I. Z. GLADCHUK, I. A. ANCHEVA

OBSTETRICS

ODESA NATIONAL
MEDICAL UNIVERSITY

I. Z. Gladchuk, I. A. Ancheva

OBSTETRICS

A manual

Odesa National
Medical University
2022

UDC 618.2(075.8)

Authors: I. Z. Gladchuk — doctor of medical sciences, professor, the head of the Obstetrics and Gynecology Department of the Odesa National Medical University,
I. A. Ancheva — doctor of medical sciences, professor of the Obstetrics and Gynecology Department of the Odesa National Medical University

Reviewers: O. V. Golianovsky — doctor of medical sciences, professor, the head of the Obstetrics and Gynaecology Department No 1 of Shupyk National Healthcare University of Ukraine

A. M. Gromova — Honoured Scientist and Engineer of Ukraine member of the Order of St. Olga Equal-to-the-Apostles Third Class, doctor of medical sciences, professor, the head of the Obstetrics and Gynecology Department No1 of the Poltava State Medical University

Recommended by the Academic Council of Odesa National Medical University as a manual for students of the higher medical establishments of the IV level of accreditation (meeting minutes of Academic Council on August 31, 2022 No 1)

Навчальний посібник створено з метою покращення засвоєння матеріалу розділу «Фізіологічний та патологічний перебіг вагітності, пологів та післяпологового періоду» з дисципліни «Акушерство і гінекологія», які передбачені програмою навчання за кредитно-модульною системою, а також з урахуванням європейських стандартів діагностики та лікування.

Для англомовних студентів медичних факультетів зі спеціальності «Лікувальна справа» вищих медичних закладів освіти України IV рівня акредитації.

Gladchuk I. Z.

Obstatrics : manual / I. Z. Gladchuk, I. A. Ancheva. — Odesa : ONMedU, 2022. — 172 p.

ISBN 978-966-443-115-3

The manual was established with the aim to improve learning the material "Physiological and pathological course of pregnancy, childbirth and postpartum period" by the discipline "Obstetrics and gynecology", which are provided by the curriculum program with taking into account European standards of diagnosis and treatment.

For English-speaking students of medical faculties by specialty "Medical care" of higher medical institutions of Ukraine of the IV accreditation level.

ISBN 978-966-443-115-3

INTRODUCTION

Despite the fact that most pregnancies and labors have an uncomplicated course, all cases of pregnancy pose a certain risk. So, according to the WHO, 15% of pregnant women have a life-threatening pathology, which requires qualified medical care, and in some cases urgent intervention.

Nowadays the demographic problem is very important in Ukraine. In the world, the birth rate is decreasing mainly at the expense of the developed countries, especially Western Europe, and therefore the infant mortality in the world becomes an increasingly urgent topic for discussion every year.

This book recommends the clinical principles of assisting women with a physiological pregnancy and with possible complications during pregnancy and childbirth.

The presented course of normal labor using partograms and active management of the third stage of labor is covered in order to provide the student with the information necessary for differential diagnosis between normal and pathological delivery. The main types of obstetric emergency conditions are also covered. In addition, there is information on primary care for newborns without any pathology. Presence of figures and tables provide simplification of the information and promote memorization.

A doctor of any specialty must be able to help a woman in labor and perform normal delivery. Therefore, after completing the study of the obstetrics cycle, the student should be able to determine the duration of the pregnancy and the childbirth, the estimated weight of the fetus, assess the condition of the fetus, and measure and evaluate the bone pelvis. One more poin: to determine the factors complicating the course of pregnancy and childbirth, to make an individual diagnosis with the indication of the period of birth, to determine the level of the head, to know the signs of placenta abscission, be able to assess physiologically acceptable blood loss in childbirth.

In order to get a clear understanding of the patient's problem, students should understand and be able to describe and record the history of pregnancy and childbirth properly.

The authors hope that the book will help English-speaking students of higher medical institutions in mastering knowledge of the obstetrics and passing the national standardized examinations in this field.

Igor GLADCHUK,
MD, PhD, Professor

LIST OF ABBREVIATIONS

AC — Abdominal circumference
ACOG — American Congress of Obstetricians and Gynecologists
ACTH — Adrenocorticotropic hormone
AFV — Amniotic fluid volume
AIDS — Acquired immune deficiency syndrome
AMTSL — Active management of third stage of labor
ANC — Antenatal care
ARDS — Acute respiratory distress syndrome
ARM — Artificial rupture of membranes
BMI — Body mass index
BPD — Biparietal diameter
CPD — True cephalopelvic disproportion
CRL — Crown rump length
CS — Cesarean section
CTG — Cardiotocography
E2 — Estradiol
EDD — The expected date of delivery
EPF — Early pregnancy factor
EVT — Extravillous trophoblast
FHR — Fetal heart rate
FHS — Fetal heart sounds
FL — Femur length
FSH — Follicle stimulating hormone
GCT — A glucose challenge test
GFR — Glomerular filtration rate
GH — Growth hormone
GnRH — Gonadotrophin releasing hormone
GTT — A glucose tolerance test
HAART — Highly active anti-retroviral treatment
HC — Head circumference
HCG — Human chorionic gonadotrophin
HCS — Human chorionic somatomammotropic hormone
HIV — Human immunodeficiency virus
HPL — Human placental lactogen
HPV — Human papillomavirus
IM — Intramuscular (administration)
IU — International unit
IUGR — Intrauterine growth restriction
JVP — Jugular venous pressure
LH — Luteinizing hormone
LMP — Last menstrual period
LSCS — Lower segment Cesarean section
MRI — Magnetic resonance imaging
MSAFP — Maternal serum alpha fetoprotein
NICE — National Institute for Health and Care Excellence
NICU — A neonatal intensive care unit
NTDs — Neural tube defects
P4 — Progesterone
PIH — Pregnancy-induced hypertension
PPH — Postpartum hemorrhage
PRBC's — Packed red blood cells
PROM — Premature rupture of the membranes
SFH — Symphysis fundal height
SVD — Spontaneous vaginal delivery
TSH — Thyroid-stimulating hormone
TVU — Transvaginal ultrasound
UE3 — Unconjugated estriol
USG — Ultrasonography
VBAC — Vaginal birth after cesarean section
WHO ANC — World Health Organization Antenatal care

Chapter 1
PHYSIOLOGY OF REPRODUCTION

REPRODUCTIVE CYCLE

Each female reproductive cycle (menstrual cycle) represents a complex interaction between the **hypothalamus, pituitary gland, ovaries,** and **endometrium.**

Cyclic changes in the *gonadotropins*, *luteinizing hormone* (LH), and *follicle-stimulating hormone* (FSH) and sex *steroid hormones*, mainly *estradiol* (E2) and *progesterone* (P4), induce functional as well as morphologic changes in the ovary, resulting in follicular maturation, ovulation, and corpus luteum formation (Fig. 1.1). Similar changes at the level of the endometrium allow for successful implantation of the fertilized ovum or a physiologic shedding of the menstrual endometrium when an early pregnancy does not occur. By convention, the normal cycle begins on the first day of menstrual bleeding and ends just before the first day of the next menses. The average length of each cycle is 28 (±7) days. The reproductive cycle can be viewed from the perspective of each of four physiologic components.

The pituitary gland lies below the hypothalamus at the base of the brain. The pituitary gland is divided into two major portions, the neurohypophysis and the adenohypophysis.

The neurohypophysis serves primarily to transport oxytocin and vasopressin (antidiuretic hormone) along neuronal projections from the supraoptic and paraventricular nuclei of the hypothalamus to their release into the circulation. The anterior pituitary contains different cell types that produce six protein hormones: FSH, LH, *thyroid stimulating hormone* (TSH), *prolactin, growth hormone* (GH), and *adrenocorticotropic hormone* (ACTH) (Fig. 1.2).

The ovarian changes are controlled mainly by the anterior pituitary.

At the end of the menstrual cycle estrogen levels are low. Low estrogen levels stimulate production of FSH by the pituitary. FSH in turn acts upon the ovary to stimulate growth of ovarian follicles. The increasing levels of estrogen produced by the developing follicles act on the pituitary to reduce FSH levels by the process of negative feedback (Fig. 1.3) [4].

In the majority of cycles only one follicle, the so-called dominant follicle, is sufficiently large and has a greater density of FSH receptors to respond to the lower FSH levels and develops to the stage of ovulation. Non-identical twinning results when more than one follicle proceeds to ovulation. Estrogen levels continue to rise. In the mid-cycle the nature of the ovarian control of pituitary function changes. Increasing estrogen levels are required to produce a positive feedback mechanism which causes a surge in FSH and LH levels. This surge evokes ovulation.

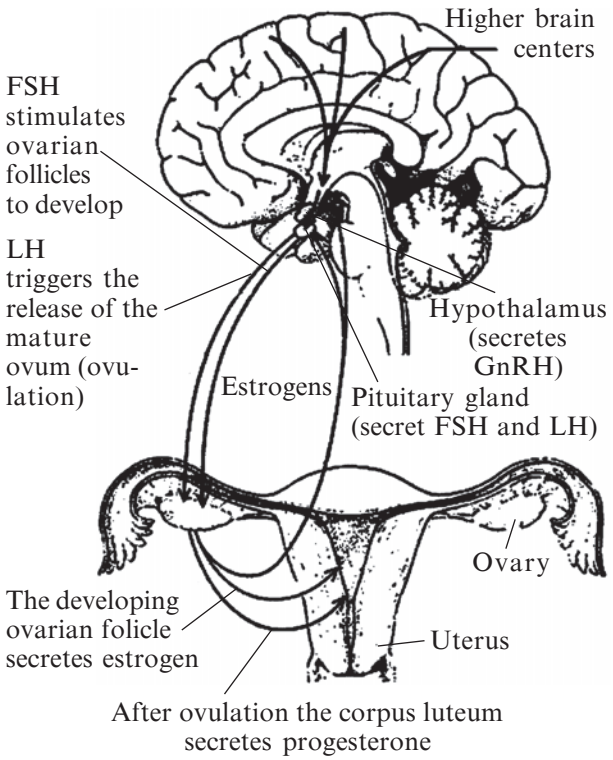


Fig. 1.1

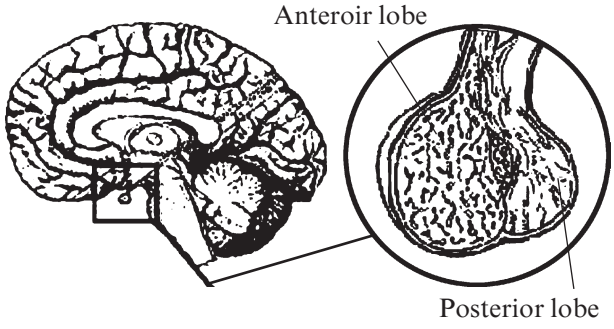


Fig. 1.2

Ovulation is a process whereby a secondary oocyte is released from the ovary following rupture of a mature Graafian follicle and becomes available for conception. LH acts to increase local production of prostaglandins and proteolytic enzymes to allow oocyte extrusion. LH is responsible for the development of the corpus luteum which produces progesterone (Fig. 1.4).

The normal functional lifespan of the corpus luteum is about 9 to 10 days. After this time, it regresses, and unless pregnancy occurs, menstruation ensues and the corpus luteum is gradually replaced by an avascular scar called a corpus albicans [33].

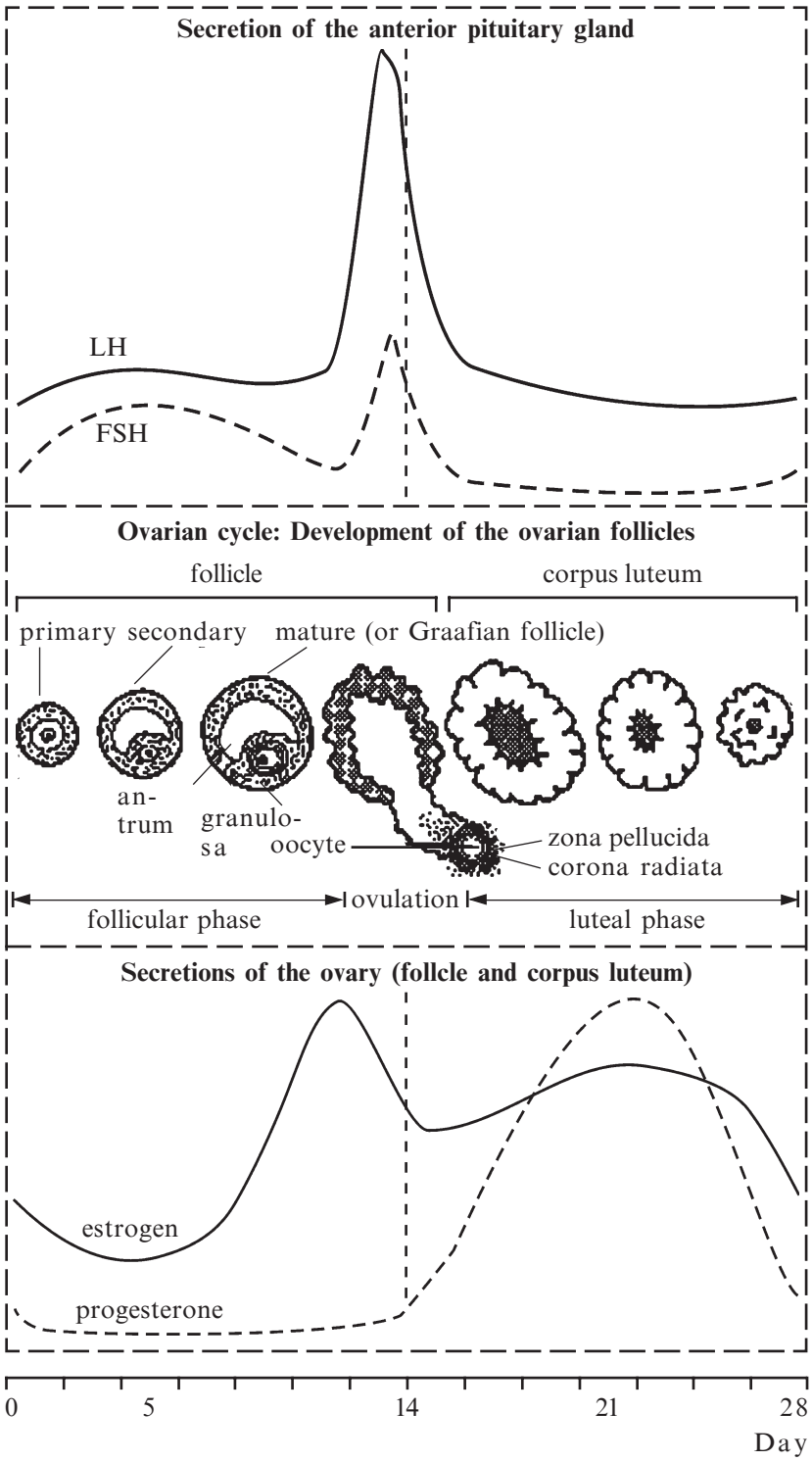


Fig. 1.3

HISTOPHYSIOLOGY OF THE ENDOMETRIUM

The endometrium is uniquely responsive to the circulating progestins, androgens, and estrogens. It is this responsiveness that gives rise to menstruation and makes implantation and pregnancy possible. At the completion of the menstrual period the endometrium is only one to two millimetres thick. Under the influence of increasing levels of estrogen this increases until by day 12 of the cycle the endometrium is 10 to 12 mm thick. This growth results from an increase in epithelial and stromal cells of the superficial layer of endometrium.

Functionally, the endometrium is divided into two zones: (1) the *outer portion*, or *functionalis*, which undergoes cyclic changes in morphology and function during the menstrual cycle and is sloughed off at menstruation; and (2) the *inner portion*, or *basalis*, which remains relatively unchanged during each menstrual cycle and, after menstruation, provides stem cells for the renewal of the functionalis. *Basal arteries* are regular blood vessels found in the basalis, whereas *spiral arteries* are specially coiled blood vessels seen in the functionalis (Fig. 1.5).

The cyclic changes in histophysiology of the endometrium can be divided into three stages: the *menstrual phase*, the *proliferative* or *estrogenic phase*, and the *secretory* or *progestational phase.*

Menstrual phase. Because it is the only portion of the cycle that is visible externally, the first day of menstruation is taken as day 1 of the menstrual cycle. The first 4 to 5 days of the cycle are defined as the menstrual phase. During this phase, there is disruption and disintegration of the endometrial glands and stroma, leukocyte infiltration, and red blood cell extravasation. In addition to this sloughing of the functionalis, there is a compression of the basalis due to the loss of ground substances. Despite these degenerative changes, early evidence of renewed tissue growth is usually present at this time within the basalis of the endometrium.

This *Proliferative phase* is characterised by an increase in estrogen receptor content and increase in size of the endometrial glands.

