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УЧЕБНОЕ ПОСОБИЕ ДЛЯ ПРАКТИЧЕСКИХ ЗАНЯТИЙ ПО АКУШЕРСТВУ

(для студентов с английским языком обучения)

TEXTBOOK FOR PRACTICAL TRAINING IN OBSTETRICS

(for students with English language of instruction)

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У 91 УЧЕБНОЕ ПОСОБИЕ ДЛЯ ПРАКТИЧЕСКИХ ЗАНЯТИЙ ПО АКУШЕРСТВУ (для студентов с английским языком обучения) = TEXTBOOK FOR PRACTICAL TRAINING IN OBSTETRICS (for students with English language of instruction) / Г.В. Долгая, Н.В. Потылицына, А.Ш. Асымбекова, Д.А. Умарбаева. – Бишкек: Изд-во КРСУ, 2022. 186 с.

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Пособие предназначено для самостоятельной подготовки студентов с английским языком обучения к практическим занятиям по акушерству. В нем рассматриваются все аспекты беременности, течение и ведение родов, новые концепции в уходе за новорожденными и родильницами. В пособии изложены учебные материалы о тактике ведения родов при многих патологических состояниях. В отдельной главе излагается течение беременности и родов при различных экстрагенитальных заболеваниях. Даны основы перинатологии. Материал представлен подробно, на современном уровне, что необходимо для подготовки и дальнейшей самостоятельной работы квалифицированных врачей.

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THEMATIC LESSON PLAN

1. Obstetric care plan for pregnant women
2. Menstrual cycle. Pregnancy planning
3. Diagnostics of pregnancy, pregnancy management in the 1st trimester
4. Pregnancy management in the 2nd and 3rd trimester
5. Obstetric terminology. The pelvis from an obstetric point of view. The fetus as an object of labor
6. Changes in a woman's body during pregnancy. Dominant pregnancy
7. Physiological delivery
8. 1 delivery period
9. Partogram
10. 2 period of labor
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20. Resuscitation of a newborn
21. Methods for assessing the fetal state (gravidogram, Doppler ultrasound, cordocentesis, amniocentesis, amnioscopy, CTG, chorion biopsy)
22. RH negative blood of the mother and the pregnancy. Management tactics. Terms of delivery. Indications for operative delivery
23. Extragenital pathology and pregnancy (asymptomatic bacteriuria, pyelonephritis, anemia)

24. Extragenital pathology and pregnancy
(viral hepatitis A, B, C, pulmonary tuberculosis)
25. Extragenital pathology and pregnancy (heart defects,
diabetes mellitus, hypothyroidism, hyperthyroidism)
26. Thiroid disease in pragnancy
27. Miscarriage. Abortions.
Threatened and inevitable. Missed and reccurent
28. Methods of termination of pregnancy
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30. Premature rupture of the membranes
31. Postterm pregnancy
32. Contracted pelvis (anatomically)
33. Contracted pelvis (clinically)
34. Abnormal uterine action
35. Face and brow presentation
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(abortion, cervical pregnancy, trophoblastic disease)
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(placenta previa, placental abruption)
39. Postpartum bleeding.
Tonus and thrombin as a cause of bleeding
40. Postpartum bleeding.
Tears and tissue as a cause of bleeding
41. Obstetric injuries (rupture of the perineum, cervix)
42. Obstetric injuries
(rupture of the uterus, eversion of the uterus)
43. Caesarean section.
Management of pregnancy with a scar on the uterus
44. Sepsis

OBSTETRIC CARE PLAN FOR PREGNANT WOMEN

Objectives of perinatal care

- To promote the health and wellbeing of the pregnant woman, fetus, infant, and family up to 1 year after birth.
- The major components of perinatal care include: (1) early and continuing risk assessment, including preconception assessment; (2) continued health promotion; and (3) both medical and psychosocial assessment and intervention.
- Three levels of perinatal care are described.

Issues that should be addressed routinely

- Establish accurate gestational age.
- Folate (400 μg daily for all reproductive age women).
- Identify and treat sexually transmitted infections (STIs), diabetes, thyroid disease, HIV, hepatitis B.
- Identify maternal phenylketonuria (PKU).
- Stop Coumadin (warfarin), vitamin A, and other teratogenic agents.
- Counsel on risks of smoking, alcohol, and illicit drug use.
- Counsel about appropriate use of seatbelts during pregnancy.
- Reassure about the safety of sexual intercourse and moderate exercise in uncomplicated pregnancy.
- Review symptoms/signs of pregnancy complications, including preterm labor and pre-eclampsia.
- Check rubella immunity status.
- Ask about chickenpox (consider checking varicella immunity status if no history of chickenpox).
- Counsel about toxoplasmosis prevention (transmitted in cat feces, uncooked meats, soil).
- Counsel about influenza vaccination in pregnancy.

- Counsel about food safety: avoid raw or undercooked meat, poultry, fish, shellfish, рвтй, raw/unpasteurized dairy (listeriosis, toxoplasmosis); avoid large amounts of large fish (mercury).
- Prescribe multivitamins (especially for smokers, women with multiple pregnancies, poor diet, vegetarians).
- Encourage breastfeeding.

What makes your pregnancy high risk?

- Twenty percent (1 in 5) of pregnancies are high risk. Risk factors for adverse pregnancy outcome may exist before pregnancy or develop during pregnancy or labor.
- The content and timing of prenatal visits should vary depending on the risk status of the pregnant woman and her fetus. In low-risk women, prenatal visits are typically every 4 weeks to 28 weeks' gestation, every 2–3 weeks to 36 weeks' gestation, and then weekly until delivery.

High-risk pregnancies

Maternal factors

- Pre-existing medical conditions (pregestational diabetes, chronic hypertension, maternal cardiac disease, chronic renal disease, chronic pulmonary disease).
- Pre-eclampsia.
- Gestational diabetes.
- Morbid obesity.
- Active venous thromboembolic disease.
- Poor obstetric history (prior preterm birth, PPROM, IUGR, placental abruption, pre-eclampsia, recurrent miscarriage).
- Extremes of maternal age.

Fetal factors

- Toxic exposure during pregnancy (to known environmental toxins, medications, illicit drugs).
- Known fetal structural or chromosomal anomaly.
- Prior baby with a structural or chromosomal anomaly.
- Family history of a genetic syndrome.
- Multiple pregnancy (especially if monochorionic).

- IUGR.
- Fetal macrosomia.
- Isoimmunization.
- Intra-amniotic infection.
- Non-reassuring fetal testing (“fetal distress”).

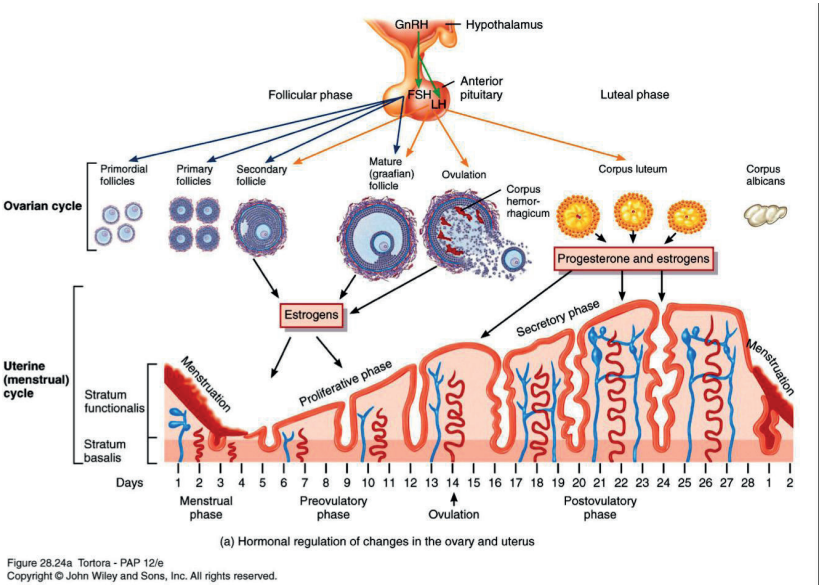
Uteroplacental factors

- Preterm premature rupture of fetal membranes.
- Unexplained oligohydramnios.
- Large uterine fibroids.
- Prior uterine surgery, especially a “classic” hysterotomy.
- Placental abruption.
- Uterine anomaly.
- Placenta previa.
- Abnormal placentation (placenta accreta, increta, or percreta).
- Vasa previa.
- Prior cervical insufficiency.

MENSTRUAL CYCLE. PREGNANCY PLANNING

1. The concept of the reproductive system.
2. Anatomy of the reproductive system.
3. What is the menstrual cycle?
4. Criteria for a normal menstrual cycle.
5. Phases of the menstrual cycle.
6. Ovarian cycle.
7. Uterine cycle.
8. Function of hormones: estrogen, progesterone, testosterone.
9. Partner screening before pregnancy planning.
10. Preparing partners before planning a pregnancy.

Scheme see Fig. 1.



(a) Hormonal regulation of changes in the ovary and uterus

Fig. 1. Scheme of Menstrual levels

Definitions

- *Puberty* is a general term encompassing the entire transition from childhood to sexual maturity.
- *Menarche* (the onset of menstruation) occurs at an average age of 12 years (normal range 8–16 years).
- *Menstrual cycles* or "periods" are often irregular through adolescence before settling into a more consistent ovulatory pattern ranging from 24 days to 35 days (average 28 days). The average duration of menstruation is 3–7 days and blood loss is typically about 80 ml.
- *Mittelschmerz* refers to periovulatory, unilateral, pelvic pain that some women consistently experience.
- *Menopause* is defined as the cessation of menses and usually occurs around age 50. Women with continued bleeding beyond age 55 should be sampled to rule out malignancy.

Hormonal regulation of ovulation

The cyclopentenophenanthrene ring structure is the basic carbon skeleton for all steroid hormones. Cholesterol is the parent steroid from which all the glucocorticoids, mineralocorticoids, and gonadal steroids are derived.

Phases of the menstrual cycle

- The *menstrual phase* begins on day 1 with the onset of bleeding and continues for several more days until the shedding of the endometrial lining stops (usually day 4–5).
- The *proliferative phase* begins at the end of the menstrual phase and ends at ovulation (usually day 13 or 14). This phase is characterized by endometrial thickening and ovarian follicular maturation.
- The *luteal (secretory) phase* starts at ovulation and lasts through day 28 before the entire process starts over again on day 1.

Biologic basis of menstruation

Coordination of the menstrual cycle depends on a complex interaction of the brain, pituitary, ovaries, and endometrium.

Brain

- The hypothalamus is located at the base of the brain and essentially functions as the central processing unit of the reproductive system.
- Neuronal stimuli from the cerebral cortex are converted by the hypothalamus into pulses of neuropeptides (gonadotropin-releasing hormone, GnRH).
- Hypothalamic GnRH production is modulated by *negative feedback* of steroid hormones (estradiol-17β, progesterone).

Pituitary

- Located just below the hypothalamus, the pituitary gland consists of the neurohypophysis (posterior lobe) and the adenohypophysis (anterior lobe). It is the anterior pituitary that gives rise to gonadotropin (luteinizing hormone [LH] and follicle-stimulating hormone [FSH]) production.
- Pulsatile GnRH from the hypothalamus initiates the synthesis and secretion of LH and FSH. Similar to the hypothalamus, the anterior pituitary is subject to negative feedback regulation by the steroid hormones.
- In women of reproductive age, LH and FSH levels generally remain in the 10–20 mIU/mL range. After the menopause or surgical oophorectomy, estradiol-17β levels decline and LH and FSH are released from negative feedback, achieving circulating concentrations of more than 50 mIU/mL.

Ovaries

- Primitive germ cells (oogonia) divide by mitosis during fetal embryogenesis, peaking at around 7 million by 5 months of gestation.
- Meiotic division then begins, resulting in formation of primary oocytes. However, rapid atresia reduces the number of available follicles to 2 million at birth. At puberty, only around 300,000–400,000 follicles remain.
- Oocytes remain "resting" in *meiotic prophase* untilpuberty. Resting ovarian follicles are surrounded by thecal and

granulosa cells: FSH stimulates the granulosa cells and LH stimulates the thecal cells.

- Only a single "dominant follicle" develops each menstrual cycle.
- When it produces enough estrogen to sustain a circulating estradiol – 17β concentration of about 200 pg/mL for 48 hours, the hypothalamic– pituitary axis responds by secreting a surge of gonadotropins, primarily LH. This *LH surge* precedes ovulation by 24–36 hours.
- Following ovulation, the follicle collapses to form the *corpus luteum*. This endocrine organ mainly synthesizes progesterone to prepare the endometrium for pregnancy.
- If implantation does not occur, the corpus luteum will degenerate, resulting in a precipitous decline in circulating steroid hormone levels and the onset of menstruation. The decreasing steroid hormone levels release the negative feedback mechanism, inducing the pituitary to increase gonadotropin secretion. As a result, a new cycle of follicular recruitment is initiated.
- If implantation does occur, the embryo will rescue the corpus luteum by producing human chorionic gonadotropin (hCG) to prevent menstruation. At 7–9 weeks of gestation, the placenta takes over the production of progesterone from the corpus l uteum.

Endometrium

- Dramatic monthly cyclic changes occur in the endometrium under the control of steroid hormones produced by the ovaries.
- Estradiol-17β production by the ovarian follicles induces endometrial proliferation. Progesterone synthesis by the corpus luteum then acts to mature the estrogen-primed endometrium in preparation for blastocyst implantation.
- Lowered steroid hormone levels in the late secretory phase cause a collapse of the endometrial vasculature, resulting in menstruation.

DIAGNOSTICS OF PREGNANCY, PREGNANCY MANAGEMENT IN THE 1ST TRIMESTER

1. Signs of pregnancy.
2. Methods of pregnancy diagnosis: clinical, laboratory, instrumental.
3. Scope of examination of pregnant women at registration.
4. Calculating the duration of pregnancy.
5. Calculating the delivery time.
6. Examinations of pregnant women in the 1st trimester.
7. Screening tests for various pathologies.

Diagnosis of pregnancy

Symptoms

Amenorrhoea

The monthly shedding of the endometrium is prevented by higher progesterone levels from the persistence of the corpus luteum. Pregnancy is dated from the first day of the last normal menstrual period (LNMP) even though conception does not occur until about 14 days later. Any bleeding after the LNMP should be considered as abnormal.

Nausea and vomiting

Nausea occurs in 80% of nulliparous and 60% of multiparous women. For many pregnant women this is the first sign of pregnancy with the symptoms occurring even before the first period is missed. The nausea and vomiting usually disappears by 16 weeks' gestation and lessens in severity after about 12 weeks. Although some women are sick first thing in the morning, it is not unusual to find that vomiting may occur at any time of the day. Commonly some biscuits or sweets help prevent nausea. There is usually no accompanying metabolic upset, women do not feel ill all the time, and it does not affect their daily activities. They do not usually require hospitalization.

Specific causes may include urinary tract infection or ingestion of iron tablets.

Breast symptoms

Breast enlargement accompanied by tingling of the skin and nipples. Montgomery's tubercles develop from between six and eight weeks' gestation and colostrum may be secreted from the nipples after about 12 weeks' gestation.

Urinary symptoms

From six weeks' gestation onwards, many women experience increased frequency of micturition.

This is due to:

- Increased renal blood flow in the early stages.
- Pressure on the bladder from the growing uterus in later pregnancy.

Signs

Uterus

- An increased softness and enlargement of the uterus can be felt on bimanual vaginal examination from 6–8 weeks of gestation.

Breasts

- Increased in size and feel warm.
- The areolae darken.
- Montgomery's tubercles develop.
- Tortuous skin veins dilate.

Investigations

Pregnancy test

Animal pregnancy tests and early crude immunological tests have now been replaced by accurate, sensitive tests involving monoclonal antibodies.

Human chorionic gonadotrophin (hCG) is a glycoprotein hormone that contains two carbohydrate side chains: alpha (a) and beta (b).

The a subunit is identical to that of follicle stimulating hormone (FSH), luteinizing hormone (LH) and thyrotrophin (TSH). The b subunit is immunologically specific. hCG is secreted by the trophoblast cells of the fertilized ovum and later by the definitive placenta.

Modern tests can detect hCG levels as low as 25iu/l, before the time of the missed menses. Such tests can be performed in two minutes and are unaffected by urine contaminated by proteinuria, or bacterial contamination. Only a few drops of urine are required. The tests come in a variety of kits which can be bought in any chemist and are based on a colour change occurring if hCG binds to the monoclonal antibody embedded in the absorbent paper. Two main sorts are available: a double band of blue or a central spot of pink indicates a positive test while a single band of blue or absence of a pink spot indicates a negative pregnancy test.

Ultrasound

Real-time ultrasound machines will detect an intrauterine gestation sac from five weeks of amenorrhoea, with fetal heart activity becoming visible at six weeks and a fetal pole visible at seven weeks. Transvaginal probes enable a better resolution image than transabdominal ultrasound allowing the diagnosis of an intrauterine pregnancy to be made one week earlier (5–6 weeks).

Antenatal visits

The current method of antenatal care was established 80 years ago but is now subject to change. In particular, the visits in mid-pregnancy (12–34 weeks) may be reduced. Traditionally, the woman is seen monthly from the booking visit until 28 weeks, fortnightly until 36 weeks and then weekly until delivery. A reduction in the number of visits does not affect the outcome of pregnancy and is very popular with women.

The aim of the visits is to screen the low-risk population by means of history, examination and investigation; then antenatal care for high-risk women may be carried out on a more frequent basis.

The following scheme applies to all women and is an attempt to identify risk factors.

The first visit

Ideally the booking visit should be at 8–12 weeks' gestation. More frequently now the woman's history is being taken in her own home by a community midwife.

History

1 Establish the reliability of the LNMP.

- Was the woman sure of the dates?
- Was the cycle regular?
- Was the woman on oral contraceptives within two months?
- Was there bleeding in early pregnancy?

Any of the above circumstances render prediction of expected date of delivery (EDD) from LNMP unreliable and later ultrasound examination is needed to determine dates.

2 History of maternal disease, e.g. hypertension, diabetes mellitus.

3 Family history, e.g. diabetes mellitus, tuberculosis, hypertension, multiple pregnancy or the birth of a congenitally abnormal baby, inherited disorders.

4 Past obstetric history. This involves listing all the pregnancies in chronological order together with the following details:

(I) Deliveries after 24 weeks regardless of outcome.

(II) Miscarriages and ectopic pregnancies:

- First trimester (less than 12 weeks) or second trimester.
- If second trimester, were they:

(a) Relatively painless, associated with early rupture of the membranes suggesting cervical incompetence.

(b) Associated with pain and bleeding suggestive of premature placental separation.

(c) Associated with the delivery of a dead, macerated baby, an intra-uterine death.

(III) List all therapeutic abortions, their reason, gestation and method by which they were performed.

5 Drug history. Note all drugs taken in the pregnancy so far.

6 Allergies. Note allergies to medication, food or Elastoplast.

7 Social history:

- Detail the woman's alcohol, tobacco and illicit drug intake giving appropriate advice.
- The woman's marital status, her occupation and that of her partner.
- The living conditions.
- Social support – family, friends.

Examination

In the absence of a relevant history and with the routine use of ultrasound, there is little need to examine the pregnant woman's pelvis. Most doctors and midwives, however, would still perform the following examinations:

1 Maternal blood pressure.

2 The respiratory system.

3 The breasts to check for:

- Lumps.
- Inverted nipples, which may require advice for breast feeding.

4 The spine for kyphosis or scoliosis.

5 The abdomen looking for scars, masses and, if the pregnancy is sufficiently advanced, the size of the uterus.

6 The legs looking for varicose veins.

7 Vaginal examination is not usually required at a booking visit but sometimes is done:

- To confirm the pregnancy. If ultrasound.

- To check uterine size. not available.
- To exclude uterine or ovarian masses.
- To take a smear if the patient has not had one within the last three years, although this is often left until the postpartum visit.

Investigations

Urine

1 Proteinuria – renal disease.

2 Glucose – diabetes.

3 White blood cells – response to infection.

4 Nitrite – bacteria.

Blood

1 Haemoglobin.

2 Red cell indices, particularly Anaemia. the mean corpuscular volume (MCV).

3 ABO and Rhesus (Rh) group (if negative need for Anti-D).

4 The presence of atypical antibodies.

5 Sickle cell screen particularly if the patient is Afro-Caribbean.

6 Haemoglobin electrophoresis, looking for thalassaemia if the patient is Asian or Mediterranean in origin.

7 Test for hepatitis antigens.

8 Test for rubella antibodies.

9 Human immunodeficiency virus (HIV) test. All women after appropriate counselling should be offered an HIV antibody test but thefollowing patients are at high risk:

- Women from or with partners from sub- Saharan Africa.
- Drug abusers or partners of drug abusers.
- Women who have bisexual partners.
- Women with haemophiliac partners.
- Women who have had a blood transfusion overseas.

In places where HIV is more prevalent, universal testing is performed with an opt-out policy. In the UK this includes London and Edinburgh.

10 Screening test for syphilis (usually VDRL, Venereal Disease Research Laboratory test). If positive, more specific tests are required.

General advice for healthy pregnancy

1 Establish a rapport between the woman and the antenatal clinic staff.

2 Show the woman where she can discover more about her pregnancy and delivery from:

- books available;
- parentcraft classes;
- relaxation classes;
- video and TV programmes.

3 Discuss the social welfare benefits available.

4 Make arrangements for the medical social worker to see the woman if there are any difficulties such as care of the other children or housing.

5 Advise a visit to the dentist reasonably soon, as dental care in pregnancy is free, and there is an increased prevalence of tooth decay and gingivitis in pregnancy.

6 Give dietary advice. This should be simple advice as most people in the UK have a more than adequate diet. The idea of eating for two should be discouraged and, in general, pregnant women need only an additional 500 kcal (2100 J) a day to ensure normal fetal growth. Vegans may require specialized advice from the dietitian in order to ensure adequate nutrition throughout the pregnancy especially for certain amino acids. Similarly, some Asian women may need dietary advice or supplements of vitamin D as a consequence of living in the cloudy Northern Hemisphere.

7 Advise the woman to stop smoking since it increases the risk of intrauterine growth retardation and delayed fetal maturation.

8 Advise the woman to stop drinking alcohol or cut down on her intake.

9 Advise the woman to avoid unpasteurized products, soft cheese and paté as these have been associated with intrauterine death secondary to listeriosis.

10 Advise the woman to be careful when dealing with cats' litter by avoiding emptying the tray and using rubber gloves because of the risk of acquiring toxoplasmosis which may lead to mental retardation in the fetus.

11 Consider providing iron supplementation. The routine of prophylactic iron supplements in pregnancy is controversial. Many obstetricians only provide iron if the woman has a haemoglobin of less than 10.5 g/dl or a MCV of less than 84fl at the booking visit. Additional indications may be for multiple pregnancies or the previous pregnancy within 2 years. Most women's haemoglobin level will fall by about 1g/dl due to haemodilution that occurs in pregnancy. If iron is given, it should be taken with meals as it is only absorbed in the ferrous state and this is best achieved in the presence of vitamin C. In the non-pregnant state, about 10% of iron is absorbed and this is thought to double in pregnancy. When supplementation is given, you should aim to give at least 100mg of elemental iron a day.

12 Vitamin supplements. These are not usually required by women receiving an adequate diet. An exception is folic acid as it is often only barely sufficient in many diets. The requirements in pregnancy rise from 50mg a day to 300mg a day. Many women are therefore given prophylactic iron tablets that also contain folic acid (500mg a day.) Folic acid supplements have been shown to reduce the incidence of neural tube defects (NTDs) when taken preconceptually and up to 14 weeks' gestation.

Special visits

To be performed at all visits

1 Check the history of recent events and ensure that the baby is moving.

2 Examine:

- Blood pressure.
- The growth of the uterus and its contents can be assessed by measuring the symphysiofundal height. Normal growth is 1cm per week ±2cm, e.g. at 32 weeks the SFH should be 30–34 cm. Less than 30cm may indicate growth retardation or oligohydramnios; greater than 34cm may indicate a multiple pregnancy, polyhydramnios or macrosomia. Clinical assessment by palpation is rather a crude method with only 40% of small babies accurately detected and of these only 60% will still be small for dates at birth.

The SFH is measured by palpating the fundus, a tape measure is placed under the left index finger and laid over the abdomen to the top of the symphysis which should be felt for gently as it can be tender. The tape measure should be face down to prevent cheating (making the measurement fit the gestation). Beyond 26 weeks the following should be noted in the notes:

- lie (longitudinal, oblique, transverse);
- presentation (cephalic, breech, none);
- engagement (one-fifth palpable;
- liquor volume (normal, polyhydramnios, oligohydramnios);
- fetal heart auscultation with a hand-held Doppler (from 14 weeks) or pinard stethoscope (beyond 30 weeks).
- Test the urine for protein and glucose. This is also traditional and, in the absence of hypertension, it is less worthwhile testing for protein.

Additionally at 26–28 weeks

- Check the haemoglobin.
- If the woman is Rh-negative also check for the presence of Rh antibodies. Give anti-D.
- Many now screen for gestational diabetes by doing random blood sugar at 28 weeks' gestation.
- Check the lie and presentation of the fetus.

Additionally at 34 weeks

- Check haemoglobin level.
- If the patient is Rh-negative, check for the presence of antibodies. Give anti-D.
- If the presentation is cephalic, is the head engaged?

Between 41 and 42 weeks

- Examine the cervix to assess the chances of success of induction if this is needed and do a membrane sweep if the cervical os is open; 70% of women will go into spontaneous labour within 48 hours.

Advice to mothers

Apart from the dietary and social welfare information that should be available to the woman when she books, the following should be enquired about specifically.

Intercourse

There is no restriction to intercourse during pregnancy unless the woman bleeds from the vagina or has placenta praevia. Mechanical problems may occur in late pregnancy so that alterations in the position of intercourse may become necessary, for example the woman may be more comfortable on top.

PREGNANCY MANAGEMENT IN THE 2ND AND 3RD TRIMESTER

1. Frequency of visits to the doctor of a pregnant woman.
2. Necessary examinations of pregnant women during every visit.
3. Position, presentation, position and type of fetal position.
4. Palpation of the abdomen (4 Leopold – Levitsky maneuvers).
5. Auscultation of the fetal heartbeat.
6. Necessary examinations of pregnant women in the 2nd and 3rd trimester.

Routine anomaly ultrasound scanning (18–20 weeks)

Most hospitals in the UK now offer a routine ultrasound examination at 18–20 weeks' gestation. The aim of this ultrasound examination is:

- To establish gestational age.
- To exclude major structural abnormalities of the fetus.
- To diagnose multiple pregnancy.

A small pulse of ultrasound is sent into the tissues and a recorder in the same transducer, then detects the echoes. The distance between tissue boundaries can be assessed by determining the differences in time taken for the echoes to return from each boundary.

At the 18–20-weeks routine ultrasound visit, the following are assessed:

- The biparietal diameter (BPD).
- The head circumference (HC).
- The abdominal circumference (AC).
- Femur length (FL).

These measurements are used to confirm the gestational age of the fetus. The EDD may be changed if the measurements are more than two weeks greater or smaller than the 50th centile at 18–20 weeks.

The later the scan is done the less accurate dating by ultrasound becomes and caution should be exercised before changing the EDD if the first scan is done after 22 weeks.

The ultrasound scan will detect the following:

- Multiple pregnancy.
- Placental site – particularly low-lying placenta (5% at 20 weeks but 0.5% at 34 weeks when the scan should be repeated).
- Fetal congenital abnormalities:
- Anencephaly (absent top of the head and brain).
- Spina bifida.
- Double bubble of dilated stomach and duodenum in duodenal atresia (common in Down's syndrome).
- Some cardiac abnormalities (Fallot's tetralogy, ventricular septal defect (VSD), atrial septal defect (ASD)).
- Hydrocephaly.
- Renal pelvic dilatation (outflow obstruction – urethral valves in boys).
- Sacral agenesis (insulin-dependent diabetics).
- Major limb defects (dwarfism).
- The ultrasound scan is the first time that parents see their baby and it is known to increase the bonding that they feel towards their baby.

LIE: **The lie refers to the relationship of the long axis of the fetus to the long axis of the centralized uterus or maternal spine**, the commonest lie being longitudinal (99.5%). The lie may be transverse or oblique; sometimes the lie is unstable until labor sets in, when it becomes either longitudinal or transverse.

PRESENTATION: **The part of the fetus which occupies the lower pole of the uterus** (pelvic brim) is called the presentation of the fetus. Accordingly, the presentation may be cephalic (96.5%), podalic (3%) or shoulder and other (0.5%). When more than one part of the fetus present, it is called **compound presentation.**

POSITION: **It is the relation of the denominator to the different quadrants of the pelvis**. For descriptive purpose, the pelvis is divided into equal segments of 45° to place the denominator in each segment. Thus, theoretically, there are 8 positions with each presenting part.

Fundal height: The distance between the umbilicus and the ensiform cartilage is divided into three equal parts. The fundal height corresponds to the junction of the upper and middle third at 32 weeks, upto the level of ensiform cartilage at 36th week and it comes down to 32 weeks level at 40th week because of engagement of the presenting part. To determine whether the height of the uterus corresponds to 32 weeks or 40 weeks, engagement of the head should be tested. **If the head is floating, it is of 32 weeks pregnancy and if the head *is engaged*, *it is of 40 weeks pregnancy.***

Symphysis fundal height (SFH). The upper border of the fundus is located by the ulnar border of the left hand and this point is marked. The distance between the upper border of the symphysis pubis upto the marked point is measured by a tape in centimeter. After 24 weeks, the SFH measured in cm corresponds to the number of weeks upto 36 weeks. A variation of ± 2 cm is accepted as normal. Variation beyond the normal range needs further evaluation

Obstetric grips (Leopold maneuvers): Palpation should be conducted with utmost gentleness. Clumsy and purposeless palpation is not only uniformative but may cause undue uterine irritability. **During Braxton-Hicks contraction or uterine contraction in labor**, **palpation should be suspended**.

