
c) Unexplained perinatal loss
d) Presence of polyhydramnios or recurrent vaginal candidiasis in present pregnancy
e) Persistent glycosuria
f) Age over 30 years
g) Obesity
h) Ethnic group (East Asian, Pacific island ancestry).

GDM diagnostics tests – see Fig.13.

Table 19.2: Criteria for diagnosis of impaired glucose tolerance and diabetes with 75 gm oral glucose (American Diabetic Assocation)

Plasma (mg %)			
Time	Normal tolerance	Impaired glucose tolerance	Diabetes
Fasting	<100	≥100 and <126	≥126
2 hour post glucose	<140	≥140 and <200	≥200

- Venous whole blood values are 15% less than the plasma
- m mol/l = mg% × 0.0555

Fig. 13. Diagnostics tests

HAZARDS:

- Increased perinatal loss is associated with fasting hyperglycemia. Fetal anomalies are however not increased
- Increased incidence of macrosomia
- Polyhydramnios
- Birth trauma
- Recurrence of GDM in subsequent pregnancies is about 50 percent.

Management:

The patient needs more frequent antenatal supervision with periodic check up of fasting plasma glucose level which should be less than 90 mg percent. The control of high blood glucose is done by restriction of diet, exercise with or without insulin. Diet with 2000–2500 Kcal/day for normal weight woman and restriction to 1200–1800 Kcal/day for over weight woman is recommended. Exercise (aerobic, brisk walking) programs are safe in pregnancy and may obviate the need of insulin therapy.

OVERT DIABETES: According to American Diabetic Association diagnosis is positive if

- the fasting plasma glucose exceeds 126 mg/dL
- the 2 hours post glucose (75 gm) value exceeds 200 mg/dL.

EFFECTS OF PREGNANCY ON DIABETES: Vascular changes, specially retinopathy, nephropathy, coronary artery disease and neuropathy may be worsened during pregnancy.

EFFECTS OF DIABETES ON PREGNANCY

Maternal

During pregnancy:

– Abortion: Recurrent spontaneous abortion may be associated with uncontrolled diabetes.

– Preterm labor (20%) may be due to infection or polyhydramnios.

– Infection: Urinary tract infection and vulvo vaginitis.

– Increased incidence of pre-eclampsia (25%).

– Polyhydramnios (25–50%) is a common association. Large baby, large placenta, fetal hyperglycemia leading to polyuria, increased glucose concentration of liquor irritating the amniotic epithelium or increased osmosis, are some of the probabilities.

– Maternal distress may be due to the combined effects of an oversized fetus and polyhydramnios.

– Diabetic retinopathy, microaneurysms, hemorrhages and proliferative retinopathy. Laser photocoagulation is the preferred treatment.

– Diabetic nephropathy—may lead to renal failure

– Ketoacidosis

During labor: There is increased incidence of:

- Prolongation of labor due to big baby.
- Shoulder dystocia is due to disproportionate growth with increased shoulder/head ratio.
- Perineal injuries.
- Postpartum hemorrhage.
- Operative interference.

Puerperium: (1) Puerperal sepsis. (2) Lactation failure.

FETAL AND NEONATAL HAZARDS:

– Fetal macrosomia (30–40%)

– Congenital malformation (6–10%)

Early detection of fetal anomalies:

- Estimation of glycosylated hemoglobin A (HbA1c) before 14 weeks. Chance of major congenital malformation is about 8% and 23% when the values are 9.5 and 10 respectively.
- Maternal serum α fetoprotein level at 16 weeks and a detailed high resolution ultrasonography of the fetus including fetal echocardiography at 20–22 weeks are advocated
- A comprehensive ultrasound examination—including fetal echocardiography is done at 20–22 weeks to detect any cardiac anomaly along with other structural malformation.

- Birth injuries (brachial plexus) are associated with prolonged labor and shoulder dystocia due to macrosomic baby.

- Growth restriction is less commonly observed and is associated with maternal vasculopathy.

- Fetal death has got multifactorial pathogenesis but the final event being hypoxia and lactic acidemia.

- Neonatal complications include—
  - hypoglycemia (< 37 mg/dL)
  - respiratory distress syndrome
  - hyperbilirubinemia
  - Polycythemia
  - hypocalcemia (< 7 mg/dL)
  - hypomagnesemia (< 7 mg/dL)
  - cardiomyopathy

- Longterm effects – childhood obesity, neuropsychological effects and diabetes.

Management:

Principles in the management are:

(1) Careful antenatal supervision and glycemic control, so as to maintain the glucose level as near to physiological level as possible

(2) To find out the optimum time and method of delivery

(3) Arrangement for the care of the newborn.

ANTENATAL CARE: Antenatal supervision should be at monthly intervals up to 20 weeks and thereafter at 2 weeks intervals.

Insulin therapy: A post prandial (2 hours) plasma glucose level of more than 140 mg% even on diet control is an indication of insulin therapy. The patient should receive three to four daily injections of a regular (human act rapid) and an intermediate acting insulin (isophane), the latter is to be given before dinner.

ADMISSION: Early hospitalization facilities:

- Stabilization of diabetes
- Minimizes the incidence of pre-eclampsia, polyhydramnios and preterm labor
- To select out the appropriate time and method of delivery.

Induction of labor:

The indications are:

- Diabetic women controlled on insulinare considered for induction of labor after 38 completed weeks.

- Women with vascular complications (pre-eclampsia, IUGR) often require induction after 37 weeks.

Epidural analgesia is ideal for pain relief.

Cesarean section: The indications are – (1) Elderly primigravidae (2) Multigravidae with a bad obstetric history (3) Diabetes with complications or difficult to control (4) Obstetric complications like pre-eclampsia, polyhydramnios, malpresentation (5) Fetal macrosomia (> 4 kg).

As such 50% of diabetic mothers are delivered by cesarean section.

THYROID DYSFUNCTION WITH PREGNANCY

Hyperthyroidism: Physiological changes during pregnancy such as increase in cardiac output, oxygen consumption and heat production may mimic mild thyrotoxicosis.

The maternal and fetal/neonatal complications in untreated hyperthyroidism:

Maternal: Miscarriage, preterm delivery, pre-eclampsia, congestive cardiac failure, placental abruption, thyroid storm and infection.

Fetal/Neonatal: prematurity, stillbirth, hyperthyroidism, hypothyroidism, increased perinatal morbidity and mortality.

Clinical diagnosis of hyperthyroidism: free T4 (FT4 – high), free T4 index (FT4 1– high), FT3 (high) and TSH (suppressed) levels.

The main stay of treatment is use of antithyroid drugs [propylthiouracil (PTU) or methimazole (MM)]. Both the drugs are effective. Methimazole is preferably avoided in the first trimester of pregnancy. Carbimazole is given orally with a daily dose of 10–40 mg and maintained at this dose until the patient becomes euthyroid. Then it is progressively reduced to a maintenance of between 5 and 15 mg daily. Propylthiouracil is given at a daily dose of 300–450 mg and continued till the patient becomes euthyroid – the maintenance dose being 50 and 150 mg daily. Normalization of TSH is an indicator to reduce

the dose of drugs. Patients having marked tachycardia or arrhythmias should also have propranolol (β blocking agent).

HYPOTHYROIDISM: May be subclinical (elevalated TSH and normal FT4) or overt (elevated TSH and low FT4).

Myxedema rarely presents in pregnancy because they tend to be infertile. Untreated hypothyroidism in early pregnancy has a high fetal wastage in the form of abortion, stillbirth and prematurity and deficient intellectual development of the child. However, pregnancy complications like pre-eclampsia and anemia are high. Serum thyroid peroxidase antibodies (anti TPO) or antimicrosomal antibodies are elevated in autoimmune thyroiditis.

The dose of levo-thyroxine needs to be increased in pregnancy. Generally, therapy is started as 0.1 mg/day and increased by 0.05 mg at 2 weeks intervals depending on free T4 and TSH levels.

PYELONEPHRITIS IN PREGNANCY

There is increased chance of urinary tract infection in females as compared to males due to:

1. Short urethra (4 cm)
2. Close proximity of the external urethral meatus to the areas (vulva and lower third of vagina) contaminated heavily with bacteria
3. Catheterization (iv) Sexual intercourse.

INCIDENCE: The overall incidence of pyelonephritis in pregnancy is between 1–3%.

ETIOLOGY:

- It is more common in primigravidae than multiparae
- Previous history of urinary tract infection increases the chance by 50%
- Presence of asymptomatic bacteriuria increases the chance by 25%

- Abnormality in the renal tract is found in about 25%
- Stasis–due to compression of the ureters (mainly the right) by gravid uterus.

CLINICAL TYPES:

ACUTE PYELONEPHRITIS: Clinical features—the onset is acute and usually appears beyond the 16th week. The involvement is bilateral but if unilateral, it is more frequent on the right side. Clinical features are mainly due to endotoxemia.

Acute aching pain over the loins, often radiating to the groin and costovertebral angle tenderness, dysuria, hematuria. Fever (spiky 40°C) with chills and rigor followed by hypothermia (34°C); anorexia, nausea, vomiting and myalgias; respiratory distress and pulmonary edema due to endotoxin induced alveolar injury.

Investigations: Apart from the routine ones, serum level of creatinine, electrolytes and culture studies of urine and blood should be done.

COMPLICATIONS:

Fetal: abortion, preterm labor, intrauterine fetal death caused by hyperpyrexia and low birth weight babies (prematurity and dysmaturity).

Maternal: Anemia, Septicemia, renal dysfunction and pulmonary insufficiency.

MANAGEMENT – The outlines of management are:

- Intravenous fluid (crystalloid) for adequate hydration.
- Evaluate hemogram, serum electrolytes, creatinine.
- Acetaminophen is given for the fever.
- Monitor urine output (> 60 mL/hr), temperature and BP.
- IV antibiotics – Cephalosporins, aminoglycosides (gentamicin), Cefazoline or Ceftriaxone, for 48 hours till culture report is available and then changed to oral therapy for another 10–14 days.
- Repeat urine culture after 2 weeks of antimicrobial therapy and is repeated at each trimester of pregnancy.

- If the symptoms recur or the dip stick test for nitrate and leukocyte esterase is positive, urine culture is repeated. The woman needs retreatment if the culture is positive.
- Patient not responding with this therapy needs to be evaluated (sonography, CT scan, radiography) for urinary tract obstruction.
- Antimicrobial suppression therapy is continued till the end of pregnancy to prevent recurrence (30–40%). Nitrofurantoin 100 mg daily at bed time is effective.

ASYMPTOMATIC BACTERIURIA (ASB)

The term asymptomatic bacteriuria is used when a bacterial count of the same species over 10*5/mL in mid stream cleancatch specimen of urine on two occasions is detected without symptoms of urinary infection. E. coli is the offending organism in over 90% cases.

Asymptomatic bacteriuria if recurrent is associated with high incidence of urinary tract abnormality (20%), congenital or acquired. The woman runs a greater risk of developing chronic renal lesion in later life.

Treatment: The antimicrobial agents should be appropriate to the mother and the fetus:

-ampicillin (500 mg qid), nitrofurantoin (100 mg qid), cephalexin (500 mg tid) or amoxycillin–clavulanic acid (375 mg tid). A course of 10–14 days will cure 70–100% of cases. A single dose therapy of nitrofurantoin 0.2 gm or amoxycillin 3 gm has been found effective. Long term prophylaxis with nitrofurantoin (50 mg) or amoxycillin (250 mg) at night may have to be continued until delivery when the infection is recurrent, 3 months postpartum.

PRETERM LABOR (Syn: Premature Labor)

Definition: Preterm labor (PTL) is defined as one where the labor starts before the 37th completed week (< 259 days), counting from the first day of the last menstrual period.

Etiology: In about 50%, the cause of preterm labor is not known. Often it is multifactorial. The following are, however, related with increased incidence of preterm labor.

High risk factors:

(A) History: There is an increased incidence of preterm labor in cases such as:

- Previous history of induced or spontaneous abortion or preterm delivery;
- Pregnancy following assisted reproductive techniques (ART);
- Asymptomatic bacteriuria or recurrent urinary tract infection;
- Smoking habits;
- Low socioeconomic and nutritional status;
- Maternal stress.

(B) Complications in present pregnancy: May be due to maternal, fetal or placental.