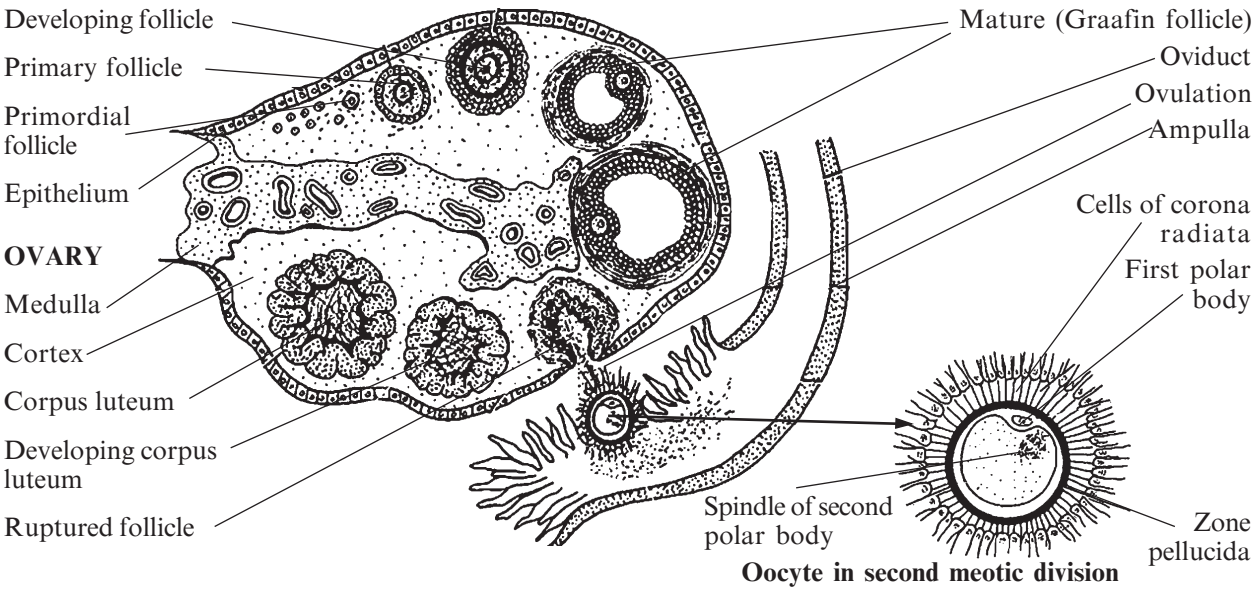

Developing follicle
Primary follicle
Primordial follicle
Epithelium
OVARY
Medulla
Cortex
Corpus luteum
Developing corpus luteum
Ruptured follicle
Mature (Graafin follicle)
Oviduct
Ovulation
Ampulla
Cells of corona radiata
First polar body
Spindle of second polar body
Zone pellucida
Oocyte in second meotic division

Fig. 1.4

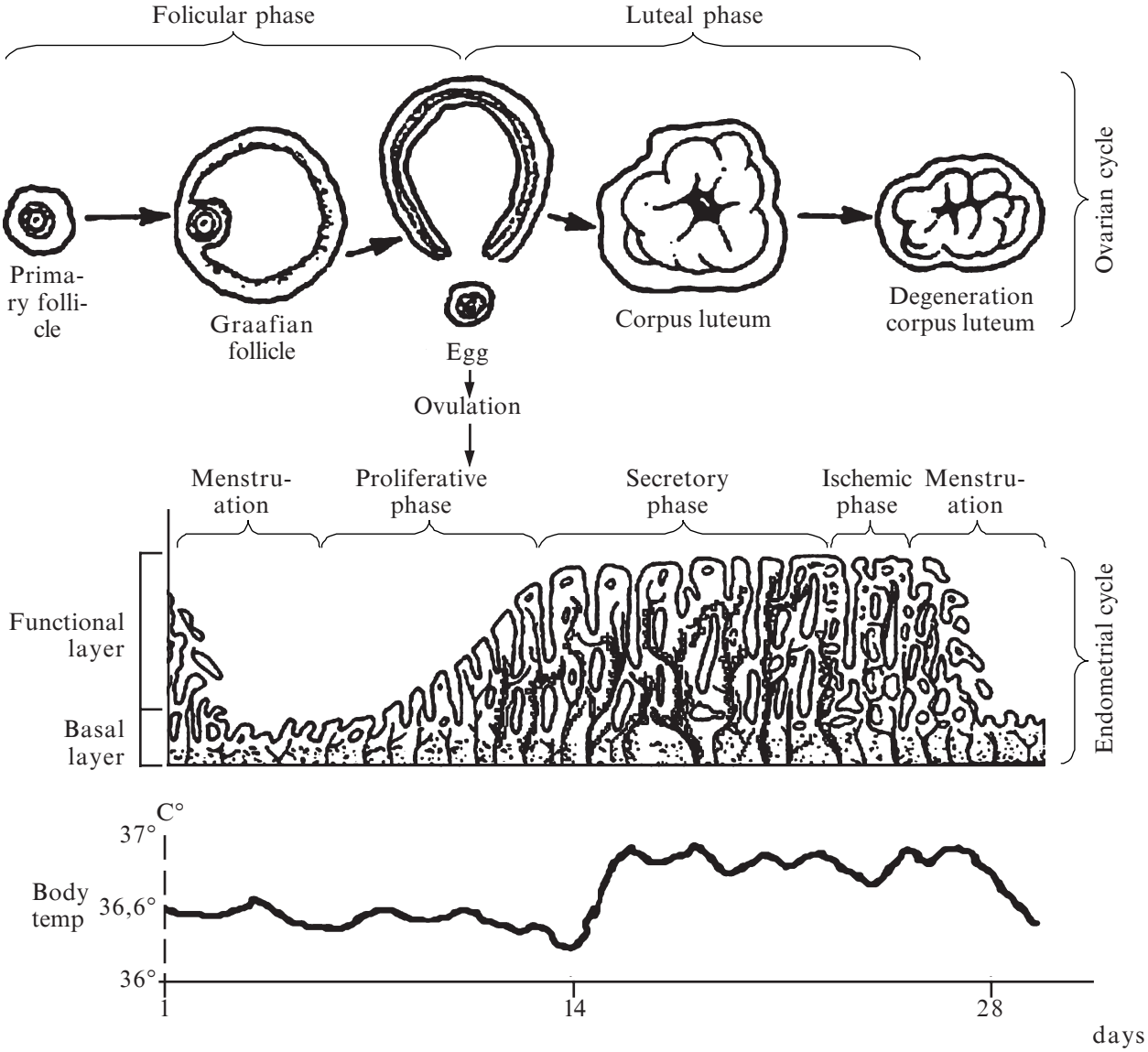

Folicular phase
Luteal phase
Prima-ry folli-cle
Graafian follicle
Egg
Corpus luteum
Degeneration corpus luteum
Ovarian cycle
Ovulation
Menstru-ation
Proliferative phase
Secretory phase
Ischemic phase
Menstru-ation
Functional layer
Basal layer
Endometrial cycle
C°
37°
Body temp
36,6°
36°
1
14
28
days

Fig. 1.5

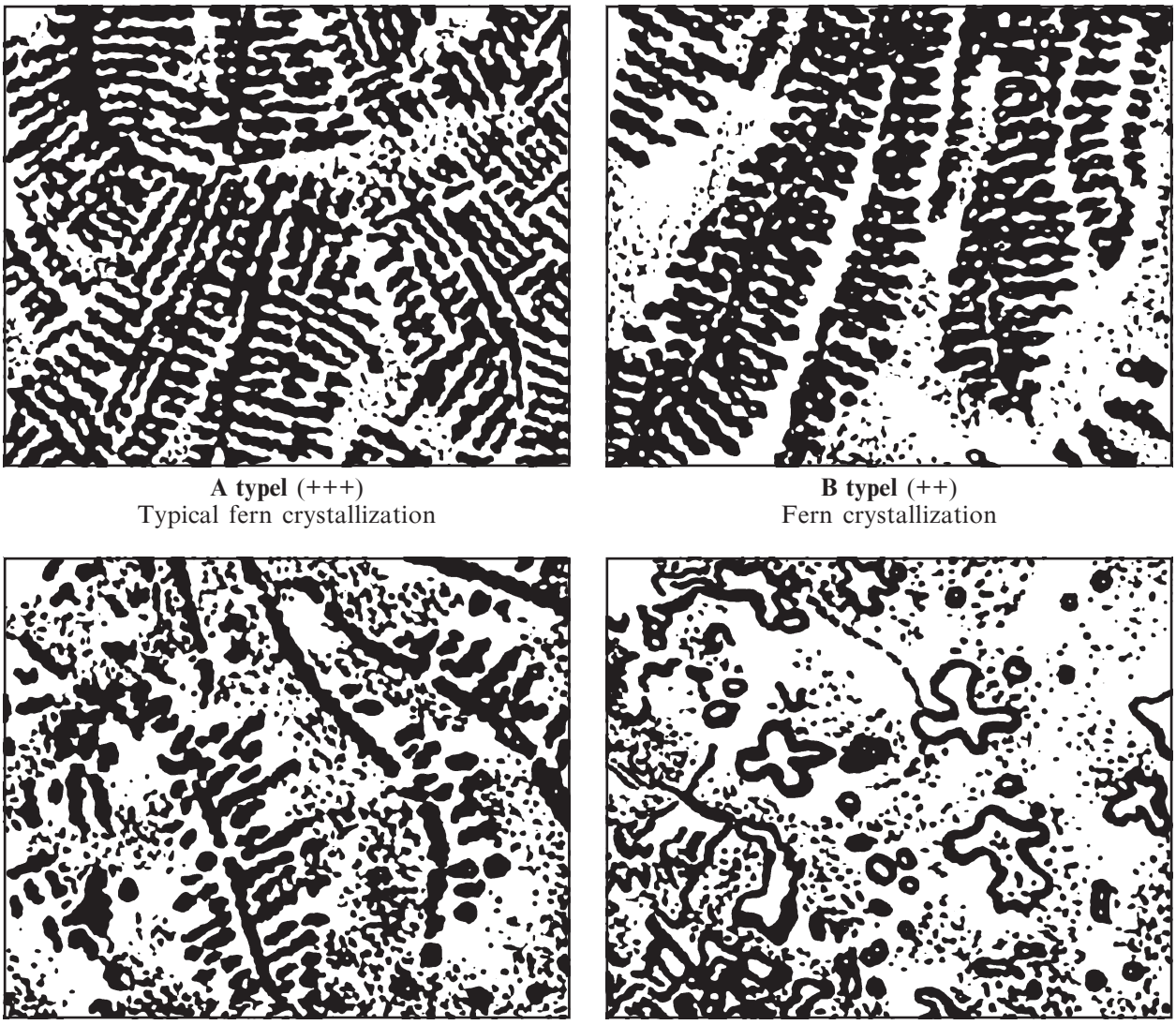
As ovulation approaches, the progesterone receptor content increases. Within two days of ovulation the effect of ovarian production of progesterone becomes apparent as the endometrium enters the *Secretory phase* of the cycle. During this phase the mitotic activity in the epithelium ceases and the glands become dilated and tortuous. The blood vessels become more coiled. Glycogen accumulation in the endometrium reaches a peak under the combined influence of estrogen and progesterone. These processes prepare the endometrium for embedding of the embryo. If fertilisation does not occur, progesterone and estrogen levels decline and menstruation occurs (the endometrium involution) [13, 16].

ANOVULATORY CYCLES

In some instances, ovulation fails to occur during the menstrual cycle. Such anovulatory cycles are common for the first 12–18 months after menarche and again before the onset of menopause. When ovulation does not occur, no corpus luteum is formed, and the effects of progesterone on the endometrium are absent. Estrogens continue to cause growth, however, and the proliferative endometrium becomes thick enough to break down and begin to slough. The time it takes for bleeding to occur is variable, but it usually occurs less than 28 days from the last menstrual period.

CYCLIC CHANGES IN THE UTERINE CERVIX

Although it is contiguous with the body of the uterus, the cervix of the uterus is different in a number of ways. The mucosa of the uterine cervix does not undergo cyclic desquamation, but there are regular changes in the cervical mucus. Estrogen makes the mucus thinner and more alkaline, changes that promote the survival and transport of sperms. Progesterone makes it thick, tenacious, and cellular. The mucus is thinnest at the time of ovulation, and its elasticity, or spinnbarkeit, increases so that by mid cycle, a drop can be stretched into a long, thin thread that may be 8–12 cm or more in length. In addition, it dries in an arborizing, a fern



A typel (+++)
Typical fern crystallization

B typel (++)
Fern crystallization

C typel (+)
Atypical fern crystallization

D typel (-)
Ellipsoid

Fig. 1.6. The cervical mucus smear a progestational effect — fern crystallization

like pattern when a thin layer is spread on a slide (Fig. 1.6) [1, 15].

After ovulation and during pregnancy, it becomes thick and fails to form the fern pattern. It also may provide indirect evidence of ovulation and fertility, however this test does not predict the time of ovulation. The test may provide indirect evidence of ovulation and fertility, however this test does not predict the time of ovulation.

FERTILISATION

Fertilization is the process of fusion of the spermatozoon with the mature ovum (Fig. 1.7, 1.8).

It begins with sperm egg collision and ends with production of a mononucleated single cell called the zygote. Its objectives are:

1. To initiate the embryonic development of the egg.
2. To restore the chromosome number of the species.

Ova are usually fertilized within 12 hours of ovulation. Almost always, fertilization occurs in the ampullary part of the uterine tube. The developing embryo begins to differentiate into the tissue which will become the fetus and that which will form the placenta and fetal membranes. The primitive precursor of the chorionic membrane produces human chorionic gonadotrophin (HCG). HCG has a biological action very similar to LH and takes over its luteinising function.

For the first fourteen days after fertilisation uterine growth and the development of the decidua (the endometrium of pregnancy) are dictated by the corpus luteum under the influence of the pituitary. Thereafter the pituitary LH levels are reduced in response to the increasing levels of HCG.

Under the influence of chorionic gonadotrophin, the corpus luteum continues to grow and to secrete ovarian steroids for the maintenance of uterine growth. HCG levels reach a peak around 10 to 12 weeks and thereafter decline to a lower constant level throughout pregnancy. The response to this reduction is a decrease in ovarian estrogen and progestogen output. As the ovarian contribution to maintaining the pregnancy declines, the placenta increases steroid production. Placental steroid production is impressive and analogues of both hypothalamic and pituitary hormones are produced. The capacity to produce these hormones increases as the early placenta develops.

Of hundreds of millions of sperms deposited in the vagina at single ejaculation, only thousands of capacitated spermatozoa enter the uterine tube while only 300–500 reach the ovum. The tubal transport is facilitated by muscular contraction and aspiration action of the uterine tube. It takes only a few minutes for the sperm to reach the fallopian tube.

DEVELOPMENT OF THE EMBRYO

The differentiation of the embryo itself into those tissues which will become the fetus and those which will form the placenta occurs soon after fertilisation; the fertilised ovum divides repeatedly as it travels through the fallopian tube.

The male and the female pronuclei unite at the center with restoration of the diploid number of chromosomes (46) which is constant for the species. The zygote, thus formed, contains both the paternal and maternal genetic materials. Sex of the child is determined by the pattern of the sex chromosome supplied by the spermatozoon. If the spermatozoon contains an 'X' chromosome, a female embryo (46, XX) is formed; if it contains an 'Y' chromosome, a male embryo (46, XY) is formed.

Following fertilization, cleavage occurs. This consists of a rapid succession of mitotic divisions that produce a mulberry-like mass known as a morula. Fluid is secreted by the outer cells of the morula, and a single fluid filled cavity develops, known as the blastocyst cavity.