(I) **Fundal grip:** The palpation is done facing the patient's face. The whole of the fundal area is palpated using both hands laid flat on it to find out which pole of the fetus is lying in the fundus: (a) broad, soft and irregular mass suggestive of breech, or (b) smooth, hard and globular mass suggestive of head. In transverse lie, neither of the fetal poles are palpated in the fundal area.

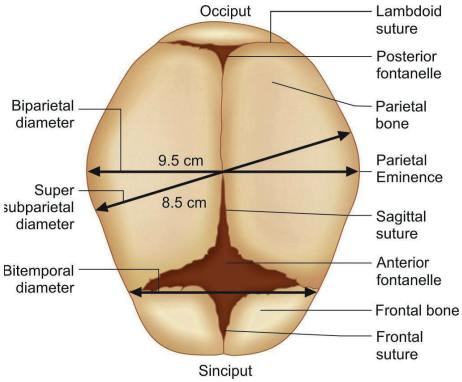
(II) **Lateral or umbilical grip:** The palpation is done facing the patient's face. The hands are to be placed flat on either side of the umbilicus to palpate one after the other, the sides and front of the uterus **to find out the position of the back**, **limbs and the anterior shoulder**. The back is suggested by smooth curved and resistant feel. The 'limb side' is comparatively empty and there are small knob like irregular parts. After the identification of the back, it is essential to note its position whether placed anteriorly or towards the flank or placed transversely. Similarly, the disposition of the small parts, whether placed to one side or placed anteriorly occupying both the sides, is to be noted. **The position of the anterior shoulder is to be sought for**. It forms a well marked prominence in the lower part of the uterus above the head. It may be placed near the midline or well away from the midline.

(III) **Pawlik's grip (Third Leopold):** The examination is done facing towards the patient's face. The overstretched thumb and four fingers of the right hand are placed over the lower pole of the uterus keeping the ulnar border of the palm on the upper border of the symphysis pubis. When the fingers and the thumb are approximated, the presenting part is grasped distinctly (if not engaged) and also the mobility from side to side is tested. **In transverse lie**, **Pawlik's grip is empty**.

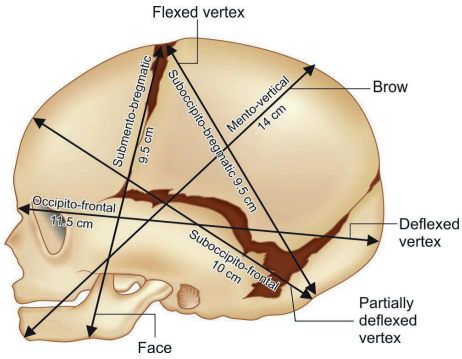
(IV) **Pelvic grip (Fourth Leopold): The examination is done facing the patient's feet**. Four fingers of both the hands are placed on either side of the midline in the lower pole of the uterus and parallel to the inguinal ligament. The fingers are pressed downwards and backwards in a manner of approximation of finger tips to palpate the part occupying the lower pole of the uterus (presentation). **If it is head**, **the characteristics to note are**: (1) precise presenting area (2) attitude and (3) engagement.

OBSTETRIC TERMINOLOGY. THE PELVIS FROM AN OBSTETRIC POINT OF VIEW. THE FETUS AS AN OBJECT OF LABOR (see Fig. 2)

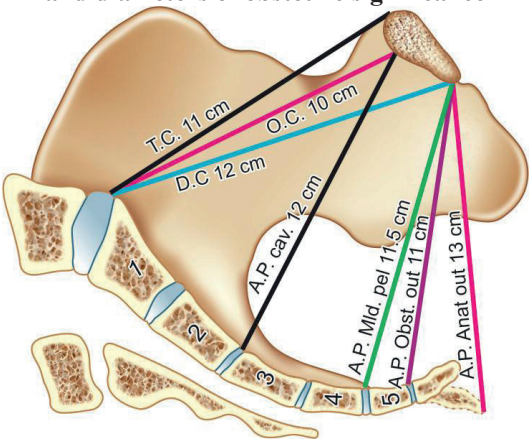
1. Pelvic bones.
2. Pelvic planes.
3. Methods for diagnosing the size of the bone pelvis.
4. Skull bones.
5. Sutures and fontanels on the head of the fetus
6. Fetal head dimensions.



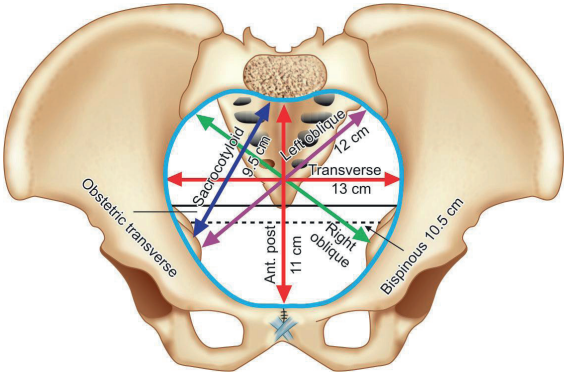
Fetal skull showing important sutures, fontanelles



The important landmarks of fetal skull and diameters of obstetric significance



Sagittal section of the pelvis showing antero-posterior diameters in different planes



Different diameters of the inlet of obstetrical significance. Bispinous diameter is also demonstrated

Fig. 2: a, b, c, d

CHANGES IN A WOMAN'S BODY DURING PREGNANCY. DOMINANT PREGNANCY

1. Dominant pregnancy.
2. Changes in the reproductive system.
3. Changes in the cardiovascular system.
4. Changes in the digestive system.
5. Changes in the endocrine system.
6. Changes in the urinary system.
7. Changes in the respiratory system.
8. Changes in the immune system.

Maternal adaptations to pregnancy

- Physiologic adaptations in the mother occur in response to demands created by pregnancy. These include:

1 support of the fetus (volume, nutritional and oxygen support, clearance of fetal waste).

2 protection of the fetus (from starvation, drugs, toxins).

3 preparation of the uterus for labor.

4 protection of the mother from potential cardiovascular injury at delivery.

- Maternal age, ethnicity, genetic factors, and maternal comorbidities affect the ability of the mother to adapt to pregnancy.
- All maternal organ systems are required to adapt to the demands of pregnancy. The quality, degree, and timing of the adaptation vary from one individual to another and from one organ system to another.

Respiratory system

- Respiratory adaptations during pregnancy are designed to optimize maternal and fetal oxygenation, and to facilitate transfer of CO2 waste from the fetus to the mother.

- Many pregnant women report a subjective perception of shortness of breath (dyspnea) in the absence of pathology. The reason for this is unclear.
- The mechanics of respiration change with pregnancy. The ribs flare outward and the level of the diaphragm rises 4 cm.
- During pregnancy, tidal volume increases by 200 mL (40%), resulting in a 100–200 mL (5%) increase in vital capacity and a 200 mL (20%) decrease in the residual volume, thereby leaving less air in the lungs at the end of expiration. The respiratory rate does not change. The end-result is an increase in minute ventilation and a drop in arterial *P*CO2 (see table below). Arterial *P*O2 is essentially unchanged. A compensatory decrease in bicarbonate enables the pH to remain unchanged. Pregnancy thus represents a state of *compensated respiratory alkalosis*.

Cardiovascular system

- Progesterone decreases systemic vascular resistance early in pregnancy, leading to a decline in blood pressure. In response, cardiac output increases by 30–50%.
- Activation of the renin–angiotensin system results in increased circulating angiotensin II, which encourages sodium and water retention (leading to a 40% increase in blood volume) and directly constricts the peripheral vasculature.

Gastrointestinal tract

- Nausea ("morning sickness") occurs in >70% of pregnancies. Symptoms usually resolve by 17 weeks.
- Progesterone causes relaxation of gastrointestinal smooth muscle, resulting in delayed gastric emptying and increased reflux.
- Pregnancy predisposes to cholelithiasis (gallstones). Most gallstones in pregnancy are cholesterol stones.
- Pregnancy is a "diabetogenic state" with evidence of insulin resistance and reduced peripheral uptake of glucose (due to increased levels of placental anti-insulin hormones, primarily

human chorionic soma tolactotropin [hCS]). These mechanisms are designed to ensure a continuous supply of glucose to the fetus.

Genitourinary system

- Glomerular filtration rate (GFR) increases by 50% early in pregnancy, leading to an increase in creatinine clearance and a 25% decrease in serum creatinine and urea concentrations.
- Increased GFR results in an increase in filtered sodium. Aldosterone levels increase two- to threefold to reabsorb this sodium.
- Increased GFR also results in decreased resorption of glucose. As such, 15% of normal pregnant women exhibit glycosuria.
- Mild hydronephrosis and hydroureter are common sonographic findings that are due to high progesterone levels and partial obstruction from the gravid uterus.
- Five percent of pregnant women have bacteria in their urine. Pregnancy does not increase the incidence of asymptomatic bacteriuria, but such women are more likely to develop pyelonephritis (20–30%).

Hematologic system

- Increased intravascular volume results in dilutional anemia. Elevated erythropoietin levels lead to a compensatory increase in total red cell mass, but never fully correct the anemia.
- A modest increase in white blood cell count (leukocytosis) can be seen during pregnancy, but the differential count should not change.
- Mild thrombocytopenia (<150,000 platelets/mL) is seen in 10% of pregnant women. This is probably dilutional and rarely clinically significant.
- Pregnancy represents a hypercoagulable state with increased circulating levels of factors II (fibrinogen), VII, IX, and X. These changes protect the mother from excessive blood loss at delivery, but also predispose to thromboembolism.

Endocrine system

- Estrogen increases hepatic production of thyroid-binding globulin, leading to an increase in total thyroid hormone concentration. However, thyroid-stimulating hormone (TSH), free triiodothyronine (T3), and free thyroxine (T4) levels remain unchanged.
- Serum calcium levels decrease in pregnancy leading to an increase in parathyroid hormone, which encourages conversion of cholecalciferol (vitamin D3) to its active metabolite, 1,25-dihydroxycholecalciferol (DHCC), by 1α-hydroxylase in the placenta. This leads to increased intestinal absorption of calcium.
- Aldosterone and cortisol are increased in pregnancy.
- Prolactin increases in pregnancy, but its function is unknown. It is probably more important for lactation after delivery.

Immune system

Cellular immunity is depressed during pregnancy. As a result, pregnant women may be at increased risk for contracting viral infections and tuberculosis.

Musculoskeletal and dermatologic systems

- A shift in posture (exaggerated lumbar lordosis) and lower back strain are common in pregnancy.
- Increased estrogens and melanocyte-stimulating hormone may cause hyperpigmentation (darkening) of the umbilicus, nipples, abdominal midline (linea nigra), and face (chloasma).
- Increased estrogen may also lead to skin changes such as spider angioma and palmar erythema.

PHYSIOLOGICAL DELIVERY

Dominant of pregnancy, dominant of childbirth. Mechanisms of birth activity. Harbingers of labor. The maturity of the cervix, the scale of Bishop. Periods and phases of labor. Duration of labor.

1. The concept of labor.
2. Dominant of labor.
3. Mechanisms of birth activity.
4. Harbingers of labor.
5. The maturity of the cervix, the scale of Bishop.
6. Periods and phases of labor.
7. Duration of labor.

Endocrinology of pregnancy

The placenta is a rich source of hormones, including human chorionic gonadotropin, human chorionic somatolactotropin, steroid hormones, oxytocin, growth hormone, corticotropin-releasing hormone, proopiomelanocortin, prolactin, and gonadotropin-releasing hormone. A few are discussed here.

Human chorionic gonadotropin

- Human chorionic gonadotropin (hCG) is a heterodimeric protein hormone that shares a common α-subunit with luteinizing hormone (LH), follicle-stimulating hormone (FSH), and thyroid-stimulating hormone (TSH), but has a unique β-subunit. It is most closely related to LH.
- Human CG is produced exclusively by the syncytiotrophoblast cells and can be detected in maternal serum 8–9 days after conception. It is the basis of all standard pregnancy tests.
- Human CG levels double every 48 hours in the first several weeks of pregnancy, reaching a peak of 80,000–100,000 mIU/mL at around 8–10 weeks' gestation. Thereafter, hCG

concentrations fall to 10,000–20,000 mIU/mL, and remain at that level for the remainder of pregnancy.

- The primary function of hCG appears to be maintenance of progesterone production from the corpus luteum of the ovary, until the placenta can take over progesterone production at around 6–8 weeks' gestation. Progesterone is essential for early pregnancy success, eg, surgical removal of the corpus luteum or administration of a progesterone receptor antagonist (such as RU 486, mifepristone) before 7 weeks (49 days) of gestation will cause abortion.
- Human CG also has thyrotropic activity (0.025% of TSH), which only becomes clinically significant if hCG levels are markedly elevated such as in complete molar pregnancies.

Human chorionic somatolactotropin

- Human chorionic somatolactotropin (hCS) – previously known as human placental lactogen (hPL) – is a family of protein hormones produced exclusively by the placenta which are closely related to both prolactin and growth hormone.
- Human CS production is directly proportional to placental mass and levels rise steadily throughout pregnancy.
- The function of hCS is not known, but it has anti-insulin-like activity and may be involved in the development of insulin resistance, which characterizes pregnancy.

Steroid hormones

- The placenta is the major source of progesterone and estrogen production during pregnancy.
- In the placenta, estrogen is synthesized from androgen precursors and is important for preparing the uterus for labor. Progesterone is derived primarily from maternal substrate (cholesterol) and may be important for maintaining uterine quiescence before labor.

Endocrine control of labor

- Reproductive success is critical for survival of the species. Each species has solved the problem of labor in a different way.

Such differences may reflect the evolutionary status of the organism in question or may represent solutions to inherent obstacles to reproduction faced by each species (such as differences in placentation, gestational length, and the number of offspring per pregnancy).

- The slow progress in our understanding of the mechanisms responsible for the process of labor in humans reflects the lack of an adequate animal model and the difficulty of extrapolating from endocrine mechanisms in many animal species to the paracrine/autocrine mechanisms of parturition in humans.

Initiation of labor

- Considerable evidence suggests that, in most viviparous animals, the fetus is in control of the timing of labor. It is likely that this is achieved through activation of the fetal hypothalamic–pituitary–adrenal (HPA) axis before the onset of labor, and that this is common to all viviparous species.
- A proposed "parturition cascade" is outlined in Figures 37.1 and 37.2.
- The human placenta is an incomplete steroidogenic organ, and estrogen production by the placenta has an obligatory need for androgen precursor. This excess androgen is supplied by the fetus in the form of dehydroepiandrostenedione sulfate (DHEAS).
- Activation of the fetal HPA axis at term results in excess DHEAS release from the intermediate (fetal) zone of the fetal adrenal. DHEAS is 16-hydroxylated in the fetal liver and passes via the fetal circulationto the placenta where it is converted almost exclusively to estriol (16-hydroxyestradiol-17β).
- Human pregnancy is characterized by a hyperestrogenic state of unparalleled magnitude in the entire mammalian kingdom. The placenta is the primary source of estrogens. The concentration of estrogens in the maternal circulation increases with gestational age. Placental estrone and estradiol-17β are derived primarily from maternal C19 androgens (testosterone and androstenedione), whereas estriol is derived almost exclusively

from fetal DHEAS. Estrogens do not cause uterine contractions, but do promote a series of myometrial changes (including increasing the number of prostaglandin receptors, oxytocin receptors, and gap junctions) that enhance the capacity of the myometrium to generate contractions.

- In addition to DHEAS, the enlarged fetal adrenal glands also produce cortisol, which has two actions:

1 It prepares fetal organ systems for extrauterine life.

2 It promotes expression of a number of placental products, including corticotropin-releasing hormone (CRH), oxytocin, and prostaglandins (especially prostaglandin E2 – PGE2).

- Placental CRH initiates a *positive feedback loop* by stimulating the fetal HPA axis to produce more DHEAS and more cortisol, which then further upregulates placental CRH expression. (This stimulatory effect of cortisol on placental CRH should be contrasted with the feedback inhibition of cortisol on maternal CRH.)
- Placental oxytocin acts directly on the myometrium to cause contractions and indirectly by upregulating prostaglandin production (especially prostaglandin F2α – PGF2α) by the decidua.
- PGF2α is produced primarily by the maternal decidua and acts on the myometrium to upregulate oxytocin receptors and gap junctions, and thereby promote uterine contractions.
- PGE2 is primarily of fetoplacental origin and is probably more important in promoting cervical "ripening" (maturation) and spontaneous rupture of the fetal membranes (SROM).

1 DELIVERY PERIOD

1. Course of labor.
2. Management of labor.
3. Amniotomy.
4. Complications.

Normal labor and delivery I

Definition

Labor is the physiologic process by which a fetus is expelled from the uterus to the outside world. It is a clinical diagnosis requiring two elements: (1) regular phasic uterine contractions increasing in frequency and intensity, and (2) progressive effacement and dilation of the cervix. Normal labor occurs at term (defined as 37–0/7 to 42–0/7 weeks' gestation).

The endocrine control of labor

Labor may be regarded physiologically as a release from the inhibitory effects of pregnancy on the myometrium rather than as an active process mediated by uterine stimulants. In vivo, however, both inhibitory and stimulatory factors appear to be important. It is likely that there is a "parturition cascade" at term that removes the mechanisms maintaining uterine quiescence and recruits factors promoting uterine activity. Regardless of whether the trigger for labor begins within the fetus or outside the fetus, the final common pathway ends in the maternal tissues of the uterus, and is characterized by the development of regular phasic uterine contractions.

Myometrial contractility

As in other smooth muscles, myometrial contractions are mediated through the ATP-dependent binding of thick filaments (myosin) to thin filaments (actin). Electrical stimuli (action potentials) must be generated and propagated in the myometrium to cause contractions,

which are achieved through the rapid shift of ions (especially calcium) through membrane ion channels. The frequency of contractions correlates with the frequency of action potentials, the force of the contractions correlates with the number of spikes in the action potential and the number of cells activated together, and the duration of contractions correlates with the duration of the action potentials. The transition of the uterus from a quiescent entity to a contractile one comes in part through an increase in gap junctions leading to recruitment and improved communication between adjacent myometrial cells. In contrast to vascular smooth muscle, myometrial cells have a sparse innervation which is further reduced during pregnancy.

The regulation of uterine contractility is therefore largely humoral and/or dependent on intrinsic factors within myometrial cells.

Mechanics of normal labor

The ability of the fetus to successfully negotiate the pelvis is dependent on the interaction of three variables (known as "the 3 Ps"): powers, passenger, and passage. The "powers" refers to the forces generated by the uterine musculature, the "passenger" is the fetus, and the "passage" consists of the bony pelvis and resistance provided by soft tissues, specifically the cervix and pelvic floor musculature.

Powers

- Several techniques are available to assess uterine activity. Uterine activity is characterized by frequency, intensity (amplitude), and duration of contractions.
- Despite technological advances, the definition of "adequate" uterine activity remains unclear. Classically, three to five strong contractions in 10 minutes has been used to define adequate labor. This contraction pattern is seen in 95% of women in normal labor at term. Remember that the external uterine monitor is a tonometer (it measures muscle tone). It provides an accurate measure of the timing of contractions, but not the intensity. If an intrauterine pressure catheter (IUPC) is used, 150–200 Montevideo units (strength of contractions in millimeters of mercury multiplied by the frequency per 10 minutes)

are deemed adequate. The ultimate barometer of uterine activity is the rate of cervical dilation and descent of the presenting part.

Passenger

- Two main fetal variables influence the course of labor: *fetal size* and *attitude* (degree of flexion or extension of the head). When the fetal head is optimally flexed, the smallest possible diameter (suboccipitobregmatic diameter 9.5 cm) presents at the pelvic inlet.
- The lie, presentation, position, and station of the fetus can be assessed on clinical examination. *Lie* refers to the long axis of the fetus relative to the long axis of the uterus, and can be longitudinal, transverse, or oblique. *Presentation* can be either cephalic or breech, referring to the pole of the fetus that overlies the pelvic inlet.

Position refers to the relationship of a nominated site on the presenting part of the fetus to a nominated location on the maternal pelvis, and can be assessed most accurately on bimanual examination. In a cephalic presentation, the nominated site is usually the occiput. In the breech, the nominated site is the sacrum. *Station* refers to the leading bony edge of the presenting part relative to the maternal pelvis (specifically the ischial spines) as assessed on bimanual examination. The vertex is said to be *engaged* when the widest diameter has entered the pelvic inlet, which is best assessed on abdominal examination.

- *Fetal weight* can be estimated clinically or by ultrasound. Using birth weight as the gold standard, both techniques are equally accurate with an error of 15–20%.

Passage

- The bony pelvis is composed of the sacrum, ilium, ischium, and pubis. The shape of the pelvis can be classified into one or more four broad categories: gynecoid, android, anthropoid, and platypelloid. The gynecoid pelvis is the classic female shape.

- Clinical pelvimetry can be used to estimate the shape and adequacy of the bony pelvis, but has not been shown to accurately predict the course of labor or to change clinical management.
- Pelvic soft tissues (cervix and pelvic floor musculature) can provide resistance in labor. In the second stage, the pelvic musculature may play an important role in facilitating rotation and descent of the head.

Excessive resistance may, however, contribute to failure to progress in labor.

PARTOGRAM (Fig. 3)

1. Definition.
2. Parts of the partogram.
3. Description of each part of the program.

ANNEX 2: Partograph

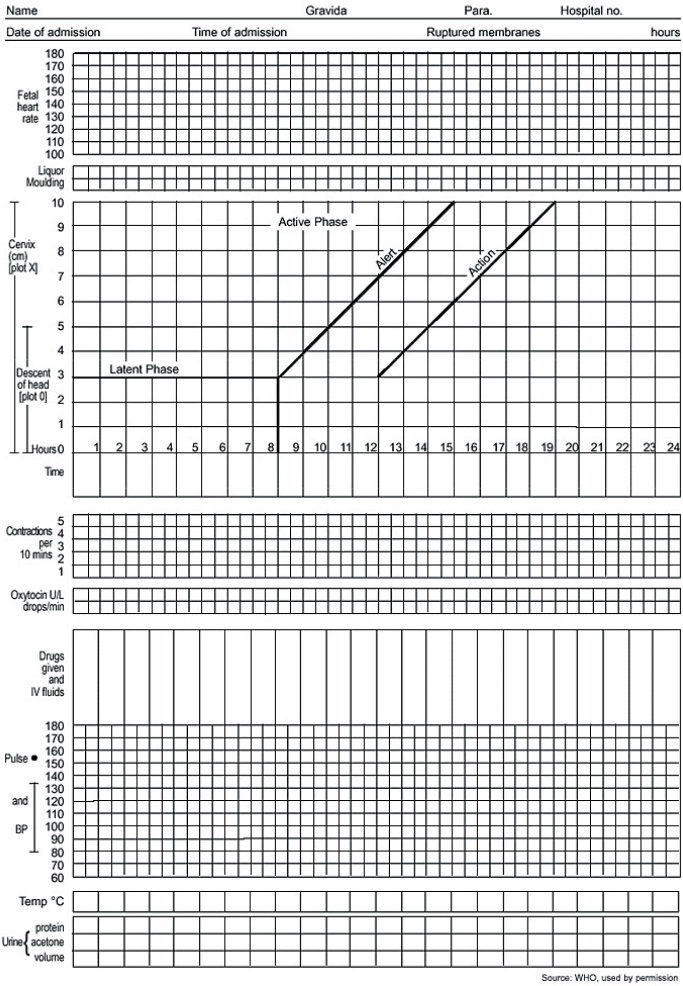


Fig. 3

2 PERIOD OF LABOR

1. Course of labor.
2. Management of labor.
3. Complications.
4. Biomechanism of labor in anterior and posterior occipital presentation.
5. Newborn care.
6. Apgar score.

Normal labor and delivery II

Stages of labor

Labor is a continuous process. For clinical purposes, however, it is divided into three stages:

1 The *first stage* refers to the interval between the onset of labor and full cervical dilation. It is further divided into the *latent phase* (the period between the onset of labor and a point at which a change in the slope of the rate of cervical dilation is noted) and the *active phase* (which is associated with a greater rate of cervical dilation and usually beings at around 3–4 cm dilation). The partogram (Friedman curve) is a graphic representation of the normal labor curve against which a patient's progress is plotted. Normal latent phase is <20 hours in nullipara and <14 hours in multipara. In active phase, the cervix should dilate a minimum of >1.2 cm/h in nullipara (>1.5 cm/h in multipara). A delay in cervical dilation in the active phase of ≥2 hours over that expected suggests labor dystocia and requires further evaluation.

2 The *second stage* commences when the cervix achieves full dilation (10 cm) – not when the mother starts to push – and ends with delivery of the fetus. Prolonged second stage refers to >3 hours with or >2 hours without regional analgesia in a nullipara and >2 hours with or >1 hour without regional analgesia in a multipara.

3 The *third stage* refers to delivery of the placenta and fetal membranes and usually lasts <10 min. In the absence of excessive bleeding, up to 30 min may be allowed before intervention.

Cardinal movements in normal labor

The cardinal movements refer to the changes in position of fetal head required for the fetus to successfully negotiate the birth canal and include the following:

1 *Engagement* refers to passage of the widest diameter of the presenting part to a level below the plane of the pelvic inlet. In a cephalic fetus with a well-flexed head, the largest transverse diameter is the biparietal diameter (9.5 cm). In nulliparas, engagement of the fetal head usually occurs by 36 weeks. Failure of the head to engage by this.

time may be a sign of cephalopelvic disproportion (CPD). In multipara, engagement can occur later or even during labor.

2 *Descent* refers to the downward passage of the presenting part through the pelvis.

3 *Flexion* of the fetal head on to the chest occurs passively as it descends due to the shape of the bony pelvis and the resistance of the pelvic floor. Although flexion of the head is present to some degree in most fetuses before labor, complete flexion occurs only during labor. The result of complete flexion is to present the smallest diameter of the fetal head (suboccipitobregmatic diameter) for optimal passage through the pelvis.

4 *Internal rotation* refers to rotation of the presenting part from its original position (transverse with regard to the birth canal) to the anteroposterior position as it passes through the pelvis. As with flexion, internal rotation is a passive movement resulting from the shape of the pelvis and the pelvic floor musculature. As the head descends, the occiput of the fetus rotates towards the symphysis pubis (or, less commonly, towards the hollow of the sacrum), thereby allowing the widest portion of the fetus to negotiate the pelvis at

its widest dimension. Due to the angle of inclination between the maternal lumbar spine and pelvic inlet, the fetal head engages in an asynclitic fashion (ie, with one parietal eminence lower than the other). With uterine contractions, the leading parietal eminence descends and is first to engage the pelvic floor. As the uterus relaxes, the pelvic floor musculature causes the fetal head to rotate until it is no longer asynclitic.

5 *Extension* occurs once the fetus has descended to the level of the introitus. This descent brings the base of the occiput into contact with the inferior margin at the symphysis pubis. At this point, the birth canal curves upwards. The fetal head is delivered by extension and rotates around the symphysis pubis. The forces responsible for this motion are the downward force exerted on the fetus by the uterine contractions along with the upward forces exerted by the muscles of the pelvic floor.

6 *External rotation*, also known as restitution, refers to the return of the fetal head to the correct anatomic position in relation to the fetal torso. This can occur to either side depending on the orientation of the fetus. This is again a passive movement resulting from a release of the forces exerted on the fetal head by the maternal bony pelvis and its musculature, and mediated by the basal tone of the fetal musculature.

7 *Expulsion* refers to delivery of the rest of the fetus. After delivery of the head and external rotation, further descent brings the anterior shoulder to the level of the symphysis pubis. The anterior shoulder is delivered in much the same manner as the head, with rotation of the shoulder under the symphysis pubis. After the shoulder, the rest of the body is usually delivered without difficulty.

Clinical assistance at delivery

The goals of clinical assistance at delivery are to support the mother psychologically, reduce maternal trauma, prevent fetal injury, and resuscitate the newborn if required.

- As the fetal head crowns, the clinician's hand is used to control delivery and prevent precipitous expulsion (which has been associated with perineal injury in the mother and intracranial hemorrhage in the neonate).
- Mouth and pharynx can be gently suctioned, although this maneuver has largely fallen out of favor as it has not been shown to change perinatal outcome. Vigorous suctioning can cause a vagal response and fetal bradycardia, and should be avoided.
- If a nuchal cord is present, it should be reduced at this time.
- Following restitution of the fetal head, a hand is placed on each parietal eminence and the anterior shoulder delivered by gentle downward traction.
- The posterior shoulder and torso are then delivered by upward traction.
- The umbilical cord should be double clamped and cut. Delayed cord clamping has been shown to increase blood flow to the infant and thus increase its hematocrit. It may have clinical benefit in preterm infants, but has not been shown to significantly improve perinatal outcome at term.
- The infant should be supported at all times.
- The third stage of labor can be managed either passively or actively
- The placenta and fetal membranes should be examined, and the number of blood vessels in the umbilical cord recorded. If indicated, the placenta should be sent for pathologic examination

3 PERIOD OF LABOR

1. Course of the labor.
2. Management of labor.
3. Complications.

Third stage of labor

Definition

- Begins with delivery of the fetus and ends with delivery of the placenta and fetal membranes.

Duration

- Median duration of the third stage of labor is 10 min.
- Of pregnant women 3–5% have a third stage lasting ≥30 min.