Maternal:

- Pregnancy complications: Preeclampsia, antepartum hemorrhage, premature rupture of the membranes, polyhydramnios;
- Uterine anomalies: Cervical incompetence, malformation of uterus;
- Medical and surgical illness: Acute fever, acute pyelonephritis, diarrhea, acute appendicitis, toxoplasmosis and abdominal operation. Chronic diseases: Hypertension, nephritis, diabetes, decompensated heart lesion, severe anemia, low body mass index (LBMI);
- Genital tract infection: Bacterial vaginosis, beta-hemolytic streptococcus, bacteroides, chlamydia, mycoplasma.

Fetal: Multiple pregnancy, congenital malformations, intrauterine death.

Placental: Infarction, thrombosis, placenta previa or abruption.

(C) Iatrogenic: Indicated preterm delivery due to medical or obstetric complications.

(D) Idiopathic: (Majority) – Premature effacement of the cervix with irritable uterus and early engagement of the head are often associated.

Diagnosis:

- Regular uterine contractions with or without pain (at least one in every 10 minute);
- Dilatation (> 2 cm) and effacement (80%) of the cervix;
- Length of the cervix (measured by TVS) < 2.5 cm and funneling of the internal os;
- Pelvic pressure, backache and or vaginal discharge or bleeding.

MANAGEMENT OF PRETERM LABOR

The management includes: (1) To prevent preterm onset of labor, if possible; (2) To arrest preterm labor, if not contraindicated; (3) Appropriate management of labor; (4) Effective neonatal care. (See Fig. 14, Fig. 15).

– Bed rest – The patient is to lie preferably in left lateral position though the benefits are doubtful.

– Adequate hydration is maintained. Prophylactic antibiotic is not routinely given. It is recommended when infection is evident or culture report suggests.

– Prophylactic cervical circlage for women with prior preterm birth and short cervix in the present pregnancy may be beneficial.

– Tocolytic agents: Various drugs including progesterone (micronized) have been used to inhibit uterine contractions.

– Glucocorticoid therapy: Either betamethasone (Betnesol) 12 mg IM 24 hours apart for two doses or dexamethasone 6 mg IM every 12 hours for 4 doses is given. Betamethasone is the steroid of choice.

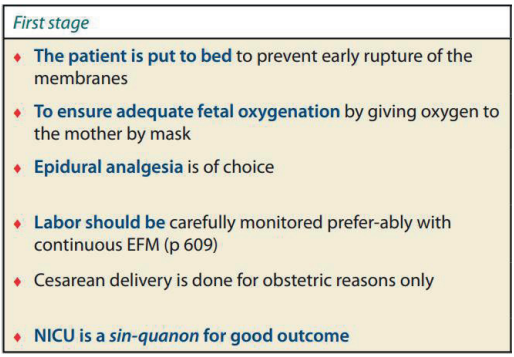


Fig. 14. Management of preterm labor

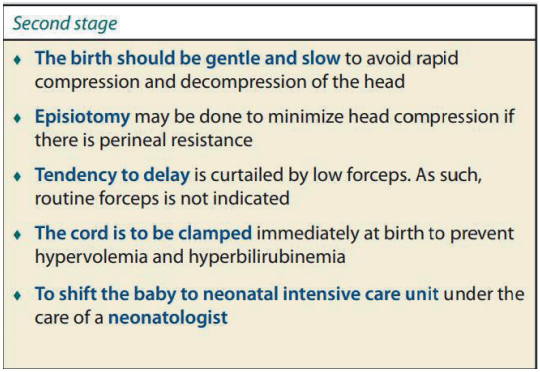


Fig. 15. Management of preterm labor

PRELABOR RUPTURE OF THE MEMBRANES (PROM) (Syn: Premature Rupture of Membranes)

Definition: Spontaneous rupture of the membranes any time beyond 28th week of pregnancy but before the onset of labor is called prelabor rupture of the membranes (PROM). Rupture of membranes for > 24 hours before delivery is called prolonged rupture of membranes.

Incidence: PROM occurs in approximately 10% of all pregnancies.

Dangers: The implications are less serious when the rupture occurs near term than earlier in pregnancy.

- In term PROM labor starts in 80–90% of cases within 24 hours. PROM is one of the important causes of preterm labor and prematurity;
- Chance of ascending infection is more if labor fails to start within 24 hours. Liquor gets infected (chorioamnionitis) and fetal infection supervenes;
- Cord prolapse specially when associated with malpresentation;
- Continuous escape of liquor for long duration may lead to dry labor;
- Placental abruption;
- Fetal pulmonary hypoplasia specially in preterm PROM is a real threat when associated with oligohydramnios;
- Neonatal sepsis, RDS, IVH and NEC in preterm PROM;
- Perinatal morbidities (cerebral palsy) are high.

Management (see Fig.16.)

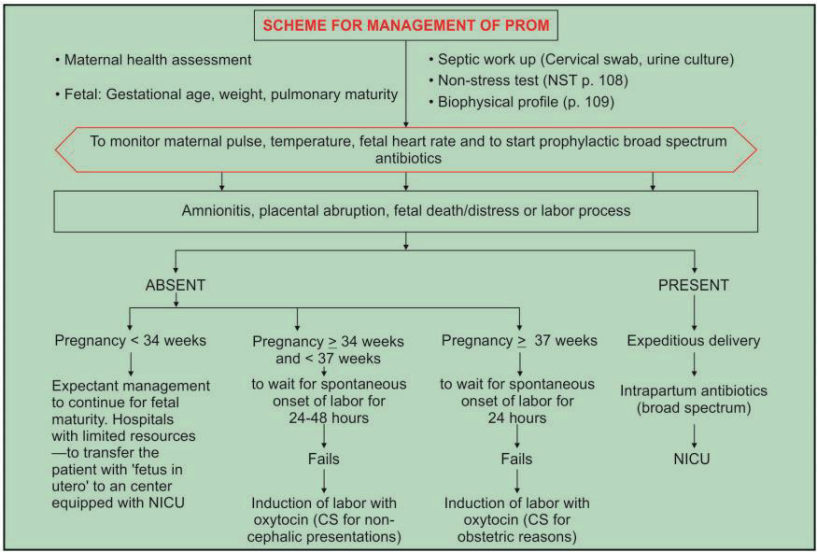


Fig. 16. Management of Premature Rupture of Membranes

POST-TERM PREGNANCY (Syn: Postmaturity)

Definition: But for clinical purposes, a pregnancy continuing beyond two weeks of the expected date of delivery (> 294 days) is called post-maturity or post-term pregnancy.

Incidence: The incidence of pregnancies continuing beyond 42 completed weeks (> 294 days) ranges between 4 and 14 percent.

Etiology:

- Wrong dates – due to inaccurate LMP (most common).
- Biological variability (Hereditary) may be seen in the family.
- Maternal factors: Primiparity, previous prolonged pregnancy, sedentary habit, elderly multiparae.
- Fetal factors: Congenital anomalies: Anencephaly → abnormal fetal HPA axis and adrenal hypoplasia → diminished fetal cortisol response.
- Placental factors: Sulphatase deficiency → low estrogen.

Diagnosis: It is indeed difficult to diagnose postmaturity when the case is first seen beyond the expected date.

- Menstrual history – If the patient is sure about her date with previous history of regular cycles, it is a fairly reliable diagnostic aid in the calculation of the period of gestation.
- The suggested clinical findings when a pregnancy overruns the expected date by two weeks are:

– Weight record: Regular periodic weight checking reveals stationary or even falling weight.

– Girth of the abdomen: It diminishes gradually because of diminishing liquor.

– History of false pain: Appearance of false pain followed by its subsidence is suggestive.

– Obstetric palpation: The following findings, taken together are helpful. These are: height of the uterus, size of the fetus and hardness

of the skull bones. As the liquor amnii diminishes, the uterus feels "full of fetus"— a feature usually associated with postmaturity.

– Internal examination: While a ripe cervix is usually suggestive of fetal maturity, to find an unripe cervix does not exclude maturity. Feeling of hard skull bones either through the cervix or through the fornix usually suggests maturity.

Baby:

- General appearance: Baby looks thin and old. Skin is wrinkled. There is absence of vernix caseosa. Body and the cord are stained with greenish yellow color. Head is hard without much evidence of moulding. Nails are protruding beyond the nail beds;
- Weight often more than 3 kg and length is about 54 cm. Both are variable and even an IUGR baby may be born.

Liquor amnii: Scanty and may be stained with meconium.

Placenta: There is evidence of ageing of the placenta manifested by excessive infarction and calcification.

Cord: There is diminished quantity of Wharton's jelly which may precipitate cord compression.

COMPLICATIONS OF POST-TERM PREGNANCY: When pregnancy overruns the expected date, there is risk of placental insufficiency due to placental aging. This is manifested by placental calcification and infarction. Associated complications like hypertension and diabetes aggravates the pathology.

Fetal:

During pregnancy – There is diminished placental function, oligohydramnios and meconium stained liquor. These lead to fetal hypoxia and fetal distress.

During labor:

- Fetal hypoxia and acidosis;
- Labor dysfunction;

- Meconium aspiration;
- Risks of cord compression due to oligohydramnios;
- Shoulder dystocia;
- Increased incidence of birth trauma due to big size baby and non-moulding of head due to hardening of skull bones;
- Increased incidence of operative delivery. The main clinical significance of post-term pregnancy is dysmaturity or macrosomia.
- Following birth;
- Chemical pneumonitis, atelectasis and pulmonary hypertension are due to meconium aspiration;
- Hypoxia (low Apgar scores) and respiratory failure;
- Hypoglycemia and polycythemia;
- Increased NICU admissions.

Management

Uncomplicated:

– Selective induction: In this regime, the pregnancy may be allowed to continue till spontaneous onset of labor. Fetal surveillance is continued with modified biophysical profile twice a week.

– Routine induction: The expectant attitude is extended for 7–10 days past the expected date and thereafter labor is induced. Induction: Induction of labor reduces the rate of cesarean delivery and perinatal mortality. If the cervix is favorable (ripe), induction is to be done by stripping of the membranes or by low rupture of the membranes. If the liquor is found clear, oxytocin infusion is added to be more effective. Careful fetal monitoring is mandatory.If the cervix is unripe, it is made favorable by vaginal administration of PGE2 gel. This is followed by low rupture of the membranes. Oxytocin infusion is added when required.

Cervical length (TVS) < 25 mm is a predictor of successful induction of labor.

Complicated group:

(Associated with complicating factors)

– Elective cesarean section is advisable when postmaturity is associated with high risk factors like: elderly primigravidae, preeclampsia, Rh-incompatibility, fetal compromise or oligohydramnios.

Associated complications that are likely to produce placental insufficiency – Ideally pregnancy should not be allowed to go past the expected date.

Care during labor: If fetal distress appears, prompt delivery either by cesarean section.

ABNORMAL UTERINE ACTION

Normal labor is characterized by coordinated uterine contractions associated with progressive dilatation of the cervix and descent of the fetal head. Normal labor is associated with cervical dilatation > 1 cm/h in a nulliparous woman and is likely to end in successful vaginal delivery. Any deviation of the normal pattern of uterine contractions affecting the course of labor is designated as disordered or abnormal uterine action.

Etiology:

(1) Prevalent in first birth specially with elderly women.
(2) Prolonged pregnancy.
(3) Overdistension of the uterus (twins and fibroids).
(4) Emotional factor (anxiety, stress).
(5) Constitutional factor (obesity).
(6) Contracted pelvis and malpresentation.
(7) Injudicious administration of sedatives, analgesics and oxytocics.
(8) Premature attempt at vaginal delivery or attempted instrumental vaginal delivery under light anesthesia. Normal baseline tonus is between 5 and 20 mm of Hg and peak pressure is around 60 mm of Hg (8 kPa).

UTERINE INERTIA (HYPOTONIC UTERINE DYSFUNCTION)

Uterine contraction: The intensity is diminished; duration is shortened; good relaxation in between contractions and the intervals are increased; intrauterine pressure during contraction is below 25 mm of Hg.

Diagnosis:

(1) Patient feels less pain during uterine contraction.
(2) Hand placed over the uterus during uterine contraction reveals less hardening of the uterus.
(3) Uterine wall is easily indentable at the acme of a pain.

(4) Uterus becomes relaxed after the contraction; fetal parts are well palpable and fetal heart rate remains normal.
(5) Internal examination reveals:
(a) Poor dilatation of the cervix (Normal rate of dilatation in primigravida should be at the rate of 1 cm per hour beyond 3 cm dilatation)
(b) Associated presence of contracted pelvis, malposition, deflexed head or malpresentation may be evident
(c) Membranes usually remain intact.