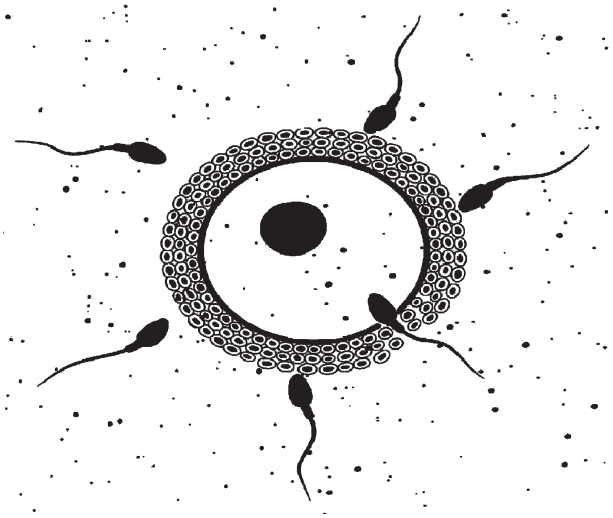


Fig. 1.7

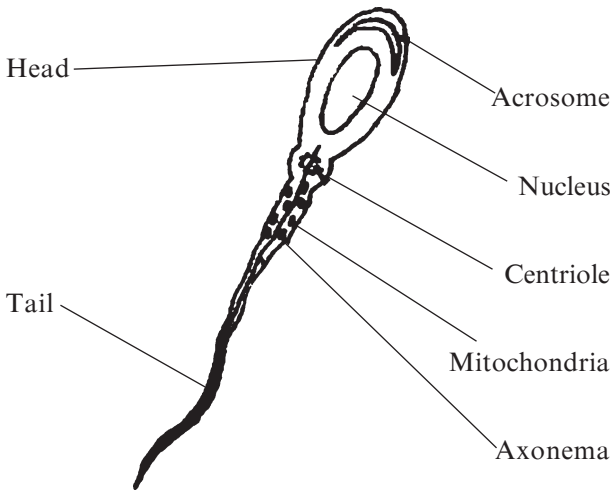


Fig. 1.8. Spermatozoid

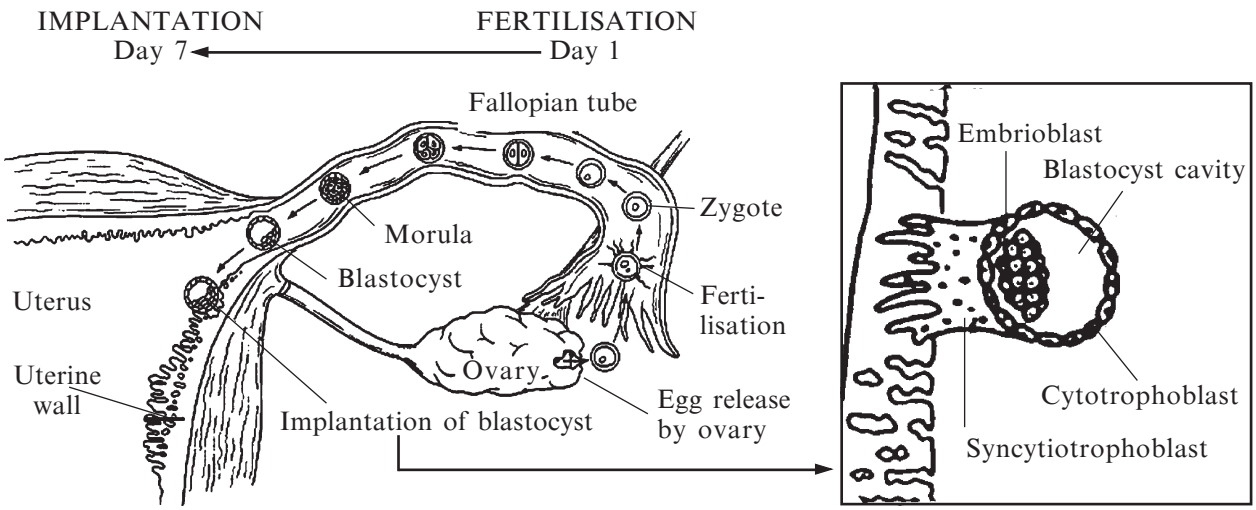


Fig. 1.9

At the so-called blastocyst stage, the cavity is lined predominantly by primitive trophoblast cells. It is these cells which are so active in hormone production in early pregnancy. It is also believed that these cells produce an immunosuppressive protein EPF (Early Pregnancy Factor) which acts to prevent rejection of the antigenically foreign conceptus (Fig. 1.9).

At this stage implantation is required for the zygote to obtain sufficient oxygen and nutrition. The inner cell mass develops into an ectodermal layer and endodermal layer. A mesodermal layer develops between them and grows outwards to form the extra-embryonic mesoderm. Two cavities appear around this stage, the ***yolk sac*** *(vitelline sac)* and the ***amniotic cavity***. The amniotic sac is derived from ectoderm and the yolk sac from the endoderm. At this stage the amniotic cavity is very much smaller [8, 16, 22].

The two cavities, covered by mesoderm, move into the middle of the blastocyst cavity. A mesodermal stalk appears which will eventually form the umbilical cord. The embryonic area, comprising the ectoderm, endoderm and interposed mesoderm will become the fetus.

As development and growth continue the amniotic cavity enlarges to reach the wall of the blastocyst. In doing this, part of the yolk sac becomes enclosed within the embryo whilst the remainder forms a vestigial tube applied to the mesodermal stalk (Fig. 1.10).

Blood vessels develop in the embryonic mesoderm and in the mesoderm of the trophoblast. Extension of these vessels along the connecting stalk results in the formation of the two arteries and single vein of the umbilical cord [33].

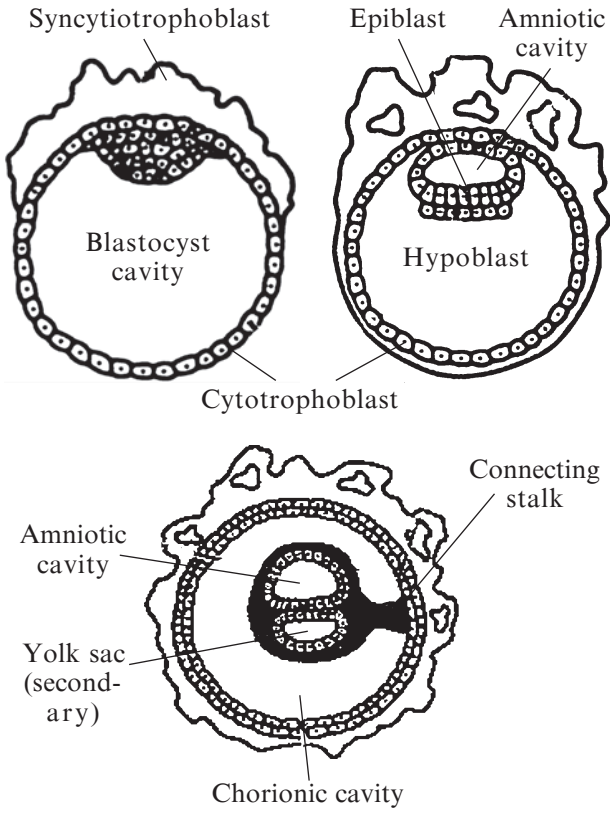


Fig. 1.10

DEVELOPMENT OF INNER CELL MASS

During the embryonic stage which extends from the fourth to eighth week, individual differentiation of the germ layers and formation of the folds of the embryo occur. Most of the tissues and organs develop during this period. The embryo can be differentiated as human at 8th week.

Ectodermal layer: Central and peripheral nervous system, epidermis of skin with its appendages, pituitary gland, chromaffin organs, salivary glands; mucous lining of the nasal cavity, paranasal sinus, roof of the mouth etc. (Fig. 1.11).

Mesodermal layer: Bones, cartilage, muscles, cardiovascular system, kidney, gonads, suprarenals, spleen, most of the genital tract; mesothelial lining of pericardial, pleural and peritoneal cavity etc.

Endodermal layer: Epithelial lining of the gastrointestinal tract, liver, gallbladder, pancreas; epithelial lining of respiratory tract and most of the mucous membrane of urinary bladder and urethra; bulbo-urethral and greater vestibular glands etc.

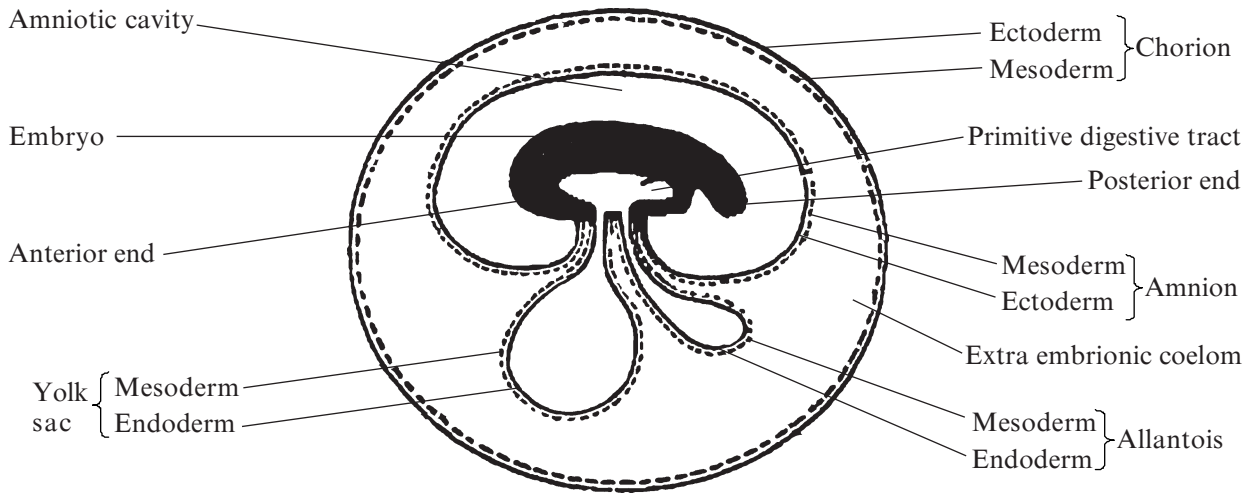


Fig. 1.11

THE PLACENTA AND FETAL MEMBRANES

The placenta is the most important accessory fetal structure and brings the fetal and maternal circulations into close relationship. The human placenta is discoid, because of its shape; hemochorial, because of direct contact of the chorion with the maternal blood and decidua, because some maternal tissue is shed at parturition. The placenta is attached to the uterine wall and establishes connection between the mother and fetus through the umbilical cord (Fig. 1.12). The fact that maternal and fetal tissues come in direct contact without rejection suggest immunological acceptance of the fetal graft by the mother [5, 33].

The placenta is developed from two sources. The principal component is fetal which develops from the chorion frondosum and the maternal component consists of decidua basalis.

When the interstitial implantation is completed on 11th day, the blastocyst is surrounded on all sides by lacunar spaces around cords of syncytial cells, called trabeculae. From the trabeculae develops the stem villi on the 13th day, which connect the chorionic plate with the basal plate. Primary, secondary and tertiary villi are successively developed from the stem villi. Arterio-capillary-venous system in the mesenchymal core of each villus is completed on the 21st day. This ultimately makes connection with the intraembryonic vascular system through the body stalk. Simultaneously, lacunar spaces become confluent with one another and by the 3rd and 4th week, form a multilocular receptacle lined by syncytium and filled with maternal blood. This space becomes the future intervillous space (Fig. 1.13).

As the growth of the embryo proceeds, decidua capsularis becomes thinner beginning at 6th week and both the villi and the lacunar spaces in the abembryonic area get obliterated, converting the chorion into chorion laeve. This is, however, compensated by exuberant growth and proliferation of the decidua basalis and

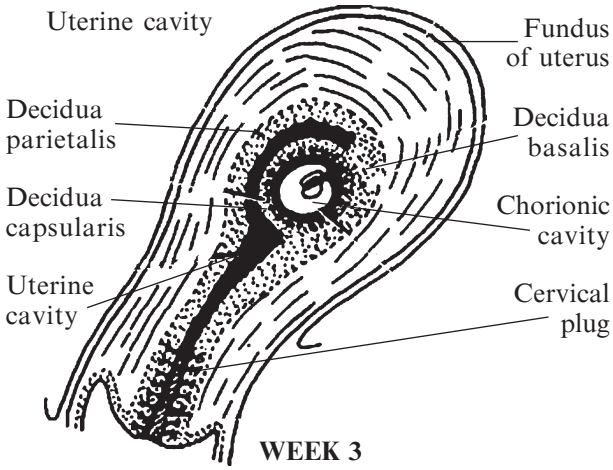


Fig. 1.12

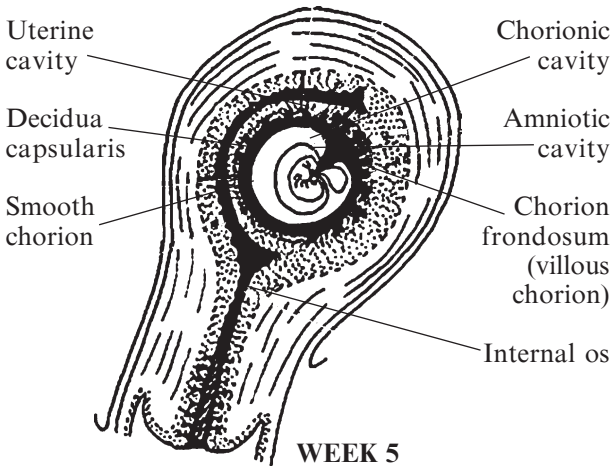


Fig. 1.13

enormous and exuberant division and subdivision of the chorionic villi in the embryonic pole (chorion frondosum). These two, i.e. chorion frondosum and the decidua basalis form the discrete placenta. It begins at 6th week and is completed by the 12th week (Fig. 1.14).

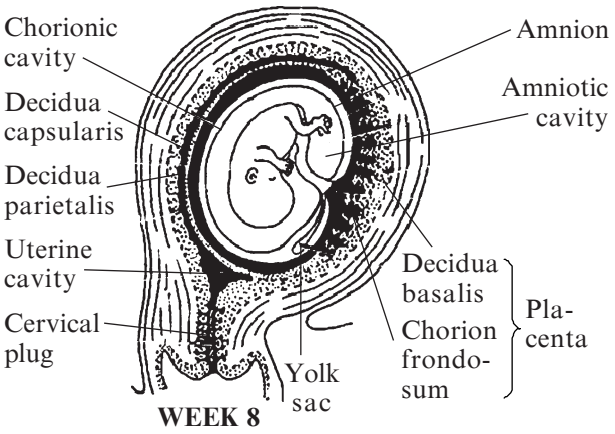


Fig. 1.14

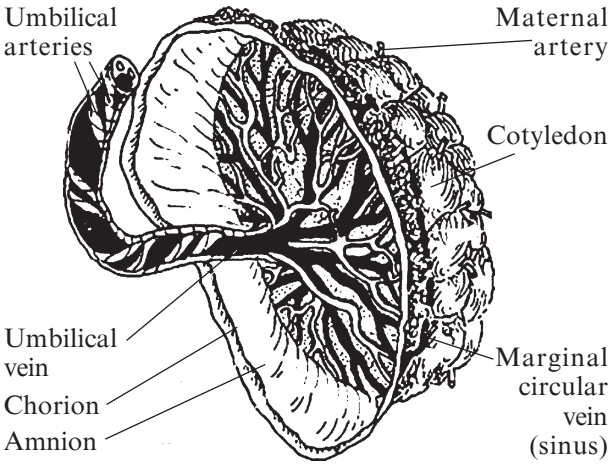


Fig. 1.15. Fetal face of placenta

Until the end of the 16th week, the placenta grows both in thickness and circumference due to growth of the chorionic villi with accompanying expansion of the intervillous space. Subsequently, there is little increase in thickness but it increases circumferentially till term. The human hemochorial placenta derived its name from hemo (blood) that is in contact with the syncytiotrophoblasts of chorionic tissue.

The placenta at term is a disk about 20 cm in diameter, 3 cm thick, and weighs about 500 g, the proportion to the weight of the baby being roughly 1 : 6 at term (a ratio markedly different than this indicates a pathologic condition) and occupies about 30% of the uterine wall. It presents two surfaces, *fetal* and *maternal*, and *a peripheral margin* [31, 33].

FULL-TERM PLACENTA SUMMARY

Fetal surface:

The umbilical cord attaches to this surface. The amniotic covering of the cord is continuous, with the amnion adherent to this surface of the placenta. The vessels radiating from the umbilical cord are clearly seen through the transparent amnion (Fig. 1.15).

The umbilical cord, containing the two umbilical arteries and the single umbilical vein normally attaches to the placenta near its center. Usually they immediately divide to provide a very rich blood supply on the fetal side, which, coupled with the maternal blood in the intervillous spaces, gives it its colour (Fig. 1.16).

The two arteries and single vein of the umbilical circulation lie in a supporting myxomatous tissue derived from the mesoderm and termed 'Wharton's Jelly'. This jelly acts as a buffer, attenuating any pressure on the vessels, resisting occlusion and preventing kinking of the cord.

Maternal surface:

The maternal surface is rough and spongy. Cobblestone appearance caused by 10 to 38 cotyledons separated by grooves formerly occupied by the placental septa. The cotyledon surface is covered by shreds of the decidua basalis (Fig. 1.17). The maternal portion of the placenta amounts to less than one fifth of the total placenta. Only the decidua basalis and the blood in the intervillous space are of maternal origin.