Management

- The third stage of labor is usually managed expectantly. Uterine contractions result in cleavage of the placenta between the zona basalis and zona spongiosum.
- The three clinical signs of placental separation include:

1 a sudden gush of blood ("separation bleed")

2 apparent lengthening of the umbilical cord

3 elevation and contraction of the uterine fundus.

- Placental separation can be encouraged by "controlled cord traction" using either the Brandt–Andrews maneuver (where the uterus is secured and controlled traction is applied to the cord) or the Cred maneuver (where the cord is secured and the uterus elevated). Care should be taken to avoid uterine placental inversion.

Complications

- Postpartum hemorrhage
- Retained placenta is defined as failure of the placenta to deliver within 30 min. If there is excessive bleeding, manual removal may be required earlier. Failed manual removal of the placenta suggests abnormal placentation.

POSTPARTUM PERIOD

1. Classification.
2. The involution of organs.
3. Complications.

Lactation.

1. Advantages for the mother and for the baby.
2. The regime of a nursing woman.

The puerperium

Physiology

- The *puerperium* is the 6-week period after delivery when the reproductive tract returns to its non-pregnant state.
- Immediately after delivery, the uterus shrinks down to the level of the umbilicus. By 2 weeks postpartum, it is no longer palpable above the symphysis. By 6 weeks, the uterus has returned to its non-pregnant size.
- Decidual sloughing after delivery results in a physiologic vaginal discharge, known as *lochia*.
- The abdomen will resume its pre-pregnancy appearance, with the notable exception of *abdominal striae* ("stretch marks"). These fade with time.
- Most women will experience the return of menstruation by 6–8 weeks postpartum.

Postpartum care

- In the *immediate postpartum period*, maternal vital signs should be taken frequently, the uterine fundus should be palpated to ensure it is well contracted, and the amount of vaginal bleeding should be noted.
- Early ambulation is encouraged regardless of route of delivery.

Adequate pain management is essential.

- Shortly after birth, neonates should receive topical ophthalmic prophylaxis (to prevent ophthalmia neonatorum) and vitamin K (to prevent hemorrhagic disease of the newborn due to a physiologic deficiency of vitamin K-dependent coagulation factors).
- Prior to discharge, skilled nursing staff should be made available to prepare the mother for care of the newborn. The mother should receive anti-D immunoglobulin (if she is rhesus [Rh] negative and her baby Rh positive) and MMR (measles, mumps, rubella) vaccine (if she is rubella non-immune).
- Coitus can be resumed 2–3 weeks after delivery depending on the patient's desire and comfort. Contraception is necessary to prevent conception.
- A *routine visit* is recommended 6 weeks postpartum. Contraceptive counseling and breastfeeding should be addressed.

Lactation and breastfeeding

- *Advantages.* Breastfed infants have a lower incidence of allergies, gastrointestinal infections, otitis media, respiratory infections, and (possibly) higher intelligence quotient (IQ) scores. Women who breastfeed appear to have a lower incidence of breast cancer, ovarian cancer, and osteoporosis. Breastfeeding is also a bonding experience between infant and mother.
- *Contraindications.* HIV, cytomegalovirus, and possibly chronic hepatitis B or C. Most drugs given to the mother are secreted to some extent into breast milk, but the amount of drug ingested by the infant is typically small. There are some drugs, however, in which breastfeeding is contraindicated (radioisotopes, cytotoxic agents).
- *Physiology.* Prolactin is essential for lactation. Women with pituitary necrosis (Sheehan syndrome) do not lactate. Cigarette smoking, diuretics, bromocriptine, and combined oral contraceptives (not the progestin-only pill) decrease milk production.
- *Colostrum* is a lemon-colored fluid secreted by the breasts during the first 4–5 days postpartum. It contains more minerals

and protein than mature milk, but less sugar and fat. *Mature milk* production is established within a few days. It contains high concentrations of lactose, vitamins (except vitamin K), immunoglobulins, and antibodies.

Complications of the puerperium

Breast engorgement

- May occur on days 2–4 postpartum in women who are not nursing or at any time if breastfeeding is interrupted.
- Conservative measures (tight-fitting brassiere, ice packs, analgesics) are usually effective. Bromocriptine may be indicated in refractory cases.

Mastitis

- Refers to a regional infection of the breast parenchyma, usually by

Staphylococcus aureus.

- *Incidence*. Uncommon. More than 50% of cases occur in primiparas.
- Mastitis is a *clinical diagnosis* with fever, chills, and focal unilateral breast erythema, edema, and tenderness. It usually occurs during the third or fourth week postpartum.
- *Treatment*. Overcome ductal obstruction (by continuing breast-feeding or pumping), symptomatic relief, and oral antibiotics (usually flucloxacillin). Ten percent of women will develop an abscess requiring surgical drainage.

Endometritis

- Refers to a polymicrobial infection of the endometrium that often invades the underlying myometrium.
- *Incidence*. Less than 5% after vaginal delivery, but 5- to 10-fold higher after cesarean section delivery.
- *Risk factors*. Cesarean section delivery, prolonged rupture of membranes, multiple vaginal examinations, manual removal of the placenta, and internal fetal monitoring.

- Endometritis is a *clinical diagnosis* with fever, uterine tenderness, a foul purulent vaginal discharge, and/or increased vaginal bleeding. It occurs most commonly 5–10 days after delivery.
- *Treatment.* Broad-spectrum antibiotics (until the patient is clinically improved and afebrile for 24–48 hours) and dilation and curettage (if retained products of conception are suspected).
- *Complications.* Abscess, septic pelvic thrombophlebitis.

Necrotizing fasciitis

- Refers to necrotic infection of the superficial fascia that spreads rapidly along tissue planes to the abdominal wall, buttock, and/or thigh, leading to septicemia and circulatory failure. Maternal mortality rate approaches 50%.
- *Diagnosis.* Skin edema, blue–brown discoloration, or frank gangrene with loss of sensation or hyperesthesia.
- *Treatment.* Early diagnosis, antibiotics, aggressive surgical debridement.

Psychiatric complaints

- A mild *transient depression* ("postpartum blues") is common after delivery, occurring in >50 % of women.
- *Postpartum depression* occurs in 8–15% of women. Risk factors include a history of depression (30%) or prior postpartum depression (70–85%). Symptoms develop 2–3 months postpartum and resolve slowly over the next 6–12 months. Supportive care and monthly follow-up are necessary.
- *Postpartum psychosis* is rare (1–2 per 1,000 live births). Risk factors include young age, primiparity, and a personal or family history of mental illness. Symptoms typically start 10–14 days postpartum. Hospitalization, pharmacologic and/or electroconvulsive therapy (ECT) may be necessary. Recurrence of postpartum psychosis is high (25–30%).

POSTPARTUM CONTRACEPTION

1. Classification of methods.
2. Advantages.
3. Disadvantages.

- *Definition*. The voluntary prevention of pregnancy ("birth control").
- *Contraceptive options* (Figure 12.1) depend primarily on the motivation of the user, but none is 100% effective, immediately available, and low cost. Of the methods, only male and female condoms reduce the risk of sexually transmitted infections.

Oral contraceptives (OCs).

The most popular method of reversible contraception in the USA and the UK.

- *Composition*. Most are combinations that contain both a synthetic estrogen and a progestin. The progestin-only pill ("minipill") is less popular since it is associated with a higher incidence of irregular bleeding.
- *Administration*. For simplicity, the first pill is often taken on the first day of the period, or on the first Sunday after the first day of the period. Thereafter, one tablet is taken every day for a total of 21 days, followed by 7 days of a placebo pill. An anovulatory (withdrawal) bleed will then occur. Most OC preparations contain 28 tablets to allow a woman to take a tablet every day, which reduces mistakes and missed pills. Alternative contraception (typically condoms) is generally advisable when starting OCs.
- *Formulations*. Recently, some OCs have been designed to induce menses quarterly instead of monthly (Seasonale). Other combined hormone alternatives include a weekly transdermal patch (OrthoEvra), a vaginal ring (NuvaRing)

changed every 3 weeks, or a monthly injection (Lunelle; not available in the USA).

- *Mechanism of action.* All types prevent ovulation through central inhibition of the mid-cycle luteinizing hormone (LH) surge, act peripherally to decrease oviductal function, and thicken cervical mucus.
- *Health benefits.* OCs reduce menstrual cramps and decrease uterine bleeding. They protect against benign breast disease, prevent formation of ovarian cysts, and reduce the incidence and severity of pelvic inflammatory disease (PID). In addition, OCs reduce the risk of endometrial and ovarian cancer, and possibly endometriosis.
- *Side effects.* Irregular breakthrough bleeding (especially if doses are missed), nausea, headache, elevated blood pressure, weight gain, breast pain.
- *Absolute contraindications.* Thromboembolic disease, chronic liver disease, undiagnosed uterine bleeding, pregnancy, and estrogendependent neoplasia.
- *Relative contraindications.* Smoking in women >35 years, migraine headaches, cardiac disease, diabetic complications.

Long-acting injectable contraception

Depomedroxyprogesterone acetate (DMPA, Depo-Provera)

- *Dosing.* This is 150 mg intramuscularly every 12 weeks.
- *Mechanism.* Prevents ovulation by blocking the mid-cycle LH surge.
- *Side effects.* markedly irregular vaginal bleeding, amenorrhea, weight gain, alopecia, reduced libido, depression, and osteopenia.

Subdermal etonogestrel (Nexplanon/Implanon)

- *Dosing.* Single-rod subdermal implant inserted beneath the skin of the upper arm is effective for 3 years.
- *Mechanism.* Prevention of ovulation in addition to impaired oocyte maturation and thickened cervical mucus.

- *Side effects.* Similar to DMPA. Removal usually takes longer than placement and may be mildly uncomfortable due to fibrosis.

Barrier contraception

Works by blocking sperm from getting into the female reproductive tract.

Male condoms (prophylactics, rubbers)

- The most popular barrier method, a latex or polyurethane sheath placed over the penis, prevents deposition of semen in the vagina.
- Disposable, convenient to use, inexpensive, widely available, and prevents the spread of sexually transmitted infections (STIs).

Intravaginal devices

- The *diaphragm* is a circular patch of latex rubber held in place by a collapsible metal frame. It prevents passage of sperm into the cervical canal. It must be used with a spermicidal gel and left in place for 6 hours after intercourse.
- The *female condom* fits loosely inside the vagina and covers the perineum. It is used infrequently.

Spermicides

- Nonoxynol-9, a non-toxic detergent that destroys the cell membrane of sperm, is the main active ingredient.
- Available without prescription as foam, cream, or suppositories.
- Most commonly used with a diaphragm.

Intrauterine device (IUD), also known as LARC (long-acting reversible contraception)

The most commonly used reversible contraceptive worldwide and endorsed as a first-line product regardless of parity.

- *Terminology.* In the USA, all devices that are placed in the uterus to prevent pregnancy are referred to as IUDs. In the UK,

only coppercontaining devices are called IUDs and hormonal intrauterine contraceptives are referred to by the term intrauterine system (IUS).

- *Dosing.* May be inserted at any time in the menstrual cycle once a pre-existing pregnancy has been excluded. Uterine perforation may occur at the time of insertion, but is rare. The expulsion rate is 5% in the first year.
- *Mechanism.* Prevents fertilization and implantation by inducing a local, sterile, inflammatory reaction that is hostile to the oocyte, sperm, and zygote.
- *Side effects.* Menorrhagia and dysmenorrhea are the primary reasons for early removal of the copper T-380A (ParaGard) IUD.

Conversely, the levonorgestrel (Mirena) IUS reduces menstrual blood flow and cramping. There is a very low risk of uterine infection for the first 3 days after insertion of the device.

Emergency contraception (morning-after pill)

- Not recommended as a first-line method. It is typically used after failure of another method or after unprotected sex.
- The risk of pregnancy can be reduced by 75% if taken within 72 hours of unprotected intercourse.
- *Dosing.* Single dose of 1.5 mg levonorgestrel (UK standard) or two pills (each: 0.05 mg ethinylestradiol, 0.25 mg levonorgestrel) followed by a second dose 12 hours later (USA standard). Alternatively, an IUD may be placed to prevent implantation.

Failure rates

The *rhythm method* (periodic abstinence), c*oitus interruptus* (withdrawal of the penis before ejaculation), *postcoital douching*, and *prolonged breastfeeding* are unreliable and not considered methods of contraception because of their high failure rates.

MULTIPLE PREGNANCY

1. Definition.
2. Etiology.
3. Classification.
4. Diagnostics.
5. Management of multiple pregnancies.
6. Complications.
7. Indications for operative delivery.

Multiple pregnancy

Incidence

- This is 1–2% of all deliveries.
- The majority (97–98%) are twin pregnancies; 80% of twin pregnancies are dizygous (derived from two separate embryos).
- Multiple pregnancies are becoming increasingly common, primarily as a result of assisted reproductive technology (ART). This is especially true of higher-order multiple pregnancies (triplets and up) which now constitute 0.1–0.3% of all births.

Diagnosis

- Multiple pregnancy should be suspected in women with risk factors, excessive symptoms of pregnancy, or uterine size greater than expected.
- Ultrasound will confirm the diagnosis.

Chorionicity

- Chorionicity refers to the arrangement of membranes in multiple pregnancies. It has important prognostic implications.
- Perinatal mortality rate is higher with monozygous (30–50%) than with dizygous twins (10–20%), and is especially high with monochorionic/monoamniotic twins (65–70%).

- Chorionicity is determined most accurately by examination of the membranes after delivery. Antenatal diagnosis is more difficult. Identification of separate sex fetuses or two separate placentas confirm dichorionic/diamniotic placentation.

Complications

Antepartum complications develop in 80% of multiple pregnancies compared with 30% of singleton pregnancies.

1 Multiple pregnancies account for 10% of all *perinatal deaths.*

2 *Preterm delivery* increases as fetal number increases: the average length of gestation is 40 weeks in singletons, 37 weeks in twins,33 weeks in triplets, and 29 weeks in quadruplets.

3 *Preterm premature rupture of membranes* occurs in 10–20% of multiple pregnancies.

4 *Fetal growth discordance* (defined as a ≥20% difference in estimated fetal weight between fetuses) occurs in 5–15% of twins and 30% of triplets. Perinatal mortality is increased sixfold.

5 *Intrauterine demise* of one twin.

6 *Twin polyhydramnios/oligohydramnios sequence* results from an imbalance in blood flow from the "donor" twin to the "recipient." Both twins are at risk for adverse events. Twin–twin transfusion syndrome is a subset of polyhydramnios/oligohydramnios sequence seen in 15% of monochorionic pregnancies, and is due to vascular communications between the fetal circulations. After delivery, a difference in birth weight of ≥20% or a difference in hematocrit ≥5 g/dL confirms the diagnosis. Prognosis depends on gestational age, severity, and underlying etiology. Overall perinatal mortality rate is 40–80%. Treatment options include expectant management, serial amniocentesis, indomethacin (to decrease fetal urine output), laser obliteration of the placental vascular communications, or selective fetal reduction.

7 *"Stuck-twin" syndrome* is an ultrasound diagnosis with severe oligohydramnios of the affected fetus which appears "vacuum packed"

in its membranes. In 40% of cases, this represents severe polyhydramnios/oligohydramnios sequence. Perinatal mortality is very high.

8 *Twin reversed arterial perfusion (TRAP) sequence* is a rare complication of monozygotic twinning (1 in 35,000 deliveries) in which vascular communications within the umbilical cord or placenta cause blood to flow from one twin retrograde up the umbilical arteries to its co-twin before returning to the placenta. As a result, the co-twin (known as the "acardiac" twin) develops multiple congenital anomalies, including absent head and trunk regions, absent cardiac structures, and reduction anomalies in other organ systems. Prognosis for the normal twin may be improved if the acardiac twin is removed.

9 *Cord entanglement* is rare (1 in 25,000 births), but may occur in up to 70% of monochorionic/monoamniotic pregnancies and account for >50% of perinatal mortality in this subgroup. As such, delivery is usually by cesarean section. The risk of death due to cord entanglement appears to decrease after 32 weeks, although there is an overall increase in perinatal mortality in the third trimester. For this reason, monochorionic/monoamniotic twin pregnancies are usually delivered at 32–34 weeks.

Management issues specific to multiple pregnancy

Selective fetal reduction

- Of higher-order multiple pregnancies 10–15% will reduce spontaneously during the first trimester. For those that do not reduce, selective fetal reduction to twins at 13–15 weeks has been recommended.
- The procedure-related loss rate before 20 weeks is 15% (range 5–35%), which is comparable to the background risk for higher-order multiple pregnancies.
- The benefits of selective reduction include increased gestational length, increased birthweight, and reduced prematurity and perinatal mortality. For quadruplet pregnancies and upward, the benefits of selective reduction clearly outweigh the risks. In the absence of fetal anomaly, no clear benefit has been demonstrated for reduction of twins to a singleton.

Whether triplet pregnancies benefit from selective reduction to twins, however, remains controversial. Overall, reduction of triplets to twins seems to result in a more satisfactory pregnancy outcome.

Screening for congenital anomalies

- Second trimester maternal serum analyte screening for aneuploidy and/or maternal serum α-fetoprotein (MS-AFP) for open neural tube defect is available for twins (not triplets), as it is for singletons at 15–20 weeks' gestation. *First trimester aneuploidy screening* (nuchal translucency + serum pregnancy-associated plasma protein A [PAPPA] and β human chorionic gonadotropin [βhCG]) is rapidly becoming the preferred aneuploidy screening test for multiple pregnancies.
- In dizygous twin pregnancies, the risk of *aneuploidy* (genetic abnormality) is independent for each fetus. As such, the chance that one or both fetuses have a karyotypic abnormality is greater than for a singleton. US practice favors offering amniocentesis when the probability of aneuploidy is equal to or greater than the procedure-related pregnancy loss rate (quoted as 1 in 400). In singleton pregnancies, this balance is reached at a maternal age at delivery of 35 years. In twin pregnancies, this balance is reached at a maternal age at delivery of around 32 years.

Route of delivery

- Recommended route of delivery of twins depends on presentation, gestational age (or estimated fetal weight), and maternal and fetal wellbeing.
- Cesarean section delivery has traditionally been recommended for multiple pregnancies in which the presenting fetus is not vertex and for all higher-order multiple pregnancies, although vaginal delivery may be appropriate in selected patients.

OLIGOAMNIOS. HYDRAMNION

1. Reasons.
2. Diagnostics.
3. Management during delivery.
4. Complications.

Disorders of amniotic fluid volume

Embryology of the amniotic cavity

The *amnion* is a thin fetal membrane that begins to form on the eighth post-conceptional day as a small sac covering the dorsal surface of the embryonic disc. The amnion gradually encircles the growing embryo.

Amniotic fluid fills the amniotic cavity.

Amniotic fluid dynamics

Maintenance of amniotic fluid volume is a dynamic process that reflects a balance between fluid production and absorption.

Fluid production

- Before 8 weeks, amniotic fluid is produced by passage of fluid across the amnion and fetal skin (transudation).
- At 8 weeks, the fetus begins to urinate into the amniotic cavity. Fetal urine quickly becomes the primary source of amniotic fluid production.

Near term, 800–1000 mL of fetal urine is produced each day.

- The fetal lungs produce some fluid (300 mL per day at term), but much of it is swallowed before entering the amniotic space.

Fluid absorption

- Before 8 weeks' gestation, transudative amniotic fluid is passively reabsorbed.

- At 8 weeks' gestation, the fetus begins to *swallow*. Fetal swallowing quickly becomes the primary source of amniotic fluid absorption.

Near term, 500–1000 mL of fluid are absorbed each day by fetal swallowing.

- A lesser amount of amniotic fluid is absorbed through the fetal membranes and enters the fetal bloodstream. Near term, 250 mL of amniotic fluid is absorbed by this route every day.
- Small quantities of amniotic fluid cross the amnion and enter the maternal bloodstream (10 mL per day near term).

Changes in volume during pregnancy

Amniotic fluid volume is maximal at 34 weeks (750–800 mL) and decreases thereafter to 600 mL at 40 weeks. The amount of fluid continues to decrease beyond 40 weeks.

The role of amniotic fluid

Amniotic fluid has a number of critical functions, including:

- Cushioning the fetus from external trauma.
- Protecting the umbilical cord from compression.
- Allowing unrestricted fetal movement, thereby promoting the development of the fetal musculoskeletal system.
- Contributing to fetal pulmonary development.
- Lubricating the fetal skin.
- Preventing maternal chorioamnionitis and fetal infection through its bacteriostatic properties.
- Assisting in fetal temperature control.

Measurement of amniotic fluid volume

Ultrasonography is a more accurate method of estimating amniotic fluid than measurement of fundal height. Several techniques are described:

- Subjective assessment of amniotic fluid volume.
- Measurement of the single deepest pocket free of umbilical cord, known as maximal vertical pocket (MVP).

- Amniotic fluid index (AFI) is a semiquantitative method for estimating amniotic fluid volume, which minimizes inter- and intraobserver error. AFI refers to the sum of the maximum vertical pockets of amniotic fluid (in centimeters) in each of the four quadrants of the uterus.

Normal AFI beyond 20 weeks' gestation ranges from 5 cm to 20 cm.

Clinical importance of amniotic fluid volume

- Amniotic fluid volume is a marker of *fetal wellbeing.*
- Normal amniotic fluid volume suggests that uteroplacental perfusion is adequate. An abnormal amount of amniotic fluid volume is associated with an unfavorable perinatal outcome.

Oligohydramnios

- *Definition.* An abnormally low amount of amniotic fluid around the fetus.
- *Incidence.* This is 5–8% of all pregnancies.
- *Diagnosis.* Oligohydramnios should be suspected if the fundal height is significantly less than expected for gestational age. It is defined sonographically as a total amniotic fluid volume <300 mL, the absence of a single 2-cm vertical pocket, or an AFI <5 cm at term or <5th percentile for gestational age.
- *Causes.*
- *Management.* Antepartum treatment options are limited, unless a structural defect (such as posterior urethral valve in a male infant) is amenable to *in utero* surgical repair. The timing of delivery depends on gestational age, etiology, and fetal well-being. During labor, infusion of crystalloid solution into the amniotic cavity (*amnioinfusion*) may improve abnormal fetal heart rate patterns (particularly in the setting of repetitive variable decelerations) and, possibly, decrease the cesarean delivery rate.
- *Outcome.* Oligohydramnios is associated with increased perinatal morbidity and mortality at any gestational age.
- *Complications.* Amniotic band syndrome (adhesions between the amnion and fetus causing serious deformities, including

limb amputation) or musculoskeletal deformities due to uterine compression (such as clubfoot) may develop in some cases.

Polyhydramnios

- *Definition*. An abnormally large amount of amniotic fluid surrounding the fetus.
- *Incidence.* This is 0.5–1.5% of all pregnancies.
- *Diagnosis.* Polyhydramnios should be suspected if the fundal height is significantly more than expected for gestational age. It is defined sonographically as a total amniotic fluid volume >2 L, a single vertical pocket ≥10 cm, or an AFI >20 cm at term or >95th percentile for gestational age.
- *Causes*
- *Management.* Antepartum treatment options are limited. Nonsteroidal anti-inflammatory drugs (indomethacin) can decrease fetal urine production, but may cause premature closure of the fetal ductus arteriosus. Removal of fluid by amniocentesis is only transiently effective. During labor, controlled amniotomy may reduce the incidence of complications resulting from rapid decompression (placental abruption, cord prolapse).
- *Outcome.* Polyhydramnios has been associated with increased maternal morbidity as well as perinatal morbidity and mortality.
- *Complications.* Uterine overdistension may result in maternal dyspnea or refractory edema of the lower extremities and vulva. During labor, polyhydramnios can result in fetal malpresentation, dysfunctional labor, and/or postpartum hemorrhage.

LARGE FETUS

1. Etiology.
2. Diagnostics.
3. Complications in labor.

Disorders of fetal growth

Definitions

- *Low birthweight* (LBW) refers to infants with an absolute birth weight <2500 g regardless of gestational age.
- *mall-for-gestational-age* (SGA) fetuses are <10th percentile for gestational age. Fetuses >90th percentile are termed "*large for gestational age*" (LGA). Fetuses between the 10th and 90th percentiles are referred to as "appropriate for gestational age" (AGA). Correct assignment of fetal weight category is dependent on accurate dating of the pregnancy because birthweight is a function of both gestational age and rate of fetal growth.

Intrauterine growth restriction

- *Definition*. Intrauterine growth restriction (IUGR) refers to any fetus that fails to reach its full growth potential.
- *Incidence*. Of fetuses 4–8% are diagnosed with IUGR.
- *Classification*. IUGR can be classified as *symmetric* (in which the fetus is proportionally small, suggesting long-term compromise) or *asymmetric* (in which the fetal head is proportionally larger than the body, suggesting short-term compromise with "sparing" of the brain).

This distinction is, however, of little clinical value.

- *Causes*. IUGR represents the clinical end-point of many different fetal, uteroplacental, and maternal conditions. An attempt should be made to determine the cause before delivery in order to provide counseling, perform ultrasonographic evaluation for

fetal growth and delineation of anatomy, and obtain neonatal consultation. Frequently, the cause is readily apparent.

- *Risk factors.* Numerous pre-existing and acquired conditions predispose the fetus to IUGR.
- *Diagnosis.* The clinical diagnosis of IUGR is unreliable, but a fundal height measurement significantly less than expected (3–4 cm) for gestational age may suggest the diagnosis. IUGR is confirmed by sonographic measurements.
- *Pathophysiology.* IUGR most commonly results from compromise of uteroplacental blood flow.
- *Prevention.* Bed rest and low-dose aspirin have been used to prevent IUGR in women at high risk, but with little or no benefit.
- *Management.* Principles of management include:

1 the identification of women at high risk for IUGR;

2 early antepartum diagnosis;

3 determination of etiology;

4 regular (usually weekly) fetal testing with non-stress test (NST) or cardiotocography;

5 appropriate timing of delivery.

- *Complications.* IUGR infants have higher rates of perinatal morbidity and mortality at any given gestational age, but have a better prognosis than infants with the same birthweight delivered at earlier gestational ages. Unfortunately, neonatal morbidity (meconium aspiration syndrome, hypoglycemia, polycythemia, pulmonary hemorrhage) will be present in 50% of IUGR neonates. Long-term studies show a 38-fold increase in the incidence of cerebral dysfunction (ranging from minor learning disabilities to cerebral palsy) in term IUGR infants and even more so if the infant was born preterm.

Fetal macrosomia

- *Definition.* Fetal macrosomia is defined as an estimated weight (not birthweight) of ≥4500 g.

- *Incidence.* In developing countries, 5% of infants weigh >4000 g at delivery and 0.5% weigh >4500 g.
- *Risk factors.* Although a number of factors have been associated with macrosomia, most women with risk factors have normal weight babies:

1 *Maternal diabetes* (35–40% of all macrosomic infants) is the most common risk factor.

2 *Post-term pregnancy* (10–20%) is another common risk factor. Of all infants born at or beyond 42 weeks, 2.5% weigh >4500 g.

3 *Maternal obesity* (10–20%), defined as a pre-pregnancy body mass index (BMI) >30 kg/m2, predisposes to fetal macrosomia. Moreover, clinical and ultrasound estimates of fetal weight in obese women are technically more difficult and may be less accurate.

4 *Other risk factors* include multiparity, a prior macrosomic infant, a male infant, increased maternal height, advanced maternal age, and Beckwith–Wiedemann syndrome.

- *Diagnosis.* Clinical estimates of fetal weight based on the Leopold maneuvers or fundal height measurements are often unreliable. Ultrasound is generally used to estimate fetal weight

However, currently available ultrasonographic techniques are accurate only to within 15–20% of actual fetal weight.

- *Prevention.* Meticulous control of maternal diabetes throughout pregnancy reduces the incidence of fetal macrosomia.
- *Management:*

1 *Antepartum.* Women at high risk for having a macrosomic infant or who have a known LGA fetus should be followed with serial ultrasound examinations at 3–4 weeks to chart fetal growth.

2 *Induction of labor.* Despite the association between fetal macrosomia, and both birth trauma and cesarean section delivery, early induction of labor is not often recommended in patients with suspected fetal macrosomia at term. Induction of labor in this setting doubles the risk of cesarean section delivery without reducing shoulder

dystocia or neonatal morbidity. However, induction of labor for "impending macrosomia" does not decrease the cesarean section rate. As such, this approach should not be encouraged.

3 To prevent birth trauma, *elective (prophylactic) cesarean section delivery* should be offered to diabetic women with an estimated fetal weight >4500 g and non-diabetic women with estimated fetal weight >5000 g.

4 *Vaginal delivery* of a macrosomic infant should take place in a controlled fashion, with immediate access to anesthesia staff and a neonatal resuscitation team. It is prudent to avoid assisted vaginal delivery in this setting.

- *Fetal morbidity and mortality.* Macrosomic fetuses have an increased risk of intrauterine and neonatal death and birth trauma, especially shoulder dystocia and brachial plexus palsy. Other neonatal complications include hypoglycemia, polycythemia, hypocalcemia, and jaundice.
- *Maternal morbidity.* The increased maternal morbidity associated with the birth of a macrosomic infant is due primarily to a higher incidence of cesarean section delivery. Other maternal complications include postpartum hemorrhage, perineal trauma, and puerperal infection.

BREECH PRESENTATION

1. Definition.
2. Etiology.
3. Classification.
4. Diagnosis and differential diagnosis.
5. Management of a breech presentation in pregnancy.
6. Management of a breech presentation in labor.
7. Mechanism of labor.
8. Methods, which help to deliver big parts of the fetus.
9. Complications during pregnancy and labor.
10. Indications for Caesarean Section.

Breech presentation

It is the situation, the lie is longitudinal and pre-lying part of the fetus are buttocks or legs.