Place of cesarean section:
(1) Presence of contracted pelvis
(2) Malpresentation
(3) Evidences of fetal or maternal distress.

Vaginal delivery –
(A) General measures:
(1) To keep up the morale of the patient. Maternal stress and emotion appear to inhibit uterine contractions through endogenous adrenaline.
(2) Posture of the woman is changed. Supine position is avoided.
(3) To empty the bladder, catheterization is made.
(4) To maintain hydration by infusion of Ringer's solution.
(5) Adequate pain relief.

(B) Active measures: Acceleration of uterine contraction can be brought about by low rupture of the membranes followed by oxytocin drip. The drip rate is gradually increased until effective contractions are set up. The drip is to be continued till one hour after delivery.

INCOORDINATE UTERINE ACTION

The hypertonic state of the uterus arises from any of the conditions such as spastic lower uterine segment, colicky uterus, asymmetrical uterine contraction, constriction ring or generalized tonic contraction of the uterus and all these states are collectively called

incoordinate uterine action. Increased frequency and or duration of uterine contractions. New pacemakers appear all over the uterus.

SPASTIC LOWER SEGMENT – UTERINE CONTRACTION:

(1) Fundal dominance is lacking and often there is reversed polarity.
(2) The pacemakers do not work in rhythm.
(3) The lower segment contractions are stronger.
(4) Inadequate relaxation in between contractions.
(5) Basal tone is raised above the critical level of 20 mm Hg.

Diagnosis:

(1) The patient is in agony with unbearable pain referred to the back.
(2) Bladder is frequently distended.
(3) There are premature attempts to bear down.
(4) Abdominal palpation reveals: (a) Uterus is tender and gentle manipulation excites hardening of the uterus with pain (b) palpation of the fetal parts is difficult
(5) Fetal distress appears early.
(6) Internal examination may reveal: (a) Cervix which is thick, edematous hangs loosely like a curtain; (b) Inappropriate dilatation of the cervix (c) Absence of the membranes; (d) Meconium stained liquor amnii may be there.

Management:

There is no place of oxytocin augmentation with this abnormality. Cesarean section is done in majority of cases. Prior correction of dehydration and ketoacidosis must be achieved by rapid infusion of Ringer's solution.

CONSTRICTION RING *(Syn: Contraction ring, Schroeder's ring)*:

It is usually situated at the junction of the upper and lower segment around. The common causes are:

1. injudicious administration of oxytocics;
2. premature rupture of the membranes;
3. premature attempt at instrumental delivery.

Treatment:

Delivery is usually done by cesarean section.

CERVICAL DYSTOCIA: Failure of cervical dilatation may be due to:

Inefficient uterine contractions

(a) Malpresentation, malposition (abnormal relationship between the cervix and the presenting part)
(b) Spasm (contractions) of the cervix. Cervical dystocia may be primary or secondary.

Primary: Commonly observed during the:

1. First birth where the external os fails to dilate.
2. Rigid cervix.
3. Inefficient uterine contractions and the others. **Treatment:** In presence of associated complications (malpresentation, malposition), cesarean section is preferred.

Secondary cervical dystocia:

1. Post delivery.
2. Postoperative scarring.
3. Cervical cancer.
4. **Geniralized tonic contraction** *(Syn: Uterine tetany)*:
5. Thus, there is no physiological differentiation of the active upper segment and the passive lower segment of the uterus. The whole uterus undergoes a sort of tonic muscular spasm holding the fetus inside. New pacemakers appear all over the uterus.

Clinical features: The patient is in prolonged labor having severe and continuous pain. Abdominal examination reveals the uterus to be somewhat smaller in size, tense and tender. Fetal parts are neither well defined, nor is the fetal heart sound audible. Vaginal examination reveals jammed head with big caput; dry and edematous vagina.

Treatment:

– Correction of dehydration and ketoacidosis – by rapid infusion of Ringer's solution

– Antibiotic – to control infection

– Adequate pain relief. Cesarean delivery is done in majority of the cases.

PRECIPITATE LABOR

A labor is called precipitate when the combined duration of the first and second stage is less than two hours. Labor is short as the rate of cervical dilatation is 5 cm/h or more for the nulliparous women.

Maternal risks include:

(1) Extensive laceration of the cervix, vagina and perineum (to the extent of complete perineal tear).
(2) Uterine rupture.
(3) Infection.
(4) Amniotic fluid embolism. The fetal risks include – intracranial stress and hemorrhage.

Treatment: Delivery of the head should be controlled.

TONIC UTERINE CONTRACTION AND RETRACTION *(Syn: Bandl's ring, Pathological retraction ring).*

A circular groove encircling the uterus is formed between the active upper segment and the distended lower segment, called pathological retraction ring (Bandl's ring).

Clinical features:

(1) Patient is in agony from continuous pain and discomfort and becomes rest-less.
(2) Features of exhaustion and ketoacidosis are evident.
(3) Abdominal palpation reveals:
(a) Upper segment is hard and tender.
(b) Lower seg-ment is distended and tender.

Treatment:

– Rupture of uterus is to be excluded

– Internal version is contraindicated
– Correction of dehydration and ketoacidosis by infusion of Ringer's solution
– Adequate pain relief
– Parenteral antibiotic is given (Ceftriaxone 1 g IV)
– Cesarean delivery is done in majority of the cases. Rupture of uterus must be excluded before attempting destructive operation.

See ***the differential diagnosis*** in Fig. 16, Fig. 17.

Abdominal examination	(a) Uterus feels normal and not tender (b) Fetal parts are easily felt (c) Ring is not felt (d) Round ligament is not felt (e) FHS is usually present	(a) Uterus is tense and tender (b) Not easily felt (c) Ring is felt as a groove placed obliquely (d) Round ligaments are taut and tender (e) Usually absent
Vaginal examination	(a) Lower segment is not pressed by the presenting part (b) Ring is felt usually above the head (c) Features of obstructed labor are absent	(a) Lower segment is very much pressed by the forcibly driven presenting part (b) Ring cannot be felt vaginally (c) Features are present
End result	(a) Maternal exhaustion is a late feature (b) Fetal anoxia usually appear late (c) Chance of uterine rupture is absent	(a) Maternal exhaustion and sepsis appear early (b) Fetal anoxia and even death are usually early (c) Rupture uterus in multigravidae is common
Principle of treatment	To relax the ring followed by delivery of the baby or to cut the ring during cesarean section. Cesarean delivery and to cut the ring, if needed	Cesarean delivery after excluding rupture uterus

Fig. 17. Differential diagnosis

	CONSTRICTION RING	RETRACTION RING
Situation	Usually at the junction of upper and lower segment but may occur in other places. Once formed the position does not alter	Always situated at the junction of upper and lower segment The position progressively moves upwards
Uterus	Upper segment contracts and retracts with relaxation in between; lower segment remains thick and loose. (Fig. 24.3) • Polarity is abnormal (p 358)	Upper segment is tonically contracted with no relaxation The wall becomes thicker; lower segment becomes distended and thinned out (Fig. 24.5) • Polarity is normal
Maternal condition	Almost unaffected unless the labor is prolonged	Features of maternal exhaustion, sepsis appear early

Fig. 18. Differential diagnosis (cont.)

FACE PRESENTATION

Face is a rare variety of cephalic presentation where the presenting part is the face. The attitude of the fetus shows complete flexion of the limbs with extension of the spine. There is complete extension of the head so that the occiput is in contact with the back. The denominator is mentum.

Incidence: Its frequency is about 1 in 500 births. It occurs more frequently in multiparae (70%).

Etiology:

Maternal:

(1) Multiparity with pendulous abdomen,
(2) Lateral obliquity of the uterus especially,
(3) Contracted pelvis is associated in about 40% cases. Flat pelvis favors face presentation,
(4) Pelvic tumors.

Fetal:

1. Congenital malformations (15%):
   (a) The commonest one is anencephaly.
   (b) Congenital goiter – prevalent in endemic areas,
   (c) Dolichocephalic head with long anteroposterior diameter,
   (d) Congenital branchocele.
2. Twist.
3. Increased tone of the extensor group of neck muscles.

MECHANISM OF LABOR

MENTOANTERIOR 60–80% (LMA OR RMA):

Engagement: the mentumand the glabella to the opposite sacroiliac joint. The engaging diameter of the head is submento-bregmatic 9.5 cm. Internal rotation – Further descent occurs till the submentum hinges under the pubic arch. Delivery of the head – The head

is born by flexion delivering the chin, face, brow, vertex and lastly the occiput. This follows delivery of the anterior shoulder followed by the posterior shoulder and the rest of the trunk by lateral flexion.

MENTOPOSTERIOR (20–25%) (RMP OR LMP):

The salient differentiating features are –

(1) In the mentoposterior position, anterior rotation of the mentum occurs in only 20–30% cases.

(2) In the rest (70–80%), there is no possibility of spontaneous delivery in persistent mentoposterior. This is because the relatively short neck cannot clear off the total length of the sacrum (12 cm).

Diagnosis (see Fig. 19)

	Mentoanterior	*Mentoposterior*
Lateral grip	(1) Fetal limbs are felt anteriorly. (2) Back is on the flank and is difficult to palpate. (3) The chest is thrown anteriorly against the uterine wall and is often mistaken for back	(1) Back is felt to the front and better palpated only towards the podalic pole because of extension of spine.
Pelvic grip	(1) Head seems big and is not engaged. (2) Cephalic prominence is to the side towards which back lies (3) Groove between the head and back is not so prominent.	(1) Same (2) Same (3) The groove is prominent.
Auscultation	FHS is distinctly audible anteriorly through the chest wall of the fetus towards the side of limbs	FHS is not so distinct and is audible on the flank towards the side of limbs.

Fig. 19. Differential diagnosis

VAGINAL EXAMINATION

1. the mouth and the malar eminences are not in a line;
2. sucking effect of mouth;
3. hard alveolar margins and;
4. absence of meconium staining on the examination fingers.

The clinical course of the latter is adversely affected because of the following:

- Irregular face ill fits.
- Chance of cord prolapse is more.

- Delay of labor, in all the stages, is common. The causes are –

(a) weak uterine contractions,
(b) absence of moulding of the facial bones,
(c) delayed engagement – the distance between the biparietal plane to chin is 7 cm and to occiput is only 3 cm;
(d) late internal rotation and
(e) arrest and at times.

- Chance of perineal damage.
- Postpartum hemorrhage

Management

Indications of elective or early cesarean section:

(1) Contracted pelvis,
(2) Big baby,
(3) Associated complicating factors.

Vaginal delivery

MENTOANTERIOR

First stage: In uncomplicated cases, a wait and watch policy is adopted.

Second stage: Perineum should be protected with liberal mediolateral episiotomy.

BROW PRESENTATION

Brow is the rarest variety of cephalic presentation where the presenting part is the brow and the attitude of the head is short of that degree of extension necessary to produce face presentation, i.e. the head lies in between full flexion and full extension.

Vaginal examination: The position is to be confirmed on vaginal examination by palpating supraorbital ridges and anterior fontanelle.

MECHANISM OF LABOR: diameter of the head is mento-vertical (14 cm), there is no mechanism of labor in an average size baby with normal pelvis. However, if the baby is small and the pelvis is roomy with good uterine contractions, delivery can occur in mento-anterior brow position. There is no mechanism in posterior brow position.

COURSE AND PROGNOSIS: It is an important cause of rupture of uterus in multiparae. On occasion (10%), there may be spontaneous conversion of brow into face or vertex presentation.

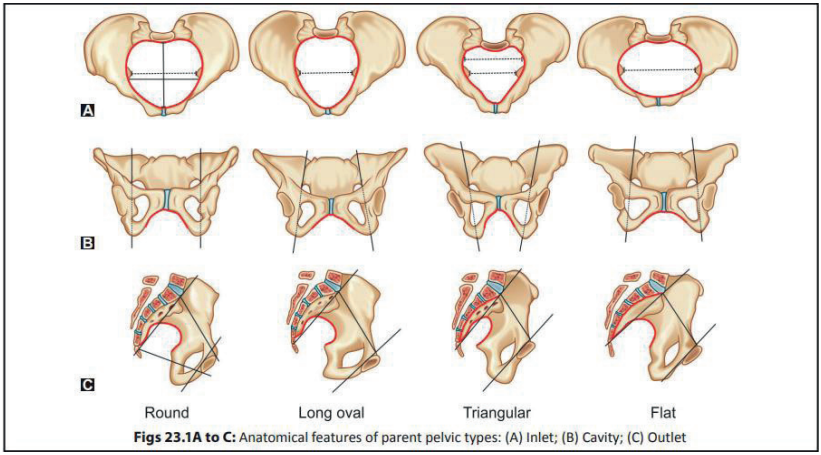
Cases with persistent brow presentation are delivered by elective cesarean section.