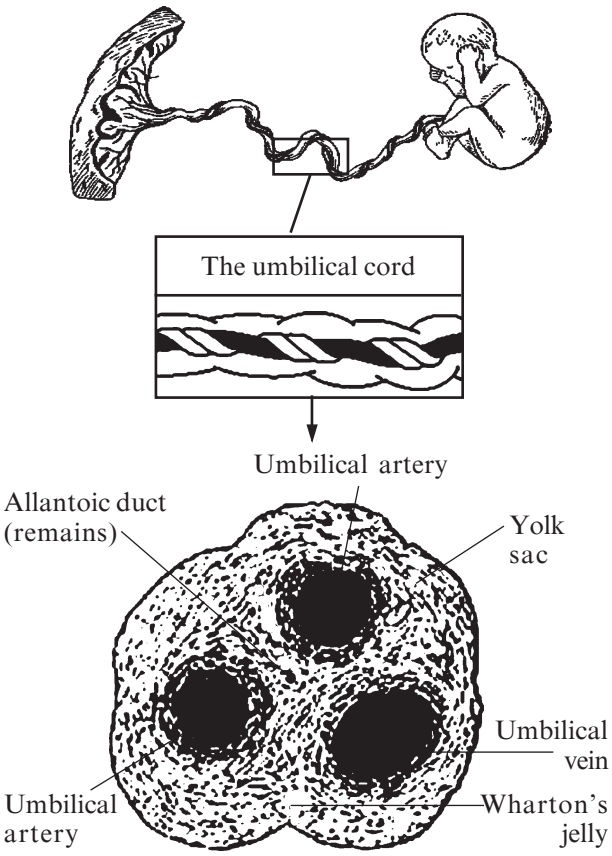


Fig. 1.16

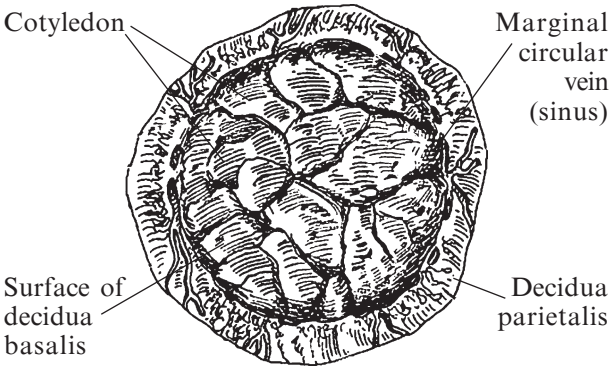


Fig. 1.17. Uterine face of placenta

Margin: Peripheral margin of the placenta is limited by the fused basal and chorionic plates and is continuous with the chorion laeve and amnion. Essentially, the chorion and the placenta are one structure but the placenta is a specialized part of the chorion.

Attachment: The placenta is usually attached to the upper part of the body of the uterus encroaching to the fundus adjacent to the anterior or posterior wall with equal frequency. The attachment to the uterine wall is effective due to anchoring villi connecting the chorionic plate with the basal plate and also by the fused decidua capsularis and vera with the chorion laeve at the margin.

Separation: Placenta separates after the birth of the baby and the line of separation is through the decidua spongiosum.

Examination of the placenta provides information about placental dysfunction, fetal growth, retardation, neonatal illness, and infant death. Retention of a cotyledon in the uterus may lead to late puerperal hemorrhage, but, more often, retained placental tissue is the cause.

STRUCTURE OF PLACENTA

The placenta consists of two plates.

The *chorionic plate* lies internally. It is lined by the *amniotic membrane* (Fig. 1.18). The umbilical cord is attached to this plate. The basal plate lies in the maternal aspect. Between the two plates lies the intervillous space containing the stem villi with their branches, the space being filled with maternal blood.

Amniotic membrane: It consists of single layer of cuboidal epithelium loosely attached to the adjacent chorionic plate. It takes no part in formation of the placenta.

Chorionic plate: From within outwards, it consists of primitive mesenchymal tissue containing branches of umbilical vessels a layer of cytotrophoblast and syncytiotrophoblast. The stem villi arises from the plate. It forms the inner boundary of the choriodecidual space.

Basal plate (it consists of the following structures from outside inwards): Part of the compact and spongy layer of the decidua basalis; nitabuch's layer of fibrinoid degeneration of the outer syncytiotrophoblast at the junction of the cytotrophoblastic shell and decidua; cytotrophoblastic shell; syncytiotrophoblast (Fig. 1.19).

The basal plate is perforated by the spiral branches of the uterine vessels through which the maternal blood flows into the intervillous space. At places, placental or decidual septa project from the basal plate into the intervillous space but fail to reach the chorionic plate. The septum consists of decidual elements covered by trophoblastic cells. The areas between the septa are known as cotyledons (lobes), which are observed from the maternal surface.

Intervillous space: It is bounded on the inner side by the chorionic plate and the outer side by the basal plate, limited on the periphery by the fusion of the two plates. It is lined internally on all sides by the syncytiotrophoblast and is filled with slow flowing maternal blood. Numerous branching villi which arise from the stem villi project into the space and constitute chief content of the intervillous space (Fig. 1.20) [33].

Stem villi arise from the chorionic plate and extend to the basal plate. With the progressive development — primary, secondary and tertiary villi are formed. Functional unit of the placenta is called a fetal cotyledon or placentome, which is derived from a major primary stem villus. These major stem villi pass down through the intervillous space to anchor onto the basal plate. Functional subunit is called a lobule which is derived from a tertiary stem villi. Each cotyledon (totalling 15–29) contains 3–4 major stem vil-

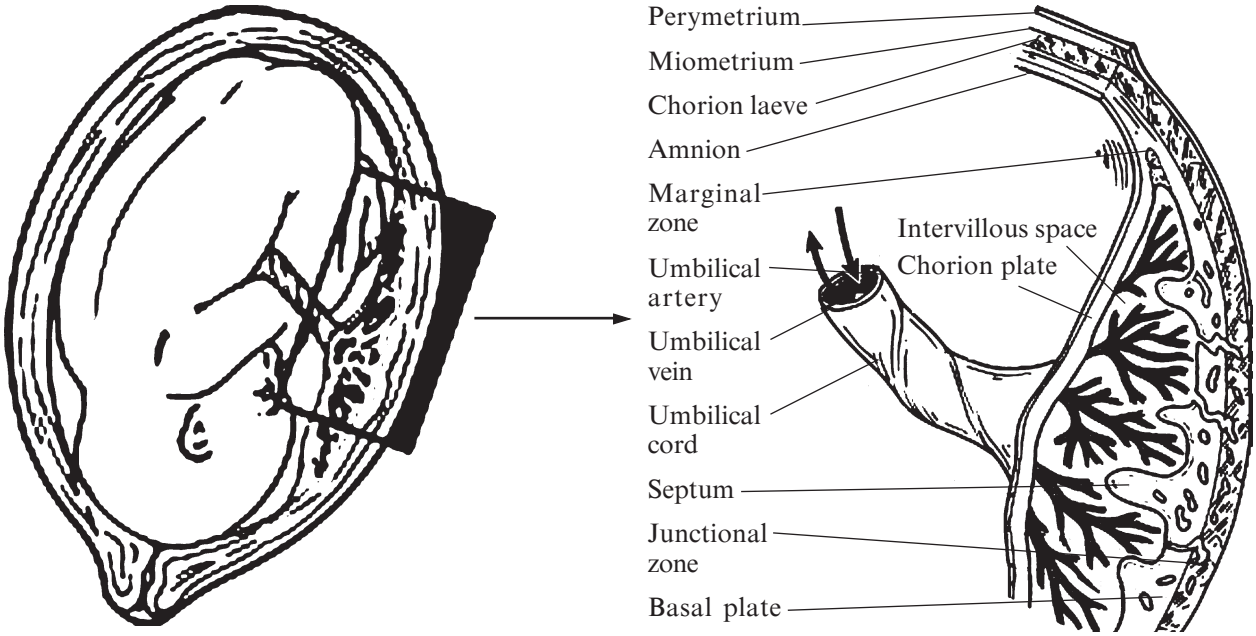


Fig. 1.18

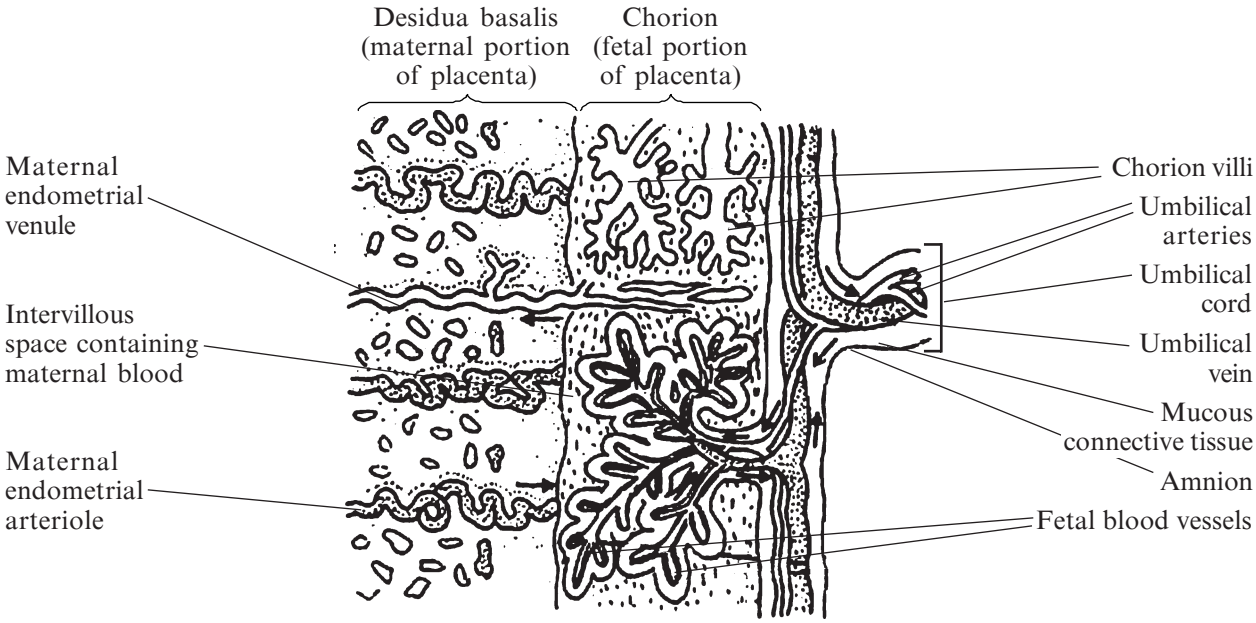


Fig. 1.19. Diagram demonstrates structure of the placenta and umbilical cord

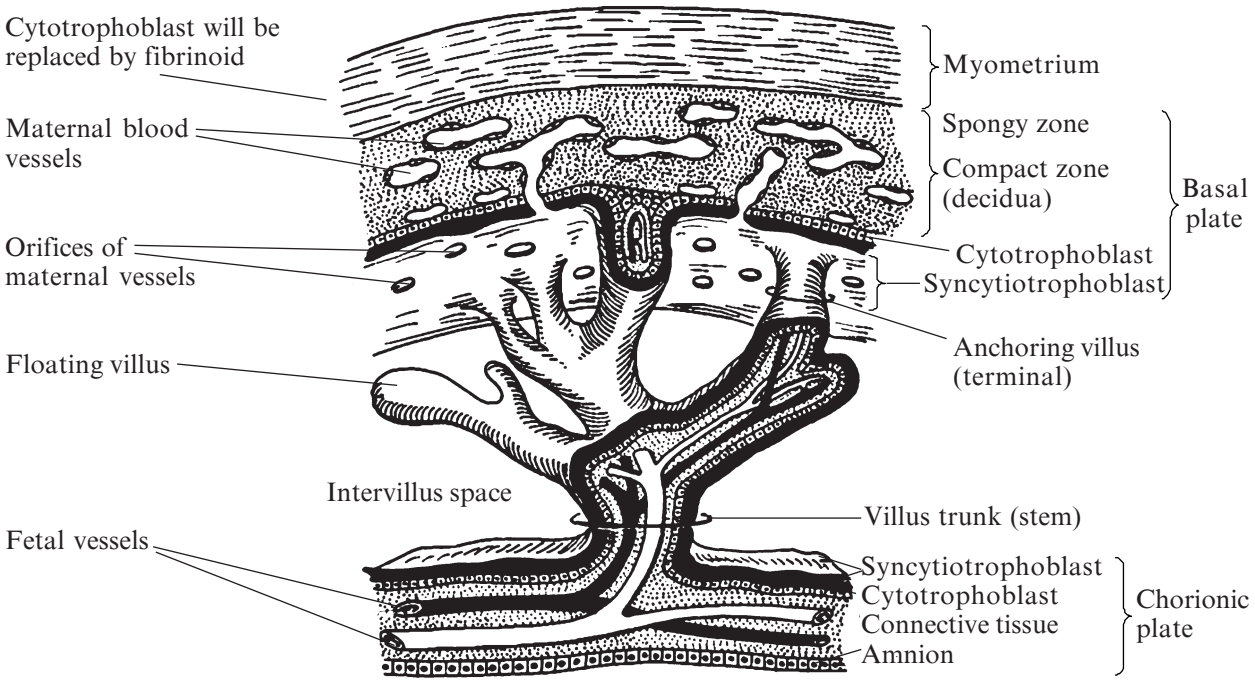


Fig. 1.20

li. The villi are the functional unit of the placenta. While some of the villi are anchoring the placenta to the decidua, the majority are free within the intervillous space and are called nutritive villi. Blood vessels within the branching villi do not anastomose with the neighboring one.

PLACENTAL CIRCULATION

Placental circulation consists of independent circulation of blood in two systems:

— Uteroplacental circulation.

— Fetoplacental circulation.

Uteroplacental circulation (Maternal circulation): It is concerned with the circulation of the maternal blood through the intervillous space. Maternal circulation results from a difference in pressure which is very high in the artery (about 70 mm Hg) and relatively low in the intervillous space (about 10 mm Hg). Blood spurts up to the chorionic plate, then comes toward the basal plate and is taken up by the uterine veins where the pressure is even lower than that found in the intervillous space (Fig. 1.21).

Arterial circulation: About 120–200 spiral arteries open into the intervillous space by piercing the basal plate randomly at numerous sites. Normally,

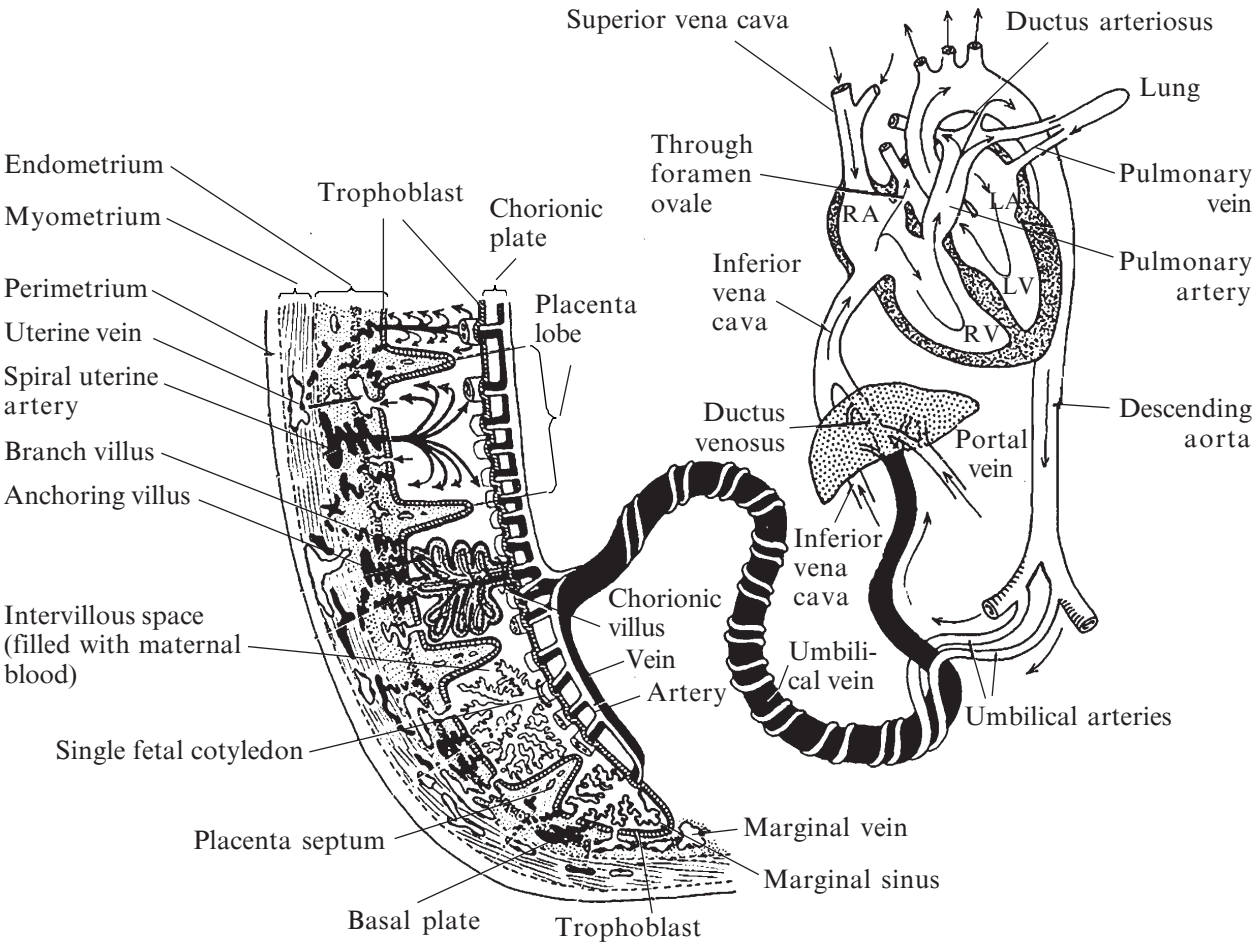


Fig. 1.21

there is cytotrophoblastic invasion into the spiral arteries initially upto the intra-decidual portion within 12 weeks of pregnancy. Not only the endothelial lining is replaced but also the muscular elastic media is destroyed and replaced by fibrinoid material. There is a secondary invasion of trophoblast between 12–16 weeks extending up to radial arteries within the myometrium (Fig. 1.22).