Etiology

Maternal
Fetal
Placental

Classification

Complete
Incomplete (Frank breech, Footling presentation, Knee presentation)

Diagnosis and differential diagnosis

Abdominal examination
Auscultation of the fetal heart
Vaginal examination
Ultrasound
Amnioscopy
Differential diagnosis-between breech and face presentation.

Management of a breech presentation in pregnancy

Keep a diet
Make exercises to move baby
External cephalic version at 36 weeks of the pregnancy

Management of a breech presentation in labor

Hospitalization 2 weeks before the labor
Assessment conditions for vaginal labor
Vaginal examination according to indication
Bed – rest during 1 stage of the labor
Delivery from umbilical cord before head must last not more than 3–5 min.

Mechanism of labor

Sacro-anterior position

Descent of the buttocks and internal rotation. The diameter of engagement of the buttock is one of the oblique diameters of the inlet. The engaging diameter is bi-trochanteric (10 cm) with the sacrum directed towards the ilio-pubic eminence.

Lateral flexion of the trunk and delivery of the trunk and the lower limbs

Restitution. Bisacromial diameter (12 cm) engages in the same oblique diameter as that occupied by the buttocks at the brim soon after the delivery of the breech.

Bisacromial diameter is in the anteroposterior diameter of the pelvic outlet.

Anterior flexion of the trunk. Delivery of the posterior shoulder, then anterior one.

Internal rotation of the occiput occurs anteriorly. The engaging diameter of the head is suboccipitobregmatic (9.5 cm).

Flexion of the head. The head is born by flexion—the chin, mouth, nose, forehead, vertex and occiput appearing successively.

Methods, which help to deliver big parts of the fetus

Episiotomy

Burns-Marshall method

Forceps delivery

modified Mauriceau-SmellieVeit technique

LOVSET'S MANEUVER

Complications during pregnancy and labor

Risks for the mother

Risks for the fetus

Indications for Caesarean Section

Post-term pregnancy

Abnormalities in development of the genital tract

Contracted pelvis

Hypoxia

Weight of the fetus > 3500 and <2000 gr

The unfolded head of the fetus

Long infertility

Anamnesis about dead children during labor.

HYPERTENSIVE DISORDERS DURING PREGNANCY

1. Classification.
2. Etiopathogenesis.
3. Gestational hypertension.
4. Preeclampsia, diagnostic criteria. Management tactics.
5. Eclampsia. Stages of an attack of convulsions. Management tactics.
6. HELLP syndrome. Management tactics.

Hypertensive disorders of pregnancy

Hypertensive disorders of pregnancy are the second most common cause of maternal death in developed countries (after embolism), accounting for 15% of all maternal deaths.

Effects of pregnancy on maternal cardiovascular system

- Blood volume increases 800 mL by 12 weeks (1.5 L in twins).
- Blood pressure (BP) decreases in early pregnancy (due primarily to a decrease in systemic vascular resistance secondary to progesterone), nadirs in mid-pregnancy, and returns to baseline by term.

Classification

1 Chronic hypertension

- *Definition*. Hypertension before pregnancy. The diagnosis should also be entertained in women with BP ≥140/90 mmHg before 20 weeks' gestation.
- *Complications.* Such pregnancies are at increased risk of superimposed pre-eclampsia, intrauterine fetal growth restriction (IUGR), placental abruption, and stillbirth.
- *Management.* Continue antihypertensive medications with the exception of angiotensin-converting enzyme (ACE) inhibitors. These drugs have been associated with progressive

and irreversible renal injury and possibly other structural anomalies in the fetus. Diuretic therapy is generally discouraged.
- Fetal testing (serial ultrasound examinations for fetal growth with or without fetal non-stress testing) should be initiated after 32 weeks' gestation. Delivery should be achieved by 40 weeks.

2 Chronic hypertension with superimposed pre-eclampsia (see Preeclampsia below)

3 Gestational hypertension
- Also known as gestational non-proteinuric hypertension.
- *Diagnosis.* Persistent elevation of BP ≥140/90 mmHg in the third trimester without evidence of pre-eclampsia. It is a diagnosis of exclusion that is best made retrospectively.
- *Etiology.* It probably represents an exaggerated physiologic response of the maternal cardiovascular system to pregnancy.
- Rarely associated with adverse maternal or fetal outcome.

4 Preeclampsia
- Also known as gestational proteinuric hypertension, preeclamptic toxemia (PET).
- *Definition.* A multisystem disorder specific to pregnancy and the puerperium. More precisely, it is a disease of the placenta because it occurs in pregnancies where there is trophoblast but no fetal tissue (complete molar pregnancies).
- *Incidence.* Six to eight percent of all pregnancies.
- *Risk factor.*
- *Diagnosis.* A clinical diagnosis with two elements:

1 *New-onset hypertension* defined as a sustained sitting BP ≥140/90 mmHg in a previously normotensive woman (a prior definition included an elevation in systolic BP ≥30 or diastolic BP ≥15 mmHg over first trimester BP, but these criteria have now been dropped).

2 *New-onset significant proteinuria* defined as >300 mg/24 h or ≥1+ on a clean-catch urine in the absence of urinary tract infection.

Note. A definitive diagnosis of preeclampsia should only be made after 20 weeks' gestation. Evidence of gestational proteinuric hypertension

before 20 weeks should raise the possibility of an underlying molar pregnancy, drug withdrawal, or (rarely) chromosomal abnormality in the fetus.

- *Classification.* Preeclampsia is classified as "mild" or "severe." There is no category of "moderate" pre-eclampsia.
- *Etiology.* The cause of preeclampsia is not known. Theories include an abnormal maternal immunologic response to the fetal allograft, an underlying genetic abnormality, an imbalance in the prostanoid cascade, and the presence of circulating toxins and/or endogenous vasoconstrictors.

What is known is that the blueprint for the development of preeclampsia is laid down early in pregnancy. The primary event is a failure of the second wave of trophoblast invasion from 8 weeks to 18 weeks, which is responsible for remodeling of the spiral arterioles in the myometrium adjacent to the developing placenta, and establishment of the definitive uteroplacental circulation. As pregnancy progresses and the metabolic demand of the fetoplacental unit increases, the spiral arterioles are therefore unable to accommodate the necessary increase in blood flow. This then leads to the development of "placental dysfunction" which manifests clinically as preeclampsia. Although attractive, this hypothesis remains to be validated. Whatever the placental abnormality, the end-result is widespread vasospasm and endothelial injury.

- *Complications*). Eclampsia – defined as one or more generalized convulsions or coma in the setting of preeclampsia and in the absence of other neurologic conditions – was thought to be the end stage of preeclampsia, hence the nomenclature. It is now clear, however, that seizures are but one clinical manifestation of "severe" pre-eclampsia; 50% of eclampsia occurs preterm. Of those at term, 75% occur either intrapartum or within 48 hours of delivery.
- *Management.* Delivery is the only effective treatment for preeclampsia, and is recommended:

1 in women with "mild" preeclampsia once a favorable gestational age has been reached (>36–37 weeks).

2 in all women with "severe" preeclampsia regardless of gestational age (with the exception of "severe" preeclampsia due to proteinuria alone or intrauterine growth restriction [IUGR] remote from term with good fetal testing). There has also been a recent trend toward expectant management of "severe" preeclampsia by BP criteria alone at <32 weeks' gestation.

- There is no proven benefit to routine delivery by cesarean section. However, the probability of vaginal delivery in a patient with preeclampsia remote from term with an unfavorable cervix is only 15–20%.
- BP control is important to prevent cerebrovascular accident (usually associated with BP ≥170/120 mmHg), but does not affect the natural course of preeclampsia.
- Intravenous magnesium sulfate should be given intrapartum and for at least 24 hours postpartum to prevent eclampsia.
- *Prevention.* Despite promising early studies, low-dose aspirin (acetylsalicylic acid or ASA) and/or supplemental calcium does not prevent preeclampsia in either high- or low-risk women.
- *Prognosis.* Preeclampsia and its complications always resolve after delivery (with the exception of cerebrovascular accident). Diuresis (>4 L/day) is the most accurate clinical indicator of resolution. Fetal prognosis is dependent largely on gestational age at delivery and problems related to prematurity.

FETO-PLACENTAL INSUFFICIENCY

1. Classification.
2. Etiology.
3. Diagnostics.
4. Treatment.

Placental insufficiency or **feto/utero-placental insufficiency** is the failure of the placenta to deliver sufficient nutrients to the fetus during pregnancy, and is often a result of insufficient blood flow to the placenta. The term is also sometimes used to designate late decelerations of fetal heart rate as measured by cardiotocography or an NST, even if there is no other evidence of reduced blood flow to the placenta, normal uterine blood flow rate being 600ml/min.

Causes

The following characteristics of placentas have been said to be associated with placental insufficiency, however all of them occur in normal healthy placentas and full term healthy births, so none of them can be used to accurately diagnose placental insufficiency:

- Abnormally thin placenta (less than 1 cm).
- Circumvallate placenta (1% of normal placentas).
- Amnion cell metaplasia, (amnion nodosum) (present in 65% of normal placentas).
- Increased syncytial knots.
- Calcifications.
- Infarcts due to focal or diffuse thickening of blood vessels.
- Vill capillaries occupying about 50% of the villi volume or when <40% of capillaries are on the villous periphery.

Placental insufficiency should not be confused with complete placental abruption, in which the placenta separates off the uterine wall, which immediately results in no blood flow to the placenta, which leads to immediate fetal demise. In the case of a marginal, incomplete placental abruption of less than 50%, usually weeks

of hospitalization precedes delivery and outcomes are not necessarily affected by the partial abruption.

Maternal effects

Several aspects of maternal adaptation to pregnancy are affected by dysfunction of placenta. Maternal arteries fail to transform into low-resistance vessels (expected by 22–24 weeks of gestation). This increases vascular resistance in fetoplacental vascular bed eventually leading to reduction in metabolically active mass of placenta like a vicious cycle.

Fetal effects

Placental insufficiency can affect the fetus, causing fetal distress. Placental insufficiency may cause oligohydramnios, preeclampsia, miscarriage or stillbirth. Placental insufficiency is most frequent cause of asymmetric IUGR.

Diagnosis

The following tests have been promoted as supposedly diagnosing placental insufficiency, but all have been unsuccessful at predicting stillbirth due to placental insufficiency:

- Placental grading.
- Amniotic fluid index.
- Fetal biophysical profile test scoring.
- Doppler velocimetry.
- Routine ultrasound scanning.
- Detection and management of maternal diabetes mellitus.
- Antenatal fetal heart rate monitoring using cardiotocography.
- Vibroacoustic stimulation, fetal movement counting.
- Home vs. hospital-based bed rest and monitoring in high-risk pregnancy.
- In-hospital fetal surveillance unit.
- Use of the partograph during labor.
- Cardiotocography during labor with or without pulse oximetry.

Treatment

Placental insufficiency can be treated so as to ensure as few risks as possible during the pregnancy. The kind of treatment required for placental insufficiency will depend on how far along a woman is into her pregnancy. For example, babies past 37 weeks in the womb are considered to be fully developed, and so the doctor may decide to induce labour or perform a C-section. For cases where the condition develops and is detected early on in the pregnancy, other methods of treatment are taken into condition. Some doctors may suggest steroid shots to strengthen the baby's lungs. Proper management of this condition will depend on further tests and the woman's health. If the mother has diabetes or problems with blood pressure, these will need to be monitored and brought under control during pregnancy.

RESUSCITATION OF A NEWBORN (See Fig. 4)

1. Step A.
2. Step B.
3. Step C.
4. Step D.

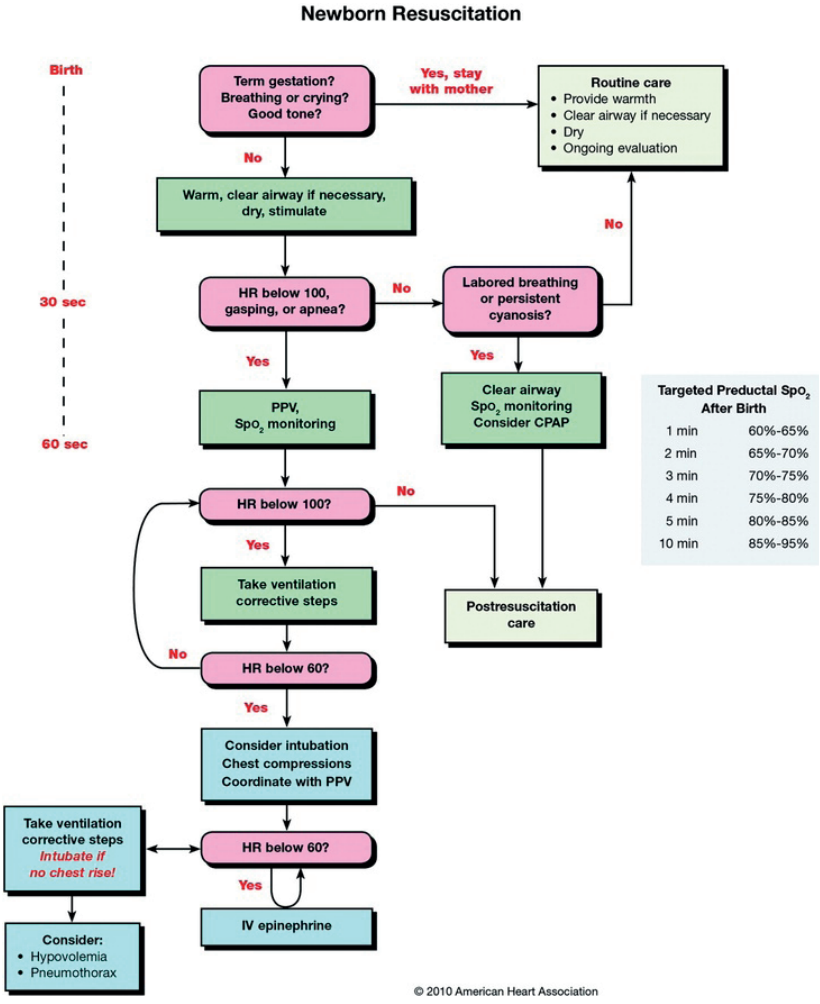


Fig. 4

METHODS FOR ASSESSING THE FETAL STATE (gravidogram, Doppler ultrasound, cordocentesis, amniocentesis, amnioscopy, CTG, chorion biopsy)

1. Gravidogram.
2. Doppler ultrasound.
3. Cordocentesis.
4. Amniocentesis.
5. CTG.
6. Amnioscopy.
7. Chorion biopsy.

Antepartum fetal surveillance

Introduction

Obstetric care providers have two patients: the mother and the fetus.

Assessment of maternal wellbeing is relatively easy, but fetal wellbeing is far more difficult to assess. Several tests have been developed to confirm fetal wellbeing before labor and delivery.

Goal

- There are many causes of irreversible neonatal cerebral injury, including congenital abnormalities, intracerebral hemorrhage, hypoxia, infection, drugs, trauma, hypotension, and metabolic derangements (hypoglycemia, thyroid dysfunction).
- Antenatal fetal testing cannot predict or reliably detect all of these causes. The goal of antepartum fetal surveillance is early identification of a fetus at risk for preventable morbidity or mortality due specifically to uteroplacental insufficiency.
- Antenatal fetal tests make the following assumptions:

1 that pregnancies may be complicated by progressive fetal asphyxia which can lead to fetal death or permanent handicap;

2 that current antenatal tests can adequately discriminate between asphyxiated and non-asphyxiated fetuses;

3 that detection of asphyxia at an early stage can lead to an intervention,

which is capable of reducing the likelihood of an adverse perinatal outcome.

It is not clear whether any of these assumptions are true. At most, 15% of cerebral palsy is due to birth asphyxia.

Note. All antepartum fetal tests should be interpreted in light of the gestational age, the presence or absence of congenital anomalies, and underlying clinical risk factors.

Antepartum fetal tests

Non-stress test (NST)

- Also known as cardiotocography (CTG).
- NST refers to changes in the fetal heart rate pattern with time. It reflects maturity of the fetal autonomic nervous system. NST is non-invasive, simple to perform, readily available, and inexpensive.

However, interpretation is largely subjective.

- *Is a "reactive" NST (R-NST) reassuring?* R-NST is defined as an NST with normal baseline heart rate (110–160 beats/min), moderate variability, and at least two accelerations in 20 minutes each lasting ≥15 s and peaking at ≥15 beats/min above baseline (≥10 beats/min for ≥10 s if <32 weeks). Weekly R-NST after 32 weeks' gestation has been shown to decrease perinatal mortality. R-NST is therefore reassuring.
- *Is a non-reactive NST (NR-NST) worrisome?* NR-NST should be interpreted in light of gestational age: 65% of fetuses will have R-NST by 28 weeks, 95% by 32 weeks. Once R-NST has been documented in a given pregnancy, it should remain so throughout delivery. NR-NST at term is associated with poor perinatal outcome in only 20% of cases. The significance of NR-NST depends on the clinical end-point. If the end-point is a 5-min Apgar score <7, NR-NST at term has a sensitivity of 57%, positive predictive value of 13%, and negative

predictive value of 98% (assuming a prevalence of 4%). If the end-point is permanent cerebral injury, then NR-NST at term has a 99.8% falsepositive rate.

- *Is there a place for vibroacoustic stimulation?* Refers to the response of the fetal heart rate to a vibroacoustic stimulus. Acceleration on NST (≥15 beats/min for ≥15 s) is a positive result. It is a useful adjunct to decrease the time to achieve R-NST and to decrease the proportion of NR-NST at term, thereby precluding the need for further testing.

Biophysical profile

- Biophysical profile (BPP) refers to a sonographic scoring system designed to assess fetal wellbeing.
- The five variables described in the original BPP were: NST, fetal movement, fetal tone, amniotic fluid volume, and fetal breathing. Twopoints are awarded if the variable is present or normal, 0 points if absent or abnormal. Amniotic fluid volume is the most important variable.

More recently, BPP is interpreted without the NST.

- Recommended management based on the original BPP:

Fetal movement charts ("kickcharts")

- Maternal appreciation of fetal movement is reliable.
- Fetal movement decreases with advancing gestational age, oligohydramnios, smoking, and antenatal corticosteroid therapy.
- "Kickcharts" involve either counting all fetal movements in 1 hour or counting the time that it takes the fetus to kick 10 times ("countto- ten"). Measurements should be repeated at least twice daily.
- Use of "kickcharts" in high-risk pregnancies can decrease perinatal mortality fourfold.

Doppler velocimetry

- Umbilical artery Doppler velocimetry measurements reflect resistance to blood flow from the fetus to the placenta.

- Absent or reversed diastolic flow is associated with poor perinatal outcome in the setting of intrauterine growth restriction (IUGR), and urgent delivery should be considered. It is unclear how to interpret these data in the setting of a normally grown fetus.
- Abnormal flow in the middle cerebral artery (MCA) and ductus venosus may help in the timing of delivery of IUGR fetuses.

Contraction stress test (CST)

- CST refers to the response of the fetal heart rate to artificially induced uterine contractions. A minimum of three contractions in 10 minutes are required to interpret the test. A negative CST (no decelerations with contractions) is reassuring. A positive CST (severe variable or late decelerations with ≥50% of contractions) is associated with adverse perinatal outcome in 35–40% of cases. However, the falsepositive rate exceeds 50%. An equivocal CST should be repeated in 24–72 hours. More than 80% of repeat tests will be negative.
- As this test is time-consuming, requires skilled nursing care, and may precipitate "fetal distress" needing emergency cesarean section delivery, it is not routinely used in clinical practice.

Intrapartum fetal monitoring

Introduction

- Fetal morbidity and mortality can occur as a consequence of labor.

A number of tests have been developed to assess fetus wellbeing.

- Attention has focused on *hypoxic ischemic encephalopathy* (HIE) as a marker of birth asphyxia and a predictor of long-term outcome. HIE is a clinical condition that develops within the first hours or days of life. It is characterized by abnormalities of tone and feeding, alterations in consciousness, and convulsions. In order to attribute such a state to birth asphyxia, the following four criteria must all be fulfilled:

1 profound metabolic or mixed acidemia (pH <7.00) on an umbilical cord arterial blood sample, if obtained;

2 Apgar score of 0–3 for longer than 5 min;

3 neonatal neurologic manifestations (seizures, coma);

4 multisystem organ dysfunction.

• At most, only 15% of cerebral palsy and learning disability can be attributed to HIE.

Intrapartum fetal monitoring

Non-stress test (NST) or fetal cardiotocography (CTG)

A fetal scalp electrode for the continuous monitoring of the fetal heart rate during labor was introduced by Hon and Lee in 1963. A year later, Doppler technology made external fetal heart analysis possible. Continuous intrapartum CTG is now recommended for all high-risk pregnancies and is commonly used in low-risk pregnancies too.

Characteristics of intrapartum fetal heart rate patterns

- *Baseline fetal heart rate* refers to the dominant reading taken over ≥10 min. Normal baseline fetal heart rate is 110–160 beats/min. Bradycardia is a baseline rate <110 beats/min. Tachycardia is a baseline rate >160 beats/min.
- *Fetal heart rate variability* is classified as moderate (which refers to peak-to-trough excursions of 5–25 beats/min around the baseline, and is a healthy sign), minimal (<5 beats/min excursions, which is concerning for hypoxia and requires further evaluation), absent (0 beats/min excursions, which is worrisome for hypoxia), or marked (>25 beats/min excursions, which suggests hypoxia without acidosis).
- *Accelerations* are periodic, transient increases in fetal heart rate of ≥15 beats/min for ≥15 s (or ≥10 beats/min for ≥10 s for fetuses <32 weeks). Accelerations are often associated with fetalactivity, and are a sign of a healthy fetus (specifically the absence of metabolic acidosis).

- *Decelerations* are periodic, transient decreases in fetal heart rate usually associated with uterine contractions. They can be further classified into early, variable, or late decelerations by their shape and timing in relation to contractions. Decelerations are regarded as "repetitive" if they occur with more than 50% of contractions.

Interpretation of NST

- Fetal heart rate patterns in labor are classified as:

1 "reactive" (defined as two or more accelerations in 20 min), which is considered reassuring;

2 suspicious or equivocal (indeterminate);

3 ominous or agonal (non-reassuring).

- Reassuring elements of the intrapartum fetal heart rate include normal baseline, moderate variability, and accelerations (class I). Nonreassuring elements include bradycardia, tachycardia, minimal or absent variability, and/or repetitive severe variable or late decelerations (class III). Class II refers to an intermediate pattern.
- Non-reassuring patterns are seen in up to 60% of labors, suggesting that they are not specific to fetal hypoxia. Severely abnormal fetal heart rate patterns (specifically, repetitive severe variable or late decelerations), on the other hand, occur in only 0.3% of intrapartum fetal heart rate tracings.
- NST interpretation is largely subjective and should always take into account gestational age, the presence or absence of congenital anomalies, and underlying clinical risk factors. Fetuses that are premature or growth restricted are less likely to tolerate episodes of decreased placental perfusion and, as such, may be more prone to hypoxia and acidosis. Drugs can also affect heart rate and variability.
- Only two intrapartum fetal heart rate patterns have been associated with poor perinatal outcome, namely repetitive severe variable (defined as decreasing to <70 beats/min and lasting for ≥ 60 s) and repetitive late decelerations.

- When compared with intermittent fetal heart rate auscultation, continuous fetal heart rate monitoring during labor is associated with a decrease in the incidence of seizures before 28 days of life, but no difference in other measures of short-term perinatal morbidity or mortality. Moreover, the increase in neonatal seizures does not translate into differences in long-term morbidity (cerebral palsy, learning disability, or seizures after 28 days of life). However, continuous fetal heart rate monitoring is associated with a significant increase in obstetric intervention, including surgical vaginal and cesarean section delivery.
- Several unusual fetal heart rate patterns have been described:

1 A *salutatory* pattern (in which there are large oscillations in baseline) is of unclear clinical significance. It may indicate intermittent cord occlusion.

2 A *lambda* pattern (an acceleration followed by a deceleration) is attributed to fetal movement. It is not felt to be of pathological significance.

3 A *sinusoidal* pattern (one with normal baseline, decreased variability, and a cyclic sinusoidal pattern with a frequency of 2–5 cycles/min and amplitude of 5–15 beats/min) is associated most strongly with fetal anemia. It may also be seen in the setting of chorioamnionitis, impending fetal demise, and maternal drug administration (especially opiate analgesics).

Fetal scalp sampling

- The pH of fetal capillary blood lies between that of fetal arterial and venous blood (see table below).
- Fetal scalp blood sampling was introduced by Saling in 1962. It is most useful in labor when alternative non-invasive tests are unable to confirm fetal wellbeing.
- Suggested management based on fetal scalp pH:

RH NEGATIVE BLOOD OF THE MOTHER AND THE PREGNANCY. MANAGEMENT TACTICS. TERMS OF DELIVERY. INDICATIONS FOR OPERATIVE DELIVERY

1. Management during pregnancy.
2. Management during labor.
3. Terms of delivery.
4. Indications for operative delivery.

Introduction

Description: Isoimmunization to any fetal blood group not possessed by the mother is possible. The most common example is the Rh (D) factor. What was once a common cause for intrauterine fetal death has been largely eradicated by prophylactic administration of immunoglobulins to those at a risk.

Prevalence: Uncommon in developed countries because of theroutine use of D immunoglobulin therapy.

Predominant Age: Reproductive age.

Genetics: Mothers who are Rh (D) negative. The genes for the CDE blood groups are separately inherited from the ABO groups and are located on the short arm of chromosome 1.

Etiology and Pathogenesis

Causes: Antibody formation against the D antigen.

Risk Factors: Any process that exposes the woman to blood carrying the D antigen including blood transfusion, miscarriage, ectopic or normal pregnancy, trauma, amniocentesis during pregnancy, and others.

Signs and symptoms

- Elevated maternal serum titers of anti-D immunoglobulin (IgM)

- Fetal hydrops, erythroblastosis fetalis, hemolytic disease of the newborn
- Intrauterine fetal demise

Diagnostic approach

Differential Diagnosis

- Other isoimmunizations (most frequently Lewis, Kell, or Duffy antigens).
- Iron-deficiency anemia (maternal).
- Hemoglobinopathy.

Associated Conditions: Polyhydramnios.

Workup and Evaluation

Laboratory: Serum antibody titers (at first visit, 20 weeks, and approximately every 4 weeks thereafter), testing of baby's father's antibody status. Emerging data suggest that the Rh status of the fetus can be directly determined from fetal cells circulating in maternal blood.

Imaging: Ultrasonography is useful to establish gestational age and monitor amniotic fluid volume and fetal growth. Some studies have assessed the ability of ultrasonography to monitor the degree of fetal anemia, but this technique has not gained wide usage.

Special Tests: Amniocentesis or umbilical cord blood sampling if titers are elevated or there has been a prior affected pregnancy.

Diagnostic Procedures: Serum titers, amniocentesis, or umbilical cord blood sampling.

Management and therapy

Nonpharmacologic

General Measures: Evaluation, increased surveillance.

Specific Measures: When antibody titers are <1:8, no intervention is required. When titers are >1:16 in albumin or 1:32 by an indirect Coombs test, amniocentesis or umbilical cord blood sampling should be considered. In severely affected fetuses, intrauterine transfusion may be required.

Diet: No specific dietary changes indicated.

Activity: No restriction.

Patient Education: American College of Obstetricians and Gynecologists Patient Education Pamphlet AP027 (The Rh Factor: How It Can Affect Your Pregnancy)

Drug(s) of Choice

None, if isoimmunization has occurred. Prophylaxis (with Rh-positive father): D immunoglobulin — 50 mcg for miscarriage before 13 weeks of gestation or after chorionic villus sampling; 300 mcg after amniocentesis or ectopic pregnancy; at 28-30 weeks of gestation in unsensitized patients or after normal delivery (20 mcg/1 mL of D-positive cells [2 mL of whole blood] infused or lost into the patient's circulation).

Contraindications: Patients who are already sensitized to the D antigen should not receive D immunoglobulin.

Follow-up

Patient Monitoring: Normal prenatal care with increased surveillance of fetal growth and health.

Prevention/Avoidance: All patients should have their Rh type established and be tested for isoimmunization (indirect Coombs test) at the first prenatal visit. Those who are Rh negative should receive D immunoglobulin after delivery, amniocentesis, fetal demise, miscarriage, ectopic pregnancy, or any other time exposure to Rh-positive cells may have occurred. Prophylactic administration between 28 and 30 weeks of gestation is also standard.

Possible Complications: Isoimmunization with subsequent immune damage to fetal red cells leading to lysis, anemia, hydrops, and fetal death.

Expected Outcome: With prophylaxis, the risk for isoimmunization is estimated to be 0.3%.

EXTRAGENITAL PATHOLOGY AND PREGNANCY (asymptomatic bacteriuria, pyelonephritis, anemia)

1. Features of the course of the disease during pregnancy.
2. Acceptable treatment.
3. Effect on the fetus.
4. Complications.
5. Method of delivery.

Renal disease in pregnancy

Asymptomatic bacteriuria

- *Incidence.* This is 4–7% of all pregnancies, which is similar to that in non-pregnant women.
- In pregnancy, asymptomatic bacteriuria is more likely to progress to pyelonephritis (20–30%).
- *Escherichia coli* is the most common causative organism.

Chronic renal failure

- *Complications.* Infertility (usually due to chronic anovulation), spontaneous abortion, pre-eclampsia, IUGR, fetal death, and preterm birth.
- Pregnancy outcome is dependent on baseline renal function (below), and presence and severity of hypertension. The degree of proteinuria does not correlate with pregnancy outcome.
- In women with end-stage renal disease, renal transplantation offers the best chance of a successful pregnancy (especially if renal function is stable for 1–2 years and there is no hypertension). Triple-agent immunosuppression (cyclosporin, azathioprine, prednisone) should be continued in pregnancy.