CONTRACTED PELVIS

The obstetric definition which states that alteration in the size and/or shape of the pelvis of sufficient degree so as to alter the normal mechanism of labour in an average size baby.

Classificasion:

- Gynecoid (50%)
- Anthropoid (25%)
- Android (20%)
- Platypelloid (5%) (see Fig. 20.)



Figs 23.1A to C: Anatomical features of parent pelvic types: (A) Inlet; (B) Cavity; (C) Outlet

Fig. 20. Types of pelvis

Diagnostics: (see Fig. 21, Fig. 22).

Table 23.1: Anatomical features of parent pelvic types (Figs 23.1A to C)					
		Gynecoid	*Anthropoid*	*Android*	*Platypelloid*
Inlet	• Shape	Round	Antero-posteriorly oval	Triangular	Transversely oval
	• Anterior and posterior segment	Almost equal and spacious	Both increased with slight anterior narrowing	Posterior segment short and anterior segment narrow	Both reduced-flat
	• Sacrum	Sacral angle (SA) more than 90°. Inclined backwards. Well curved from above down and side to side	SA more than 90°. Inclined posteriorly. Long and narrow. Usual curve	Sacral angle less than 90°. Inclined forwards and straight	SA more than 90°. Inclined posteriorly. Short and straight
Cavity	• Sacrosciatic notch	Wide and shallow	More wide and shallow	Narrow and deep	Slightly narrow and small
	• Side walls	Straight or slightly divergent	Straight or divergent	Convergent	Divergent
Outlet	• Ischial spines	Not prominent	Not prominent	Prominent	Not prominent
	• Pubic arch	Curved	Long and curved	Long and straight	Short and curved
	• Subpubic angle	Wide (85°)	Slightly narrow	Narrow	Very wide (more than 90°)
	• Bituberous diameter	Normal	Normal or short	Short	Wide

Fig. 21. Differential diagnosis

Table 23.2: Obstetric outcome in parent pelvic types					
Inlet	• Position	Occipitolateral or oblique occipito-anterior	Direct occipito-anterior or posterior	Occipito-lateral or oblique occipito-posterior	Occipito-lateral
	• Diameter of engagement	Transverse or oblique	Anteroposterior	Transverse or oblique	Transverse
	• Engagement	No difficulty. Usual mechanism	No difficulty except flexion is delayed	Delayed and difficult	Difficult by exaggerated parietal presentation
Cavity	• Internal rotation	Easy anterior rotation	Non-rotation common	Difficult anterior rotation. Not occurs early above the ischial spines, chance of arrest	Anterior rotation usually occurs late in the perineum
Outlet	• Delivery	No difficulty	More incidence of face-to-pubis delivery	Difficult delivery with increased chance of perineal injuries	No difficulty

Fig. 22. Differential diagnosis (cont.)

ETIOLOGY OF CONTRACTED PELVIS:

Common causes of contracted pelvis are:

Nutritional and environmental defects –

- Minor variation: common
- Major: Rachitic and osteomalacic – rare.

Diseases or injuries affecting the bones of the pelvis – fracture, tumours, tubercular arthritis; Spine – Kyphosis, scoliosis, spondylolisthesis, coccygeal deformity; Lower limbs – Poliomyelitis, hip joint disease.

Development defects – Naegele's pelvis, Robert's pelvis; High or low assimilation pelvis.

RACHITIC FLAT PELVIS:

Inlet: Sacral promontory is pushed downwards and forwards producing a "reniform" shape of the inlet with marked shortening of the antero-posterior diameter without affecting the transverse diameter, which is often increased.

Cavity: Sacrum is flat and tilted backwards. There may be sharp angulation at the sacrococcygeal joint.

Outlet: Body weight transmitted through the ischium in sitting position results in widening of the transverse diameter of the outlet and the pubic arch.

OSTEOMALACIC PELVIS:

The promontory is pushed downwards and forwards and the lateral pelvic walls are pushed inwards causing the anterior wall to form a beak. The shape of the inlet thus becomes triradiate. Approximation of the two ischial tuberosities occurs. Sacrum is markedly shortened, Coccyx is pushed forward. Vaginal delivery is unlikely and cesarean section is ideal.

ASYMMETRICAL OR OBLIQUELY CONTRACTED PELVIS

Naegele's pelvis: This type of pelvis is extremely rare. It is produced due to arrested development of one ala of the sacrum. It may be (i) Congenital or (ii) Acquired (osteitis of sacroiliac joint). Congenital variety may be associated with urinary tract and may of the same side. The pelvis is obliquely contracted at all levels but more marked in the outlet. Ilio-pectineal line on the affected side is almost straight. Method of delivery is by cesarean section.

Scoliosis involving only the lumbar region will cause deformity of the pelvis. Cesarean section is the only safe method of delivery.

Robert's pelvis (transversely contracted pelvis): This is an extremely rare abnormality. Ala of both the sides are absent and the sacrum is fused with the innominate bones. Delivery is done by cesarean section.

Kyphotic pelvis: This pelvic deformity is secondary to the kyphotic changes of the vertebral column either following tuberculosis or rickets. Cesarean section.

FLAT PELVIS

The head negotiates the brim by the following mechanism:

The head engages with the sagittal suture in the transverse diameter.

Head remains deflexed and engagement is delayed.

If the anteroposterior diameter is too short, the occiput is mobilized to the same side, to occupy the sacral bay. The biparietal diameter is thus placed in the sacro-cotyloid diameter (9.5 cm or 8.5 cm) and the narrow bitemporal diameter is placed in the narrow conjugate. If lateral mobilization is not possible, there is a chance of extension of the head leading to brow or face presentation.

Engagement occurs by exaggerated parietal presentation so that the super-subparietal diameter (8.5 cm), instead of the biparietal diameter (9.5 cm), passes through the pelvic brim.

Moulding may be extreme and often there is an indentation or even a fracture of one parietal bone. However, the caput that forms is not big.

Once the head negotiates the brim, there is no difficulty in the cavity and outlet and normal mechanism follows.

DISPROPORTION

Definition: The disparity in the relation between the head and the pelvis is called cephalopelvic disproportion.

Pelvic inlet contraction is considered when the obstetric conjugate is < 10 cm or the greatest transverse diameter is < 12 cm or diagonal conjugate is < 11 cm. Contracted Midpelvis: Midpelvis is considered contracted when the sum of the inter-ischial spinous and posterior sagittal diameters of the mid pelvis (normal: 10.0 + 5 = 15.0 cm) is 13.0 cm or below.

Contracted outlet is suspected when the inter ischial tuberous diameter is 8 cm or less.

DIAGNOSIS OF CEPHALOPELVIC DISPROPORTION (CPD) AT THE BRIM

Clinical –

- Abdominal method;
- Abdominovaginal (Muller-Munro Kerr);
- Imaging pelvimetry (see above).

Cephalometry –

- Ultrasound;
- Magnetic Resonance Imaging;
- X-ray

Inferences:

– The head can be pushed down in the pelvis without overlapping of the parietal bone on the symphysis pubis – no disproportion.

– Head can be pushed down a little but there is slight overlapping of the parietal bone evidenced by touch on the under surface of the

fingers (overlapping by 0.5 cm or 1/4" which is the thickness of the symphysis pubis) – moderate disproportion.

– Head cannot be pushed down and instead the parietal bone overhangs the symphysis pubis displacing the fingers – severe disproportion.

Abdominovaginal method (Muller-Munro Kerr):

Muller introduced the method by placing the vaginal finger tips at the level of ischial spines to note the descent of the head.

EFFECTS OF CONTRACTED PELVIS ON PREGNANCY AND LABOR

Labor:

– Increased incidence of early rupture of the membranes;

– Incidence of cord prolapse is increased;

– Cervical dilatation is slowed;

– There is increased tendency of prolonged labor and in neglected cases, obstructed labor with features of exhaustion, dehydration, keto-acidosis and sepsis;

– There is increased incidence of operative interference, shock, postpartum; and hemorrhage and sepsis.

Maternal injuries:

The injuries of the genital tract may occur spontaneously or following operative delivery. There is increased maternal morbidity and mortality.*Fetal hazards:* Fetal risks are due to trauma and asphyxia. The net effect leads to increased perinatal mortality and morbidity.

MIDPELVIC AND OUTLET DISPROPORTION

Cephalopelvic disproportion at the outlet is defined as one where the biparietal-suboccipitobregmatic plane fails to pass through the bispinous and anteroposterior planes of the outlet.

To allow vaginal delivery:

Labor progress should be mapped with a partograph to make an early diagnosis of dysfunctional labor due to disproportion. Oxytocin may be used to augment labor for adequate uterine contractions.

If there is no dilatation of cervix or descent of the fetal head after a period of 2 hours in the active phase of labor, arrest of labor is considered. Once arrest disorder is diagnosed, cesarean delivery is the option.

CASES SEEN LATE IN LABOR

The principles of management rest on:

– Cesarean section to avoid difficult forceps;

– Forceps with deep episiotomy;

– Symphysiotomy followed by ventouse or

– Craniotomy if the fetus is dead.

HEMORRHAGE IN EARLY PREGNANCY

The causes of bleeding in early pregnancy are broadly divided into two groups:

Those related to the pregnant state: This group relates to abortion (95%), ectopic pregnancy, hydatidiform mole and implantation bleeding.

Those associated with the pregnant state: The lesions are unrelated to pregnancy—either pre-existing or aggravated during pregnancy. Cervical lesions such as vascular erosion, polyp, ruptured varicose veins and malignancy are important causes.

SPONTANEOUS ABORTION (MISCARRIAGE)

Definition: Abortion is the expulsion or extraction from its mother of an embryo or fetus weighing 500 g or less when it is not capable of independent survival. This 500 g of fetal development is attained approximately at 22 weeks (154 days) of gestation. The term miscarriage is the recommended terminology for spontaneous abortion. 75% abortions occur before the 16th week and of these, about 75% occur before the 8th week of pregnancy.

CLASSIFICATION OR VARIETIES: (see Fig. 23.)

Etiology:

- The etiology of miscarriage is often complex and obscure. The following factors (embryonic or parental) are important:
- Genetic
- Endocrine and metabolic
- Anatomic
- Infection
- Immunological
- Antifetal antibodies
- Thrombophilias
- Others

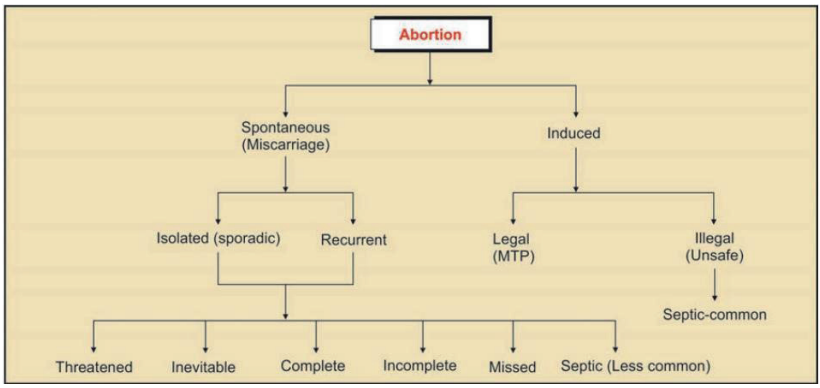


Fig. 23. Classification and differential diagnosis

Blood group incompatibility:

Rh incompatibility is a rare cause of death of the fetus before 28th week. Couple with group 'A' husband and group 'O' wife have got higher incidence of abortion.

THREATENED MISCARRIAGE

Definition: It is a clinical entity where the process of miscarriage has started but has not progressed to a state from which recovery is impossible.

Clinical features:

Bleeding per vaginam;

Pain: Bleeding is usually painless but there may be mild backache or dull pain in lower abdomen. Pain appears usually following hemorrhage.