So, spiral arteries are converted to large bore uteroplacental arteries. The net effect is funnelling of the arteries which reduces the pressure of the blood to 70–80 mm Hg before it reaches the intervillous space. So, it increases the blood flow.

Trophoblast cells that do not take part in villous structure are known as extravillous trophoblast (EVT). EVT are of two types: endovascular that migrates down the lumen of the spiral arteries and replaces the endothelium (Fig. 1.23).

Interstitial that invades as far as the inner third of the myometrium. Further invasion is limited by the natural killer cells to prevent morbid adhesion of placenta (placenta accreta). Defects in trophoblast invasion and

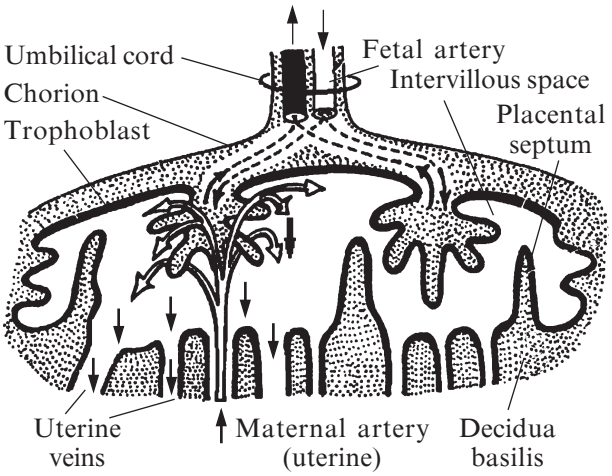


Fig. 1.22

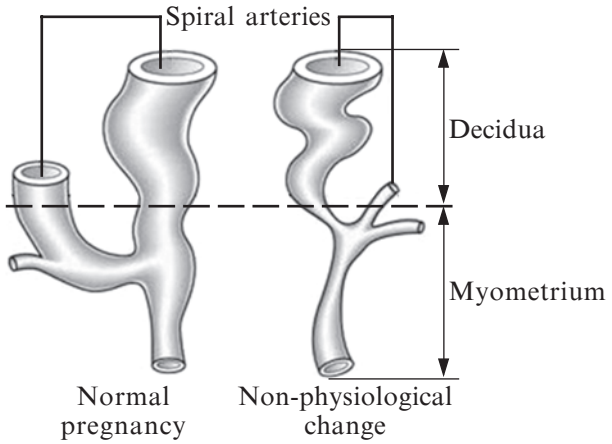


Fig. 1.23

failure to establish maternal circulation correctly leads to complications of pregnancy (PIH, IUGR).

Venous drainage: The venous blood of the intervillous space drains through the uterine veins which pierce the basal plate randomly like the arteries [9, 33].

Circulation in the intervillous space: the arterial blood enters the space under pressure. Lateral dispersion occurs, after it reaches the chorionic plate. Villi help in mixing and slowing the blood flow. Mild stirring effect by the villi pulsation aided by uterine contraction help migration of the blood toward the basal plate and thence to the uterine veins. During uterine contraction, the veins are occluded, but the arterial blood is forced into the intervillous space; while uterine relaxation facilitates venous drainage. This is brought about by the fact that the spiral arteries are perpendicular and the veins are parallel to the uterine wall. Thus during contraction, a greater volume of blood is available for exchange even though the rate of flow is decreased. The blood in the intervillous space is protected from clotting by some fibrinolytic enzyme activity of the trophoblast.

The fetal circulation can be compared to the pulmonary circulation of the adult in that desaturated blood enters through the fetal arteries and oxygenated blood returns by way of the veins.

Blood arrives via the 2 umbilical arteries which are branches of the iliac arteries of the fetus. It is dispersed in a highly dense network which penetrates even the smallest villous division. Blood is returned via the umbilical vein and finally reaches the inferior vena cava system of the fetus. Fetal circulation is carried out in a closed vascular system where the average pressure is about 30 mm Hg, which is much higher than that seen in the intervillous space where it is about 10 mm Hg. The difference in pressure prevents the collapse of the villous vessels.

PLACENTAL FUNCTION

The main functions of the placenta are: transfer of nutrients and waste products between the mother and fetus, barrier and immunological function, endocrine function.

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

Respiratory function. Although the fetal respiratory movements are observed as early as 11 weeks, there is no gaseous exchange. Intake of oxygen and output of carbon dioxide take place by simple diffusion across the fetal membrane.

Excretory function. Waste products from the fetus such as urea, uric acid, and creatinine are excreted to the maternal blood by simple diffusion.

Nutritive function. The fetus obtains its nutrients from the maternal blood and when the diet is inadequate, then only, depletion of maternal tissue storage occurs.

Barrier function. Fetal membrane has long been considered as a protective barrier to the fetus against noxious agents circulating in the maternal blood. The race of drug transfer is increased in late pregnancy. Maternal infection during pregnancy by virus (rubella, chickenpox, measles, mumps, poliomyelitis), bacteria *(Treponema pallidum, Tubercle bacillus)* or protozoa (*Toxoplasma gondii*, malaria parasites) may be transmitted to the fetus across the so called placental barrier and affect the fetus in utero. Similarly, almost any drug used in pregnancy can cross the placental barrier and may have deleterious action on the fetus.

Immunological function. The fetus and the placenta contain paternally determined antigens, which are foreign to the mother. In spite of this, there is no evidence of graft rejection. Placenta offers immunological protection against rejection. Placental barrier and may have deleterious action on the fetus.

Endocrine function. A large number of hormones are produced by the placenta. They include hormones analogous to adult hypothalamic and pituitary hormones and steroid hormones.

A variety of other products are produced by the placenta. Many of these are glycoproteins such as Pregnancy Associated Proteins A to D, Pregnancy Specific Glycoprotein (SP1) and Placental Protein 5 (PP5).

Hormone	Properties
Human Chorionic Somatomammotropin →	Similar to growth hormone and prolactin
Human Chorionic Gonadotrophin →	Stimulates adrenal and placental steroidogenesis. Analogous to LH
Human Chorionic Thyrotropin →	Analogous to Thyrotropin
Corticotrophin Releasing Hormone →	Without CRH, tlie embryo may be rejected by the mother, resulting in a miscarriage (spontaneous abortion)
Estrogen →	Complex. Stimulates uterine blood flow and growth
Progestogens →	Enables implantation and relaxes smooth muscle
Adrenocorticoids →	Induction of fetal enzyme systems and fetal maturity

Fig. 1.24

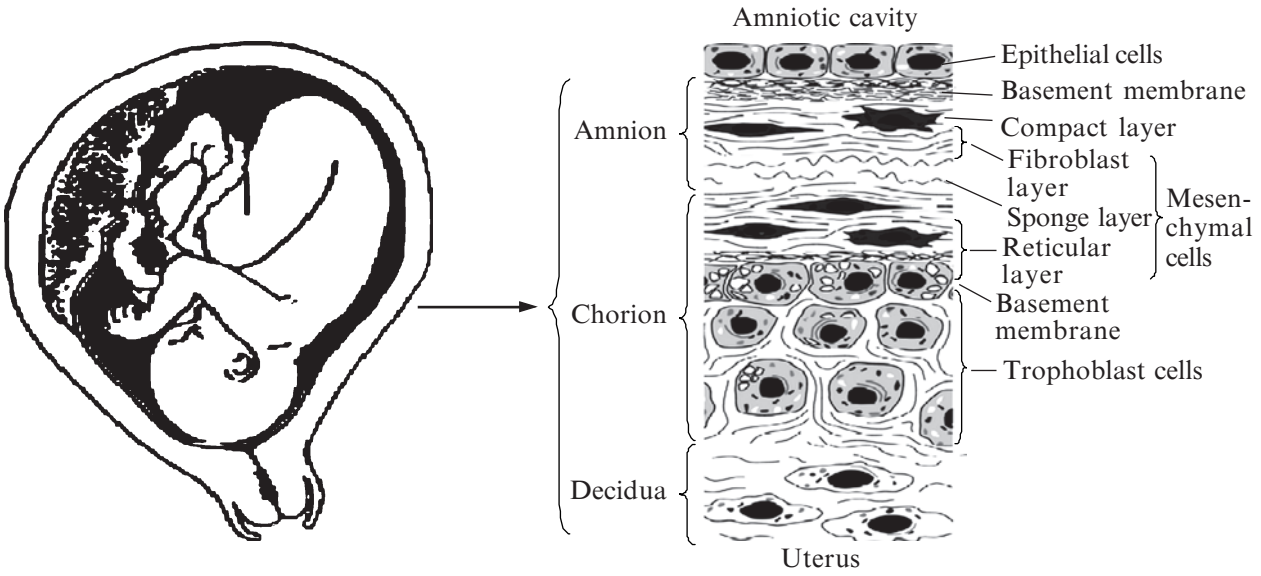


Fig. 1.25

Some products of placental and fetal metabolism may be used to screen for fetal disease. Measurement of alpha fetoprotein, produced by the fetal liver, gut and yolk sac, may be used to screen for a number of anatomical abnormalities. Together with measurement of maternal serum HCG the risk of the fetus having a trisomy may also be calculated (Fig. 1.24) [10, 23].

AMNIOTIC CAVITY, AMNION AND AMNIOTIC FLUID

Amnion is the inner layer of the fetal membranes. Its internal surface is smooth and shiny and is in contact with liquor amnii. The outer surface consists of a layer of connective tissue and is opposed to the similar tissue on the inner aspect of the chorion from which it can be peeled off. The normal amniotic fluid volume increases from around 250 ml at 16 weeks gestation to 800 ml around 38 weeks (Fig. 1.25).

Fully formed amnion is 0.02–0.5 mm in thickness. The amnion has got neither blood nor nerve supply nor any lymphatic system.

In early pregnancy, it is colorless but near term it becomes pale straw colored due to the presence of exfoliated lanugo and epidermal cells from the fetal skin. It may look turbid due to the presence of vernix caseosa. Study of the amniotic fluid provides useful information about the well being and also maturity of the fetus.

Its main function is to protect the fetus. Its resilience to rupture is vitally important to successful pregnancy outcome. Indeed, preterm rupture of fetal membranes is a major cause of preterm delivery.

Chapter 2
MATERNAL PHYSIOLOGY DURING PREGNANCY

Pregnancy involves a number of changes in anatomy, physiology, and biochemistry, which can challenge maternal reserves. Maternal changes start from very early on in the first trimester of the pregnancy. A basic knowledge of these adaptations is critical for understanding normal laboratory measurements.

CARDIOVASCULAR SYSTEM

Anatomic Changes

With uterine enlargement and diaphragmatic elevation, the heart rotates on its long axis in a left-upward displacement. As a result of these changes, the apical beat (point of maximum intensity) shifts laterally. Overall, the heart size increases by about 12%, which results from both an increase in myocardial mass and intracardiac volume. Vascular changes include hypertrophy of smooth muscle and a reduction in collagen content.

CARDIAC OUTPUT

Cardiac output increases approximately 40% during pregnancy, with maximum values achieved at 20–24 weeks' gestation. This rise in cardiac output is thought of as a result of the hormonal changes in the pregnancy as well as the arteriovenous-shunt effect of uteroplacental circulation (Fig. 2.1).

Blood Volume

Blood volume expansion begins early in the first trimester, increases rapidly in the second trimester, and plateaus at about the 30th week. The approximately 50% elevation in plasma volume, which accounts for most of the increment, results from a cascade of effects triggered by pregnancy hormones. For example, increased estrogen production by the placenta stimulates the renin-angiotensin system, which, in turn, leads to higher circulating levels of aldosterone. Aldosterone promotes renal Na^+ reabsorption and water retention. Progesterone also participates in plasma volume expansion through a poorly understood mechanism; increased venous capacitance is another important factor. Human chorionic somatomammotropin, progesterone, and perhaps other hormones promote erythropoiesis, resulting in about 30% increase in red cell mass. The magnitude of the increase in blood volume varies according to the size of the woman, the number of prior pregnancies, and the number of fetuses she is carrying. This hypervolemia of pregnancy compensates for maternal blood loss at delivery, which averages 500–600 ml for vaginal and 1000 ml for cesarean delivery.

Local vascular changes

Local changes are most apparent in the lower limbs and are due to pressure exerted by the enlarging uterus on the pelvic veins. Since one third of the total circulating blood is distributed to the lower limbs the increased venous pressure may produce varicosities and oedema of the vulva and legs. These changes are most marked during the daytime due to the upright posture. They tend to be reversed at night when the woman retires to bed: oedema fluid is reabsorbed, venous return increased and renal output rises, resulting in nocturnal frequency. If the patient adopts the supine position, however, the uterine pressure on the veins increases and this may lead to reduced venous return to the heart. This in turn leads to a reduction in cardiac output (Fig. 2.2).

An extreme example of this occurs when the uterus compresses the vena cava (the supine position) and reduces cardiac output to the point where the

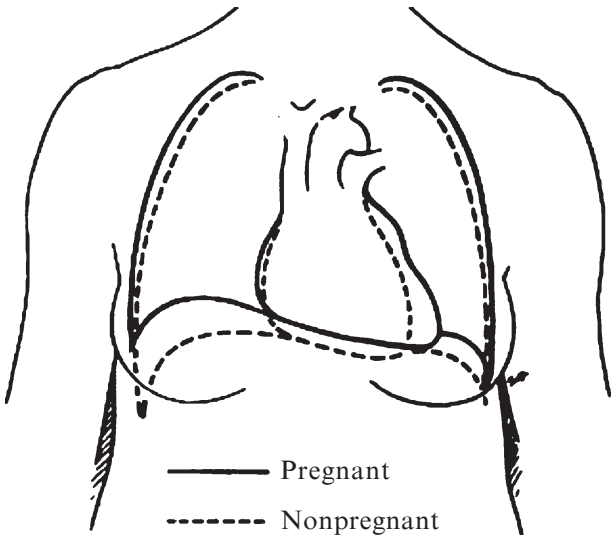


Fig. 2.1

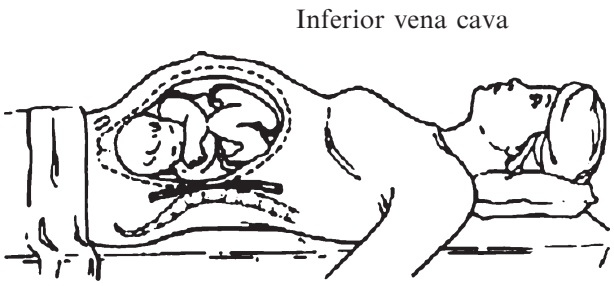


Fig. 2.2. The gravid uterus compresses the vena cava in supine position

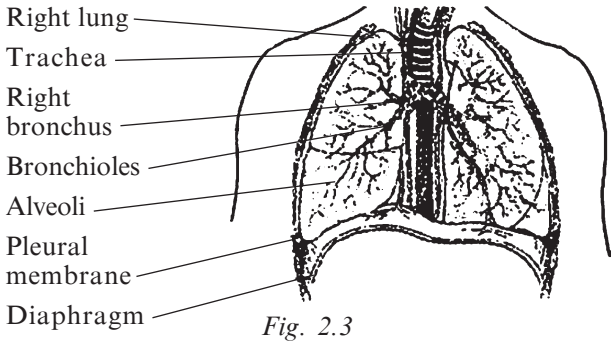


Fig. 2.3

mother feels faint and may become unconscious. A sensation of nausea also occurs and vomiting may result. This condition, *supine hypotension syndrome*, should be borne in mind when examining women in late pregnancy [11, 18].

HAEMATOLOGICAL CHANGES

The increasing plasma volume, however, produces an apparent reduction in hemoglobin. The hemoglobin concentration falls throughout pregnancy until the last four weeks when there might be a slight rise. The fall is apparent by the 12th week of pregnancy and the minimum value is reached at 32 weeks.

Leukocytes. There is a marked increase in white cells during pregnancy. The increase is almost entirely due to an increase in neutrophil polymorphonuclear cells. The white cell count may rise markedly during labor. *Platelets* decline progressively through pregnancy. The mean platelet size increases slightly and the lifespan of platelets is shortened.

Clotting Factors

Pregnancy is a hypercoagulable state. This is a protective mechanism for the mother to be able to minimise blood loss after delivery of the placenta. There is an increase in fibrinogen, factor VIII and von Willebrand factor, and a decrease in anticoagulants such as antithrombin III. There is also an increase in fibrinolytic activity. While this hypercoagulable state is protective against bleeding, it results in an increased risk of thrombosis during pregnancy, especially when there are other risk factors present (e.g. increasing age, smoking, high BMI, operative delivery, prolonged immobilisation).

PULMONARY SYSTEM

The changes that occur in the respiratory system during pregnancy are necessitated by the increased oxygen demand of the mother and fetus. These changes are primarily mediated by progesterone (Fig. 2.3).

ANATOMIC CHANGES

As the uterus enlarges, the diaphragm is elevated by as much as 4 cm.

The rib cage is displaced upward, increasing the angle of the ribs with the spine. These changes increase the lower thoracic diameter by about 2 cm and the thoracic circumference by up to 6 cm. Elevation of the diaphragm does not impair its function. Abdominal muscles have less tone and activity during pregnancy, causing respiration to be more diaphragm dependent (Fig. 2.4).