Anaemia

Anaemia can follow:

1 Lack of production of blood: haemopoietic.

2 Increased breakdown of blood: haemolytic.

3 Blood loss: haemorrhagic.

In pregnancy, most anaemia is haemopoietic when it may be due to lack of:

1 Iron: iron deficiency anaemia.

2 Folic acid: megaloblastic anaemia.

3 Protein: iron deficiency anaemia.

Iron deficiency anaemia

Aetiology

POOR INTAKE

- Diet deficient in iron-containing foods.

POOR ABSORPTION

- Vomiting in pregnancy affects absorption.
- Increased pH of gastric juice.
- Ferric ions in gut instead of ferrous.
- Lack of vitamin C.

INCREASED UTILIZATION

Demands of pregnancy. Total body iron measures about 3500mg. Includes:

- Fetus and uterus: 500mg.
- Increased maternal blood volume: 500mg.

More if:

- Multiple pregnancies.
- Grand multiparity.
- Pregnancies close together.
- Vegetarian (particularly vegans).

DIAGNOSIS

- Rarely made clinically unless woman is severely affected.
- May show pallor of conjunctivae.

- May have tiredness and oedema.
- Hb estimates must be done on all pregnant women at booking, and twice later in pregnancy, at 26 and 34 weeks.
- If level below 10 g/dl, diagnose anaemia, look for cause and treat.

Treatment

PREVENTATIVE

Regular iron-bearing foods in diet (Box 11.2). If needed, iron tablet supplements. Daily requirements are 100mg elemental iron with 300mg folic acid.

- See she gets them—give them to her at the clinics.
- See she takes them—ask at each visit.
- See they are effective—check Hb levels.

CURATIVE

Depends on:

1 Degree of anaemia.

2 Duration of pregnancy.

3 Cause of iron deficiency:

- Mild anaemia: Hb below 10 g/dl.
- Severe anaemia: Hb below 8 g/dl.

Mild:

1 Check that the woman is being given, and is taking, oral iron.

2 If so, increase oral iron; add vitamin C to aid absorption or try another preparation.

3 If she cannot swallow tablets, use liquid preparation.

4 If change of oral therapy does not improve, use intramuscular (i.m.) or i.v. preparation. Give total dose i.v. as a transfusion in 1000 ml of saline. Alternatively, iron dextran 250 mg is associated with a rise of Hb of about 1 g/dl. Give on alternate days, i.m. for six doses, with small test dose first to check anaphylaxis.

Severe and early:

1 Admit to hospital and check that anaemia is solely iron deficient:
- Blood film.
- Serum iron.
- Total iron-binding capacity.
- Serum ferritin.
- Serum folate.
- Sickle/thalassaemia status.

2 Treat with oral, i.m. or i.v. therapy as required.

3 Check protein and vitamin intake adequate.

4 Check that improvement is maintained for rest of pregnancy.

Severe and late:

1 If after 36 weeks, too late to rely on haemopoiesis to provide red cells in time to cover labour. The refore transfuse slowly with packed red cell blood.

2 If Hb below 4g/dl consider exchange transfusion.

3 Build up iron stores for puerperium by i.m. therapy.

Folic acid deficiency anaemia

Aetiology

Folic acid required for building DNA in all tisses. Hence demands are maximal when fetal tissue being made.

1 Poor intake:
- Diet deficient in folates.
- Vomiting in pregnancy.

2 Increased utilization:
- Demands of pregnancy.
- Rapid growth of fetal, placental and uterine tissues.
- Worse if:

(a) Multiple pregnancy.

(b) Grand multiparity.

(c) Fetal haemolysis (in Rh effect).

(d) Infection.

Commoner in underdeveloped countries and combined with other forms of malnutrition.

Diagnosis

Sometimes made clinically.

- May be tired, breathless, oedematous.
- May have other signs of malnutrition.

Treatment

PREVENTATIVE

Folic acid supplements in last 20 weeks of pregnancy (300mg/day).

Theoretical risk of masking pernicious anaemia (PA) and its uncommon accompanying subacute combined degeneration (SCD) of the spinal cord. In practice, PA is very rare in those under 35 years and SCD almost unknown in the pregnancy age group.

CURATIVE

- Mild or moderate anaemia – folic acid 5–10mg/day orally only.
- Severe anaemia – folic acid 5–10 mg/day i.m.:

(a) oral iron both should be

(b) blood transfusion given with care.

EXTRAGENITAL PATHOLOGY AND PREGNANCY (viral hepatitis A, B, C, pulmonary tuberculosis)

1. Features of the course of the disease during pregnancy.
2. Acceptable treatment.
3. Effect on the fetus.
4. Complications.
5. Method of delivery.
6. Bacterial infection

Group B streptococcus

- *Incidence.* In developed countries, neonatal group B streptococcus (GBS) sepsis complicates 1.8/1,000 live births.
- *Maternal signs/symptoms.* Of all pregnant women 20% are asymptomatically colonized in the vaginal or perianal region.
- *Fetal/neonatal effects.* Two clinically distinct neonatal GBS infections have been identified:

1 *Early onset*, neonatal GBS infection (80%) results from transmission during labor or delivery. Signs of serious infection (respiratory distress, septic shock) usually develop within 6–12 hours of birth. The mortality rate is 25% and surviving infants frequently exhibit neurologic sequelae.

2 *Late-onset* GBS infection (20%) is a nosocomial or community-acquired infection. It presents more than a week after birth, usually as meningitis. The mortality rate is lower than for early onset disease, but neurologic sequelae are equally common.

- *Prevention.* Strategies to prevent early onset neonatal GBS infection vary. In the UK, a risk factor-based protocol is used. Patients are treated in labor if one of the following risk factors is present: a prior affected infant (not GBS positive in a prior pregnancy), GBS urinary tract infection (UTI) in index pregnancy, preterm labor, fever, or rupture of membranes ≥18 hours.

This protocol results in the treatment of 15–20% of pregnant women and prevents 65–70% of early onset GBS sepsis. US practice favors a universal screening protocol. All women are screened for GBS carrier status at 35–37 weeks. Women who are GBS carriers receive intrapartum antibiotics. The latter protocol results in the treatment of 25–30% of pregnant women and prevents 85–90% of cases of early onset GBS sepsis. Patients with unknown GBS carrier status in labor should be treated according to the risk factor-based protocol.

- *Treatment.* Intrapartum penicillin (second-generation cephalosporin, erythromycin, or clindamycin if penicillin allergy and sensitivities available; vancomycin if penicillin allergy and no sensitivities available).

Chorioamnionitis

- *Incidence.* One to ten percent of pregnancies.
- *Maternal signs/symptoms.* Chorioamnionitis is a clinical diagnosis. Definitive diagnosis requires a positive amniotic fluid culture. Maternal complications may include sepsis, adult respiratory distress syndrome (ARDS), pulmonary edema, and death.
- *Fetal/neonatal effects.* Neonatal sepsis, pneumonia, death.
- *Prevention.* Avoidance of rupture of membranes >18 hours.
- *Treatment.* Prompt administration of broad-spectrum antibiotics and expedite delivery. Chorioamnionitis is not an indication for cesarean section delivery; however, the cesarean section rate is increased due to dysfunctional labor dystocia and non-reassuring fetal testing.

Listeriosis

- Listeriosis is an uncommon cause of neonatal sepsis that may beacquired transplacentally. Cervical and blood cultures should be obtained in women with suspicious symptoms. Listeriosis is a common cause of intrauterine fetal demise and neonatal mortality rate is high.
- *Treatment.* Ampicillin and gentamicin.

Tuberculosis

- *Incidence.* Tuberculosis (TB) in pregnant women is rare in developed countries. Cases occur most commonly among recent immigrants.
- *Maternal signs/symptoms.* Most infected women are asymptomatic. Active disease at presentation is rare.
- *Fetal/neonatal effects.* Congenital or neonatal TB is a highly morbid condition that may be fatal if misdiagnosed.
- *Prevention.* Intradermal placement of purified protein derivative (PPD) is an accurate way to screen for TB. Interpretation of the PPD test depends on the risk status of the patient
- *Treatment.* A positive PPD necessitates a chest radiograph. If the radiograph is normal, 6 months of isoniazid (INH) is recommended in women aged <35 years (can be deferred until after delivery). If the chest radiograph is abnormal, immediate treatment with INH and ethambutol is indicated, and three early morning sputum cultures should be sent to exclude active pulmonary TB.

Bacterial vaginosis

Bacterial vaginosis (BV) is the most common cause of vaginal discharge in pregnancy. It is associated with preterm delivery in high-risk women. However, it remains unclear whether treatment for asymptomatic BV will reduce the risk of preterm delivery.

Chlamydia infection and gonorrhea

- *Incidence.* Very prevalent sexually transmitted infections.
- *Maternal signs/symptoms.* Usually asymptomatic.
- *Fetal/neonatal effects.* Untreated maternal chlamydia and gonorrhea infections are associated with increased neonatal morbidity.
- *Prevention.* Cervical cultures in early pregnancy in high-risk women reliably detect infection. Instillation of prophylactic antibiotic ointment into the eyes of all newborns prevents eye infection.

- *Treatment. Chlamydia*: oral erythromycin or azithromycin; gonorrhea: intramuscular ceftriaxone, oral cefuroxime, or ceftriaxone.

Protozoan infections

Toxoplasmosis

- *Incidence.* Acute toxoplasmosis during pregnancy is rare.
- *Maternal signs/symptoms.* Most patients are asymptomatic, but some have flu-like symptoms.
- *Fetal/neonatal effects.* Only acute toxoplasmosis in pregnancy is capable of being transmitted to the fetus; 10% of infected newborns will have clinical evidence of disease.
- *Prevention.* Toxoplasmosis is acquired through ingestion of encysted organisms in raw or undercooked meat or through contact with infected cat feces. Avoid cleaning litter box. Strict hygiene.
- *Treatment.* Sulfadiazine with pyrimethamine.

Trichomoniasis

- *Vaginal trichomoniasis* is very common.
- *Treatment*. Metronidazole.

Viral infections

Rubella

- *Incidence.* Rare in developed countries.
- *Transmission*. Airborne.
- *Maternal signs/symptoms.* Rubella ("German measles") is usually a mild viral illness.
- *Diagnosis.* Serologic diagnosis requires either the presence of IgM or a significant rise in IgG antibody titer (fourfold rise over 4–6 weeks).
- *Fetal/neonatal effects.* The risk of congenital rubella syndrome is 90% if maternal infection is acquired <11 weeks, 33% if 11–12 weeks, 11% if 13–14 weeks, 4% if 15–16 weeks, 0% if >16 weeks.

- *Prevention.* Measles/mumps/rubella (MMR) immunization. MMR is a live vaccine and is not recommended in pregnancy.
- *Management.* There is no treatment.

Cytomegalovirus (CMV)

- *Incidence.* One to two percent of all births.
- *Transmission.* Contact with body fluids, sexual contact.
- *Maternal signs/symptoms.* Of women 20% have a non-specific viral syndrome (fever, pharyngitis, lymphadenopathy).
- *Diagnosis.* The high prevalence of CMV seroreactivity (>50%) and multiple CMV serotypes limits the value of serologic screening.
- *Fetal/neonatal effects.* Of infected newborns 90% are asymptomaticat birth, but many later demonstrate deafness, learning disability, and/or delayed psychomotor development.
- *Prevention.* There is no vaccine.
- *Management.* There is no treatment.

Human immunodeficiency virus

- *Incidence.* Rare in developed countries, but very high prevalence in developing countries (eg, almost one in three pregnant women in South Africa is HIV positive).
- *Transmission.* Sexual contact, intravenous drug use, vertical transmission.
- *Maternal signs/symptoms.* Variable.
- *Diagnosis.* Serum enzyme-linked immunosorbent assay (ELISA) and confirmatory western blot.
- *Fetal/neonatal effects.* HIV-positive infants may develop AIDS with high perinatal mortality rate.
- *Prevention.* Safe sexual practices, avoidance of high-risk drug behavior, serial serum viral load measurements, and antiviral therapy, if indicated.
- *Management.* Prenatal HIV testing. Zidovudine (AZT) therapy reduces the risk of vertical transmission from 25–33% to 8%, with a further reduction to 0–2% if the viral load is <1,000 copies/mL. Elective cesarean delivery may reduce

vertical transmission if viral load is ≥1,000 copies/mL. Continue highly active antiretroviral therapy (HAART) if the patient is already on it or consider initiating such treatment in pregnancy.

Varicella-zoster virus

- *Incidence.* One in 7,500 pregnancies.
- *Transmission.* Airborne (highly infectious).
- *Maternal signs/symptoms.* "Chickenpox." The maternal mortality rate approaches 50% for adults with pneumonitis or encephalitis.
- *Diagnosis.* Clinical suspicion. Confirmatory serologic tests.
- *Fetal/neonatal effects.* First trimester varicella-zoster virus (VZV) has a 2–3% risk of congenital varicella syndrome. Near-term infections resemble benign childhood infection.
- *Prevention.* Only 5% of adults are not immune to VZV. Consider VZIG (varicella-zoster immune globulin) or acyclovir if a susceptible woman is exposed in pregnancy.
- *Management.* Delivery should be avoided at the time of acute maternal infection. At-risk neonates should receive VZIG. Acyclovir may also be helpful.

Herpes simplex virus

- *Incidence.* Neonatal herpes simplex virus (HSV) infection occurs in 2–4 per 10,000 births.
- *Transmission.* Direct contact.
- *Maternal signs/symptoms.* First-episode primary genital HSV may be associated with systemic symptoms. Both primary and recurrent HSV are characterized by painful, vesicular lesions.
- *Fetal/neonatal effects.* Neonatal herpes is acquired from passage through an infected birth canal. The risk of vertical transmission is 50% for primary HSV infection and 0–4% in women with recurrent disease.
- *Prevention.* Cesarean section delivery is recommended for all pregnancies complicated by primary genital HSV in labor. The management of women with a recurrent genital HSV outbreak

(either lesion and/or symptoms) in labor is less clear. In the UK, such women are allowed to deliver vaginally. US practice favors cesarean section delivery for such women.

- *Management.* Prophylactic acyclovir from 35 weeks to 36 weeks may be useful in preventing active lesions in labor in some high-risk women.

Hepatitis B and C

- *Incidence.* One to two percent of pregnancies.
- *Transmission.* Sexual contact, intravenous drug use, vertical transmission.
- *Maternal signs/symptoms.* Usually mild/moderate viral illness.
- *Diagnosis.* Serologic testing.
- *Fetal/neonatal effects.* Hepatitis B and C are not teratogenic, but affected infants may become carriers. Vertical transmission rates of hepatitis B range from 15% (in women who are e-antigen negative) to 80% (e-antigen positive), and of hepatitis C from 0–5% (HIVnegative women) to 35–50% (HIV-positive women).
- *Prevention.* Safe sexual practices, avoidance of high-risk drug behavior. Breastfeeding is controversial, but is best avoided. Hepatitis B has an effective vaccine.
- *Management.* Infants born to women with detectable hepatitis B surface antigen (HBsAg) should receive hepatitis B immunoglobulin (HBIg) and hepatitis B vaccine within 12 hours of birth. There is no effective treatment for hepatitis C.

Spirochete infections

Syphilis

- *Incidence.* Rare in developed countries.
- *Transmission.* Sexual contact.
- *Maternal signs/symptoms.* Patients may exhibit primary, secondary, or tertiary syphilis.
- *Diagnosis.* Serum rapid plasma reagin (RPR) or Venereal Disease Research Laboratory (VDRL) test. Confirmatory tests are required before instituting treatment.

- *Fetal/neonatal effects.* Affected infants may be stillborn or exhibit signs of early or late congenital syphilis.
- *Prevention.* Safe sexual practices. Congenital syphilis is unusual if the mother is treated.
- *Management.* Penicillin.

EXTRAGENITAL PATHOLOGY AND PREGNANCY (heart defects, diabetes mellitus, hypothyroidism, hyperthyroidism)

1. Features of the course of the disease during pregnancy.
2. Acceptable treatment.
3. Effect on the fetus.
4. Complications.
5. Method of delivery.

Maternal heart disease in pregnancy

Incidence

One percent of pregnancies.

Etiology

- Congenital lesions account for >50% of heart disease in pregnancy.
- Other common causes include coronary artery disease, hypertension, and thyroid dysfunction. Rare causes include myocarditis, cor pulmonale, cardiomyopathy, constrictive pericarditis, and cardiac dysrhythmias. Historically, rheumatic fever accounted for 90% of heart disease in pregnancy, but is now rare.

Prognosis

Prognosis depends on four factors:

1 *Cardiac function*. A clinical classification was developed by the New York Heart Association (NYHA) in 1928 (*see table below*):

2 *Clinical conditions* that may further increase cardiac output (multiple gestation, anemia, thyroid disease).

3 *Medications.*

4 The specific nature of the *cardiac lesion*.

Management

- Allow spontaneous labor at term. Scheduled induction is indicated for women requiring invasive cardiac monitoring.
- Adequate pain relief (regional analgesia is preferred).
- Left lateral positioning with supplemental oxygen.
- Maternal pulse oximetry and ECG monitoring.
- Fluid intake and output monitoring.
- Consider invasive hemodynamic monitoring for women with NYHA class III and IV disease.
- Consider elective shortening of the second stage of labor.

Diabetes mellitus in pregnancy

Gestational diabetes

Physiology

Pregnancy is a "diabetogenic state" with increased insulin resistance and reduced peripheral uptake of glucose (due to placental hormones with anti-insulin activity). In this way, the fetus has a continuous supply of glucose.

Incidence

Three to five percent of pregnancies.

Maternal complications

- Gestational diabetes poses little risk to the mother. Such women are not at risk of diabetic ketoacidosis (DKA), which is a disease resulting from an absolute deficiency of insulin.
- Care should be taken to avoid iatrogenic hypoglycemia due to excessive insulin administration.
- Gestational diabetes is a good screening test for insulin resistance; 50% will develop gestational diabetes in a subsequent pregnancy, and 40–60% will develop diabetes later in life.

Fetal complications

Fetuses of women with poorly controlled gestational diabetes are exposed to high concentrations of glucose and, as a result, grow large. Fetal macrosomia is associated with an increased risk of cesarean section delivery and birth injury.

Screening

- *Glucose load test* (GLT) is used to screen for gestational diabetes. In the UK, screening is recommended only for high-risk women at approximately 28 weeks. Risk factors include women with a family history of diabetes, sustained glycosuria, obesity, or a history of gestational diabetes, fetus macrosomia, or unexplained fetal demise. US practice favors screening all pregnant women at 24–28 weeks, and high-risk women in the first trimester at 16–20 weeks and again at 24–28 weeks.
- GLT is a non-fasting test, but women should not eat after their 50-g glucose load until a venous blood sample is drawn 1 hour later. A positive test should be followed by a *glucose tolerance test* (GTT). A GLT cut-off of ≥7.8 mmol/L (≥140 mg/dL) will detect 80% of women with gestational diabetes with a false-positive rate of 14–18%; a cut-off of ≥7.2 mmol/L (≥130 mg/dL) will detect 90% with a falsepositive rate of 20–25%.
- A definitive diagnosis of gestational diabetes requires a GTT; there is no GLT cut-off that is diagnostic. In the UK, a 2-hour 75-g GTT is used. Fasting glucose >5.5 mmol/L (>100 mg/dL) and at 2 hours >7.9 mmol/L (>140 mg/dL) will confirm the diagnosis. In the USA, a GTT involves a 100-g glucose load administered after an overnight fast. Venous plasma glucose is measured fasting and at 1, 2, and 3 hours. Gestational diabetes requires two or more abnormal values, defined by the NDDG as ≥105 (≥5.8), ≥190 (≥10.5), ≥165 (≥9.1), and ≥145 mg/dL (≥8.0 mmol/L), respectively. Other cutoffs exist (such as the Carpenter and Coustan criteria).

Antepartum management

- The primary aim is to prevent fetal macrosomia and its complications by maintaining blood glucose at desirable levels (defined as fasting, <95 mg/dL [<5.2 mmol/L] and 1 hour postprandial, <l40 mg/ dL [<7.8 mmol/L]).
- A diabetic diet is recommended for all such women.
- Treatment may be required. If fasting glucose levels are >95 mg/dL (>5.2 mmol/L), therapy can be initiated right away because "you cannot diet more than fasting." Although insulin remains the "gold standard" for the treatment of gestational diabetes in pregnancy, the use of oral hypoglycemic agents is becoming more common.

Intrapartum management

- Cesarean section delivery may be appropriate if the estimated fetal weight is excessive because of the risk of birth injury
- As the primary source of anti-insulin hormones is the placenta, no further management is required in the immediate postpartum period.
- All women with gestational diabetes should have a standard (nonpregnant) 75-g GTT 6–8 weeks postpartum, because such women are at increased risk of developing diabetes in later life.

Pregestational diabetes

Pathophysiology

Results from either an absolute deficiency of insulin (type 1 or insulindependent diabetes mellitus) or increased peripheral resistance to insulin (type 2 or non-insulin-dependent diabetes mellitus).

Incidence

Less than 1% of women of childbearing age.

Classification

- The age of onset and duration of diabetes (White classification) does not correlate with pregnancy outcome.

- Poor prognostic features include DKA, poor compliance, hypertension, pyelonephritis, and vasculopathy.

Complications

In contrast with gestational diabetes, pregestational diabetes is associated with significant maternal and perinatal mortality and morbidity

Antepartum management

- Diabetic women should ideally be seen before conception. Pregnancy complications such as fetal congenital anomalies and spontaneous abortion correlate directly with the degree of diabetic control at conception.
- Intense antepartum management can reduce the perinatal mortality rate from 20% to 3–5%.
- Approximately 5% of maternal hemoglobin is glycated (bound to glucose), known as hemoglobin A1 (HbA1). HbA1c refers to the 80–85% of HbA1 that is irreversibly glycated. As red blood cells have a lifespan of 120 days, HbA1c measurements reflect the degree of glycemic control over the previous 3–4 months. HbA1c measurements should be checked before conception, at first prenatal visit, and every 4–6 weeks throughout pregnancy.

Intrapartum and postpartum management

- If metabolic control is good, spontaneous labor at term can be awaited. As a result of the risk of unexplained fetal demise, women with pregestational diabetes should be delivered by 39–40 weeks.
- If the estimated fetal weight is excessive (likely ≥4,500 g), elective cesarean section may be appropriate to avoid birth injury.
- Women may not eat during labor. As such, intravenous glucose should be administered (5% dextrose at 75–100 mL/h) and blood glucose levels checked every 1–2 hours. Regular insulin should be given by subcutaneous injection or intravenous infusion to maintain blood glucose levels at 100–120 mg/dL (5.5–6.6 mmol/L).

- During the first 48 hours postpartum, women may have a “honeymoon period” during which their insulin requirement is decreased. Blood glucose levels of 150–200 mg/dL (8.2–11.0 mmol/L) can be tolerated during this period. Once a woman is able to eat, she can be placed back on her usual regular insulin regimen.

THYROID DISEASE IN PREGNANCY

Thyroid physiology

- Circulating thyroxine (levothyroxine, T4) and l-triiodothyronine (T3) are bound primarily to thyroxine-binding globulin (TBG) with <1% circulating as free (biologically active) hormone.
- Iodine is required for thyroid hormone production and fetal thyroid function is dependent on iodine from the mother.
- Non-thyroid medical illnesses and select drugs can affect thyroid function.

Thyroid function during pregnancy

- Estrogen has two effects on thyroid function in pregnancy:

1 It increases circulating TBG concentrations resulting in elevated levels of total T4 and T3.

2 It increases TBG sialylation which reduces hepatic clearance of T4 and T3.

Despite these changes, circulating levels of free T4 and T3 remain unchanged.

- Less than 0.1% of thyroid hormone crosses the placenta. As such, tests of fetal thyroid function (although rarely, if ever, indicated) are reliable and independent of maternal thyroid status.
- Thyroid hormone can be measured in fetal blood as early as 12 weeks' gestation.

Maternal hyperthyroidism (thyrotoxicosis).

Incidence

This is 0.05–0.2% of pregnancies.

Diagnosis

A definitive diagnosis requires thyroid function testing .

Etiology

- *Graves disease* is the most common cause of maternal hyperthyroidism in pregnancy (95%). It results from the presence of circulating thyroid-stimulating antibodies. Eye signs (ophthalmopathy) are specific to Graves disease. As IgG antibodies cross the placenta, the fetus is at risk of thyroid dysfunction.
- *Toxic multinodular goiter* is characterized by hyperthyroidism and the presence of a large, palpable thyroid gland.
- *Hyperemesis gravidarum* is often associated with elevated human chorionic gonadotropin (hCG) levels; 50–70% of women will have biochemical studies suggestive of hyperthyroidism, but no symptoms or signs.
- Hyperthyroidism in the setting of *gestational trophoblastic neoplasia* is probably secondary to elevated levels of hCG.
- Metastatic *follicular cell carcinoma* of the thyroid (rare).
- *Exogenous* T3 or T3.
- *De Quervain thyroiditis* (rare) is acute and painful.

Complications

- *Maternal complications.* Infertility, recurrent pregnancy loss, cardiac failure (10–20%), thyroid storm (<0.1%).
- *Fetal complications.* Preterm delivery, intrauterine growth restriction (IUGR), increased perinatal mortality.

Management

- The goal during pregnancy is to control thyrotoxicosis while avoiding fetal and/or transient neonatal hypothyroidism.
- *Antithyroid drugs* are the treatment of choice during pregnancy.

Propylthiouracil (PTU) is preferred because it blocks the release of hormone from the thyroid gland and – unlike carbimazole – also blocks peripheral conversion of T4 to T3. Carbimazole has also been associated with a rare congenital abnormality (aplasia cutis congenita). PTU treatment is initiated at 100–150 mg three times daily, but it takes 3–4 weeks before a clinical response is seen.

Thyroid-stimulating hormone (TSH) levels should be checked every 4–6 weeks and treatment adjusted accordingly.

- Radioactive iodine to ablate the thyroid gland is absolutely contraindicated in pregnancy.
- Surgery is best avoided during pregnancy but, if indicated for failed medical therapy, is best performed in the second trimester.
- Regular fetal testing is recommended after 32 weeks to look for evidence of fetal thyroid dysfunction. Fetal tachycardia (>160 beats/min) is a sensitive index of fetal hyperthyroidism.

Maternal hypothyroidism

Incidence

This is 0.6% of all pregnancies.

Diagnosis

- Thyroid function testing is required for a definitive diagnosis.
- Subclinical maternal hypothyroidism during pregnancy may be associated with long-term cognitive deficits in the offspring. However, routine TSH screening of all pregnant women is not as yet recommended.

Etiology

- *Hashimoto thyroiditis* (chronic lymphocytic thyroiditis) is characterized by hypothyroidism, a firm goiter, and the presence of circulating anti-thyroglobulin or anti-microsomal antibodies. In women with existing Hashimoto disease, pregnancy may result in a transient improvement of symptoms.
- Women *previously treated* for hyperthyroidism may manifest with hypothyroidism and require thyroid hormone replacement.
- *Infectious (suppurative) thyroiditis* is characterized by fever and a painful, swollen thyroid gland.
- *Subacute thyroiditis* is similar to suppurative thyroiditis with a painful, swollen thyroid with or without fever. It is usually the result of a viral infection, and is self-limiting.

- *Iodine deficiency* (rare).

Management

- Early diagnosis is essential to avoid antepartum complications (placental abruption, IUGR, stillbirth) and impaired neonatal and childhood development (cretinism).
- Levothyroxine (thyroxine) treatment should be initiated at 100–150 μg daily. TSH levels should be measured every 4–6 weeks, and the dose adjusted accordingly.
- Women on thyroxine before conception should have their TSH levels monitored every 4–6 weeks. Most women will need to increase their dose by 30–50% during pregnancy.

Postpartum thyroiditis

- *Incidence.* Four to ten percent of all postpartum women.
- *Etiology.* Unknown, but may be an autoimmune phenomenon.
- *Clinical features.* characterized by a transient hyperthyroid state occurring 2–3 months postpartum (with dizziness, fatigue, weight loss, palpitations) or a transient hypothyroid state 4–8 months postpartum (with fatigue, weight gain, and depression).
- *Treatment.* Therapy may be indicated to control symptoms, and can usually be tapered within 1 year.

MISCARRIAGE. ABORTIONS. THREATENED AND INEVITABLE. MISSED AND RECCURENT

1. Clinical manifestations.
2. Diagnosis.
3. Management.
4. Complications.
5. Rehabilitation.

- *Definition*. Expulsion or removal of an embryo or fetus from the uterus before it is capable of independent survival.
- Of all conceptions 50–75% abort spontaneously. Most are unrecognized because they occur before or at the time of the next expected menses.

Spontaneous miscarriage

- *Definition*. Loss of a clinically recognized pregnancy before 20 weeks' gestation or <500 g.
- Make up 15–20% of clinically diagnosed pregnancies.
- Risk factors include advanced maternal age, increasing gravidity, prior miscarriage, and smoking.
- *History*. Vaginal bleeding is the most common presenting complaint, followed by crampy abdominal pain.
- *Examination*. Initially, vital signs should be taken to rule out hemodynamic instability. Speculum examination allows visualization of the cervix and potentially the location of products of conception. Bimanual examination may help estimate gestational age.
- *Laboratory tests*. Serum human choronic gonadotropin (hCG), blood count, and rhesus (Rh) typing should be sent (virtually all Rh-negative patients should receive anti-D immunoglobulin prophylaxis).