Treatment:

Rest: The patient should be in bed for few days until bleeding stops.

Drugs: Relief of pain may be ensured by diazepam 5 mg tablet twice daily.ADVICE ON DISCHARGE: The patient should limit her

activities for at least two weeks and avoid heavy work. Coitus is avoided during this period. She should be followed up with repeat sonography at 3–4 weeks time.

Prognosis: In isolated spontaneous threatened miscarriage, the following events may occur. If the pregnancy continues, there is increased frequency of preterm labor, placenta previa, intrauterine growth restriction of the fetus and fetal anomalies.

INEVITABLE MISCARRIAGE

Definition: It is the clinical type of abortion where the changes have progressed to a state from where continuation of pregnancy is impossible.

CLINICAL FEATURES:

- Increased vaginal bleeding
- Aggravation of pain in the lower abdomen which may be colicky in nature
- Internal examination reveals dilated internal os of the cervix through which the products of conception are felt.

Management is aimed:

- to accelerate the process of expulsion
- to maintain strict asepsis.

COMPLETE MISCARRIAGE

Definition: When the products of conception are expelled en masse, it is called complete miscarriage.

Management: Transvaginal sonography is useful to see that uterine cavity is empty, otherwise evacuation of uterine curettage should be done.

Rh-negative women: A Rh-negative patient without antibody in her system should be protected by Anti-D gamma globulin-50 microgram or 100 microgram intramuscularly in cases of early miscarriage or late miscarriage respectively within 72 hours.

INCOMPLETE MISCARRIAGE

Definition: When the entire products of conception are not expelled, instead a part of it is left inside the uterine cavity, it is called incomplete miscarriage.

Complication: The retained products may cause:

- profuse bleeding
- sepsis or
- placental polyp.

Management: She should be resuscitated before any active treatment is undertaken.

- Early abortion: Dilatation and evacuation under analgesia or general anesthesia is to be done.
- Late abortion: The removed materials are subjected to a histological examination.
- Medical management of incomplete miscarriage may be done. Tablet Misoprostol 200 μg is used vaginally every 4 hours.

MISSED MISCARRIAGE

Definition: When the fetus is dead and retained inside the uterus for a variable period, it is called missed miscarriage or early fetal demise.

Clinical features:

- Persistence of brownish vaginal discharge
- Subsidence of pregnancy symptoms
- Retrogression of breast changes
- Cessation of uterine growth which in fact becomes smaller in size
- Nonaudibility of the fetal heart sound even with Doppler ultrasound if it had been audible before
- Cervix feels firm
- Immunological test for pregnancy becomes negative
- Real time ultrasonography reveals an empty sac early in the pregnancy or the absence of fetal motion or fetal cardiac movements.

Management:

Uterus is less than 12 weeks:

- Expectant management – Many women expel the conceptus spontaneously
- Medical management: Prostaglandin E1 (Misoprostol) 800 mg vaginally in the posterior fornix is given and repeated after 24 hours if needed. Expulsion usually occurs within 48 hours
- Suction evacuation or dilatation

Uterus more than 12 weeks: Induction is done by the following methods:

(a) Prostaglandin E1 analogue (misoprostol) 200 µg tablet is inserted into the posterior vaginal fornix every 4 hours for a maximum of 5 such.
(b) Oxytocin—10–20 units of oxytocin in 500 mL of normal saline at 30 drops per minute is started. If fails, escalating dose of oxytocin to the maximum of 200 mlU/min, may be used with monitoring.
(c) Dilatation and evacuation is done.

SEPTIC ABORTION

Definition: Any abortion associated with clinical evidences of infection of the uterus and its contents, is called septic abortion. Abortion is usually considered septic when there are:

- rise of temperature of at least 100.4°F (38°C) for 24 hours or more
- offensive or purulent vaginal discharge and
- other evidences of pelvic infection such as lower abdominal pain and tenderness.

Majority of cases the infection occurs following illegal induced abortion.

The microorganisms are: (a) Anaerobic – Bacteroides group (fragilis), anaerobic Streptococci, Cl. welchii, and tetanus bacillus (b) Aerobic – Escherichia coli (E. coli), Klebsiella, Staphylococcus, Pseudomonas and hemolytic Streptococcus (usually exogenous). Mixed infection

is more common. In about 5%, there is generalized peritonitis and/or endotoxic shock.

Clinical grading:

Grade–I: The infection is localized in the uterus. Grade–II: The infection spreads beyond the uterus to the parametrium, tubes and ovaries or pelvic peritoneum. Grade–III: Generalized peritonitis and/or endotoxic shock or jaundice or acute renal failure.

Management:

Grade–I

Drugs: (1) Antibiotics. (2) Prophylactic anti-gas-gangrene serum of 8000 units and 3000 units of antitetanus serum intramuscularly are given. (3) Analgesics and sedatives.

Blood transfusion is given to improve anemia and body resistance.

Evacuation of the uterus: As abortion is often incomplete, evacuation should be performed at a convenient time within 24 hours following antibiotic therapy.

Grade–II

Drugs: Antibiotics – Mixed infections including Gram-positive, Gram-negative and anaerobic organisms are common. Ideal antibiotic regimens should cover all of them.

For Gram-positive aerobes: (I) penicillin G 5 million units IV every 6 hours, or (II) Ampicillin 0.5–1 g IV every 6 hours. B. Gram-negative aerobes: (I) Gentamicin 1.5 mg/kg IV every 8 hours, or (II) Ceftriaxone IG, IV every 12 hours. C. For anaerobes: Metronidazole 500 mg IV every 8 hours, or Clindamycin 600 mg IV every 6 hours.

Grade–III

Active Surgery: Indications are – (1) Injury to the uterus (2) Suspected injury to bowel (3) Presence of foreign body in the abdomen as evidenced by the sonography or X-ray or felt through the fornix on bimanual examination (4) Unresponsive peritonitis suggestive

of collection of pus (5) Septic shock or oliguria not responding to the conservative treatment (6) Uterus too big to be safely evacuated per vaginam. Even when nothing is found on laparotomy, simple drainage of the pus is effective.

RECURRENT MISCARRIAGE

Definition: Recurrent miscarriage is defined as a sequence of three or more consecutive spontaneous abortion before 20 weeks. Some however, consider two or more as a standard.

Etiology

- Genetic factors (3–5%): The most common abnormality is a balanced translocation.
- Endocrine and Metabolic: (1) Poorly controlled diabetic; (2) Presence of thyroid autoantibodies; (3) Luteal phase defect (LPD); (4) Hypersecretion of luteinizing hormone.
- Transplacental fetal infection can occur with most microorganisms.
- Inherited thrombophilia causes both early and late miscarriages due to intravascular (spiral artery), and placental intervillous thrombosis. Hyperhomocystinemia (autosomal recessive disorder) is also a risk factor for recurrent miscarriage.
- Immunological cause: autoimmunity; alloimmunity.

CERVICAL INCOMPETENCE (CERVICAL INSUFFICIENCY) – 20%

During pregnancy –

- Clinical digital: Painless cervical shortening and dilatation.
- Sonography: Short cervix < 25 mm; Funnelling of the internal Os > 1 cm.

Treatment:

- Rest
- Luteal Phase Defect (LPD) cases are treated with natural micronized progesterone 100 mg daily as vaginal suppository.

It is started 2 days after ovulation; is continued until 10–12 weeks gestation.

- Antiphospholipid antibody syndrome (APS): low dose aspirin (50 mg/day), and heparin (5000 units SC twice daily) up to 34 weeks.

Circlage operation:

Shirodkar – The bulging membranes, if present, must be gently reduced beforehand into the uterine cavity. The anterior and posterior incisions are repaired by interrupted stitches using chromic catgut.

McDonald – The suture starts at the anterior wall of the cervix. Taking successive deep bites (4–5 sites) it is carried around the lateral and posterior walls back to the anterior wall again where the two ends of the suture are tied.

Postoperative care: (1) The patient should be in bed for at least 2–3 days (2) Weekly injections of 17 α hydroxy progesterone caproate 500 mg IM is given in women with history of prior preterm delivery (3) Isoxsuprine (tocolytics) 10 mg tablet may be given thrice daily to avoid uterine irritability.

Advice on discharge: (a) Usual antenatal advice (b) To avoid intercourse (c) To avoid rough journey (d) To report if there is vaginal bleeding or abdominal pain (e) Periodic ultrasonographic monitoring of the fetus and the cervix.

GESTATIONAL TROPHOBLASTIC DISEASES (GTD)

Definition:

Gestational Trophoblastic Disease (GTD) encompasses a spectrum of proliferative abnormalities of trophoblasts associated with pregnancy.

HYDATIDIFORM MOLE (Syn: Vesicular mole)

Types:

- Complete
- Incomplete (partial)

Definition:

It is an abnormal condition of the placenta where there are partly degenerative and partly proliferative changes in the young chorionic villi. It is best regarded as a benign neoplasia of the chorion with malignant potential. INCIDENCE: The highest incidence is in Philippines being 1 in 80 pregnancies; in India, is about 1 in 400.

Etiology: – Its prevalence is highest in teenage pregnancies and in those women over 35 years of age and ethnic origin.

Low dietary intake of carotene is associated with increased risk.

Maternal immune mechanisms – (a) Rise in gammaglobulin level in absence of hepatic disease (b) Increased association with AB blood group which possesses no ABO antibody.

Cytogenetic abnormality – In general, complete moles have a 46, XX karyotype (85%), the molar chromosomes are derived entirely from the father. The ovum nucleus may be either absent (empty ovum) or inactivated which has been fertilized by a haploid sperm. This phenomenon is known as androgenesis.

History of prior hydatidiform mole increases the chance of recurrence (1 to 4%).

Pathology of hydatidiform mole

It is principally a disease of the chorion. Microscopic appearance: The basic findings are –

- There is marked proliferation of the syncitial and cytotrophoblastic epithelium
- Marked thinning of the stromal tissue due to hydropic degeneration
- There is absence of blood vessels in the villi which seems primary rather than due to pressure atrophy
- The villus pattern is distinctly maintained.

The contained fluid is rich in chorionic gonadotropin. It also contains estrogen and progesterone.

Diagnostics approach: signs and symptoms (see Fig. 24.)

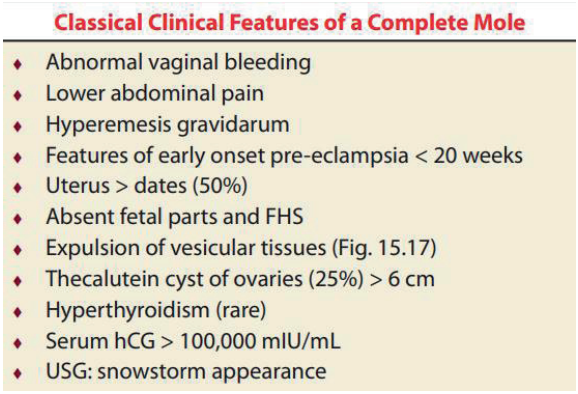


Fig. 24. Diagnostics approach: signs and symptoms

COMPLICATIONS OF MOLAR PREGNANCY

Immediate:

- Hemorrhage and shock;
- Sepsis;
- Perforation of the uterus;
- Pre-eclampsia with convulsion;
- Acute pulmonary insufficiency;
- Coagulation failure.

RISK FACIORS – see Fig. 25.

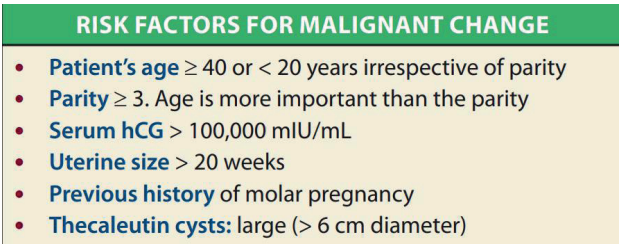


Fig. 25. Risk factors

Management:

The principles in the management are:

Suction evacuation (SE) of the uterus as early as the diagnosis is made.

Supportive therapy: Correction of anemia and infection, if there is any.

Counseling for regular follow-up.