RESPIRATION

Pregnancy has little effect on respiratory rate. Pregnancy is associated with an increase in total body oxygen consumption. Approximately 50% of this increase is consumed by the gravid uterus and its contents, 30% by the heart and kidneys, 18% by the respiratory muscles, and the remainder by the mammary tissues. Functional adaptations in the pulmonary system enhance oxygen delivery to the lungs. The consequence of diaphragmatic elevation is a 20% reduction in the residual volume and functional residual capacity plus a 5% reduction in total lung volume. Although the maternal respiratory rate is essentially unchanged, there is a 30% to 40% increase in tidal volume due to a 5% increase in inspiratory capacity, resulting in a 30% to 40% increase in minute ventilation.

Functional measurement of ventilation can also change according to posture and duration of pregnancy. For example, the peak expiratory rate, which declines throughout gestation in the sitting and standing positions, is particularly compromised in the supine position.

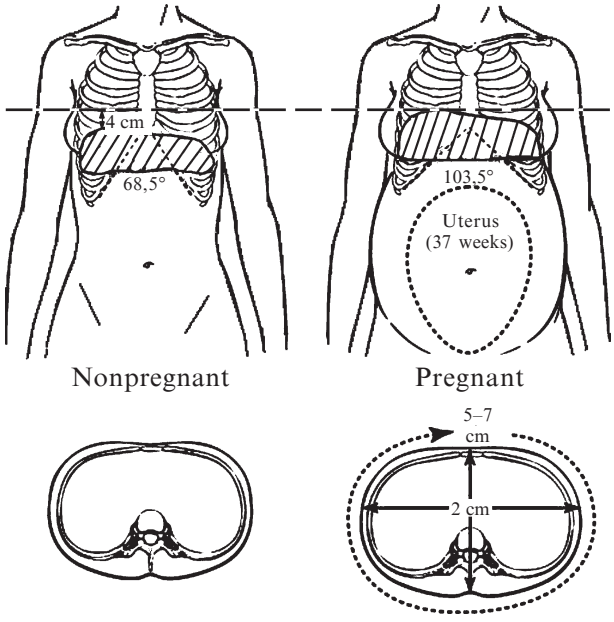


Fig. 2.4

RENAL SYSTEM

ANATOMIC CHANGES

During pregnancy, the length of the kidneys increases by 1–1.5 cm, with a proportional increase in weight. The renal calyces and pelvis are dilated in pregnancy, with the volume of the renal pelvis increased up to 6-fold compared to the nonpregnant value of 10 ml. The ureters are dilated above the brim of the bony pelvis, with more prominent effects on the right. The ureters elongate, widen, and become more curved. The entire dilated collecting system may contain up to 200 ml of urine, which predisposes to ascending urinary infections. Urinary tract dilatation disappears in virtually all women by postpartum day 4 (Fig. 2.5).

RENAL FUNCTION

Renal plasma flow increases 50–85% above nonpregnant values during the first half of pregnancy, with a modest decrease in later gestation. The changes in renal plasma flow reflect decreases in renal vascular resistance, which achieves lowest values by the end of the first trimester. Elevated renal perfusion is the principal factor involved in the rise in glomerular filtration rate (GFR), which increased by approximately 25% in the second week after conception. GFR reaches a peak increment of 40–65% by the end of the first trimester and remains high until term. Increased GFR with saturation of tubular resorption capacity for filtered glucose can result in glucosuria. In fact, more than 50% of women have glucosuria sometime during pregnancy. Increased urinary glucose levels contribute to increased susceptibility of pregnant women to urinary tract infection.

Urinary protein loss normally does not exceed 300 mg over 24 hours, which is similar to the nonpregnant state. Thus, proteinuria of more than 300 mg over 24 hours suggests a renal disorder.

BLADDER

As the uterus enlarges, the urinary bladder is displaced upward and flattened in the anteroposterior diameter. One of the earliest symptoms of pregnancy is increased urinary frequency, which may be related to pregnancy hormones. In later gestation, mechanical effects of the enlarged uterus may contribute to the increased frequency. Bladder vascularity increases and muscle tone decreases, which increases bladder capacity up to 1500 ml.

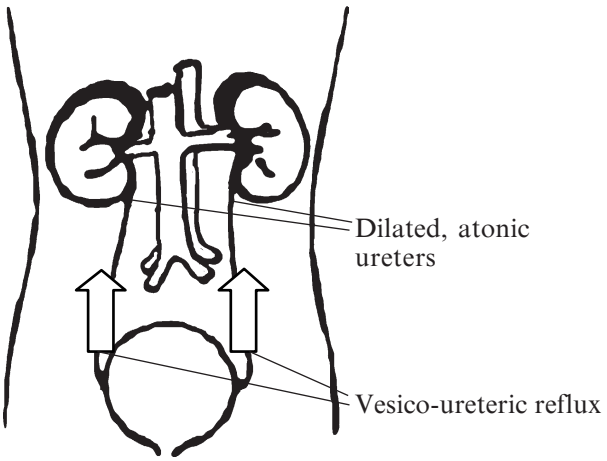


Fig. 2.5

GASTROINTESTINAL SYSTEM

ANATOMIC CHANGES

As the uterus grows, the stomach is pushed upward. The appendix is displaced superiorly in the right flank area. These organs return to their normal positions in the early puerperium.

ESOPHAGUS AND STOMACH

Reflux symptoms (heartburn) affect 30–80% of pregnant women. Gastric production of hydrochloric acid is variable and sometimes exaggerated but more commonly reduced. Pregnancy is associated with greater production of gastrin, which increases stomach volume and acidity of gastric secretions. Gastric production of mucus also may be increased. Esophageal peristalsis is decreased. Most women first report symptoms of reflux in the first trimester. Decreased motility, increased acidity of gastric secretions, and reduced function of the lower esophageal sphincter contribute to the increased gastric reflux (Fig. 2.6) [7].

INTESTINES

Intestinal transit times are decreased in the second and third trimesters, whereas first trimester and postpartum transit times are similar. Transit times return to normal within 2 to 4 days postpartum. Constipation is common in pregnancy and is associated with mechanical obstruction of the colon by the enlarging bowel, reduced motility as elsewhere in the gastrointestinal tract, and increased water absorption during pregnancy.

Generalized pruritus may result from intrahepatic cholestasis and increased serum bile acid concentrations.

GALLBLADDER

The emptying of the gallbladder is slowed in pregnancy and often incomplete. Bile stasis of pregnancy increases the risk for gallstone formation, al-

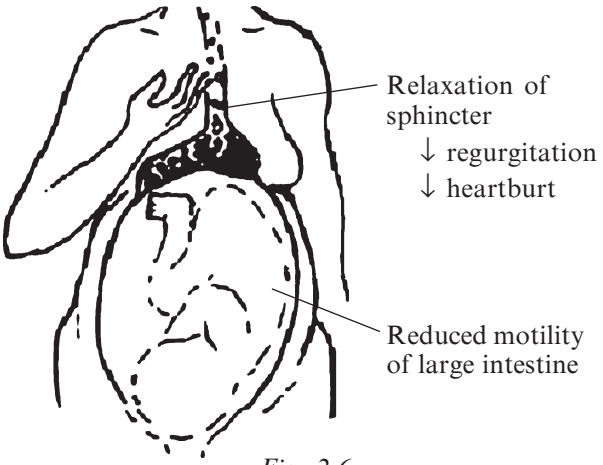


Fig. 2.6

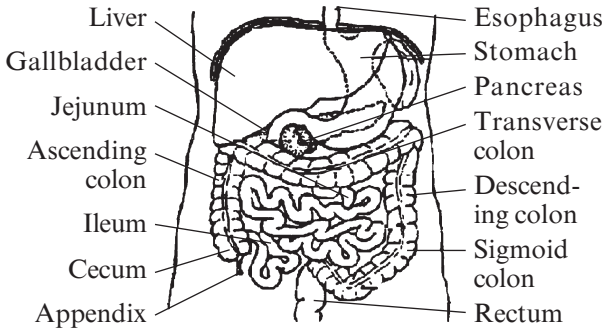


Fig. 2.7

though the chemical composition of bile is not appreciably altered (Fig. 2.7).

LIVER

Liver morphology does not change in normal pregnancy. Plasma albumin levels are reduced to a greater extent than the slight decrease in plasma globulins. This fall in the albumin/globulin ratio mimics liver disease in nonpregnant individuals. Serum alkaline phosphatase activity can double as the result of alkaline phosphatase isozymes produced by the placenta.

ENDOCRINE CHANGES IN PREGNANCY

The following is a summary of the changes in maternal and placental hormones in pregnancy and their supposed effects.

Progesterone is produced by the corpus luteum in the first few weeks of pregnancy. Thereafter it is derived from the placenta. Levels rise steadily during pregnancy with, it has been suggested, a fall towards term. Output reaches a maximum of at least 250 mg per day.

Possible actions:

1. Reduces smooth muscle tone — stomach motility diminishes — may induce nausea. Colonic activity reduced — delayed emptying — increased water reabsorption — constipation. Reduced uterine tone — diminished uterine activity — reduced bladder and ureteric tone — stasis of urine.
2. Reduces vascular tone — diastolic pressure reduced — venous dilatation.
3. Raises temperature.
4. Increases fat storage.
5. Induces over-breathing — alveolar and arterial carbon dioxide tension reduced.
6. Induces development of breasts.

Estrogens. In early pregnancy the source is the ovary. Later estrone and estradiol are probably produced by the placenta and are increased a hundredfold. Estriol, however, is a product of the interaction of the placenta and the fetal adrenals and is increased one thousandfold. The output of estrogens reaches a maximum of at least 30 to 40 mg per day. Estriol accounts for 85% of this total. Levels increase up to term.

Possible actions:

1. Induce growth of uterus and control its function.
2. Responsible, together with progesterone, for the development of the breasts.
3. Alter the chemical constitution of connective tissue, making it more pliable — stretching of cervix possible, joint capsules relax, pelvic joints mobile.
4. Cause water retention.
5. May reduce sodium excretion.

Cortisol. The maternal adrenals are the sole source in early pregnancy but later considerable quantities are thought to be produced by the placenta. Some 25 mg are produced each day. Much of this is protein bound and therefore may not be generally active.

Possible actions:

1. Increases blood sugar.
2. Modifies antibody activity.

Aldosterone is almost certainly wholly derived from the maternal adrenals. The amounts produced during pregnancy are much increased. It promotes the retention of sodium and water.

Renin. Plasma renin activity is five to ten times that in the non-pregnant state. Likewise angiotensinogen levels are increased although in normal pregnancy women show a reduced sensitivity to the hypertensive effects of angiotensin.

Human chorionic gonadotrophin (HCG) is produced by the trophoblast and peak levels are reached before 16 weeks of gestation. From 18 weeks onwards, levels remain relatively constant. Apart from the early maintenance of the corpus luteum, the physiological role of HCG remains unclear. It appears to have a thyrotrophic action and to initiate testosterone secretion from the Leydig cells.

Human placental lactogen (HPL). Levels of HPL (or chorionic somatomammotrophin) rise steadily with the growth of the placenta throughout pregnancy. It is lactogenic and antagonistic to insulin.

Relaxin is a hormone produced by the corpus luteum. It can be detected throughout pregnancy but highest levels are in the first trimester. Its physiological role is uncertain but it has been used clinically in cervical ripening.

Pituitary hormones. Maternal FSH and LH levels are suppressed during pregnancy but prolactin levels rise throughout. Lactation does not start, however, until delivery when high prolactin levels persist in association with falling oestrogen levels.

Pregnancy induces several characteristic changes in the appearance of the maternal skin. Although the exact etiology of these changes has not been established, hormonal influences appear to predominate.

SKIN

Anatomic Changes

Hyperpigmentation is one of the well-recognized skin changes of pregnancy, which is manifested in the linea nigra and melasma, the *mask of pregnancy*. The latter, which is exacerbated by sun exposure, develops in up to 70% of pregnancies and is characterised by an uneven darkening of the skin in the centrofacial-molar area. The hyperpigmentation is probably because of the elevated concentrations of

Fig. 2.8

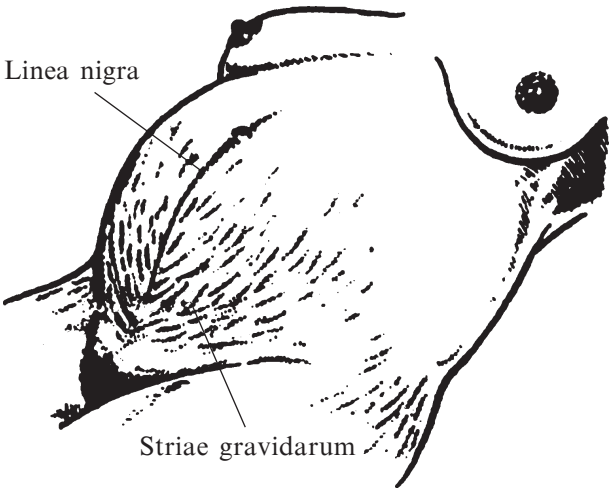


Fig. 2.9

melanocyte-stimulating hormone and/or estrogen and progesterone effects on the skin. Similar hyperpigmentation of the face can be seen in nonpregnant women who are taking oral contraceptives (Fig. 2.8).

Striae gravidarum consist of bands or lines of thickened, hyperemic skin. These "stretch marks" begin to appear in the second trimester on the abdomen, breasts, thighs, and buttocks. Decreased collagen adhesiveness and increased ground substance formation are characteristically seen in this skin condition. A genetic predisposition appears to be involved because not every gravida develops these skin changes. Effective treatment (preventive or therapeutic) has yet to be found (Fig. 2.9).

Other common cutaneous changes include spider angiomas, palmar erythema, and cutis marmorata (mottled appearance of skin secondary to vasomotor instability). The development or worsening of varicosities accompanies nearly 40% of pregnancies. Compression of the vena cava by the gravid uterus increases venous pressures in the lower extremities, which dilates veins in the legs, anus (hemorrhoids), and vulva.

The nails and hair also undergo changes. Nails become brittle and can show horizontal grooves. Thickening of the hair during pregnancy is caused by an increased number of follicles in anagen (growth) phase, and generalized hirsutism can worsen in women who already have hair that is thick or has a male pattern of distribution. The thickening of the hair ends 1 to 5 months postpartum with the onset of the telogen (resting) phase, which results in excessive shedding and thinning of hair. Normal hair growth returns within 12 months [23, 30].

Metabolism

Pregnancy increases nutritional requirements, and several maternal alterations occur to meet this demand. Pregnant women tend to rest more often, which conserves energy and thereby enhances fetal nutrition. The maternal appetite and food intake usually increase. In rare instances, women with pica may crave substances such as clay, cornstarch, soap, or even coal (Fig. 2.10).

Pregnancy is associated with profound changes in structure and metabolism.

The most obvious physical changes are weight gain and altered body shape. Weight gain results not only from the uterus and its contents, but also from increased breast tissue, blood volume, and water volume (about 6.8 l) in the form of extravascular and extracellular fluid. Deposition of fat and protein and increased cellular water are added to maternal stores. The average weight gain during pregnancy is 12.5 kg (Fig. 2.11).

Carbohydrate metabolism

Pregnancy has a diabetogenic effect on maternal carbohydrate metabolism, characterised by reduced

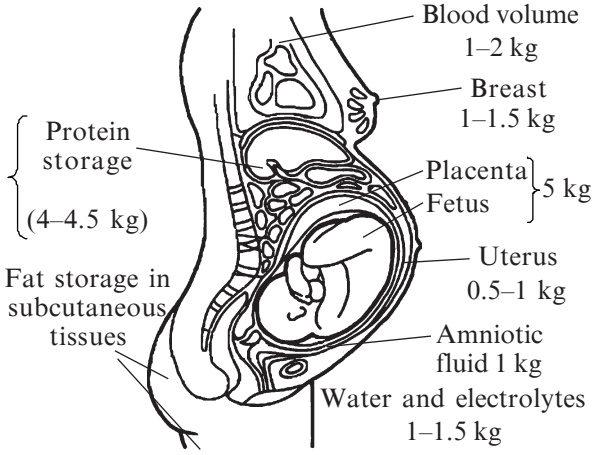


Fig. 2.10

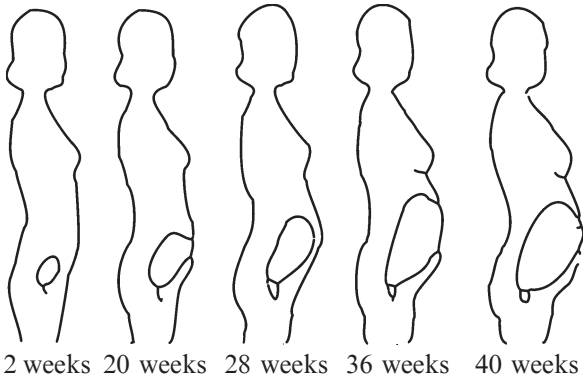


Fig. 2.11

tissue response to insulin, hyperinsulinemia, and hyperglycemia. Insulin resistance is primarily due to the action of hPL, which increases the resistance of peripheral tissues to the effects of insulin. The hormone hPL is secreted in proportion to placental mass, resulting in increased insulin resistance as pregnancy progresses. Progesterone and estrogen may also contribute to insulin resistance. Hepatic glycogen synthesis and storage is increased, and gluconeogenesis is inhibited.