Etiology

- More than 80% of all miscarriages occur in the first trimester (<12 weeks' gestation). The exact mechanisms are not always apparent, but death of the embryo or fetus nearly always *precedes* spontaneous expulsion. As a result, finding the cause of early miscarriage involves ascertaining the cause of fetal death. At least half result from chromosomal abnormalities.
- Less than 20% of all miscarriages occur in the second trimester (12–20 weeks' gestation). The fetus frequently does not die before expulsion and anatomic factors are more likely to be causative.

Fetal factors

- *Chromosomal abnormalities.* Half of embryos and early fetuses that result in first-trimester miscarriages demonstrate aneuploidy. Autosomal trisomies (either chromosome 13, 16, 18, 21, or 22) are the most frequently identified anomalies. Monosomy X (45, X), the second most frequent chromosomal abnormality, usually results in miscarriage and much less frequently in live-born female infants (Turner syndrome), Triploidy is often associated with partial hydatidiform moles.
- *Abnormal development.* First-trimester miscarriages often display a developmental abnormality of the zygote, embryo, early fetus, or even placenta. A common example is having either a degenerated or absent embryo (ie, blighted ovum).

Maternal factors

The causes of miscarriages having normal (euploid) chromosomes are poorly understood, but a various medical disorders, environmental conditions, and developmental abnormalities have been implicated.

- *Uterine defects.* Even large and multiple uterine fibroids usually do not cause miscarriage. Asherman syndrome results from vigorous curettage that results in insufficient functional endometrium to support a pregnancy. It is controversial whether abnormal мьllerian duct formation or fusion defects cause miscarriage or whether surgical correction prevents it. Such

corrective procedures should be done as a last resort, if at all. Incompetent cervix is a potential fixable cause of miscarriage.

- *Infections, endocrine factors, environmental causes, and immunologic factors.*
- *Physical trauma*. Clearly, major abdominal trauma can precipitate miscarriage, but it is a minor contribution.

Classification and treatment

Threatened miscarriage

- *Definition*. Uterine bleeding before 20 weeks with a closed cervical os and a confirmed viable intrauterine gestation.
- Vaginal spotting or heavier bleeding occurs in 20–25% of pregnant women and may persist for days or weeks. However, some bleeding near the time of expected menses may be physiologic.
- Half of these pregnancies will eventually miscarry, but the risk is substantially lower if fetal cardiac activity is demonstrated. Fetuses that do not miscarry remain at increased risk for preterm delivery, low birthweight, and perinatal death.
- *Treatment.* There are no effective therapies and patients are monitored expectantly unless the pregnancy is undesired or non-viable. Bed rest is often recommended, but does not alter the course.

Inevitable, incomplete, and complete miscarriage

- *Definition*. Imminent, partial, or complete expulsion of products of conception before 20 weeks' gestation through a dilated cervix.
- When the placenta, in whole or in part, detaches from the uterus, bleeding ensues. Typically bleeding is profuse and associated with uterine cramping. During incomplete miscarriage, the internal os remains open and allows passage of blood. Following complete detachment and expulsion of the gestational products, the internal cervical os closes.

- Incomplete miscarriages usually occur between 6 and 14 weeks gestation; complete miscarriages are most common <6 or >14 weeks.
- *Treatment.* Suction curettage is typically required for inevitable or incomplete miscarriage; complete miscarriages may be managed expectantly without evacuation.

Septic abortion

- *Definition*. Any threatened, inevitable, or incomplete miscarriage associated with a fever before 20 weeks' gestation.
- Retained products of conception after spontaneous miscarriage, legal termination of pregnancy, or illegal termination of pregnancy acts as a nidus for the development of a local infection that is potentially fatal if generalized sepsis ensues.
- Patients usually present with fever, chills, leukocytosis, uterine tenderness, and a foul-smelling cervicovaginal discharge.
- *Treatment.* Broad-spectrum intravenous antibiotics, prompt uterine evacuation, and supportive therapy in severe cases. However, a large, grossly infected uterus is at risk for perforation and may be allowed to evacuate with pitocin or prostaglandin stimulation.

Missed abortion

- *Definition*. Fetal demise before 20 weeks' gestation without detachment of the placenta – resulting in retention of products of conception for days or weeks behind a closed cervical os.
- Typically, after the demise there is an episode of bleeding that subsides and is followed by a continuous brown vaginal discharge. Many women have no other symptoms. Very rarely, prolonged retention of a dead fetus after midpregnancy can result in coagulopathy.
- *Treatment.* Management is individualized, depending on the circumstances. Expectant management is often difficult for the patient to endure after being informed of the demise. Medical management is often the safest option.

METHODS OF TERMINATION OF PREGNANCY

1. Consultation of the patient.
2. Examinations before the procedure.
3. Classification of methods of termination of pregnancy.
4. Medical methods of termination of pregnancy.
5. Surgical methods of termination of pregnancy.
6. Complications.
7. Rehabilitation.

- *Definition*. Elective or voluntary "abortion" is the interruption of pregnancy before viability at the request of the woman.
- About 1.2 million terminations are performed annually in the USA. Half the women are younger than 25 years. About 88% are conducted up to 12 weeks, 10% from 13 weeks to 20 weeks, and 1–2% beyond 20 weeks.
- Worldwide, 44 million pregnancy terminations occur annually. Abortion rates (per 1,000 women aged 15–44) vary from 12 in western Europe to 32 in Latin America.
- Half the abortions worldwide are considered unsafe. Unsafe abortion accounts for an estimated 13% of all maternal deaths.

History

- Abortion has a long history and has been induced by various methods over the centuries, including herbal abortifacients, physical trauma, and insertion of non-surgical instruments (knitting needles, clothes hangers) into the uterus. These methods are rarely seen in developed countries where medical and surgical abortion is legal and available.
- Elective pregnancy termination in the UK has been legal since the Abortion Act was passed in 1967. Abortion was legalized in the USA with the Supreme Court's *Roe v. Wade* decision in 1973. Worldwide, abortion laws vary substantially by region. Some countries allow abortion by request (the USA, Canada,

the UK, France, China, Russia). Some countries allow abortion only to save a woman's life (Ireland, Indonesia, Egypt). A few countries ban abortions without exception (Chile, Dominican Republic, Nicaragua).

Personal and social factors

- Women choose to undergo pregnancy termination for a variety of reasons, including a desire to delay or end childbearing, concern over the interruption of work or education, issues of financial or relationship stability, and perceived immaturity.
- In the USA and the UK, 1% of all abortions occur because of rape or incest, 6% because of potential health problems regarding either the mother or child, and 93% for social reasons (the pregnancy was unplanned).
- Societal pressures may also influence the decision to undergo termination.

Examples include disapproval of single motherhood, insufficient economic support for families, lack of access to or rejection of contraceptive methods, or efforts toward population control (such as China's one-child policy).

Medical termination

- About 95% effective for pregnancies up to 9 weeks' gestation.
- Nearly 10% of all terminations in the USA and the UK.
- Becoming increasingly common due to safety and convenience.
- Two medications are given in sequence:

1 *Mifepristone (RU486)* is a progesterone receptor antagonist that blocks the hormone needed to maintain the pregnancy. Within hours, the uterine lining begins to shed, the cervix begins to soften, and bleeding may occur.

2 *Misoprostol* (Cytotec, USA) or *gemeprost* (UK) are both prostaglandin E1 analogs. Side effects include profuse vaginal bleeding, uterine cramps, nausea, vomiting, diarrhea, headache, muscle weakness, and dizziness.

- US regimen: (1) mifepristone 600 mg orally followed 24–72 hours later by misoprostol 400 μg orally (Food and Drug Administration [FDA] approved for up to 7 weeks' gestation) or (2) mifepristone 200 mg orally followed 6–72 hours later by misoprostol 800 μg vaginally or buccally (evidence-based regimen).
- UK regimen: mifepristone 600 mg orally followed 48 hours later by gemeprost 1 mg vaginally.
- More than half the women will abort within a few hours of taking the gemeprost or misoprostol dose.
- In cases of failure of medical abortion, vacuum or manual aspiration is used to complete the abortion surgically.

Surgical techniques

- These are 99% effective.
- About 90% of all terminations in the USA and the UK.
- The cervix is first dilated, then the uterine cavity is mechanically evacuated.
- Surgical termination is a simple procedure and is safer than childbirth when performed before week 16.

1 *Manual vacuum aspiration (MVA)* can be performed up to 6–7 weeks' gestation without regional anesthesia. A soft, flexible, plastic cannula is attached to a handheld self-locking syringe. Evacuation is accomplished by repetitive in-and-out, rotating movements.

2 *Electric vacuum aspiration (EVA)* uses an electric pump, requires a paracervical block and cervical dilation, and is used for terminations between 6 and 15 weeks gestation.

3 *Dilation and evacuation (D&E)* is performed between 15 and 20 weeks gestation. Due to the larger fetal size, the procedure is often done in the operating room under general anesthesia. Cervical preparation and dilation precedes mechanical destruction and evacuation of fetal parts using specialized (Sopher) forceps. The placenta and remaining tissue are then removed by suction curettage.

- Regardless of technique, identification of products of conception is mandatory before the procedure is considered complete.

Late pregnancy termination (20–24 weeks)

1 *Dilation and extraction (D&X)* is usually performed after intracardiac fetal injection (KCl or digoxin) to ensure cessation of fetal cardiac activity before the procedure. The technique is similar to a D&E. Cervical preparation is required and facilitates extraction of fetal parts while minimizing uterine or cervical injury. On a living fetus, this procedure has been termed "partial birth abortion" and is banned in the USA.

2 *Induction of labor* with systemic agents such as misoprostol, mifepristone, or high-dose oxytocin is used to induce contractions and promote expulsion of the fetus and placenta. The primary advantage compared with D&X is that the fetus delivers intact.

3 *Intra-amniotic infusion* of hypertonic saline and/or prostaglandin is used to induce contractions and promote expulsion of the fetus and placenta. This method is seldom used due to the availability and safety of the above methods.

Complications

- The frequency of complications depends on operator experience and gestational age (increased if <6 weeks or >16 weeks).
- *Immediate complications.* Hemorrhage, cervical injury, anesthesia complications. The use of osmotic dilators (laminaria) significantly reduces the risk of uterine perforation.
- *Late complications.* Retained products of conception, infection (endometritis), and Rh sensitization.
- *Mortality rate.* Fewer than 1/100,000 surgical procedures when a patient is under the care of an experienced clinician.

PRETERM LABOR

1. Causes.
2. Clinical manifestations.
3. Diagnosis.
4. Management of premature labor.
5. Signs of a post-term newborn.
6. Newborn care.

Premature labor

Etiology

- Preterm labor represents a syndrome rather than a diagnosis because the etiologies are varied.
- Of all preterm births, 20% are iatrogenic (performed for maternal or fetal indications), 30% are associated with intra-amniotic infection/ inflammation, 20–25% are associated with preterm premature rupture of membranes (PPROM), and 20–25% result from spontaneous (idiopathic) preterm labor.

Prediction of preterm birth

- *Risk factors* for preterm birth have been identified However, reliance on historic/demographic risk factors alone will fail to identify >50% of pregnancies that deliver preterm.
- Although an increase in uterine activity is a prerequisite for preterm labor, *home uterine monitoring* has not been shown to decrease the incidence of preterm birth.
- Serial *cervical evaluation* is reassuring if the examination remains normal. However, an abnormal finding (dilation or effacement) is associated with preterm delivery in only 4% of low-risk and 20% of high-risk women.
- There is an strong inverse correlation between sonographic *cervical length* (CL) and preterm delivery in both high- and low-risk pregnancies. CL is not currently recommended in low-risk patients. In highrisk pregnancies, baseline CL is recommended at 16–20 weeks followed by serial CL every 2–4 weeks until 30–32 weeks.

- A number of *biochemical/endocrine markers* have been associated with preterm delivery, but only cervicovaginal fetal fibronectin (fFN) has been established as a screening tool. The value of fFN lies in its negative predictive value: 99% of women with a negative fFN at 22–24 weeks will still be pregnant in 1 week, 98% in 2 weeks, and 89% in 3 weeks. However, the positive predictive value is poor (only 25% of women with a positive fFN will deliver before 35 weeks).
- *Vaginal infections* (bacterial vaginosis, *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Trichomonas vaginalis*) have been associated with preterm birth. However, routine screening and treatment of highrisk asymptomatic women does not appear to decrease this risk and is not recommended.
- *Intra-amniotic infection* is responsible for 30% of preterm labor. A positive amniotic fluid culture is necessary for a definitive diagnosis, but biomarkers of infection (high interleukin-6, low glucose, increased C-reactive protein, and high white cell count in amniotic fluid) may suggest the diagnosis.
- A number of *endocrine assays* are also being developed to predict preterm labor. Elevated maternal salivary estriol (≥2.1 ng/mL) is predictive of preterm delivery in high-risk populations. Other endocrine assays (relaxin, corticotropin-releasing hormone) are being developed.

Management

- A firm *diagnosis* of preterm labor is necessary before treatment is considered. Diagnosis requires the presence of both uterine contractions and cervical change (or an initial cervical examination ≥2 cm and/or ≥80% effacement in a nulliparous patient).
- A *cause* for preterm labor should always be sought.
- *Absolute contraindications* to tocolytic agents (drugs that inhibit uterine contractions) include intrauterine infection, "fetal distress" (non-reassuring fetal testing), vaginal bleeding, and intrauterine fetal demise. PPROM is a relative contraindication.

Bed rest and hydration are commonly recommended, but without proven efficacy.

- Short-term *pharmacologic therapy* (Figure 57.1) remains the cornerstone of management. However, there are no reliable data to suggest that any tocolytic agent is able to delay delivery for longer than 24–48 hours. No single agent has a clear therapeutic advantage.

As such, the side-effect profile of each of the drugs will often determine which to use in a given clinical setting:

1 Calcium channel blockers (such as nifedipine) are effective, have few side effects, and are rapidly becoming the first-line tocolytic agent of choice.

2 β-Adrenergic agonists are also commonly used, but have a higher incidence of maternal adverse effects.

3 Atosiban (oxytocin receptor antagonist) is commonly used and nitroglycerin is used in some institutions in the UK.

4 Magnesium sulfate (which acts as a physiologic calcium antagonist and a general inhibitor of neurotransmission) has a wide margin of safety and is still commonly used in the USA. It has the added benefit of neuroprotection in very-low-birthweight infants (<1500 g).

5 Indometacin (a non-steroidal anti-inflammatory drug) is an effective tocolytic agent, but is associated with a number of serious neonatal complications (such as premature closure of the ductus arteriosus, persistent pulmonary hypertension, oligohydramnios). As such, it is rarely used.

- Maintenance tocolysis beyond 48 hours has not consistently been shown to delay delivery and is associated with significant adverse effects. It is therefore not generally recommended. However, recent meta-analyses suggest that maintenance tocolysis with nifedipine may be beneficial.
- The concurrent use of two or more tocolytic agents has not been shown to be more effective than a single agent alone, and

the additive risk of side effects generally precludes this course of management.

- Recent data suggest that progesterone supplementation (not treatment) from 16–20 weeks through 34–36 weeks may prevent preterm delivery in some women at high risk by virtue of a prior unexplained preterm birth or short cervix (but not multiple pregnancy). Studies are under way to better define which women will benefit, and which formulation, dose, and route of administration to use.

Cervical insufficiency (also known as cervical incompetence)

Definition

Refers to an inability to support a pregnancy to term due to a *functional* defect of the cervix.

Incidence

This is 0.05–1% of all pregnancies.

Clinical features

- Cervical insufficiency is characterized by acute, painless dilation of the cervix, usually in the mid-trimester, culminating in prolapse and/ or preterm premature rupture of the membranes (PPROM) with resultant preterm and often pre-viable delivery.
- Symptoms may include watery vaginal discharge, pelvic pressure, vaginal bleeding, and/or PPROM in the mid-trimester, but most women are asymptomatic.

Diagnosis

- Cervical insufficiency is a clinical diagnosis. It should be suspected when an advanced cervical dilation examination is noted at 16–24 weeks' gestation on pelvic (or sonographic) examination in the absence of uterine contractions. If uterine

contractions are present, the diagnosis is more likely to be preterm labor.
- Several tests have been described in an attempt to confirm the diagnosis in non-pregnant women, but are of little clinical value.

Etiology

Cervical insufficiency is likely to be the clinical end-point of many pathologic processes. In most cases, the precise etiology is unknown.

Future pregnancies

- The probability of cervical insufficiency recurring in a subsequent pregnancy is 15–30%.
- The chance of carrying a pregnancy to term with a history of two consecutive mid-trimester pregnancy losses is 60–70%.

Cervical cerclage

Indications

- *Elective (prophylactic)* cerclage should be distinguished from emergency (therapeutic) cerclage
- A prior history of cervical insufficiency is the only clear indication for prophylactic cerclage.
- Prophylactic cerclage in women with a history of *in utero* diethylstilbestrol (DES) exposure or multiple pregnancy (in the absence of prior pregnancy loss) is controversial.

Contraindications

- *Absolute contraindications* are listed in Figure 58.1.
- *Relative contraindications* include:

1 fetal membranes prolapsing through the cervical os (because of the high incidence of PPROM)

2 elevated markers of inflammation in amniotic fluid (failure rate ≥90%)

3 placenta previa

4 intrauterine fetal growth restriction

5 ≥24 weeks' gestation (the limit of fetal viability).

Complications

- Complications increase with increasing gestational age and increasing cervical dilation.
- *Short-term (<48 hours) complications*: excessive blood loss, PPROM, spontaneous pregnancy loss (3–20%).
- *Long-term complications*: cervical lacerations (3–4%), chorioamnionitis (4%), cervical stenosis (1%), other (placental abruption, migration of the suture, bladder discomfort).
- *Puerperal infection* occurs in 5–6% of patients with cerclage, twice as common as in women with no cerclage.

Types of cerclage

Transvaginal cervical cerclage

Transvaginal cerclage remains the mainstay for the management of cervical insufficiency. Shirodkar and McDonald cerclage are probably equally efficacious:

1 Shirodkar cerclage is a single suture placed around the cervix at the level of the internal os after surgically reflecting the bladder anteriorly and the rectum posteriorly. The suture is secured either anteriorly or posteriorly.

2 McDonald cerclage is one or more purse-string sutures placed around the cervix without dissection of the bladder or rectum.

Transabdominal cerclage

Transabdominal cerclage has not been shown to be superior to transvaginal cerclage, and is a far more morbid procedure requiring a laparotomy and subsequent delivery by cesarean section. It should therefore be reserved for women in whom cerclage is indicated, but who have either failed previous transvaginal cerclages or in whom transvaginal cerclage is technically impossible to place.

Technical considerations

- An ultrasound examination should be performed before cerclage placement to exclude gross structural anomalies (such as anencephaly) and/or fetal demise.
- Confirmation of fetal viability both immediately before and after the procedure (by either auscultation or ultrasound).
- Regional anesthesia is preferred.
- Prophylactic tocolysis may be used to inhibit transient uterine contractions associated with placement, but there is no objective evidence that this improves outcome.
- Prophylactic antibiotics are recommended in emergency cerclage because of the risk of chorioamnionitis. The routine use of antibiotics for elective cerclage is, however, controversial.
- If the fetal membranes are prolapsing through the external os, the risk of iatrogenic rupture of the membranes may be as high as 40–50%.

Trendelenburg position, filling the bladder, and/or amnioreduction can be used to reduce the fetal membranes before cerclage placement.

Postoperative care

- Frequent (weekly or bi-weekly) visits for cervical checks.
- Bed rest and "pelvic rest" (no coitus, tampons, or douching) until a favorable gestational age is reached.
- Remove cerclage electively at 37–38 weeks or with the onset of premature uterine contractions (to avoid cervical lacerations or uterine rupture).

PREMATURE RUPTURE OF THE MEMBRANES

Definitions

- *Premature rupture of the membranes (PROM)* refers to rupture of the fetal membranes before the onset of labor.
- *Preterm PROM (PPROM)* refers to PROM at <37 weeks.
- *Prolonged PROM* refers to PROM >24 hours and is associated with an increased risk of intra-amniotic infection.

Diagnosis

- PROM is a clinical diagnosis
- If clinical examination is equivocal and the pregnancy is remote from term, US practice favors an amnio-dye test ("tampon test") in which indigo carmine dye (not methylene blue because of an association with fetal methemoglobinemia) is instilled into the amniotic cavity, and leakage into the vagina confirmed by staining of a tampon within 20–30 min.
- *Differential diagnosis.* Leakage of urine, vaginal discharge.

Latency

- Latency refers to the interval between PROM and delivery.
- Of women with PROM at term 50% will go into labor within 12 hours, 70% within 24 hours, 85% within 48 hours, and 95% within 72 hours.
- Latency is influenced by gestational age (50% of women with PPROM will go into labor within 24–48 hours and 70–90% within 7 days), severity of oligohydramnios (severe oligohydramnios is associated with shortened latency), and multiple pregnancy (twins have a shorter latency period than singletons).

Etiology

- Near term, a focal weakness develops in the fetal membranes over the internal cervical os which predisposes to rupture at this site.

- Several pathologic processes (including bleeding, infection) may predispose to PPROM.

Term premature rupture of the membranes

Incidence

This is 8–10% of term pregnancies.

Management

- In the absence of contraindications to expectant management (intraamniotic infection, "fetal distress"/non-reassuring fetal testing, vaginal bleeding, and active labor), both expectant management and immediate augmentation of labor are acceptable options.
- If the cervix is unfavorable, cervical ripening may be required.
- Severe oligohydramnios may be associated with umbilical cord compression in labor, leading to non-reassuring fetal testing and cesarean section delivery. It is not clear whether intrapartum amnioinfusion can improve fetal testing and decrease the cesarean section delivery rate.

Preterm premature rupture of the membranes

Incidence

- This is 2–4% of singleton and 7–10% of twin pregnancies.
- PPROM is associated with 20–25% of preterm births and 10% of all perinatal mortality.

Risk factors

- Risk factors include prior PPROM (recurrence risk 20–30%), unexplained vaginal bleeding, placental abruption (seen in 10–15% of women with PPROM, but may be a result rather than a cause), cervical insufficiency, vaginal or intra-amniotic infection, amniocentesis, smoking, multiple pregnancy, polyhydramnios, chronic steroid treatment, connective tissue diseases, anemia, low socioeconomic status, and single women.
- Factors not associated with PPROM include coitus, cervical examinations, maternal exercise, and parity.

Complications

- *Neonatal complications* are related primarily to prematurity, including respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), sepsis, pulmonary hypoplasia (especially with PPROM <22 weeks), and skeletal deformities (related to severity and duration of PPROM). Overall, PPROM is associated with a fourfold increase in perinatal mortality.
- *Maternal complications* include increased cesarean section delivery (due to malpresentation, cord prolapse), intra-amniotic infection (15– 30%), and postpartum endometritis.

Management

- Management of PPROM should be individualized and depends in large part on gestational age. The risk of prematurity should be weighed against the risk of expectant management, primarily intraamniotic infection.
- Areas of controversy in the management of PPROM:

1 *Antibiotics.* Empiric, prophylactic, broad-spectrum antibiotics prolong latency in women with PPROM <34 weeks and are therefore recommended for 7 days. There is currently no evidence to recommend one antibiotic regimen over another.

2 *Tocolysis.* PPROM is a relative contraindication to the use of tocolytic agents (drugs that inhibit uterine contractions).

3 *Steroids.* Antepartum glucocorticoid administration decreases the incidence of RDS by 50%. Maximal benefit is achieved 24–48 hours after the initial dose. This effect lasts for 7 days, but it is unclear what happens thereafter. Steroids also decrease the incidence of necrotizing enterocolitis (NEC) and IVH. Intramuscular dexamethasone can also be used, but not prednisone (as it does not cross the placenta) or oral dexamethasone (because of a 10-fold increase in neonatal infection and IVH). Of note, multiple (three or more) courses of steroids may be associated with intrauterine growth restriction (IUGR), smaller head circumference, and (possibly) an increased risk of cerebral palsy. As such, repeat courses of steroids are not routinely

recommended. However, a single repeat ("rescue") course before 34 weeks may provide additional benefit if the first course was given >2 weeks previously.

4 *Fetal surveillance.* After PPROM, fetuses are at risk for ascending infection, cord accident, placental abruption, and (possibly) uteroplacental insufficiency. It is generally accepted that some form of fetal monitoring is necessary, but the type and frequency of monitoring are controversial. Options include non-stress testing and/or biophysical profile, but none has been shown to be superior to fetal kickcharts.

POSTTERM PREGNANCY

1. Causes.
2. Clinical manifestations.
3. Diagnosis.
4. Management.
5. Signs of an immature newborn.
6. Newborn care.

Induction and augmentation of labor

Induction of labor

Definition

- *Induction* refers to interventions designed to initiate labor before spontaneous onset with a view to achieving vaginal delivery.
- This should be distinguished from *augmentation* which refers to enhancement of uterine contractility in women in whom labor has already begun.

Patient assessment

- The appropriate timing for induction is the point at which benefit to mother and/or fetus is greater if pregnancy is interrupted than if pregnancy is continued, and depends on gestational age.
- Indications and contraindications

Bishop score

- The success of induction depends in large part on the status of the cervix. In 1964, Bishop designed a cervical scoring system to prevent iatrogenic prematurity. This system has since been modified and used to predict the success rate of induction. If the Bishop score is favorable (defined as ≥6), the likelihood of a successful induction and vaginal delivery is high. If unfavorable (<6), the probability of successful induction is

reduced and pre-induction cervical "ripening" (maturation) may be indicated.

- Cervical ripening describes a complex series of biochemical events that alter cervical collagen and ground substance composition, resulting in a softer and more pliable cervix. A number of agents are available to facilitate this maturation. Potential benefits include fewer failed inductions, shorter hospital stay, lower fetal and maternal morbidity, lower medical costs, and possibly lower cesarean section delivery rates.

Methods

The choice of induction regimen should be individualized. A single technique is not always rarely effective, on its own, and a combination of interventions may be required:

- *Prostaglandin E2* (PGE2) improves the rate of vaginal delivery, regardless of route of administration. Gastrointestinal side effects are lower with vaginal administration. The rate of failed induction is only 1–6%. The most commonly used local PGE2 preparation is dinoprostone gel (Prepidil). PGE1 analogs, such as misoprostol (Cytotec), are cheaper, can be administered orally with few side effects, and are as effective as PGE2 for cervical ripening and labor induction. PGE2 should be avoided in women with asthma, glaucoma, and severe renal, pulmonary, or hepatic disease. Prostaglandin induction of labor should be avoided in patients with a prior cesarean section delivery, because of a fourfold increased risk of uterine rupture.
- *Oxytocin* infusion by any protocol (low dose or high dose, continuous or pulsatile) has been shown to be effective in pre-induction cervical ripening and labor induction. Continuous low-dose infusion is as effective as other protocols, while minimizing oxytocin requirements and adverse effects (especially maternal water intoxication due to an antidiuretic hormone-like effect). Advantages of oxytocin include cost and familiarity for the clinician. Fetal monitoring is required because of the risk of uterine tachysystole and "fetal distress."

- *Progesterone receptor antagonists* (RU 486 [mifepristone], ZK98299 [onapristone]) have been shown to promote cervical ripening and lower oxytocin requirements in labor.
- A*mniotomy* (artificial rupture of the membranes – AROM) may be sufficient on its own to induce labor, but is more effective if used in combination with oxytocin. It shortens the interval from induction to delivery by 1–3 hours, but does not appear to lower the rate of cesarean section delivery. Contraindications to amniotomy include HIV, active perineal herpes infection, and viral hepatitis.
- S*weeping (stripping) of the membranes* refers to digital separation of the fetal membranes from the lower uterine segment before labor at term. It may accelerate the onset of labor by releasing endogenous prostaglandins. However, most studies show no significant increase in the proportion of women going into labor within 7 days.
- *Mechanical dilators* (transcervical Foley catheter placement, hygroscopic dilators) significantly shorten the induction to delivery interval compared with no pre-induction ripening, and are as effective as PGE2. Hygroscopic dilators rely on absorption of water to swell and forcibly dilate the cervix. A disadvantage of mechanical dilators is patient discomfort both at the time of insertion and with progressive cervical dilation.

Augmentation of labor

Indications

Augmentation of uterine activity is indicated for failure to progress in labor in the presence of inadequate contractions and in the absence of absolute cephalopelvic disproportion

Methods

These include amniotomy and/or oxytocin. It is still unclear whether such interventions improve obstetric outcome or merely produce the same outcome in a shorter period of time.

Active management of labor

- "Active management" describes a protocol of clinical management based on the premise that enhancing uterine contractility in the first stage of labor will improve obstetric outcome. It applies only to nullipara in spontaneous labor with a cephalic presentation.
- Active management protocols rely on strict criteria for the diagnosis of labor, amniotomy within 1 hour of labor onset, and high-dose oxytocin if cervical dilation is not maintained at ≥1.0 cm/h. Other components include antenatal education, one-on-one nursing care, and close supervision by a senior obstetrician.
- The National Maternity Unit in Dublin, Ireland, pioneered active management in 1968. Although the aim was to shorten the duration of nulliparous labor, it has attracted much attention for its apparent (but as yet unproven) ability to lower the cesarean section delivery rate.

Active management does decrease the duration of labor in nulliparas, but an improvement in obstetric outcome has yet to be conclusively demonstrated.

CONTRACTED PELVIS (anatomically)

1. Definition.
2. Classification.
3. Diagnosis.
4. Influence on the course of pregnancy and labor.
5. Complications.

Definition: It is indeed difficult to define precisely what constitutes a contracted pelvis. Anatomically, contracted pelvis is defined as one where the essential diameters of one or more planes are shortened by 0.5 cm. But of more importance is 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**.