Complications of vaginal evacuation – Apart from the injury to the uterus, hemorrhage and shock, there are two more rare but fatal complications –

- Acute pulmonary insufficiency due to pulmonary embolization of the trophoblastic cells. Symptoms of acute chest pain, tachycardia, tachypnea and dyspnea develop about 4–6 hours following evacuation. Medical induction (oxytocin infusion) before evacuation may increase the risk of pulmonary insufficiency.
- Thyroid storm – In presence of hyperthyroid state when evacuation is done under general anesthesia, the acute features such as hyperthermia, delirium, convulsions, coma and cardiovascular collapse develop. The condition can be managed by administration of beta adrenergic blocking agents.

Profilactic chemotherapy:

Regimes:

Methotrexate, 1 mg/kg/day IV or IM is given on Days 1, 3, 5 and 7 with Folinic acid 0.1 mg/kg IM on Days 2, 4, 6 and 8. It is to be repeated every 7 days. A total 3 courses are given.

Contraceptive advice:

The patient is traditionally advised not to be pregnant for at least one year.

Management: (see Fig. 26.)

IMPORTANT FEATURES OF COMPLETE AND PARTIAL MOLES		
Features	**Complete mole**	**Partial mole**
• Embryo/Fetus	Absent	Present
• Hydropic degeneration of villi	Pronounced and diffused	Variable and focal
• Trophoblast hyperplasia	Diffuse	Focal
• Uterine size	More than the date (30-60%)	Less than the date
• Theca lutein cysts	Common (25-50%)	Uncommon
• Karyotype	46 XX (85%), Paternal in origin	Triploid (90%) diploid (10%)
• β hCG	High (> 50,000)	Slight elevation (< 50,000)
• Classic clinical symptoms	Common	Rare
• Risk of persistent GTD	20%	< 5%

Fig. 26. Management and Patient Monitoring

PERSISTENT GESTATIONAL TROPHOBLASTIC DISEASE

A postmolar GTD may be benign or malignant. But a GTD after non-molar pregnancy is always a choriocarcinoma.

Diagnosis:

- Continued vaginal bleeding
- Persistent theca lutein cysts
- Persistently soft and enlarged uterus
- hCG titers either fail to become negative or remain plateau or there is re-elevation after a initial fall by 8 weeks post-molar evacuation. Local or systemic metastases should always be excluded (X-ray chest, CT, MRI of brain and liver). Asymptomatic patients, with a normal chest X-ray, is unlikely to have brain or other visceral metastasis.
- Pathologically this may be due to invasive mole, choriocarcinoma or placental site trophoblastic tumor.

Treatment: Low risk group receive single agent chemotherapy (usually methotrexate). High risk group receive combination chemotherapy (usually EMA-CO).

Hysterectomy – This is justified in women approaching 40 and/or who has completed her family. Following hysterectomy or chemotherapy, regular follow-up is essential.

Treatment: Low risk group receive single agent chemotherapy (usually methotrexate). High risk group receive combination chemotherapy.

Hysterectomy – This is justified in women approaching 40 and/or who has completed her family. Following hysterectomy or chemotherapy, regular follow-up is essential.

ANTEPARTUM HEMORRHAGE

Definition:

It is defined as bleeding from or into the genital tract after the 28th week of pregnancy but before the birth of the baby (the first and second stage of labor are thus included). The incidence is about 3% amongst hospital deliveries. (Fig. 27.)

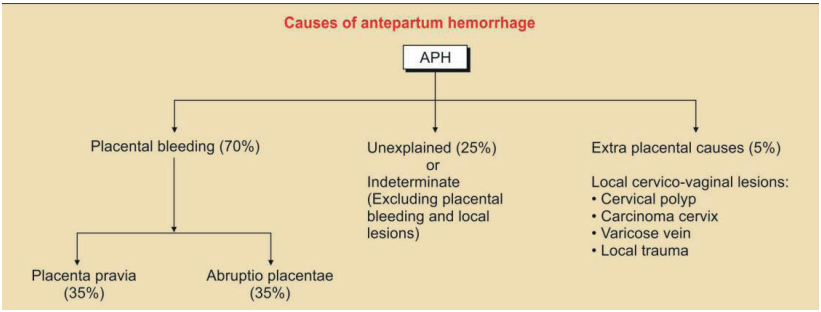


Fig. 27. Causes of antepartum hemorrhages

PLACENTA PREVIA

Definition:

When the placenta is implanted partially or completely over the lower uterine segment (over and adjacent to the internal os) it is called placenta previa.

Incidence

In 80% cases, it is found to multiparous women; beyond the age of 35.

The high-risk factors for placenta previa are —

- Multiparity
- Increased maternal age (> 35 years)
- History of previous cesarean section or any other scar in the uterus (myomectomy or hysterotomy)

- Placental size (mentioned before) and abnormality (succenturiate lobes)
- Smoking — causes placental hypertrophy to compensate carbon monoxide induced hypoxemia.
- Prior curettage.

Types or degrees:

Type – I (Low-lying): The major part of the placenta is attached to the upper segment and only the lower margin encroaches onto the lower segment but not up to the os.

Type – II (Marginal): The placenta reaches the margin of the internal os but does not cover it.

Type – III (Incomplete or partial central): The placenta covers the internal os partially (covers the internal os when closed but does not entirely do so when fully dilated).

Type – IV (Central or total): The placenta completely covers the internal os even after it is fully dilated.

Cause of bleeding:

As the placental growth slows down in later months and the lower segment progressively dilates, the inelastic placenta is sheared off the wall of the lower segment. This leads to opening up of uteroplacental vessels and leads to an episode of bleeding.

Symptoms:

The only symptom of placenta previa is vaginal bleeding. The classical features of bleeeding in placenta previa are sudden onset, painless, apparently causeless and recurrent.

Confirmation of diagnosis (see Fig. 28, Fig. 29)

I. Localization of placenta (placentography)	*II. Clinical*
• Sonography – Transabdominal ultrasound (TAS) – Transvaginal ultrasound (TVS) – Transperineal ultrasound – Color Doppler flow study • Magnetic resonance imaging (MRI)	– By internal examination (double set up examination) – Direct visualization during cesarean section – Examination of the placenta following vaginal delivery

Fig. 28. Diagnosis

Table 18.1: Distinguishing features of placenta previa and abruptio placentae

	Placenta previa	*Abruptio placentae*
♦ **Clinical features:**		
• Nature of bleeding	(a) Painless, apparently causeless and recurrent (b) Bleeding is always revealed	(a) Painful, often attributed to preeclampsia or trauma and continuous (b) Revealed, concealed or usually mixed
• Character of blood	Bright red	Dark colored
• General condition and anemia	Proportionate to visible blood loss	Out of proportion to the visible blood loss in concealed or mixed variety
• Features of preeclampsia	Not relevant	Present in one-third cases
♦ **Abdominal examination:**		
• Height of uterus	Proportionate height to gestational age	May be disproportionately enlarged in concealed type
• Feel of uterus	Soft and relaxed	May be tense, tender and rigid
• Malpresentation	Malpresentation is common. The head is high floating	Unrelated, the head may be engaged
• FHS	Usually present	Usually absent especially in concealed type
♦ **Placentography (USG)**	Placenta in lower segment	Placenta in upper segment
♦ **Vaginal examination**	Placenta is felt on the lower segment	Placenta is not felt on lower segment. Blood clots should not be confused with placenta

Fig. 29. Diferential dagnosis

COMPLICATIONS OF PLACENTA PREVIA

Material:

During pregnancy –

- Antepartum hemorrhage with varying degrees of shock
- Malpresentation
- Premature labor

During labor –

- Early rupture of the membranes
- Cord prolapse
- Slow dilatation of the cervix

- Intrapartum hemorrhage
- Increased incidence of operative interference.
- Postpartum hemorrhage is due to: Placenta previa accreta is a serious complication that may cause maternal death. Often the placenta previa and accreta is managed by hysterectomy.
- Trauma to the cervix and lower segment because of extreme softness and vascularity.
- Retained placenta is due to: (1) increased surface area and (2) morbid adhesion.

Puerperium: (1) Sepsis; (2) Subinvolution; (3) Embolism.

FETAL COMPLICATIONS IN PLACENTA PREVIA

- Low birth weight
- Asphyxia is common and it may be the effect of —
  - early separation of placenta
  - compression of the placenta or
  - compression of the cord.
- Intrauterine
- Birth injuries
- Congenital malformation

Management

AT HOME:

- The patient is immediately put to bed
- To assess the blood loss – (a) inspection of the clothings soaked with blood (b) to note the pulse, blood pressure and degree of anemia
- Quick but gentle abdominal examination to mark the height of the uterus, to auscultate the fetal heart sound and to note any tenderness on the uterus
- Vaginal examination must not be done. Only inspection is done to see whether the bleeding is present or absent and to put a sterile vulval pad.

IMMEDIATE ATTENTION:

Overall assessment of the case is quickly made as regards:

- Amount of the blood loss – by noting the general condition, pallor, pulse rate and blood pressure;
- Blood samples are taken for group, cross matching and estimation of hemoglobin;
- A large-bore IV cannula is sited and an infusion of normal saline is started and compatible cross matched blood transfusion should be arranged;
- Gentle abdominal palpation to ascertain any uterine tenderness and auscultation to note the fetal heart rate
- Inspection of the vulva to note the presence of any active bleeding.

ABRUPTIO PLACENTAE (Syn: Accidental Hemorrhage. Premature Separation Of Placenta)

Definition: It is one form of antepartum hemorrhage where the bleeding occurs due to premature separation of normally situated placenta.

Varieties:

- Revealed: Ultimately, the blood comes out of the cervical canal to be visible externally.
- Concealed: The blood collects behind the separated placenta or collected in between the membranes and decidua. The collected blood is prevented from coming out of the cervix by the presenting part which presses on the lower segment. This type is rare.
- Mixed: In this type, some part of the blood collects inside (concealed) and a part is expelled out (revealed).

- Bleeding is almost always maternal.
- The overall incidence is about 1 in 200 deliveries.

Etiology:

- high birth order pregnancies with gravida 5 and above;
- advancing age of the mother
- poor socio-economic condition

- malnutrition
- smoking (vaso-spasm).

The mechanism of the placental separation in pre-eclampsia is: Spasm of the vessels in the utero placental bed (decidual spiral artery) → anoxic endothelial damage → rupture of vessels or extravasation of blood in the decidua basalis (retroplacental hematoma); 10-50 percent.

– Trauma: Traumatic separation of the placenta usually leads to its marginal separation with escape of blood outside.

– Sudden uterine decompression: This may occur following — (a) delivery of the first baby of twins (b) sudden escape of liquor amnii in hydramnios and (c) premature rupture of membranes.

– Short cord, either relative or absolute, can bring about placental separation during labor by mechanical pull.

– Supine hypotension syndrome

– Placental anomaly

– Sick placenta

– Folic acid deficiency

– Uterine factor: Placenta implanted over a septum (Septate Uterus) or a submucous fibroid.

– Cocaine abuse is associated with increased risk of transient hypertension, vasospasm and placental abruption.

– Thrombophilias increased risk of placental infarcts or abruption.

– Previous abruption varies between 5 to 17%.

Workup and Evaluation (see Fig. 30.)

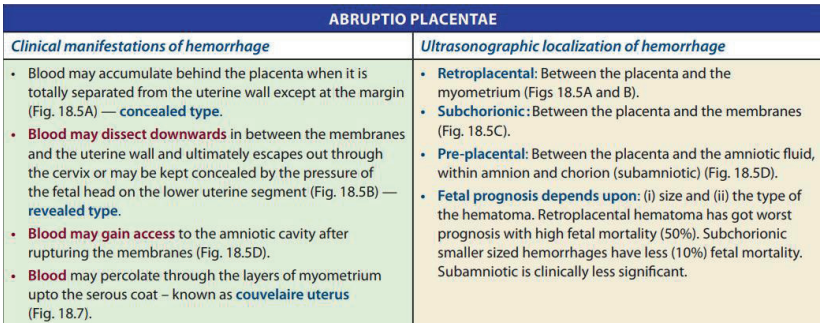


Fig. 30. Workup and Evaluation

Couvelaire uterus (uteroplacental apoplexy):

It is a pathological entity first described by Couvelaire and is met with in association with severe form of concealed abruptio placentae. There is massive intravasation of blood into the uterine musculature upto the serous coat. The condition can only be diagnosed on laparotomy.