The net effect of these changes is that the maternal response to a glucose load is blunted, producing postprandial hyperglycemia.

Additionally, the fetoplacental unit serves as a constant drain on maternal glucose levels. Glucose is the primary fuel for the placenta and fetus and, thus, delivery of glucose from the mother to the fetus occurs by facilitated diffusion. As a result, maternal hypoglycemia develops during periods of fasting.

Lipid metabolism

Pregnancy causes an increase in circulating concentrations of all lipids, lipoproteins, and apolipoproteins. During early pregnancy, fat storage in central tissues predominates. Later in pregnancy, lipolysis predominates, possibly triggered by maternal fasting hypoglycemia. In the absence of glucose, increased plasma concentrations of free fatty acids, triglycerides, and cholesterol provide energy for the mother; this has been characterised as accelerated starvation. Following delivery, the concentrations of all lipids return to non-pregnancy levels, a process accelerated by breastfeeding.

Protein metabolism

Pregnancy is characterised by the intake and utilization of approximately 1 kg of protein above the normal nonpregnant state. At term, 50% of the additional protein is utilized by the fetus and placenta, and the remainder is shared by the uterus, breasts, maternal hemoglobin, and plasma proteins [13, 34].

MUSCULOSKELETAL

As pregnancy advances, a compensatory lumbar lordosis (anterior convexity of the lumbar spine) is apparent. This change is functionally useful, because it helps keep the woman's center of gravity over the legs; otherwise, the enlarging uterus would shift it anteriorly. However, as a result of this change in posture, virtually all women complain of low back pain during pregnancy. Increasing pressure caused by intra-abdominal growth of the uterus may result in an exacerbation of hernia defects, most commonly seen at the umbilicus and in the abdominal wall (diastasis recti, a physiologic separation of the rectus abdominis muscles). Beginning early in pregnancy, the effects of relaxin and progesterone result in a relative laxity of the ligaments. The pubic symphysis separates at approximately 28 to 30 weeks. Patients often complain of an unsteady gait and may fall more commonly during pregnancy than during the nonpregnant state, as a result of both these changes and an altered center of gravity (Fig. 2.12).

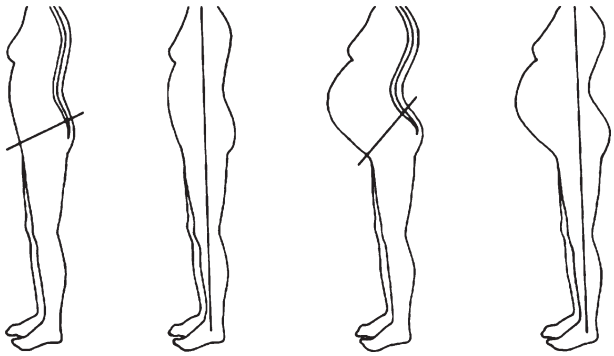


Fig. 2.12

To provide for adequate calcium supplies to the fetal skeleton, calcium stores are mobilized. Maternal serum ionized calcium is unchanged from the nonpregnant state, but maternal total serum calcium decreases. There is a significant increase in maternal parathyroid hormone, which maintains serum calcium levels by increasing absorption from the intestine and decreasing the loss of calcium through the kidney. The skeleton is well-maintained despite these elevated levels of parathyroid hormones. This may be because of the effect of calcitonin. Although the rate of bone turnover increases, there is no loss of bone density during a normal pregnancy if adequate nutrition is supplied.

REPRODUCTIVE TRACT

The effects of pregnancy on the vulva are similar to the effects on other skin (Fig. 2.13).

Because of an increase in vascularity, vulvar varicosities are common and usually regress after delivery. An increase in vaginal transudation as well as stimulation of the vaginal epithelium results in a thick, profuse vaginal discharge, called leukorrhea of

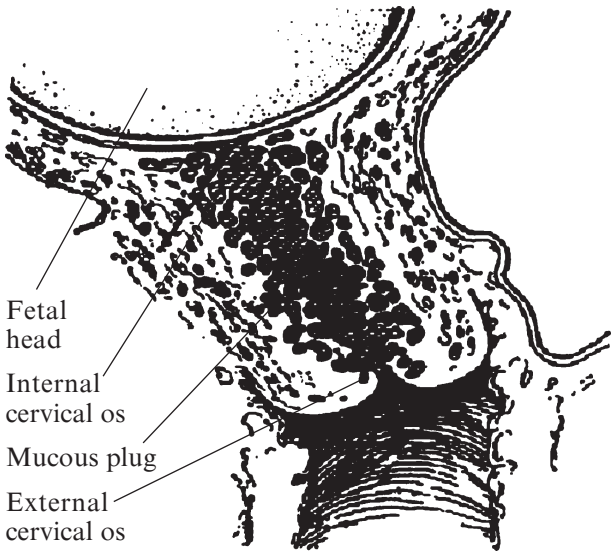


Fig. 2.13

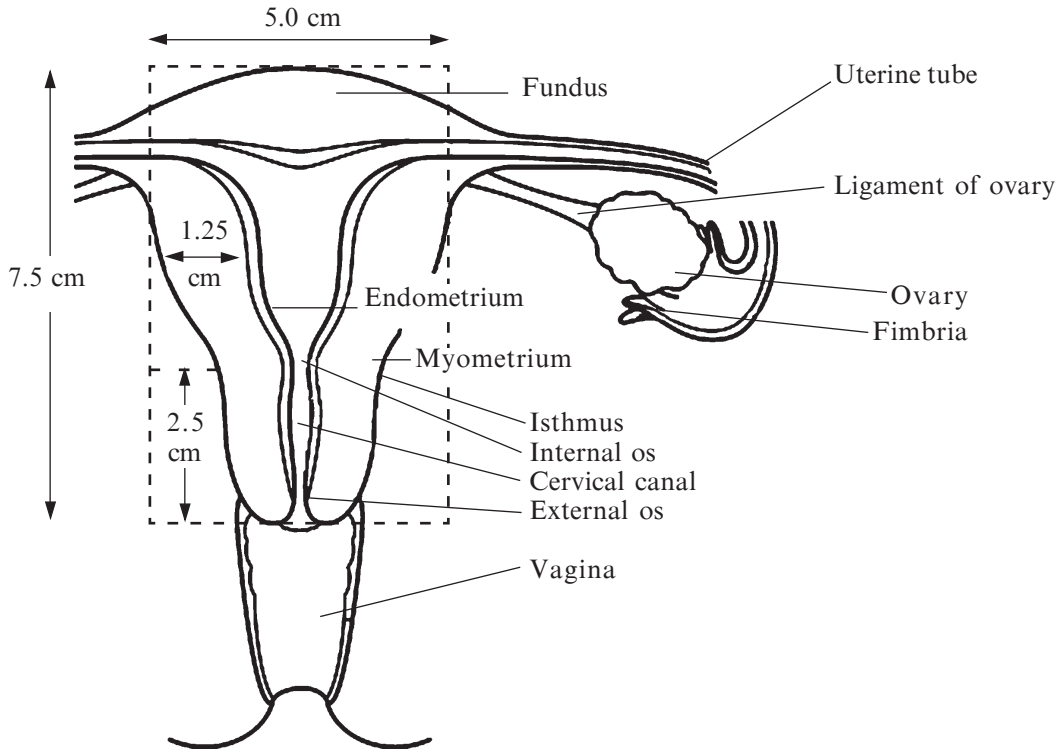


Fig. 2.14

pregnancy. The epithelium of the endocervix everts onto the ectocervix, which is associated with a mucous plug.

ISTHMUS

There are important structural and functional changes in the isthmus during pregnancy. During the first trimester, isthmus hypertrophies and elongates to about 3 times its original length. It becomes softer. With advancing pregnancy beyond 12 weeks, it progressively unfolds from above, downwards until it is incorporated into the uterine cavity. The circularly arranged muscle fibers in the region function as a sphincter in early pregnancy and thus help to retain the fetus within the uterus. Incompetency of the sphincteric action leads to mid-trimester abortion and the encirclage operation done to rectify the defect is based on the principle of restoration of the retentive function of the isthmus (Fig. 2.14).

The narrow isthmus of the uterus between the body of the uterus and the cervix becomes increasingly stretched and thinned out as the pregnancy progresses, to become the well- formed lower segment at term. This is the preferred site for the uterine incision at cesarean section, because of its relative avascularity, quiescence, better healing and lower risk of scar rupture in a subsequent pregnancy than an upper segment incision. The lower segment does not contract during labor.

As labor progresses, the cervix dilates and effaces almost completely into the lower segment. When the placenta is low lying and implanted on the lower

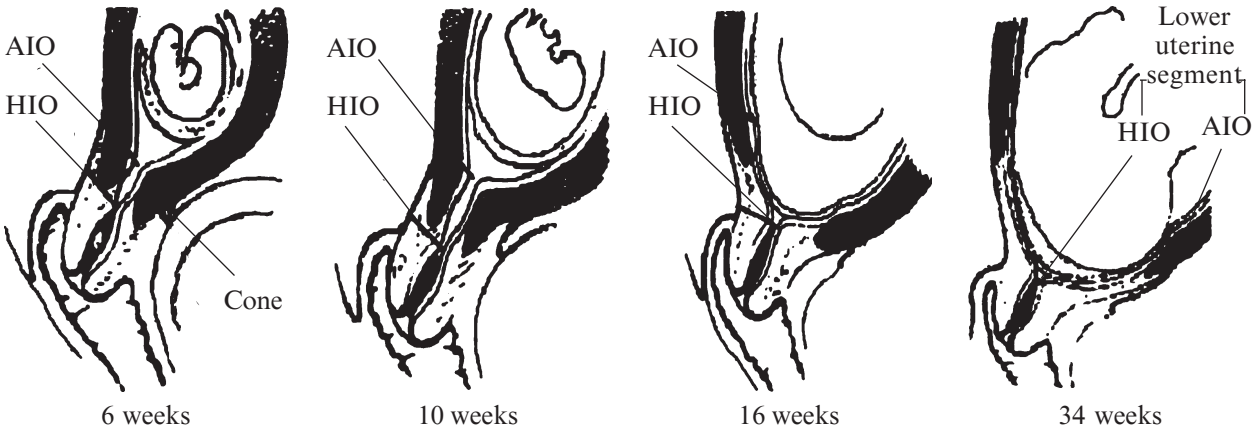


Fig. 2.15

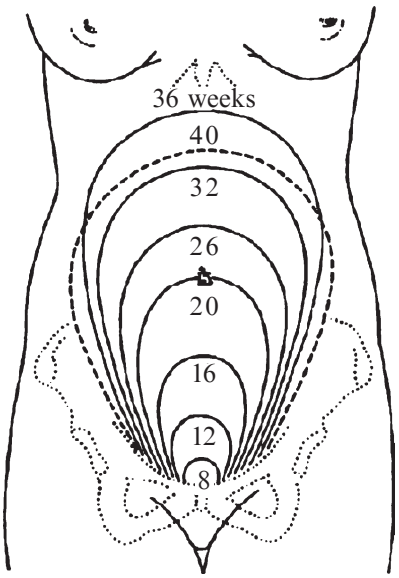


Fig. 2.16

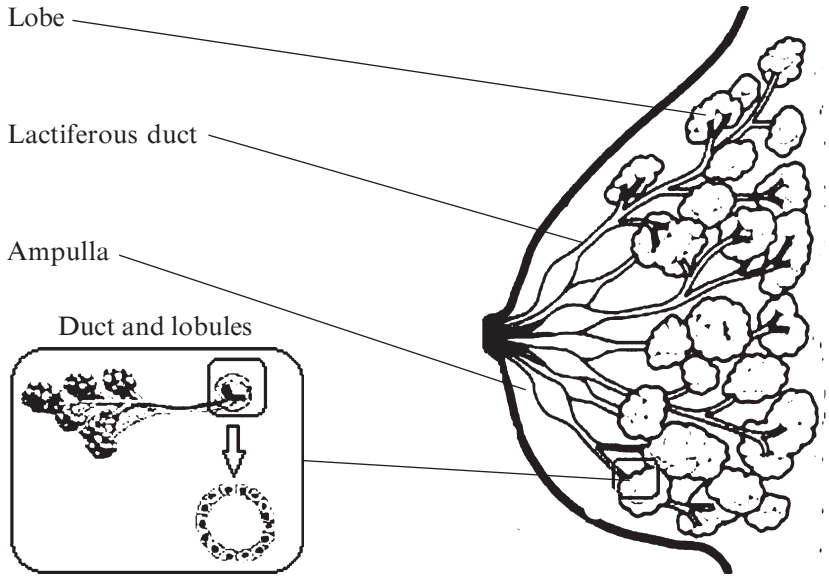


Fig. 2.17

segment, there is an increased risk of postpartum hemorrhage as the lower segment does not contract effectively after delivery of the placenta.

As pregnancy progresses, the collagen concentration in the cervix decreases and there is accumulation of glycosaminoglycans and water. This results in increasing "ripening" of the cervix in preparation for labor (Fig. 2.15).

With increasing gestation, there is increased stretching of the uterine muscle fibres. This results in the formation of the lower segment in the 3rd trimester (28–40 weeks).

UTERUS

During pregnancy, the uterus undergoes an enormous increase in weight from the 70-g non-pregnant size to approximately 1100 g at term, primarily through hypertrophy of existing myometrial cells. Changes occur in all the parts of the uterus — body, isthmus and cervix. The uterine enlargement is not a symmetrical one. The fundus enlarges more than the body. It is evident by the low down attachment of the round ligaments or insertion of the uterine end of the Fallopian tubes at term (Fig. 2.16).

After pregnancy, the uterus returns to an only slightly increased size as the actual number of cells comprising it are minimally increased. Similarly, the uterine cavity enlarges to a volume of up to as much as 5 liters, compared to less than 10 ml in the non-gravid state.

PELVIC LIGAMENTS

There is softening of the ligaments of the pelvic joints, presumably due to estrogen. The effect is to make the pelvis more mobile and increase its capacity.

BREASTS

The breasts increase in size during pregnancy, rapidly in the first 8 weeks and steadily thereafter. In most cases, the total enlargement is 25% to 50% (Fig. 2.17). The nipples become larger and more mobile and the areola larger and more deeply pigmented, with enlargement of the Montgomery glands. Blood flow to the breasts increases as they change to support lactation. Estrogen stimulation also results in ductal growth, with alveolar hypertrophy being a result of progesterone stimulation. During the latter portion of pregnancy, a thick, yellow fluid can be expressed from the nipples. This is colostrum. Ultimately, lactation depends on synergistic actions of estrogen, progesterone, prolactin, human placental lactogen, cortisol, and insulin.

Chapter 3
MATERNAL PELVIS

An understanding of the anatomy of the female pelvis is essential for obstetrical practice. Birth canal is divided into bone and soft parts: to bone belongs small pelvis and to soft parts — cervix, vagina, muscle-fascial system of the pelvic floor. Bone part of the pelvis Female pelvis with obstetric considerations are divided into two sections: the large and small pelvis. The boundary between them passes through an unmarked line (linea innominata). Large pelvis bounded on the sides of the iliac wings, back — the spine. Small pelvis formed in front branches of the pubic bones and symphysis on each side. Part bones constitute acetabular and ischial bones, behind — the sacrum and coccyx. During childbirth the small pelvis as dense bone tunnel limits and determines the size, shape and direction of the birth canal, which fetus passes, and has to adapt by changing their own configuration [50].

PELVIC BONES

The pelvis is composed of four bones: the *sacrum*, *coccyx*, and *two innominate bones*.

Each *innominate bone* is formed by the fusion of the ilium, ischium, and pubis. The innominate bones are joined to the sacrum at the sacroiliac synchondroses and to one another at the symphysis pubis.

The *sacrum* consists of five fused vertebrae (Fig. 3.1).

The anterior superior edge of the first sacral vertebra is called the *promontory*, which protrudes slightly into the cavity of the pelvis. The anterior surface of the sacrum is usually concave. It articulates with the ilium at its upper segment, with the coccyx at its lower segment, and with the sacrospinous and sacrotuberous ligaments laterally (Fig. 3.2).

The *coccyx* is composed of three to five rudimentary vertebrae. It articulates with the sacrum, forming a joint, and occasionally the bones are fused.

The pelvis is divided into the *false pelvis* above and the *true pelvis* below the linea terminalis (the edge of the pelvic inlet). The false pelvis is bordered by the lumbar vertebrae posteriorly, the iliac fossa bilaterally, and the abdominal wall anteriorly. The only obstetric function is to support the pregnant uterus.

The true pelvis is a bony canal and is formed by the sacrum and coccyx posteriorly and by the ischium and pubis laterally and anteriorly. Its internal borders are solid and relatively immobile. The posterior wall is twice the length of the anterior wall. The true pelvis is the area of concern to the obstetrician because its dimensions are sometimes not adequate to permit passage of the fetus (Fig. 3.3).