Depending upon the degree of contraction, the head may pass through the pelvis by abnormal mechanism or fail to pass due to absolute obstruction.

Variations of female pelvis: The size and shape of the female pelvis differ so widely due to morphological factors such as developmental, sexual, racial and evolutionary that it is indeed difficult to define what the features of a normal pelvis are. **However**, **on the basis of the shape of the inlet**, **the female pelvis is divided into four parent types:**

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

EFFECTS OF CONTRACTED PELVIS ON PREGNANCY AND LABOR

Pregnancy: The general course of pregnancy is not much affected. However, **the following may occur:** (1) There is more chance of incarceration of the retroverted gravid uterus in flat pelvis; (2)

Abdomen becomes pendulous specially in multigravida with lax abdominal wall; and (3) Malpresentations are increased 3–4 times and so also increased frequency of unstable lie.

Labor: The course of events in labor is greatly modified depending upon the degree of pelvic contraction and presentation of the fetus: (1) There is; increased incidence of early rupture of the membranes; (2) Incidence of cord prolapse is increased; (3) Cervical dilatation is slowed; (4) 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.

RACHITIC FLAT PELVIS

Rickets is predominantly a disease of early childhood when the bones remain soft and unossified. In childhood changes occur in the bony pelvis due to weight bearing. The classic changes in the pelvic bones are shown.

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.

FLAT PELVIS

In the flat pelvis, the head finds difficulty in negotiating the brim and once it passes through the brim, there is no difficulty in the cavity or outlet. **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.

GENERALLY CONTRACTED PELVIS: In this type of pelvis, the shape remains unaltered but all the diameters in the different planes – inlet, cavity and outlet are shortened. **There is difficulty from the beginning to the end.**

CONTRACTED PELVIS (clinically)

1. Definition.
2. Classification.
3. Diagnosis.
4. Influence on the course of pregnancy and labor.

Complications

Cephalopelvic disproportion

Classically, CPD is classified as follows:

1 *Absolute.* There is no possibility of a normal vaginal delivery even if the mechanisms of labour are completely correct. In the Western world, this condition is extremely rare; it may be due to the following:

- Fetal hydrocephalus.
- Congenitally abnormal pelvis (such as Robert's or Naegele's pelvis) in which one or both sacral ala are missing leading to a narrowing of the pelvic inlet.
- A pelvis that has been damaged usually due to a severe roll-over road traffic accident in youth.
- A pelvis that has been grossly distorted from osteomalacia.

2 *Relative CPD.* This means that the baby is large but would pass through the pelvis if the mechanisms of labour function correctly. If, however, the head is deflexed or fails to rotate in the mid-cavity, then prolonged, abnormal labour will occur.

The above definitions do not include estimates of the weight of the baby or X-ray measurements of the pelvis. CPD can only truly be diagnosed after a trial of labour. This means awaiting the onset of spontaneous labour and, if that labour becomes prolonged and abnormal, stimulating with Syntocinon as described above. CPD may be suspected antenatally in women who are less than 5¢2≤ (1.58m) in height. These women tend to have a small gynaecoid pelvis but they often also have small babies. In a cephalic presentation there is now

little evidence that X-ray pelvimetry or a CT scan helps in management. These women should have a trial of labour and in many cases will deliver vaginally. All women with a high head at term should have an obvious cause excluded by an ultrasound examination. This will diagnose placenta praevia, uterine fibroids, or an ovarian cyst as the cause. In the absence of these findings, one should suspect that the cause is CPD. Head fitting tests and X-ray pelvimetry have only a small role in the management of women with a cephalic presentation because the correct management is the proper use of a trial of labour.

ABNORMAL UTERINE ACTION

1. Definition.
2. Classification.
3. Diagnosis.
4. Description of each variant of anomalies of labor activity separately.
5. Management.
6. Complications.

Abnormal labor and delivery

Labor dystocia

- *Definition*. Abnormal or inadequate progress in labor.
- Also known as failure to progress, prolonged labor, failure of cervical dilation, failure of descent of the fetal head.
- *Causes.* Inadequate "power" (uterine contractions), inadequate "passage" (bony pelvis), or abnormalities of the "passenger" (fetal macrosomia, hydrocephalus, malpresentation, extreme extension or asynclitism (lateral tilting) of the fetal head).
- *Cephalopelvic disproportion* (CPD) is classified as absolute (where the disparity between the size of the bony pelvis and the fetal head precludes vaginal delivery even under optimal conditions) or relative (where fetal malposition, asynclitism, or extension of the fetal head prevents delivery). Absolute CPD is an absolute contraindication to attempted vaginal delivery.
- *Management.* Exclude absolute CPD. Confirm "adequate" uterine activity. If contractions are "adequate," one of two events will occur: dilation and effacement of the cervix with descent of the head, or worsening caput succedaneum (scalp edema) and molding (overlapping of the skull bones). Proceed with timely cesarean section delivery, if indicated.

Malpresentation

Breech

Transverse (shoulder presentation) or oblique lie

- *Incidence.* This is 0.3% of term pregnancies.
- *Etiology.* Prematurity, placenta previa, grandmultiparity, multiple gestation, uterine anomalies (fibroids, bicornuate uterus).
- *Management.* Consider external cephalic version. Cesarean section delivery if unsuccessful.

Other malpresentations

- Malpresentations can occur in a vertex fetus. Some can be delivered vaginally (such as occiput posterior, face with mentum [chin] anterior).

In others (brow, face with mentum posterior), conversion to occiput anterior is necessary for vaginal delivery.

- *Compound presentation* (<0.1% of all deliveries) refers to the presence of a fetal extremity alongside the presenting part. It is associated with prematurity, polyhydramnios, and multiple gestations. Vaginal delivery can often be affected.
- *Funic presentation* refers to presentation of the umbilical cord below the head. It is rare. If identified in labor, cesarean section delivery may be indicated because of the risk of cord prolapse.

Intrapartum complications

Cord prolapse

- An obstetric emergency characterized by prolapse of the umbilical cord into the vagina after rupture of the fetal membranes.
- *Incidence.* Less than 0.5% of term cephalic pregnancies.
- *Risk factors.* Malpresentation (breech, transverse lie), polyhydramnios, small fetus, prematurity.
- *Diagnosis.* Palpation of a pulsatile cord on vaginal examination with or without fetal bradycardia.

- *Prevention.* Perform amniotomy only once the vertex is well applied to the cervix and always with fundal pressure.
- *Management.* Replace cord manually and expedite delivery immediately (usually by emergency cesarean section).

Shoulder dystocia and brachial plexus injury

- *Shoulder dystocia* is an obstetric emergency associated with neonatal birth trauma (neurologic injury, fractures of the humerus, skull, clavicle) in up to 30% of cases. Immediate identification and prompt and appropriate intervention may prevent neonatal birth trauma in some cases. Shoulder dystocia complicates 0.2–2% of all vaginal deliveries. Although several risk factors are described, most cases occur in women with no risk factors.
- *Brachial plexus paralysis* is the second most common neurologic birth injury (after facial nerve palsy) complicating 0.5–3 per 1,000 deliveries. It results from "excessive" lateral traction on the head and neck at delivery with resultant injury to the brachial plexus, usually to cervical nerve roots C5–7 (Erb/Duchenne palsy). The lower brachial plexus (C8–T1) may also be involved. On examination, the arm hangs limply at the side of the body with the forearm extended and internally rotated, the classic "waiter's tip" deformity. The function of the fingers is usually retained; 95% of brachial plexus injuries resolve completely within 2 years with the help of physical therapy. Elective cesarean section delivery will prevent most (but not all) brachial plexus injuries. Given the difficulty in predicting shoulder dystocia, however, cesarean section delivery cannot be recommended for all women with identifiable risk factors.

Other congenital neurologic birth injuries

- *Facial nerve paralysis* results from pressure on the facial nerve (cranial nerve VII) as it exits the skull through the stylomastoid foramen. It is the most common neurologic birth injury (0.1–8 per 1,000 live births). It is more common after surgical

vaginal (forceps) delivery. Resolution is usually complete within a few days.

- *Injuries to the neck and spinal cord* may result from excessive traction at delivery with fracture or dislocation of the vertebrae. Such injuries may prove fatal. The true incidence of spinal injuries is not known.
- *Multicystic encephalomalacia* is a pathologic condition specific to monochorionic twin multiple pregnancies in which cerebral damage develops in the surviving fetus after intrauterine demise of its co-t win. The mechanism of cerebral injury is not known. Unfortunately, immediate cesarean section delivery does not appear to prevent neurologic injury in the surviving twin.

Intracranial hemorrhage

- Bleeding into the fetal head can occur at several anatomic sites.

Intraventricular hemorrhage (IVH), defined as bleeding into the germinal matrix within the ventricles, occurs most commonly.

- *Incidence.* Of term infants 4–5% will have sonographic evidence of IVH unrelated to obstetric factors.
- *Risk factors.* Prematurity, fetal bleeding diathesis, alloimmune thrombocytopenia. Birth trauma is an uncommon cause of intracranial hemorrhage.
- *Treatment.* Primarily supportive. Surgery is rarely indicated.
- *Prognosis* depends on gestational age at delivery, the presence and extent of ventriculomegaly, and the extent and location of the hemorrhage (parenchymal and subdural hemorrhages have a poor prognosis in 90% of cases because the hemorrhage is often more excessive; IVH has a poor prognosis in 45% of cases; only grade 3 and 4 IVH are associated with significant long-term neurologic sequelae).

FACE AND BROW PRESENTATION

1. Determination.
2. Cause.
3. Diagnosis.
4. Effect on pregnancy and labor.
5. Mechanism of delivery.
6. Complications.

Face presentation

As the fetal head gets driven down the birth canal, the front of the head can become extended. Distinguish from face-to-pubis delivery.

Incidence

0.3% of all deliveries.

Aetiology

1 Lax uterus, multiple pregnancy, polyhydramnios.

2 Deflexed fetal head.

3 Shape of fetal head:
- Dolichocephalic (long head).
- Anencephalic (no cranium).

Mechanism

Head descends with face leading. Chin (mentum) used as denominator to determine rotation. 85% engage in the mentotransverse (submentobregmatic diameter – 10 cm). With descent, most rotate to mentoanterior on the pelvic floor, the fetal chin coming behind the maternal pubis. After further descent, the chin can escape from under the lower back of the pubis and the head is then delivered over the vulva by flexion. Up to this point, the mechanisms of flexion/extension of the fetal head are the reverse of those with a vertex presentation. After

delivery of the head, however, the external rotations are the same allowing the fetal shoulders to negotiate the pelvis.

A few face presentations rotate from the transverse to mentoposterior, so that the fetal chin is in the curve of the mother's sacrum; the fetal occiput and back are crushed into each other behind the pubic bone. Further descent is unlikely for the head cannot extend further and so cannot negotiate the forward curve of the birth canal and Caesarean section is needed.

Diagnosis

Rarely made before labour and of little significance if it is.

Abdomen

- Longitudinal lie with body nearer to mid-axis of uterus.
- More head felt on the same side as the back.

Vaginal examination

- Do not expect the face to feel like the newborn baby's face. Oedema always obscures facial parts.
- Supra-orbital ridges lead to the bridge of the nose.
- Mouth has hard gums in it and may suck on the examining finger.

Management

IN PREGNANCY

- Await events.
- Membranes may rupture early (examinevaginally to exclude prolapsed cord).
- Check that pelvis is adequate and that fetus is not oversized. If either, consider Caesarean section for face presentation in labour has a higher risk.
- Check with ultrasound that the fetus is not an anencephalic for this might alter management.

IN LABOUR

- If anterior rotation to mentoanterior, a longer labour but spontaneous delivery will probably occur (90%).
- If head stays in mentotransverse, either manual rotation to mentoanterior and forceps extraction, or Kielland's forceps rotation and extraction or deliver by Caesarean section.
- If face rotates posteriorly, this is impossible to deliver vaginally. Hence, perform a Caesarean section.

Results

MOTHER

Higher morbidity associated with operative delivery.

BABY

Higher mortality:

- Abnormalities incompatible with life (anencephaly).
- In the normal, hypoxia and cerebral congestion.

Brow presentation

A very poorly flexed head may present the largest diameter of the skull: mentovertex (13 cm).

Incidence

0.1% of all deliveries.

Diagnosis

Rarely made before labour and of little significance if it is.

Abdomen

- Head feels big.
- Not well engaged.
- Groove between occiput and back. Head felt on both sides of fetus.

Vaginal examination

- Anterior fontanelle presents.
- Supraorbital ridges and base of nose can be felt at edge of field.

Management

IN PREGNANCY

- Await events. No point in trying to convert to more favourable presentation.
- Membranes may rupture early (examine vaginally to exclude prolapsed cord).

IN LABOUR

- If diagnosed early, await events for some convert spontaneously to face (by further extension) or vertex (by flexion).
- If presentation persists, it will be impossible to deliver vaginally. Hence deliver by Caesarean section.
- If fetus is dead or there is hydrocephaly, the destructive operation of perforation of head and vaginal extraction is possible provided the operator is skilled in these arts, but in the Western world these are a diminishing number.

Results

MOTHER

Higher morbidity associated with operative delivery.

BABY

Because of wider use of Caesarean section, morbidity and mortality rates are low.

SHOULDER PRESENTATION

1. Determination.
2. Cause.
3. Diagnosis.
4. Effect on pregnancy and labor.
5. Neglected shoulder presentation.
6. Complications.

Shoulder presentation (transverse lie)

Incidence

0.3% of all deliveries.

Aetiology

As for other malpresentation but most commonly:

1 Polyhydramnios causing an increased ratio of fluid to fetus.

2 Something preventing the engagement of the head in the pelvis:
- Placenta praevia.
- Fibroids.
- Contracted pelvis.

3 Abnormal shape of uterus (subseptate or arcuate uterus).

4 Second twin.

5 Grand multiparity (5+).

Diagnosis

1 Abdominal examination—the head is in one flank and the buttocks in the other. Commonly, the fetus can be rotated to a cephalic presentation quite readily but reverts back to a transverse position.

2 Vaginal examination—the pelvis is empty of presenting parts.

3 Investigation: ultrasound scan confirms diagnosis.

Management of transverse lie in pregnancy and labour

1 Before 36 weeks, ECV may be attempted or the woman referred back to the following week's clinic. The position is usually self-curing.

2 Past 37 weeks in a multiparous patient, and after 38 weeks in a primiparous one, admission to hospital should be advised, where ECV is attempted each day.

3 Should the woman go to term with the fetus still in a transverse position, management may be by either of the following:

- A *stabilizing induction*: ECV is done in the labour ward. The fetal head is held over the brim of the mother's pelvis and high membrane rupture is performed. Amniotic fluid escapes and the head often sinks into the pelvis. Labour follows in the normal fashion.
- An *elective Caesarean section.* In the Western world this may be the safer line of treatment for the fetus since it cuts down the risks of prolapsed cord during labour, but it does leave the mother with a scarred uterus for future pregnancies and an increased risk of postpartum problems.

4 Occasionally a woman is admitted in mid or late labour with a transverse lie. This would lead to an impacted shoulder presentation, the folded fetus having been driven a varying amount down the pelvis, depending on how far labour has gone. Treatment must be by immediate Caesarean section even if the fetus is dead because of the risk of uterine rupture.

BLEEDING IN THE FIRST HALF OF PREGNANCY (abortion, cervical pregnancy, trophoblastic disease)

1. Determination.
2. Cause.
3. Diagnosis.
4. Management.
5. Complications.

- *Definition*. A spectrum of histologically distinct diseases originating from the placenta: partial and complete hydatidiform mole, choriocarcinoma, and placental-site trophoblastic tumor (PSTT).
- *Tumor marker*. Serum levels of βhCG (β-human chorionic gonadotropin) are extremely accurate.

Hydatidiform moles

- *Incidence*. Japan has the highest incidence of molar pregnancy (2.0/1,000 pregnancies versus 0.6–1.1 for Europe and North America). Variations in the worldwide incidence rates result in part from discrepancies between population-based data and hospital-based data.
- *Risk factors* include maternal age >35 years (>2× increase), prior molar pregnancy (10× increase), long-term use of oral contraceptives (2× increase), and dietary deficiency (β-carotene, vitamin A).
- *Chromosomal origin*
- *Clinical presentation*. Partial moles usually present as a missed abortion during the first or early second trimester. Normal or marginally elevated βhCG levels are common. Complete moles typically have abnormal vaginal bleeding (85%) that prompts a healthcare visit. Due to earlier detection, fewer than 10% of women will have anemia, hyperemesis gravidarum,

or pre-eclampsia. Markedly elevated βhCG levels (>>100,000 mIU/mL) are characteristic.

- *Sonographic findings.* Partial moles may be suspected by visualizing a fetus with focal cystic spaces in the placenta and an increase in the transverse diameter of the gestational sac. Complete moles classically have a "snowstorm" appearance of diffuse hydropic swelling without a fetus. First-trimester sonograms may be too early to distinguish small molar villi from degenerating chorionic villi.
- *Diagnosis* of hydatidiform moles is made by histopathologic analysis. Partial moles have a non-viable fetus with malformations (syndactyly, hydrocephalus, growth restriction), variably hydropic (swollen) villi, and minimal trophoblastic hyperplasia. Complete moles have no fetal tissue and consist of diffusely hydropic villi (grape-like vesicles) with widespread trophoblastic hyperplasia. Immunostaining with p57 or ploidy analysis may be indicated in some equivocal cases.
- *Treatment.* Electric vacuum aspiration (EVA; see Chapter 16) is generally the initial treatment for molar pregnancy because patients are commonly young and desirous of future fertility. Hysterectomy is an alternative in selected patients who desire surgical sterilization.
- *Prophylaxis.* Anti-D immunoglobulin should be administered to appropriate rhesus-negative patients.
- *Surveillance.* βhCG levels should be monitored post-evacuation until they are undetectable.
- *Hormonal contraception* should be encouraged to prevent pregnancy and reduce the potential for complicating βhCG interpretation.
- *Future pregnancies.* Patients may expect normal reproductive outcome of subsequent conceptions. The risk of developing another hydatidiform mole is increased 10-fold to approximately 1%.

Gestational trophoblastic neoplasia (GTN)

- *Antecedent gestation*. Most commonly occurs after a molar pregnancy, but may occur after any gestational event (termination or spontaneous miscarriage, ectopic pregnancy, term pregnancy).
- *Diagnosis* is not uniform worldwide, but includes one of these criteria:

1 βhCG plateau of four measurements over a period of at least 3 weeks;

2 βhCG rise of three measurements over a period of at least 2 weeks;

3 βhCG level remains elevated for more than 6 months;

4 Histologic diagnosis of choriocarcinoma.

- *Choriocarcinoma* consists of sheets of anaplastic cytotrophoblast and syncytiotrophoblast cells without chorionic villi. Invasive moles may have the histologic features of either choriocarcinoma or hydatidiform mole, but metastases are always choriocarcinoma.
- *PSTT* (placental-site trophoblastic tumor) is a rare variant of choriocarcinoma that is insensitive to chemotherapy and usually requires hysterectomy.

Staging

- GTN is anatomically staged.
- The combination of a chest radiograph, abdominal/pelvic computed tomography (CT) scan, and pelvic examination is an effective strategy to determine the extent of disease. Chest and head CT scans are indicated if the chest radiograph is abnormal.
- Biopsy of suspected metastatic lesions is not recommended and may cause hemorrhage.
- The modified World Health Organization (WHO) prognostic scoring system is used to categorize patients with GTN into low-risk (score: 0–6) or high-risk (score: ≥7) groups.

Treatment

- Low-risk GTN (WHO score 0–6) is most frequently treated by single-agent methotrexate using a variety of intramuscular or intravenous regimens. If the tumor is resistant, the patient may be switched to intravenous "pulse" dactinomycin. Recently, methotrexate has been shown to have a lower response rate than dactinomycin, but most clinicians still use it preferentially due to its very mild toxicity.
- High-risk GTN (WHO score >6) and patients who fail single-agent low-risk GTN therapy are best managed by combination chemotherapy (etoposide, methotrexate, dactinomycin, cyclophosphamide, vincristine [EMA/CO]) due to the increased risk of tumor resistance to a single agent. Patients who progress through EMA/CO may be switched to EMA/EP (where EP is etoposide and cisplatin) or a paclitaxel regimen with alternating etoposide and cisplatin.
- *Surveillance.* βhCG levels are measured until undetectable and therapy is completed. Follow-up should continue for 12 (stage I–III) to 24 months (stage IV).
- *Prognosis.* Of patients 98–100% of stage I–III patients and 75–80% of stage IV patients will be cured. Few women die from GTN in the USA or the UK, but those who do generally present moribund with stage IV disease and quickly succumb.

Placental-site trophoblastic tumor (PSTT)

- Rare variant of GTN that has intermediate cytotrophoblasts.
- Diagnosis is not usually very straightforward due to lower βhCG levels and less dramatic bleeding symptoms.
- Hysterectomy is almost mandatory because the tumor tends to be insensitive to chemotherapy.

Phantom hCG

- *Definition.* Persistent mild elevations of hCG leading physicians to treat patients for GTN when in reality no true hCG or trophoblast disease is present.

- *Cause.* Some individuals have circulating factors in their serum (heterophilic antibodies) that interact with the hCG antibody and cause false-positive results using some laboratory assays.
- *Diagnosis.* Negative urine test or serial dilutions of the serum hCG.
- *Treatment.* Recognition of the false-positive test. No treatment is needed.

BLEEDING IN THE SECOND HALF OF PREGNANCY (placenta previa, placental abruption)

- Determination.
- Cause.
- Diagnosis.
- Management.
- Complications.

Antepartum hemorrhage

Definition

Vaginal bleeding after 24 weeks' gestation and before labor.

Incidence

This is 4–5% of all pregnancies.

Differential diagnosis

Placenta previa (20%)

- *Definition*. Implantation of the placenta over the cervical os in advance of the fetal presenting part.
- *Incidence.* One in 200 pregnancies.
- *Risk factors.* Multiparity, advanced maternal age, prior placenta previa, prior cesarean section delivery, smoking.
- *Classification*
- *Diagnosis.* Characterized clinically by painless, bright-red vaginal bleeding. Bleeding is of maternal origin. Fetal malpresentation is common because the placenta prevents engagement of the presenting part. May be an incidental finding on ultrasound.

Note. When a woman presents with antepartum hemorrhage, pelvic examination should be avoided until placenta previa has been excluded.

- *Ultrasound.* Ultrasound is accurate at diagnosing placenta previa. Only 5% of cases of placenta previa identified by ultrasound in the second trimester persist to term.
- *Antepartum management.* The goal is to maximize fetal maturation while minimizing risk to mother and fetus. "Fetal distress" and excessive maternal hemorrhage are contraindications to expectant management, and may necessitate immediate cesarean section irrespective of gestational age. However, most episodes of bleeding are not life threatening. With careful monitoring, delivery can be safely delayed in most cases. Outpatient management may be an option for women with a single small bleed if they can comply with restrictions on activity and maintain proximity to a hospital. Placenta previa may resolve with time, thereby permitting vaginal delivery.
- *Intrapartum management.* Elective cesarean section delivery is recommended at 36–37 weeks' gestation. Vaginal delivery is rarely appropriate, but may be indicated in the setting of intrauterine fetal demise, fetal malformation(s) incompatible with life, advanced labor with engagement of the fetal head and minimal vaginal bleeding, or an indicated delivery with a pre-viable fetus. A *double set-up examination* in labor may be appropriate when ultrasound cannot exclude placenta previa and the patient is strongly motivated for vaginal delivery. This procedure is performed in the operating room with surgical anesthesia and two surgical teams. One team is scrubbed and ready for immediate cesarean section in the event of hemorrhage or "fetal distress." The other team then performs a gentle bimanual examination, initially of the vaginal fornices and then the cervical os. If a previa is present, immediate cesarean section is indicated. If no placenta is palpated, amniotomy can be performed and labor induced.
- *Maternal complications.* Placenta accreta (abnormal attachment of placental villi to the uterine wall) is rare (1 in 2,500 pregnancies), but complicates 5% of pregnancies with placenta previa, 10–25% with placenta previa and one prior cesarean

section, and >50% with placenta previa and two or more prior cesarean sections.

- *Neonatal complications.* Preterm birth, malpresentation. Placenta previa is not associated with intrauterine growth restriction (IUGR). Placental abruption (30%)
- *Definition*. Premature separation of the placenta from the uterine side wall.
- *Incidence.* One in 120 pregnancies.
- *Risk factors.* hypertension, prior placental abruption, trauma, smoking, cocaine, uterine anomaly or fibroids, multiparity, advanced maternal age, preterm premature rupture of the membranes, bleeding diathesis, and rapid decompression of an overdistended uterus (multiple pregnancy, polyhydramnios).
- *Classification*
- *Diagnosis.* Presents clinically with vaginal bleeding (80%), uterine contractions (35%), and abdominal tenderness (70%) with or without "fetal distress" (50%). Uterine tenderness suggests extravasation of blood into the myometrium (Couvelaire uterus). The amount of vaginal bleeding may not be a reliable indicator of the severity of the hemorrhage because bleedingmay be concealed. Serial measurements of fundal height and abdominal girth are useful to monitor large retroplacental blood collections.
- *Ultrasound.* A retroplacental collection of ≥300 mL is necessary for sonographic visualization. Only 2% of abruptions can be visualized on ultrasound. Port-wine discoloration of the amniotic fluid is highly suggestive of abruption.
- *Antepartum management* Hospitalization is indicated to evaluate maternal and fetal condition. Mode and timing of delivery depend on the condition and gestational age of the fetus, condition of the mother, and state of the cervix. In the setting of hemodynamic instability, invasive monitoring and immediate cesarean section may be necessary. If the abruption is mild and pregnancy is remote from term, expectant management may be appropriate. Placental abruption is a relative contraindication to tocolysis.

- *Maternal complications.* Maternal mortality (due to hemorrhage, cardiac failure, or renal failure) ranges from 0.5% to 5%. Aggressive volume and blood replacement should be initiated. Clinically significant coagulopathy occurs in 10% of cases.
- *Fetal complications.* Fetal demise occurs in 10–35% of cases due to fetal hypoxia, exsanguination, or complications of prematurity.

Abruption is also associated with an increased rate of congenital anomalies and IUGR.

- *Recurrence.* This is 10% after one abruption, 25% after two abruptions.

Vasa previa (rare)

- *Definition.* Bleeding from fetal vessels that cross or run in close proximity to the internal cervical os umbilical vessels (fetal blood).
- *Diagnosis.* Apt test (hemoglobin alkaline elution test) involves the addition of two to three drops of an alkaline solution to 1 mL blood.

Fetal erythrocytes are resistant to rupture, and the mixture will remain red. If the blood is maternal, erythrocytes will rupture and the mixture will turn brown.

- *Complications.* Bleeding is fetal in origin. As such, fetal mortality rate is >75% due primarily to fetal exsanguination.
- *Treatment.* Emergency cesarean section if the fetus is viable.

Other causes (50%)

- Early labor.
- Lesions of the lower genital tract (cervical polyps, erosion).

POSTPARTUM BLEEDING. TONUS AND THROMBIN AS A CAUSE OF BLEEDING

1. Determination.
2. Cause.
3. Prevention.
4. Diagnosis.
5. Management.
6. Complications.

POSTPARTUM BLEEDING. TEARS AND TISSUE AS A CAUSE OF BLEEDING

1. Determination.
2. Cause.
3. Prevention.
4. Diagnosis.
5. Management tactics of.
6. Complications.

Postpartum hemorrhage
Definition

- Postpartum hemorrhage (PPH) has traditionally been defined as an estimated blood loss of ≥500 mL. However, blood loss is underestimated clinically by 30–50%. The average blood loss after vaginal delivery is 500 mL, with 5% of women losing >1,000 mL. Blood loss after cesarean section averages 1,000 mL.
- More recently, PPH has been defined as a 10% drop in hematocrit from admission or bleeding requiring blood transfusion.

Incidence

Approximately 5% of all deliveries (4% after vaginal delivery, 6–8% after cesarean section delivery).

Classification

Early PPH

- Defined as PPH <24 hours after delivery.
- Causes include uterine atony, retained placental fragments, lower genital tract lacerations, uterine rupture, uterine inversion, abnormal placentation, and coagulopathy.

Late or delayed PPH

- Defined as PPH >24 hours but <6 weeks post-delivery.
- Causes include retained placental fragments, infection (endometritis), coagulopathy, and placental site subinvolution.

Etiology and management of PPH

Uterine atony

- *Risk factors* include uterine overdistension (due to polyhydramnios, multiple pregnancy, fetal macrosomia), high parity, rapid or prolonged labor, infection, prior uterine atony, and use of uterine-relaxing agents.
- *Management*

Retained placental fragments

- May result from retention of a cotyledon or succenturiate lobe (seen in 3% of placentas). Examination of the placenta may identify defects suggestive of retained products.
- *Management*. Dilation and curettage, preferably under ultrasound guidance.

Lower genital tract lacerations

- *Risk factors* include assisted vaginal delivery, fetal macrosomia, precipitous delivery, and use of episiotomy.
- *Diagnosis* should be considered when vaginal bleeding continues despite adequate uterine tone.
- *Management*. Primary repair.

Uterine rupture

- *Incidence.* One in 2,000 deliveries.
- *Risk factors* include prior uterine surgery, obstructed labor, "excessive" use of oxytocin, abnormal fetal lie, grandmultiparity, and uterine manipulations in labor (forceps delivery, breech extraction, and intrauterine pressure catheter insertion).
- *Treatment.* Laparotomy with repair or hysterectomy.