Changes in other organs:

In the liver, apart from the changes found in pre-eclampsia, presence of fibrin knots in the hepatic sinusoids is an important finding. Kidneys may show acute cortical necrosis or acute tubular necrosis. The precise mechanism is not clear but may be due to intrarenal vasospasm as a consequence of massive hemorrhage. Shock proteinuria is probably due to renal anoxia which usually disappears two days after delivery, whereas, proteinuria due to pre-eclampsia tends to last longer.

Clinical classification:

Grade – 0: Clinical features may be absent. The diagnosis is made afterinspection of placenta following delivery.

Grade – 1 (40%): (1) Vaginal bleeding is slight (2) Uterus: irritable, tenderness may be minimal or absent (3) Maternal BP and fibrinogen levels unaffected (4) FHS is good.

Grade – 2 (45%): (1) Vaginal bleeding mild to moderate (2) Uterine tenderness is always present (3) Maternal pulse ↑, BP is maintained (4) Fibrinogen level may be decreased (5) Shock is absent (6) Fetal distress or even fetal death occurs.

Grade – 3 (15%): (1) Bleeding is moderate to severe or may be concealed (2) Uterine tenderness is marked (3) Shock is pronounced (4) Fetal death is the rule (5) Associated coagulation defect or anuria may complicate.

Diagnosis (see Fig.31., Fig. 32):

Table 18.2: Clinical features of revealed and mixed variety of abruptio placentae

	Revealed	*Mixed (Concealed features predominate)*
Symptoms:	Abdominal discomfort or pain followed by vaginal bleeding (usually slight)	Abdominal acute intenses pain followed by slight vaginal bleeding. The pain becomes continuous
• Character of bleeding	Continuous dark color (slight to moderate)	Continuous, dark color (usually slight) or blood stained serous discharge
• General condition	Proportionate to the visible blood loss, shock is usually absent.	* Shock may be pronounced which is out of proportion to the visible blood loss.
• Pallor	Related with the visible blood loss	Pallor is usually severe and out of proportion to the visible bleeding
• Features of pre-eclampsia	May be absent	Frequent association
• Uterine height	Proportionate to the period of gestation.	May be disproportionately enlarged and globular.
• Uterine feel	Normal feel with localized tenderness, contractions frequent and local amplitude.	Uterus is tense, tender and rigid
• Fetal parts	Can be identified easily	Difficult to make out
• FHS	Usually present	Usually absent
• Urine output	Normal	Usually diminished

Fig. 31. Clinical features

Laboratory:		
• Blood: Hb%	Low value proportionate to the blood loss	Markedly lower, out of proportion to the visible blood loss
• Coagulation profile	Usually unchanged	Variable changes: • Clotting time increased (> 6 min) • Fibrinogen level-low (< 150 mg/dL) • Platelet count-low • ↑ partial thromboplastin time • ↑ FDP and D-dimer
• Urine for protein	May be absent	Usually present
• Confusion in diagnosis	With placenta previa. As such vaginal examination is withheld unless certain in the diagnosis	With acute obstetrical-gynecological-surgical complications

* **Shock**: Shock is often due to blood loss and hypovolemia or due to coagulopathy. **Mild hemorrhage** (< 15% of the blood volume loss) is generally not associated with any change of vital signs. **Moderate hemorrhage** (15-30% of the blood volume loss) is associated with tachycardia, hypotension, ↓ pulse pressure and mean arterial pressure whereas **severe hemorrhage** (loss > 30-40%) is associated with features of shock.

Fig. 32. Clinical features (cont.)

COMPLICATIONS OF ABRUPTIO PLACENTAE

Maternal:

In revealed type –maternal risk is proportionate to the visible blood loss and maternal death is rare. In concealed variety –

- Hemorrhage;
- Shock;
- Blood coagulation disorders;
- Oliguria and anuria due to – (a) hypovolemia (b) serotonin liberated from the damaged uterine muscle producing renal ischemia and (c) Acute tubular necrosis (d) cortical necrosis and renal failure
- Postpartum hemorrhage due to – (a) atony of the uterus and (b) increase in serum
- Puerperal sepsis.

Fetal: The deaths are due to prematurity and anoxia due to placental separation.

Management of abruptio placentae (see Fig. 33.)

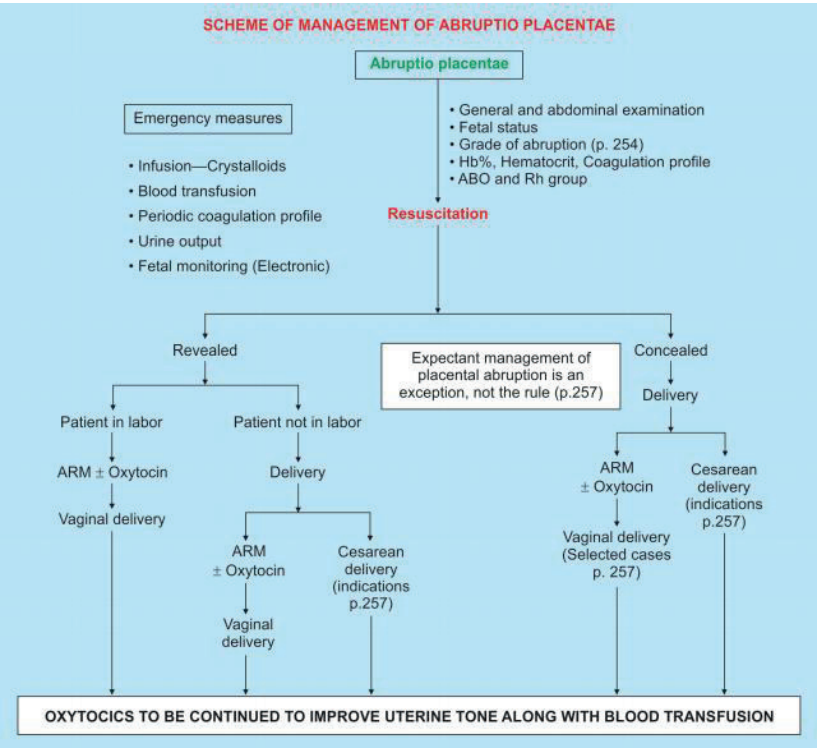


Fig. 33. Management

- Prevention of known factors likely to cause placental separation are:
- Early detection and effective therapy of pre-eclampsia and other hypertensive disorders of pregnancy.
- Needle puncture during amniocentesis should be under ultrasound guidance.
- Avoidance of trauma.
- To avoid sudden decompression of the uterus
- To avoid supine hypotension the patient is advised to lie in the left lateral position in the later months of pregnancy.
- Routine administration of folic acid from the early pregnancy – of doubtful value.

INJURIES TO THE BIRTH CANAL

PERINEUM – gross injury (third and fourth degree) is invariably a result of mismanaged second stage of labor.

Classificasion: (see Fig. 34.)

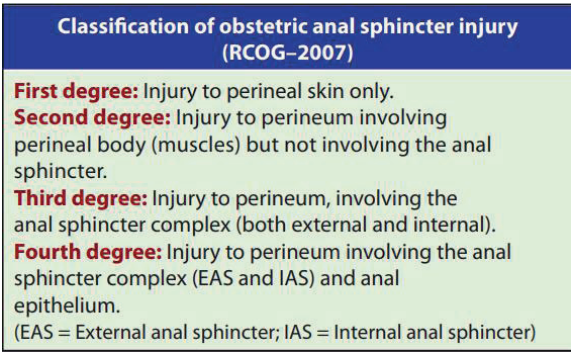


Fig. 34. Classificasion

Risk factors: (see Fig. 35.)

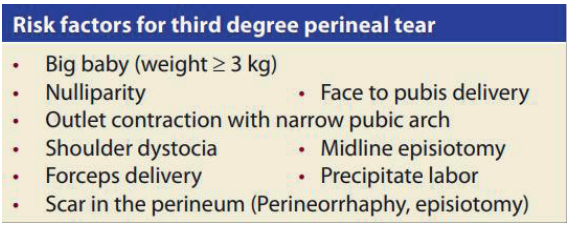


Fig. 35 Risk factors

Management

Recent tear should be repaired immediately following the delivery of the placenta. The complete tear, should be repaired after 3 months, if delayed beyond 24 hours.

Special care following repair of complete tear –

- A low residual diet consisting of milk, bread, egg, biscuits, fish, sweets, etc. is given from 3rd day onwards.
- Lactulose 8 mL twice daily beginning on the second day and increasing the dose to 15 mL on the third day is a satisfactory regime to soften the stool.
- Any one of the broad spectrum antibiotics (IV cefuroxime 1.5 g) is used during the intraoperative and the post-operative period. Metronidazole 400 mg thrice daily is to be continued for 5–7 days to cover the anerobic contamination of fecal matter. The woman is reviewed again 6–12 weeks postpartum.

RUPTURE OF THE UTERUS DEFINITION

Disruption in the continuity of the all uterine layers (endometrium, myometrium and serosa) any time beyond 28 weeks of pregnancy is called rupture of the uterus.

Rupture of the uterus ethiology (see Fig. 36.)

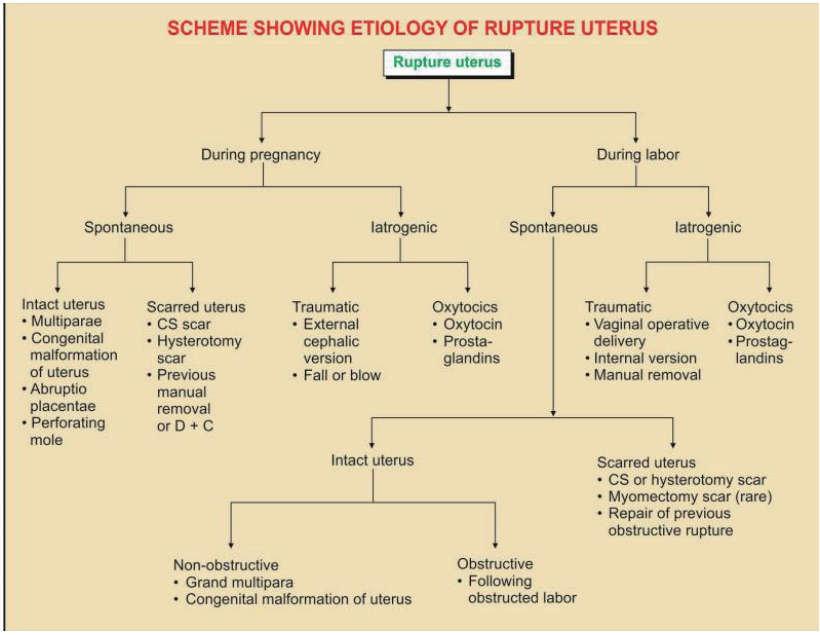


Fig. 36. Ethiology

Pathology

Types: Pathologically, it is customary to distinguish between complete and incomplete rupture depending on whether the peritoneal coat is involved or not.

Incomplete rupture usually results from rupture of the lower segment scar or extension of a cervical tear into the lower segment with formation of a broad ligament hematoma. Complete rupture usually occurs following disruption of the scar in upper segment.

Diagnosis of rupture uterus

This type of spontaneous rupture has got a distinct premonitory phase prior to rupture.

Premonitory phase: The patient is usually a multipara who is in labour with features of obstruction. Initially, the pains become severe in an attempt to overcome the obstruction and come at quick intervals. Gradually, the pains become continuous and mainly confined to the suprapubic region. On examination, the patient is dehydrated and exhausted. The pulse rate and temperature rise. Abdominal examination reveals a distended tender lower segment. Bandl's ring may be visible and there are evidences of fetal distress or FHS may be absent. On vaginal examination, the presenting part is found jammed in the pelvis and the vagina becomes dry and edematous.

Phase of rupture:

– There is a sense of something giving way at the height of uterine contraction

– The constant pain is changed to dull aching pain with cessation of uterine contractions

– General examination reveals features of exhaustion and shock

– Abdominal examination reveals – (I) superficial fetal parts (II) absence of FHS (III) absence of uterine contour and (iv) two separate swellings, one contracted uterus and the other – fetal ovoid

– Vaginal examination reveals – (I) recession of the presenting part and (II) varying degrees of bleeding.

Treatment:

- Resuscitation
- Laparotomy

Hysterectomy: Hysterectomy is the surgery for rupture uterus unless there is sufficient reason to preserve it; a quick subtotal hysterectomy, rather than total hysterectomy.

Repair: This is mostly applicable to a scar rupture where the margins are clean.