Uterine inversion

- *Incidence.* One in 2,500 deliveries.
- *Risk factors* include uterine atony, excessive umbilical cord traction, manual removal of placenta, abnormal placentation, uterine anomalies, and fundal placentation.
- *Symptoms* include acute abdominal pain and shock (30%). The uterus may be visibly extruding through the vulva.
- *Treatment.* Immediate manual or hydrostatic replacement.

Abnormal placentation

- Includes abnormal attachment of placental villi to the myometrium (accreta), invasion into the myometrium (increta), or penetration through the myometrium (percreta).
- Placenta accreta is the most common type (1 in 2,500 deliveries).
- *Risk factors* include prior uterine surgery, placenta previa, smoking, and grandmultiparity. Placenta previa alone is associated with a 5% incidence of accreta, which increases to 10–25% with placenta previa and one prior cesarean section and >50% with placenta previa and two or more prior cesarean sections.
- *Management.* Dilation and curettage, or hysterectomy. Coagulopathy
- *Congenital coagulopathy* complicates 1–2 per 10,000 pregnancies.

The most common diagnoses are von Willebrand disease and idiopathic thrombocytopenic purpura (ITP).

- *Acquired* causes include anticoagulant therapy and consumptive coagulopathy resulting from obstetric complications (such as preeclampsia, sepsis, abruption, and amniotic fluid embolism).
- *Management.* Stop ongoing bleeding and replace blood products (including platelets, coagulation factors, and red blood cells).

OBSTETRIC INJURIES
(rupture of the perineum, cervix)

1. Cause.
2. Classification.
3. Diagnosis.
4. Management.
5. Complications.

PERINEUM

While minor injury is quite common specially during first birth, **gross injury (third and fourth degree) is invariably a result of mismanaged second stage of labor**. Overall risk is 1% of all vaginal deliveries.

CAUSES: Perineal injury (mainly the third and fourth degree) results from (i) over stretching and/or (ii) rapid stretching of the perineum specially when the perineum is inelastic (elderly primigravida, perineal scar).

Classification of obstetric anal sphincter injury

First degree: Injury to perineal skin only.

Second degree: Injury to perineum involving perineal body (muscles) but not involving the anal sphincter.

Third degree: Injury to perineum, involving the anal sphincter complex (both external and internal).

Fourth degree: Injury to perineum involving the anal sphincter complex (EAS and IAS) and anal epithelium. (EAS = External anal sphincter; IAS = Internal anal sphincter)

PREVENTION: Proper conduct in the second stage of labor taking due care of the perineum when it is likely to be damaged, is essential (p. 136). The prevention of the perineal injuries in normal delivery has been outlined.

Management

Recent tear should be repaired immediately following the delivery of the placenta. This reduces the chance of infection and minimises the blood loss.

In cases of delay beyond 24 hours, the repair is to be withheld. Antibiotics should be started to prevent infection. **The complete tear**, **should be repaired after 3 months**, **if delayed beyond 24 hours**.

Repair of complete perineal tear

Step–I: Patient is put in lithotomy position. Antiseptic cleaning of the local area is done. Repair may be done with local infiltration of 1% lignocaine hydrochloride (10–15 mL) or with pudendal block or preferably under general anesthesia.

Step–II: Dissection is **not required** as in an old complete perineal tear. (a) **The rectal and anal mucosa** is first sutured from above downwards. No '00' vicryl or PDS, atraumatic needle, interrupted stitches with knots inside the lumen is used. (b) **The rectal muscles including the pararectal fascia** are then sutured by interrupted sutures using the same suture material. (c) **The torn ends of the sphincter ani externus** are then exposed by Allis's tissue forceps. The sphincter is then reconstructed with a figure of eight stitch, and it is supported by another layer of interrupted sutures. For repair of EAS either an overlapping or end to end approximation method can be used with similar outcome. IAS repair is done by interrupted suture.

Step–III: Repair of perineal muscle is done by interrupted sutures using No. '0' PDS or dexon or polyglactin (vicryl).

Step–IV: The vaginal wall and the perineal skin are apposed by interrupted sutures.

Suture material: For repair of EAS, monofilament sutures such as polydiaxanone (PDS) or polyglactin (vicryl) can be used. Repair of IAS is done with fine suture size such as 3-0 PDS and 2-0 vicryl as they cause less irritation and discomfort.

AFTER CARE: The after-care of the repaired perineal injuries is similar to that following episiotomy.

Special care following repair of complete tear— (1) A low residual diet consisting of milk, bread, egg, biscuits, fish, sweets, etc. is given from 3rd day onwards. (2) 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. (3) Any one of the broad spectrum antibiotics (IV cefuroxime 1.5 g) is used during the intra-operative 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 post-partum. In case of persistent incontinence of flatus and feces, endo anal USG and anorectal manometry should be considered to detect any residual defects (20–30%).

VAGINA

Isolated vaginal tears or lacerations without involvement of the perineum or cervix are not uncommon. These are usually seen following instrumental or manipulative delivery. In such cases, the tears are extensive and often associated with brisk hemorrhage.

TREATMENT: Tears associated with brisk hemorrhage, require exploration under general anesthesia with a good light. The tears are repaired by interrupted or continuous sutures using chromic catgut No. '0'. In case of extensive lacerations, in addition to sutures, hemostasis may be achieved by intravaginal plugging by roller gauze, soaked with glycerine and acriflavine. **The plug should be removed after 24 hours**. Selective arterial embolization may also be done if bleeding persists.

COLPORRHEXIS: **Rupture of the vault of the vagina is called colporrhexis.** It may be primary where only the vault is involved or secondary when associated with cervical tear (common). It is said to be complete when the peritoneum is opened up. Posterior fornix usually ruptures, however, cervical tear is usually associated with tear of the lateral fornix.

Treatment – If the tear is limited to the vault close to the cervix, the repair is done from below. If however, the cervical tear extends high up into the lower segment or major branches of uterine vessels are damaged, laparotomy is to be done simultaneously with resuscitative measures. Evacuation of hematoma and arterial ligation may be needed.

CERVIX

Minor degree of cervical tear is invariable during first delivery and requires no treatment. Extensive akusher cervical tear is rare. **It is the commonest cause of traumatic postpartum hemorrhage**. Left lateral tear is the commonest.

CAUSES:

– ***Iatrogenic*** **–** Attempted forceps delivery or breech extraction through incompletely dilated cervix.

– ***Rigid cervix*** **–** This may be congenital or more commonly following scar from previous operations on the cervix like amputation, conisation or presence of a lesion like carcinoma cervix.

– ***Strong uterine contractions*** as in precipitate labor or extremely vascular cervix as in placenta previa.

– ***Detachment*** – Detachment of the cervix may be annular which involved the entire circumference of the cervix. This occurs following prolonged labor in primary cervical dystocia. It may, however, involve only the anterior lip when it is nipped between the head and the symphysis pubis in association with the sacral os. In both varieties, the bleeding is minimal and healing occurs through epithelialization.

DIAGNOSIS:

Excessive vaginal bleeding immediately following delivery in presence of a hard and contracted uterus – raises the suspicion of a traumatic bleeding. Exploration of the uterovaginal canal under good light not only confirms the diagnosis but also helps to know the extent of the tear.

DANGERS:

Early – (1) Deep cervical tears involving the major vessels lead to severe postpartum hemorrhage (2) Broad ligament hematoma (3) Pelvic cellulitis (4) Thrombophlebitis.

Late – (1) Ectropion (2) Cervical incompetence with mid-trimester abortion.

TREATMENT:

Only deep cervical tear associated with bleeding should be repaired soon after delivery of the placenta. **Repair should be done under general anesthesia**, **in lithotomy position with a good light**. **The pre-requisites are** – Sims' posterior vaginal speculum, vaginal wall retractors, at least two sponge holding forceps and an assistant.

***Procedures*:** The anterior and posterior margins of the torn cervix are grasped by the sponge holding forceps. Instead of giving traction to the forceps, it is better to push down the fundus gently by the assistant. This makes the tear more accessible for effective suturing. **The apex is to be identified first and the first vertical mattress suture is placed just above the apex** using polyglactin (vicryl) or chromic catgut No. '0' taking whole thickness of the cervix. The bleeding stops immediately. The rest of the tear is repaired by similar mattress sutures. Mattress suture is preferable as it prevents rolling in of the edges. **A helpful guide for proper exposure** in such a case is to start suture at the proximal end and using the suture for traction, more distal tear area is exposed until the apex is in view and is repaired. The cervical tears extending to the lower segment or vault with broad ligament hematoma, are managed as outlined in rupture uterus.

OBSTETRIC INJURIES
(rupture of the uterus, eversion of the uterus)

1. Cause.
2. Classification.
3. Diagnosis.
4. Management.
5. Complications.

Uterine Rupture
Introduction

Description: Uterine rupture is characterized by the breach of the uterine wall (new or after previous uterine surgery such as cesarean delivery) that may result in significant maternal or fetal morbidity or mortality. This should be distinguished from uterine scar dehiscence, in which there is a separation of an old scar that does not penetrate the uterine serosa or result in complications. Rupture of an intact uterus (without scars) does occur on rare occasions (1/5700 – 1/20,000 deliveries, approximately 10% of ruptures) and is generally associated with significant uterine distention (polyhydramnios, multiple gestation).

Prevalence: Found in 0.3%-3.7% of patients with a previous cesarean delivery and 5% of patients for whom vaginal birth after cesarean delivery (VBAC) fails. Uterine rupture rates in women with previous classical incisions and T-shaped incisions range between 4% and 12%. Approximately 7% of emergency cesarean hysterectomies are for rupture.

Predominant Age: Reproductive age.

Genetics: No genetic pattern.

Etiology and pathogenesis

Causes: Abnormal healing of a previous uterine scar, mechanical disruption of the uterine wall weakened by previous surgery, congenital

anomalies (structural malformations, Ehlers-Danlos type IV), or abnormalities of placentation. The uterine wall may also be breached by injudicious manual removal of the placenta or manual exploration of the uterus after delivery of the placenta. Traumatic rupture of the uterus may occur with blunt trauma to the abdomen such as occurs to an unrestrained passenger during an automobile accident. The proper use of automobile lap and shoulder belts significantly reduces the risk for an injury to both mother and fetus. Air bags do not increase the risk for an injury.

Risk Factors: Previous uterine surgery (cesarean delivery; greatest for vertical incisions, myomectomy, septoplasty), multiple gestation, internal or external version, grand multiparity (20-fold increase), short interval between pregnancies, fetal malpresentation, polyhydramnios, oxytocin stimulation (unproved), low Bishop score, congenital anomalies, and disuse or misuse of vehicle passenger restraints. There is considerable evidence that cervical ripening with prostaglandin preparations increases the likelihood of uterine rupture (15-fold increase). Induction or augmentation of labor using mechanical means does not seem to increase the risk for rupture.

Sighns and symptoms

- Abrupt fetal distress (80% of cases)
- Abrupt loss of station (presenting part may cease to be present in the vagina)
- Vaginal bleeding (may not be present)
- Abdominal pain (may not be present; pain may be referred to the chest or diaphragm)
- Maternal circulatory collapse
- Uterine activity may persist despite expulsion of the fetus
- Hematuria (if rupture extends into the bladder)

Diagnoctic Approach

Differential Diagnosis

- Uterine dehiscence
- Placental abruption

- Umbilical cord prolapse (causing abrupt fetal distress)
- Adnexal torsion
- Pulmonary or amniotic fluid embolism
- Abdominal pregnancy

Associated Conditions: Fetal demise, maternal blood loss.

Workup and Evaluation

Laboratory: Inter- and postoperative blood counts. Evaluation of clotting when significant bleeding has occurred.

Imaging: Ultrasonography may demonstrate uterine dehiscence, but the need for clinical intervention often precludes the examination.

Special Tests: Intensive fetal and maternal monitoring may be indicated.

Diagnostic Procedures: History and physical examinations (vaginal and abdominal).

Pathologic Findings

Separation of previous uterine scar or a new failure of the uterine wall muscle.

Management and therapy

Nonpharmacologic

General Measures: Rapid evaluation, supportive measures as needed (fluids, blood products).

Specific Measures: Immediate operative delivery (most often by laparotomy), surgical exploration with the possibility of repair or hysterectomy. Ligation of one or both hypogastric arteries may be necessary.

Diet: Nothing by mouth once the diagnosis is made (pending surgical intervention).

Activity: Strict bed rest (pending surgical intervention).

Drug(s) of Choice

None. Supportive measures including fluids, blood products, and anesthetics (for immediate delivery) as needed. Prophylactic antibiotics are often recommended.

Follow-up

Patient Monitoring: Fetal and maternal monitoring must be maintained for those at a risk and intensified when the diagnosis is considered.

Prevention/Avoidance: Care in all uterine manipulations (eg, manual removal of the placenta, version, external pressure during delivery). Patients with a prior successful vaginal delivery have a greater likelihood of successful vaginal birth after cesarean delivery and a lower risk for uterine rupture than those without a successful vaginal delivery. One study has suggested that there is a lower rate of uterine rupture when a double-layer closure of the uterus is used at the time of cesarean delivery.

Possible Complications: Maternal morbidity or mortality possible (significantly reduced by fetal and maternal monitoring). Damage to the cervix, vagina, or bladder may occur as a part of the rupture. Fetal demise may occur in up to 50% – 75% of fundal incision ruptures and 10% – 15% of lower uterine segment ruptures. Longterm neurologic sequelae are common in infants who survive. Vertical uterine scars are associated with the greatest morbidity and mortality when a rupture occurs.

Expected Outcome: When diagnosed early and acted on promptly, a good outcome can be expected. If the uterus is repaired and preserved, the risk of recurrence in a subsequent pregnancy is approximately 20%.

Eversion of the uterus

Introduction

Description: Uterine inversion is the turning inside-out of the uterus immediately after delivery. Uncommon and often iatrogenic, this may

be associated with catastrophic bleeding and cardiovascular collapse. The condition has also been reported in nonpregnant patients with intrauterine pathology, accounting for 5% of inversions.

Prevalence: 1 of 25,000 deliveries (estimates range from 1 in 1200–57,000 deliveries based on definitions and selection criteria).

Predominant Age: Reproductive age.

Genetics: No genetic pattern.

Etiology and pathogenesis

Causes: Iatrogenic (traction on the umbilical cord or downward pressure on the uterine fundus to facilitate delivery of the placenta; exact role remains controversial); abnormalities of placentation (accreta, increta, percreta).

Risk factors: Uterine atony – multiparity (grand), uterine overdistention (multiple birth, polyhydramnios), prolonged labor, prolonged oxytocin stimulation, muscle-relaxant agents (MgSO4), rapid labor. Less than 50% of cases have risk factors.

Signs and simptoms

- A mass may be seen attached to or directly following the placenta as it delivers
- Bright-red vaginal bleeding
- Bradycardia from vagal stimulation
- Tachycardia, hypotension, and vascular collapse possible as a result of blood loss

Diagnostic approach

Differential Diagnosis

- Uterine atony
- Retained placental fragments
- Genital tract lacerations
- Coagulopathy
- Prolapsed leiomyoma

Associated Conditions: Uterine atony, postpartum hemorrhage.

Workup and Evaluation

Laboratory: Hemoglobin or hematocrit to monitor status and volume of blood loss. Acute loss may not be reflected by these measures until equilibration has occurred in 6–24 hours.

Imaging: Ultrasonography may be used to verify the diagnosis, but this is unnecessary and delays the implementation of therapy.

Special Tests: None indicated.

Diagnostic Procedures: Pelvic examination.

Pathologic Findings

Inversion of the uterus.

Management and therapy

Nonpharmacologic

General Measures: Rapid evaluation, fluid support or resuscitation, call for anesthesia assistance.

Specific Measures: Discontinue uterotonic agents until replacement is accomplished. Uterine-relaxant agents (see later), manual replacement of uterine fundus (may require general anesthesia with a relaxant agent [halothane]), may require operative intervention (replacement or hysterectomy). Once the uterine wall has relaxed, gentle manual pressure should be placed on the fundus to displace it inward and upward until its normal position can be restored and the uterus returned to its normal configuration. Uterotonic agents are then used to obtain uterine contraction and hemostasis.

Diet: Nothing by mouth until a diagnosis is established and effective treatment is rendered.

Activity: Bed rest until a diagnosis is established and effective treatment is rendered.

Drug(s) of Choice

- Tocolytics – terbutaline 0.25 mg IV (may repeat once) or nitroglycerin 100–250 mcg IV (may repeat to a total of 1000 mcg).

- Broad-spectrum antibiotic prophylaxis (first-generation cephalosporin or clindamycin/gentamycin) should be instituted.

Contraindications: See individual agents.

Precautions: If nitroglycerine is used, blood pressure must be closely monitored (hypotension).

Alternative Drugs

Halothane general anesthesia may be required.

Follow-up

Patient Monitoring: Normal postpartum care, follow-up complete blood count as needed.

Prevention/Avoidance: Little or no traction on the umbilical cord or fundal pressure during the delivery of the placenta.

Possible Complications: Hysterectomy, hemorrhagic shock, and cardiovascular collapse.

Expected Outcome: Generally good if recognized and acted on promptly.

CAESAREAN SECTION. MANAGEMENT OF PREGNANCY WITH A SCAR ON THE UTERUS

1. Indications and contraindications for surgery.
2. Types of surgery.
3. Anesthesia.
4. Preparation for surgery.
5. Course of surgery.
6. Complications.
7. Rehabilitation after surgery.

Management of pregnancy with a scar on the uterus

1. Pregnancy management.
2. Control of the state of the scar on the uterus.
3. Methods of delivery.
4. Complications.

Cesarean section delivery

Definition

Delivery of a fetus via the abdominal route (laparotomy) requiring an incision into the uterus (hysterotomy).

Incidence

Cesarean section delivery is the second most common surgical procedure (behind male circumcision), accounting for around 20–25% of all deliveries in the UK and 32% in the USA.

Indications

- Most indications for cesarean section are relative and rely on the judgment of the obstetric care provider.

- The most common indication for a primary (first) cesarean section is failure to progress in labor.
- Absolute cephalopelvic disproportion (CPD) refers to the clinical setting in which the fetus is too large relative to the bony pelvis to allow for vaginal delivery even under optimal circumstances. Relative CPD is where the fetus is too large for the bony pelvis because of malpresentation (brow, compound presentation).

Technical considerations

- Elective cesarean section can be performed after 39 weeks' gestation without documenting fetal lung maturity.
- Regional is preferred over general analgesia.
- Routine use of prophylactic antibiotics will decrease the incidence of postoperative febrile morbidity.
- Skin incision may be Pfannenstiel (low transverse incision, muscle separating, strong, but limited exposure), midline vertical (offers the best exposure, but is weak), or paramedian (vertical incision lateral to rectus muscles, rarely used). Pfannenstiel incisions may rarely be modified to improve exposure by dividing the rectus muscles horizontally (Maylard incision) or lifting the rectus off the pubic bone (Cherney incision).
- Types of hysterotomy are reviewed in class
- Elective surgery (such as myomectomy) should not be performed at the time of cesarean section, because of the risk of bleeding.

Puerperal (cesarean section) hysterectomy

Incidence

Around 1 in 6,000 deliveries.

Indications

- Performed primarily as an emergency procedure when the mother's life is at risk due to uncontrolled hemorrhage (30–40%).

- Other indications include abnormal placentation (see Chapter 56), severe cervical dysplasia, and cervical cancer.
- Permanent sterilization is not an acceptable indication for puerperal hysterectomy.

Technical considerations

- A highly morbid procedure usually requiring general anesthesia. As such, it should be performed only as a last resort.
- Warming blanket, three-way Foley catheter, and blood products should be available.
- Emergency puerperal hysterectomies are associated with a fourfold increased risk of complications compared with elective procedures.

Blood loss is often excessive (2–4 L) and blood transfusions are usually required (90%). Despite a high morbidity, overall maternal mortality rate is low (0.3%).

- It may be possible to leave the cervix behind (subtotal or supracervical hysterectomy), thereby minimizing complications, especially blood loss. This may not be possible if the cervix is the source of the excessive bleeding, such as with placenta previa.
- Although women will be amenorrheic and sterile, menopausal symptoms will not develop if the ovaries are left in place.

Vaginal birth after cesarean section

Background

- Of cesarean section deliveries, 30% are elective repeat procedures.
- Maternal mortality rate from cesarean section delivery is <0.1%, but is 2- to 10-fold higher than that associated with vaginal birth.
- Maternal morbidity (infection, thromboembolic events, wound dehiscence) is markedly higher with cesarean section.

Results

- Successful *v*aginal *b*irth *a*fter *cesarean* section (VBAC) can be achieved in 65–80% of women.
- Factors associated with successful VBAC include prior vaginal delivery, estimated fetal weight <4,000 g, and a non-recurrent indication for the previous cesarean section (breech, placenta previa) rather than a potential recurrent indication (such as CPD).

Contraindications

- Absolute contraindications include a prior classic (high vertical) cesarean section, "fetal distress," transverse lie, and placenta previa.
- Relative contraindications include breech presentation, prior fullthickness uterine myomectomy, prior uterine rupture, and (possibly) multiple gestations.

Complications

- *Uterine dehiscence* (subclinical separation of the prior uterine incision) occurs in 2–3% of cases. It is often detected only by manual exploration of the scar after vaginal delivery. In the absence of vaginal bleeding, no further treatment is necessary.
- *Uterine rupture* may be life threatening. Symptoms and signs include acute onset of fetal bradycardia (70%), abdominal pain (10%), vaginal bleeding (5%), hemodynamic instability (5–10%), and/or loss of the presenting part (<5%). Epidural anesthesia may mask some of these features. Risk factors include:

1 type of prior uterine incision (<1% for lower segment transvrse incision, 2–3% for lower segment vertical, and 4–8% for high vertical);

2 two or more prior cesarean sections (4%);

3 prior uterine rupture;

4 "excessive" use of oxytocin (although "excessive" is poorly defined);

5 dysfunctional labor pattern (especially prolonged second stage or arrest of dilation);

6 induction of labor using prostaglandins.

Factors NOT associated with an increased risk for rupture include epidural anesthesia, unknown uterine scar, fetal macrosomia, and indication for prior cesarean section.

Clinical considerations

- Continuous intrapartum fetal monitoring is recommended.
- Follow labor curve carefully for evidence of labor dystocia.
- The capacity to perform an emergency cesarean section should be at hand.

SEPSIS

1. Definition.
2. Triad of sepsis.
3. Septic syndrome.
4. Description of lesions of various organs.
5. Diagnosis.
6. Treatment.
7. Complications.

Introduction

Description: Although the term *puerperal infection* can be used to describe any infection during or after labor, it generally applies to the infection of the uterus and surrounding tissues after delivery. This can vary from mild to life-threatening severities. Some of the most severe infections may appear within hours of delivery and are often opportunistic and not associated with reliable risk factors. Vigilance and aggressive diagnosis and treatment are required.

Prevalence: Estimated to occur in l–3% of vaginal deliveries; approximately 15% if chorioamnionitis is present during labor. Following cesarean delivery: 2–10% if antibiotic prophylaxis is administered during delivery and 50–90% without antibiotic prophylaxis in some series.

Genetics: No genetic pattern.

Etiology and pathogenesis

Causes: Colonization and infection of the tissues of the uterus, peritoneum, or surrounding organs; typically, a polymicrobial infection (70%) that involves a mixture of two to three aerobes and anaerobes. The most common organisms are group B streptococci; other facultative streptococci; *Gardnerella vaginalis-,* and *Escherichia coli, Bacteroides,* and *Pept'ostreptococcus* species. Infection by clostridia or group A streptococci may result in rapidly progressive soft-tissue (subcutaneous tissue, muscle, or myometrial) infec-

tion. Abscesses usually contain both aerobic and anaerobic bacteria such as *Bacteroides* species *(Bacteroides bivius, B. disiens,* or *B. fragilis).* Approximately 50% of ascending uterine infections involve *Chlamydia trachomatis.*

Risk Factors: Cesarean delivery (10- to 20-fold increase), invasive procedures during labor, prolonged rupture of the membranes, prolonged labor, multiple vaginal examinations, retained placental fragments, manual removal of the placenta, urinary catheter, bacterial vaginosis, intravenous line(s), low socioeconomic or nutritional status, maternal age, anemia, and chronic disease (diabetes).

Signs and simptoms

- Fever (90%; >38.5°C by 24 hours) and tachycardia (often developing rapidly after delivery).
- Uterine tenderness (may be absent).
- Signs of septic or cardiovascular shock (hypotension, anxiety, disorientation, prostration).
- Impaired renal function (<20 mL/h urine production).
- Altered white blood count (<1000 or >25,000).
- Hemolysis or hemoconcentration.
- Uterine subinvolution and excessive bleeding.
- The United States Joint Commission on Maternal Welfare defines postpartum febrile morbidity as an oral temperature of >38.0°C (>100.4°F) on any 2 of the first 10 days postpartum, exclusive of the first 24 hours.

Diagnostic approach

Differential Diagnosis

- Urinary tract infections, including pyelonephritis (5% of patients; classical signs are routinely absent, urinalysis shows large numbers of white blood cells, and cultures are positive).
- Wound infection.

<< Atelectasis or pneumonitis

- Infection in intravenous line or site, contaminated fluids.

- Disturbed abscess (old tubo-ovarian or appendiceal abscess).
- Septic thrombophlebitis.
- Necrotizing fasciitis.
- Transfusion reaction (when applicable).
- Amniotic fluid or pulmonary embolism.
- Cardiogenic shock (drugs, cardiac disease, aortic dissection).
- Toxic shock syndrome.
- Mastitis (2% of patients).

Associated Conditions: Septic shock, adult respiratory distress syndrome, acute renal failure, and disseminated intravascular coagulation.

Workup and Evaluation

Laboratory: Complete blood count, endometrial culture obtained by protected swab (if amniotic fluid or endometrial culture obtained within 24 hours of delivery is not available). Blood cultures are positive in 15%-25% of patients who are febrile but do not reflect the severity of the infection. Tissue culture (direct or by needle aspiration, when wound infections is suspected) and Gram stain.

Imaging: Ultrasonography may be useful in evaluating the possibility of pelvic abscess or gas formation. Computed tomography (CT) and magnetic resonance imaging (MRI) are useful for a more wide-ranging assessment.

Special Tests: Frozen-section histopathologic evaluation may be useful if necrotizing fasciitis is suspected.

Diagnostic Procedures: History, physical examination, cultures. The diagnosis is generally clinical.

Pathologic Findings

Evidence of inflammation and/or necrosis (based on tissue involved and severity of infections); edema and hyperemia with marked inflammatory infiltrates of the endometrial glands, primarily by neutrophils. This may invade the myometrium and parametrium with areas of necrosis and thrombosis. Endometritis is defined as

five or more neutrophils per 400 high-power fields in the superficial endometrium and one or more plasma cells per 120 high-power fields in the endometrial stroma.

Management and therapy

Nonpharmacologic

General Measures: Evaluation, fluid replacement or resuscitation, antipyretics and analgesics (after a diagnosis has been established). Close monitoring, including intensive care, may be required when infection is severe. Consultation with an infectious-disease specialist may be desirable. Low-grade (<38°C) or intermittent fevers may not require treatment when present in the first 24 hours.

Specific Measures: Aggressive antibiotic therapy. Based on response, removal of infected products (if present), surgical exploration, abscess drainage (percutaneous or open), debridement, or hysterectomy may be required. Virtually all postpartum septic shock is caused by surgically treatable processes. Because of the expanded blood and tissue volume at and after delivery, antibiotic dosages must be increased by 40% over those used outside of pregnancy.

Diet: For patients who are acutely ill, nothing by mouth until condition is stabilized. For other patients, no specific dietary changes indicated.

Activity: Bed rest until patient's condition is stable, then a progressive return to normal activity.

Drug(s) of Choice

Antibiotics should be administered to provide protection against gram-negative facultative and anaerobic bacteria. Moderate infections require double antibiotic treatment (clindamycin/ gentamicin, 90–97% effective); severe infections should be treated with triple therapy: an aminoglycoside or first-generation cephalosporin (for facultative bacteria); clindamycin, imipenemcilastatin, or metronidazole (anaerobic bacteria); and penicillin or ampicillin (clostridia and synergistic action with aminoglycosides on enterococci). (3-Lactam

antibiotics (penicillin or cephalosporin) should be administered in dosages of 8–12 g/day.

Contraindications: See individual agents.

Precautions: Antibiotic dosages must be increased by up to 40% because of the altered physiologic state of pregnancy.

Interactions: See individual agents.

Follow-up

Patient Monitoring: When severe infections are present, intensive monitoring (including placement in an intensive care unit) may be required. This may include central venous access and monitoring, pulse oximetry, and careful (frequent if not continuous) blood pressure monitoring.

Prevention/Avoidance: Careful attention should be given to antisepsis, reduced numbers of vaginal examinations when the amniotic membranes have been ruptured, careful tissue handling during operative procedures, use of prophylactic antibiotics when risk factors are identified. Changing intravenous sites every 48 hours reduces the risk of infection. There is no evidence to support a role for vaginal antisepsis (chlorhexidine or similar) during labor, though benefit has been shown for its use prior to cesarean delivery. Parenteral prophylaxis at the time of cesarean delivery is appropriate. There are insufficient data to evaluate the role of prophylactic antibiotics after manual removal of the placenta or operative delivery.

Possible Complications: Progression of infection, abscess formation, septic thrombophlebitis, septic shock, adult respiratory distress syndrome, renal failure, cardiovascular collapse, death. If septic shock occurs, mortality rates of 20–30% are common. Coagulopathy may develop. Necrotizing fasciitis is possible.

Expected Outcome: With timely diagnosis and appropriate therapy a complete recovery with no long-term sequelae should be expected. Approximately 90% of patients rapidly respond to antibiotic therapy (and/or percutaneous drainage of abscesses).

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ПО АКУШЕРСТВУ
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