To tackle a broad ligament hematoma – To open up the anterior leaf of the broad ligament → Scoop out the blood clot → Secure the bleeding points → Replaced by ligature, taking care not to injure the ureter. Failing to secure the bleeding points → To tie the anterior division of the internal iliac artery.

PUERPERAL SEPSIS

Pueprial sepsis (Syn: Puerperal infection)

Definition: An infection of the genital tract which occurs as a complication of delivery is termed puerperal sepsis.

Puerperal sepsis is commonly due to – (i) Endometritis, (ii) Endomyometritis, or (iii) Endoparametritis or a combination of all these when it is called pelvic cellulitis.

Intrapartum factors:

- Repeated vaginal examinations,
- Prolonged rupture of membranes (> 18 hours),
- Dehydration and keto-acidosis during labor,
- Traumatic operative delivery,
- Hemorrhage – antepartum or postpartum,
- Retained bits of placental tissue or membranes,
- Placenta praevia – placental site lying close to the vagina,
- Cesarean delivery.

PATHOGENESIS

Uterus:

Endomyometritis – The incidence varies from 1–3 percent following vaginal delivery and about 10 percent following cesarean delivery. It is commonly polymicrobial (Group A or B streptococci, Clostridia). The decidua specially over the placental site is primarily affected. The risk factors for endometritis are, retained products of conception, cesarean section, chorioamnionitis, prolonged rupture of membranes, preterm labor and repeated vaginal examinations in labor.

Septicemia and septic shock — may be due to hemolytic streptococci (Streptococcal Toxic Shock syndrome) or anaerobic streptococci.

Septicemia may cause lung abscess, meningitis, pericarditis, endocarditis or multi organ failure. Death occurs in about 30% of cases

Clinical Features

UTERINE INFECTION

Mild –

- There is rise in temperature and pulse rate,
- Lochial discharge becomes offensive and copious.
- The uterus is subinvoluted and tender.
- ***Severe—***
- The onset is acute with high rise of temperature, often with chills and rigor,
- Pulse rate is rapid, out of proportion to temperature,
- Lochia may be scanty and odorless,
- Uterus may be subinvoluted, tender and softer.

*Prophylaxis***:** Ceftriaxone 1 g IV immediately after cord clamping and a second dose after 8 hours is recommended.

Treatment

General care:

- Isolation of the patient is preferred specially when hemolytic streptococcus is obtained on culture,
- Adequate fluid and calorie is maintained by intravenous infusion,
- Anemia is corrected by oral iron or if needed by blood transfusion,
- ***Antibiotics:*** Ideal antibiotic regime should depend on the culture and sensitivity report. Pending the report, Gentamicin (2 mg/kg IV loading dose followed by 1.5 mg/kg IV every eight hours) and Ampicillin (1 g IV every 6 hours) or Clindamycin (900 mg IV every 8 hours) should be started. Intravenous administration of Cefotaxime 1 g, 8 hourly is another alternative.

Metronidazole 0.5 g, IV is given at 8 hours interval to control the anaerobic group. The treatment is continued until the infection is controlled for at least 7–10 days.

- ***Surgical treatment includes:*** wound scrubbing, debridement of all necrotic tissues, and use of effective antimicrobial agents.
- ***Management of bacteremic or septic shock includes:*** Fluid and electrolyte balance, Respiratory supports (to maintain arterial PO2 and PCO2), Circulatory support (dopamine or dobutamine), Infection control (intensive antibiotic therapy, surgical removal of septic foci) and Specific management (as hemodialysis for renal failure).

CESAREAN SECTION

Definition:

It is an operative procedure whereby the fetuses after the end of 28th week are delivered through an incision on the abdominal and uterine walls

Indications (see Fig. 37., Fig. 38.)

ABSOLUTE INDICATIONS	RELATIVE INDICATIONS
Vaginal delivery is not possible. Cesarean is needed even with a dead fetus Indications are few:	Vaginal delivery may be possible but risks to the mother and/or to the baby are high More often multiple factors may be responsible
1. Central placenta previa	1. Cephalopelvic disproportion (relative) see p. 352
2. Contracted pelvis or cephalopelvic disproportion (absolute)	2. Previous cesarean delivery (p. 330)—(a) when primary CS was due to recurrent indication (contracted pelvis). (b) Previous two CS (c) Features of scar dehiscence. (d) Previous classical CS
3. Pelvic mass causing obstruction (cervical or broad ligament fibroid)	3. Non-reassuring FHR (fetal distress)
4. Advanced carcinoma cervix	4. Dystocia may be due to (three Ps) relatively large fetus (passenger), small pelvis (passage) or inefficient uterine contractions (Power)

Fig. 37. Indicasions

5. Vaginal obstruction (atresia, stenosis)	5. Antepartum hemorrhage (a) Placenta previa and (b) Abruptio placenta (p. 249, 257)
COMMON INDICATIONS Primigravidae : (1) Failed indication (2) Fetal distress (non-reassuring fetal FHR) (3) Cephalo pelvic disproportion (CPD) (4) Dystocia (dysfuctional labour p. 359) nonprogress of labour (5) Malposition and malpresentation (occipito-posterior, breech).	6. Malpresentation—Breech, shoulder (transverse lie), brow (p. 381) 7. Failed surgical induction of labor, Failure to progress in labor (p. 522) 8. Bad obstetric history—with recurrent fetal wastage 9. Hypertensive disorders—(a) Severe pre-eclampsia, (b) Eclampsia—uncontrolled fits even with antiseizure therapy (p. 229, 236)
Mutigravidae : (1) Previous Caesarean delivery (2) Antepartum haemorrhage (placenta praevia, placental abruption) (3) Malpresentation (breech, transverse lie).	10. Medical-gynecological disorders—(a) Diabetes (uncontrolled), heart disease (coarctation of aorta, Marfan's syndrome. (b) Mechanical obstruction (due to benign or malignant pelvic tumors (carcinoma cervix), or following repair of vesicovaginal fistula

Fig. 38. Indicasions (cont.)

– LOWER SEGMENT CESAREAN SECTION (LSCS)

Preoperative preparation (see Fig.39.)

- Informed written permission for the procedure, anesthesia and blood transfusion is obtained.
- Abdomen is scrubbed with soap and nonorganic iodide lotion. Hair may be clipped.
- The stomach should be emptied.
- Bladder should be emptied by a Foley catheter.
- Anesthesia – may be spinal, epidural or general.
- The patient is placed in the dorsal position.

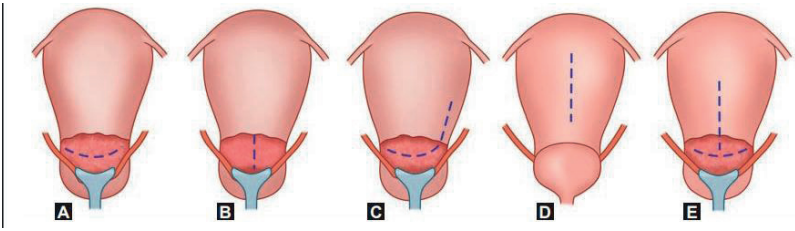


Fig. 39. Uterine incision for Caesarean section

Transverse incision, modified Pfannenstiel is made 3 cm above the symphysis pubis.

Uterine incision

- Peritoneal incision
- Muscle incision – The most commonly used incision (90%) is low transverse.
- Delivery of the head.
- Delivery of the trunk: After the delivery of the shoulders, intravenous oxytocin 20 units or methergin 0.2 mg is to be administered. The optimum interval between uterine incision and delivery should be less than 90 seconds.
- Removal of the placenta and membranes.
- Suture of the uterine wound. The uterine incision is sutured in three layers.

– First layer – The first stitch is placed on the far side in the lateral angle of the uterine incision and is tied. The suture material is No '0' chromic catgut or vicryl and the needle is round bodied. A continuous running suture taking deeper muscles excluding the decidua.

– Second layer – A similar continuous suture is placed taking the superficial muscles and adjacent fascia overlapping the first layer of suture.

Choosing the method of the operation see Fig.40, Fig.41.

Concluding part – The mops placed inside are removed and the number verified. Peritoneal toileting is done and the blood clots are removed meticulously. The tubes and ovaries are examined. Doyen's retractor is removed. After being satisfied that the uterus is well contracted, the abdomen is closed in layers. The vagina is cleansed of blood clots and a sterile vulval pad is placed.

POSTOPERATIVE CARE

First 24 hours: (Day 0)

- Observation for the first 6–8 hours is important. Periodic check up of pulse, BP, amount of vaginal bleeding and behavior of the uterus (in low transverse incision) is done and recorded.
- Fluid – Sodium chloride (0.9%) or Ringer's lactate drip is continued until at least 2 – 2.5 liters of the solution are infused. Blood transfusion is helpful in anemic mothers for a speedy postoperative recovery. Blood transfusion is required if the blood loss is more than average during the operation (average blood loss in cesarean section is approximately 0.5 to 1 liter).
- Oxytocics: Injection oxytocin 5 units IM or IV (slow) or methergin 0.2 mg IM is given and may be repeated.
- Prophylactic antibiotic (cephalosporins, metronidazole) for all cesarean delivery (see p. 642) is given for 2–3 days. Therapeutic antibiotic is given when indicated.
- Analgesics in the form of pethidine hydrochloride 75–100 mg is administered and may have to be repeated.

– Ambulation – The patient can sit on the bed or even get out of bed to evacuate the bladder, provided the general condition permits.
– Baby is put to the breast for feeding after 3–4 hours when mother is stable and relieved of pain.
– Patient may be discharged as early as third to as late as seventh postoperative day.

Table 36.4: Merits and demerits of lower segment operation over classical

	Lower segment	*Classical*
Techniques	Technically slight difficult Blood loss is less The wall is thin and as such apposition is perfect Perfect peritonization is possible Technical difficulty in placenta previa or transverse lie	Technically easy Blood loss is more The wall is thick and apposition of the margins is not perfect Not possible Comparatively safer in such circumstances
Post-operative	Hemorrhage and shock—less Peritonitis is less even in infected uterus because of perfect peritonization and if occurs, localized to pelvis Peritoneal adhesions and intestinal obstruction are less Convalescence is better Morbidity and Mortality are much lower	More Chance of peritonitis is more in presence of uterine sepsis More because of imperfect peritonization Relatively poor Morbidity and Mortality are high

Table 36.4 contd. next page...

Fig. 40. Choosing the method of the operation

Table 36.4: Merits and demerits of lower segment operation over classical

	Lower segment	*Classical*
	The scar is better healed because of:	The scar is weak because of:
Wound healing	Perfect muscle apposition due to thin margins Minimal wound hematoma The wound remains quiescent during healing process Chance of gutter formation is unlikely	Imperfect muscle apposition because of thick margins More wound hematoma formation The wound is in a state of tension due to contraction and relaxation of the upper segment. As a result, the knots may slip or the sutures may become loose Chance of gutter formation on the inner aspect is more.
During future pregnancy	Scar rupture —is less (see chapter 22) 0.5 – 1.5%	More risk of scar rupture (see chapter 22). 4 – 9%

Fig. 41. Choosing the method of the operation (cont.)

Intraoperative complications

- Extension of uterine incision to one or both the sides. This may involve the uterine vessels to cause severe hemorrhage, may lead to broad ligament hematoma formation.
- Uterine lacerations
- Bladder injury
- Ureteral injury
- Gastrointestinal tract injury is rare unless there is prior pelvic/abdominal adhesions.
- Hemorrhage
- Morbid adherent placenta (placenta accreta) is commonly seen in cases with placenta previa who had prior cesarean delivery. Total hysterectomy is often needed for such a case to control hemorrhage.

Postoperative *complications maternal:*

Immediate

- Postpartum hemorrhage.
- Shock.
- Anesthetic hazards.
- Infections.
- Intestinal obstruction.
- Deep vein thrombosis and thromboembolic disorders.
- Wound complications.
- Secondary postpartum hemorrhage.

LITERATURE

1. DC Dutta's Textbook of Obstetrics / DC Dutta, Hiralal Konar. 2013. Jaypee Brothers Medical Publishers, 698 p.
2. *Sakshi Arora Hans.* Self Assessment & Review Obstetrics, Ninth Edition, 2016. Jaypee Brothers Medical Publishers, 536 p.
3. *Smith, Roger P.* Netter's Obstetrics and Gynecology. Third edition, 2018 by Saunders, an imprint of Elsevier Inc., 616 p.

CONTENT

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