PELVIC AXES

• *Anatomical Axis* (curve of Carus):

It is an imaginary line joining the center points of the planes of the inlet, cavity and outlet. It is "C"

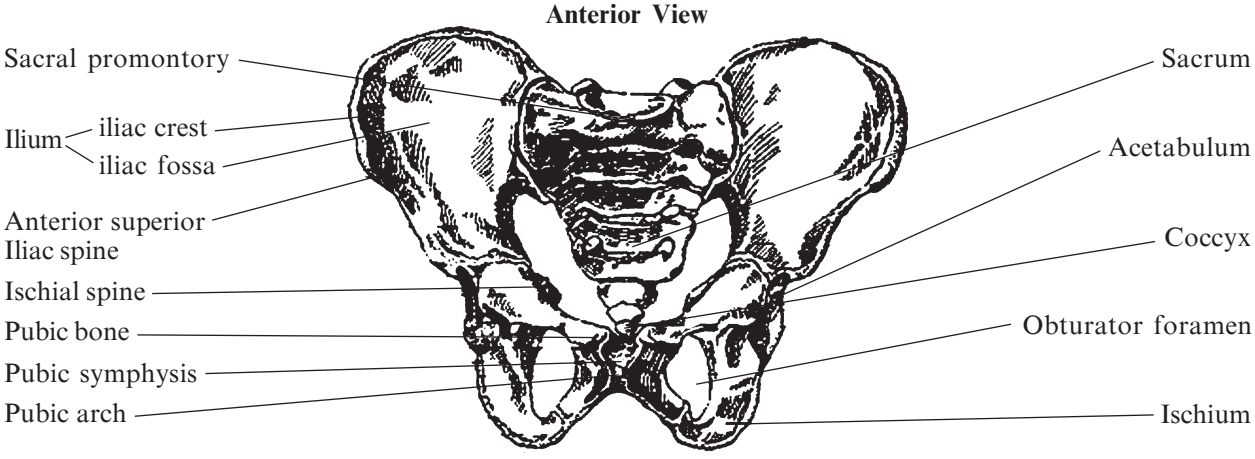


Fig. 3.1

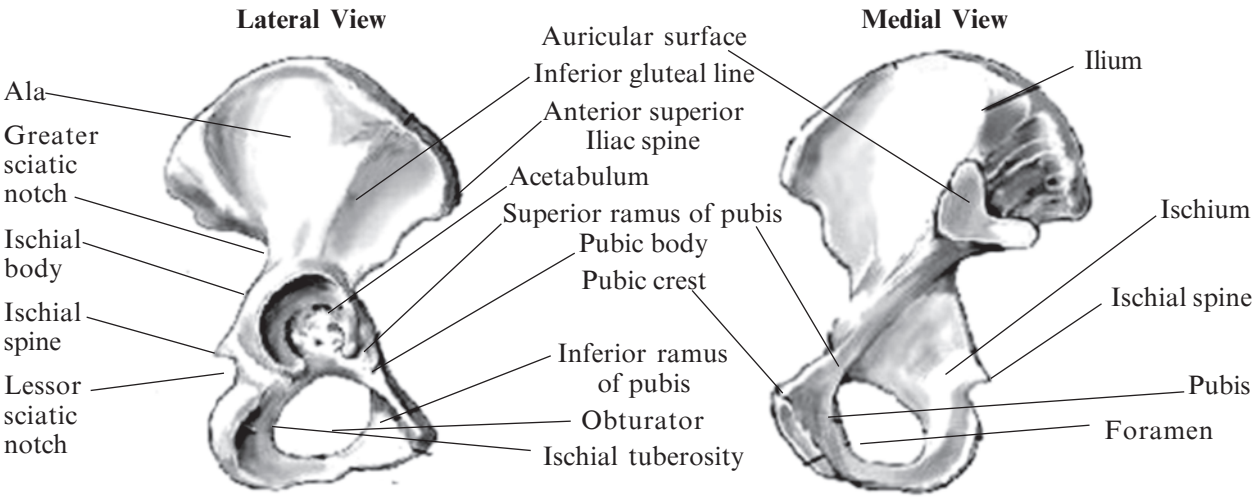


Fig. 3.2

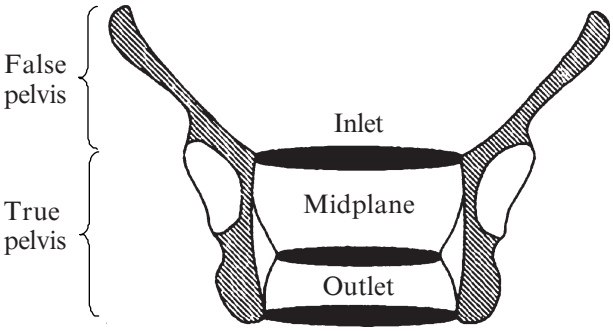


Fig. 3.3

shaped with the concavity directed forwards. It has no obstetric importance.

• *Obstetric Axis:*

The imaginary line represents the way passed by the head during labor. The "J"-shaped passes downwards and backwards along the axis of the inlet till the ischial spines where it passes downwards and formed by joining the axis of inlet, cavity and outlet.

PLANES AND DIAMETERS OF THE PELVIS

Dimensions of the pelvis, on which the course and outcomes for both mother and fetus depends are important in obstetric practice. But most of pelvis sizes can not be measured directly. The great pelvis for childbirth is not not important, but its size may indirectly point to the form and size of the pelvis. Pelvic cavity is the space between the walls, which the top and bottom limit inlet and outlet planes of the pelvis. It looks like a cylinder.

The pelvis is described as having four imaginary planes, flat surfaces that extend across the pelvis at different levels. Except for the plane of greatest diameter, each plane is clinically significant (Fig. 3.4).

The ***plane of the inlet*** is bordered by the pubic crest anteriorly, the iliopectineal line of the innominate bones laterally, and the promontory of the sacrum posteriorly. The fetal head enters the pelvis through this plane in the transverse position (Fig. 3.5).

The pelvic inlet has five important diameters. The *anterioposterior diameter* is described by one of two measurements.

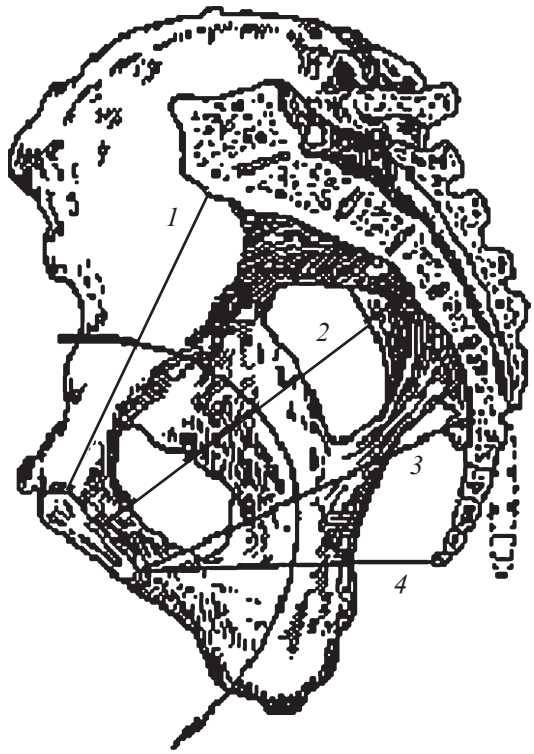


Fig. 3.4: 1 — the pelvis inlet; *2* — the plane of greatest diameter; *3* — the plane of least diameter; *4* — the pelvis outlet

The ***true conjugate*** *(anatomic conjugate)* is the anatomic diameter and extends from the middle of the sacral promontory to the superior surface of the pubic symphysis.

The ***obstetric conjugate*** represents the actual space available to the fetus and extends from the middle of the sacral promontory to the closest point on the convex posterior surface of the symphysis pubis (Fig. 3.6).

The clinically important *obstetrical conjugate* is the shortest distance between the sacral promontory and the symphysis pubis. Normally, this measures 10 cm or more, but unfortunately, it cannot be measured directly with examining fingers (Fig. 3.7).

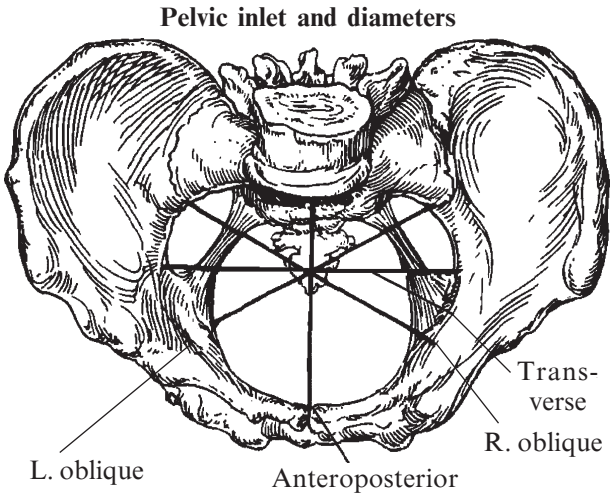


Fig. 3.5

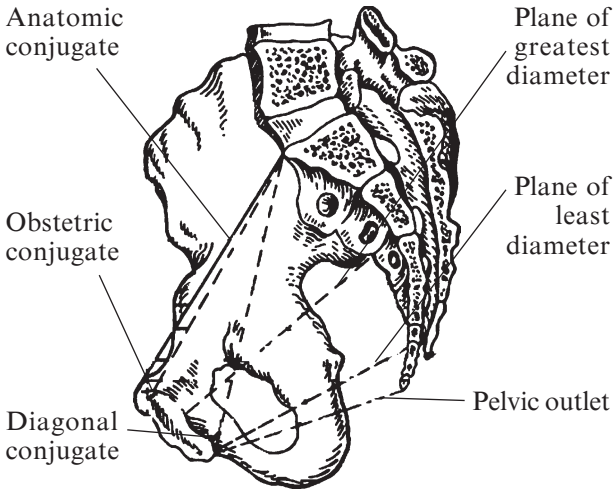


Fig. 3.6

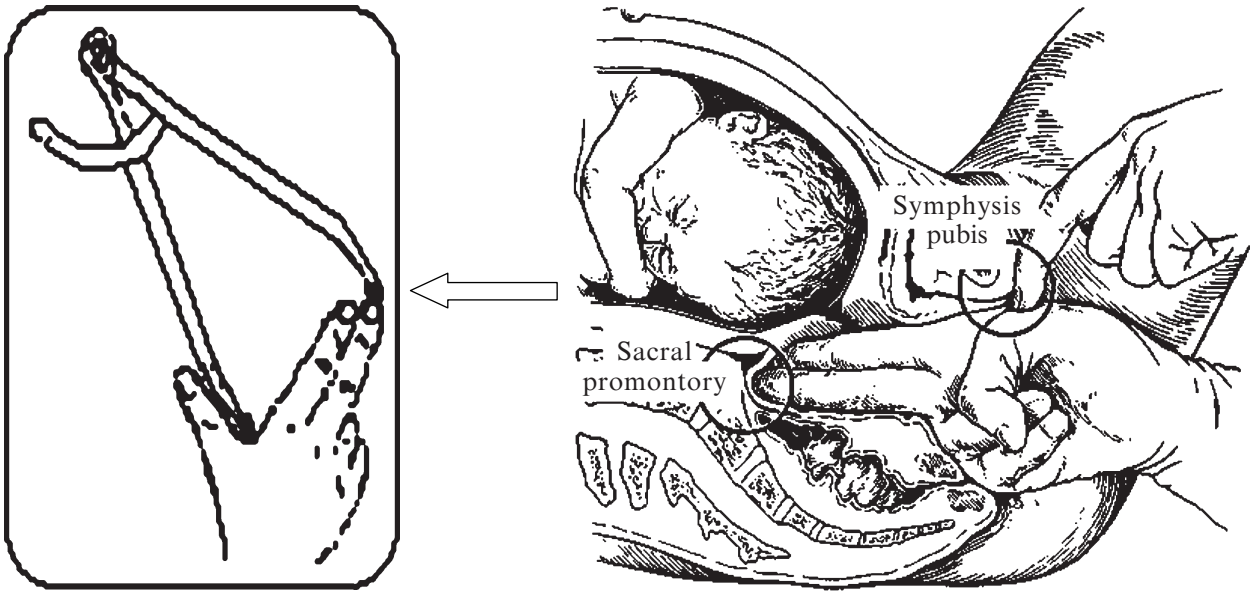


Fig. 3.7

Thus, for clinical purposes, the obstetrical conjugate is estimated indirectly by subtracting 1.5 to 2 cm from the diagonal conjugate, which is determined by measuring the distance from the lowest margin of the symphysis to the sacral promontory.

The *transverse diameter* is the widest distance between the iliopectineal lines. Each *oblique diameter extends* from the sacroiliac joint to the opposite iliopectineal eminence.

The *posterior sagittal diameter* extends from the anteroposterior and transverse intersection to the middle of the sacral promontory.

To help the obstetrician orient in the direction of the oblique diameters the following method is offered. The hands are held together at a right angle (with the palms facing each other) and the fingers are moved in the direction of the pelvic outlet of the woman in the supine position. The plane of the left hand will then coincide with the left oblique diameter and of the right hand with the right oblique diameter (Fig. 3.8).

The ***plane of greatest diameter*** is the largest part of the pelvic cavity. It is bordered by the posterior midpoint of the pubis anteriorly, the upper part of the obturator foramina laterally, and the junction of the second and third sacral vertebrae posteriorly. The fetal head rotates to the anterior position in this plane.

Fig. 3.8

The plane has two noteworthy diameters. The *anteroposterior diameter* extends from the midpoint of the posterior surface of the pubis to the junction of the second and third sacral vertebrae. The *transverse diameter* is the widest distance between the lateral borders of the plane.

The ***plane of least diameter*** is the most important from a clinical standpoint because most instances of arrest of descent occur at this level. It is bordered by the lower edge of the pubis anteriorly, the ischial spines and sacrospinous ligaments laterally, and the lower sacrum posteriorly. Low transverse arrests generally occur in this plane.

The plane has three important diameters. The *anteroposterior diameter* extends from the lower border of the pubis to the junction of the fourth and fifth sacral vertebrae. The *transverse* (bispinous) diameter extends between the ischial spines. The posterior sagittal diameter extends from the midpoint of the bispinous diameter to the junction of the fourth and fifth sacral vertebrae.

The ***plane of the pelvic outlet*** is formed by two triangular planes with a common base at the level of the ischial tuberosities. The anterior triangle is bordered by the subpubic angle at the apex, the pubic rami on the sides, and the bituberous diameter at the base. The posterior triangle is bordered by the sacrococcygeal joint at its apex, the sacrotuberous ligaments on the sides, and the bituberous diameter at the base. This plane is the site of a low pelvic arrest.

The plane has four important diameters.

The anatomic anteroposterior diameter extends from the inferior margin of the pubis to the tip of the coccyx, whereas the obstetric anteroposterior diameter extends from the inferior margin of the pubis to the sacrococcygeal joint. The transverse (bituberous) diameter extends between the inner surfaces of the ischial tuberosities, and the posterior sagittal diameter (not listed) extends from the middle of the transverse diameter to the sacrococcygeal joint [36, 45] (Fig. 3.9; Table 1).

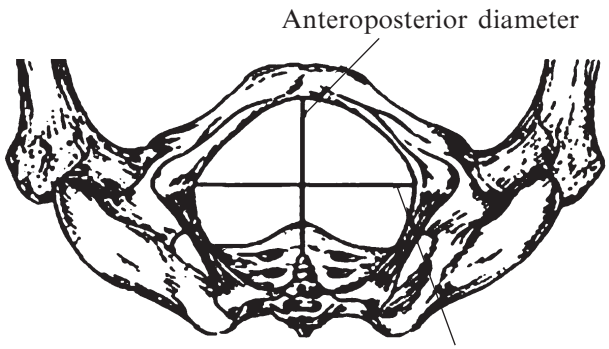


Fig. 3.9

Table 1

Average Length of Pelvic Plane Diameters

Pelvic plane	Diameter	Average length, cm
Inlet	True conjugate	11.5
	Obstetric conjugate	11
	Transverse	13.5
	Oblique	12.5
	Posterior sagittal	4.5
Greatest diameter	Anteroposterior	12.75
	Transverse	12.5
Midplane	Anteroposterior	12
	Bispinous	10.5
	Posterior sagittal	4.5–5
Outlet	Anatomic anteroposterior	9.5
	Obstetric anteroposterior	11.5
	Bituberous	11
	Posterior sagittal	7.5

PELVIC SHAPES

Based on the general bony architecture, the pelvis may be classified into four basic types: gynecoid, android, anthropoid, and platypelloid [18] (Fig. 3.10).

Four types of female pelvis (The Caldwell–Moloy's classification)

They differ in:

— Shape of the pelvic inlet.
— Shape of the side-walls.
— Character of the subpubic arch.

Four types do exist:

— Gynecoid.
— Android.
— Anthropoid.
— Platypelloid.

However, the majority of the pelvis are a mixture of all the 4 types (Fig. 3.11).

Gynecoid

The gynecoid pelvis is the classic female type of pelvis and is found in approximately 50% of women. It has the following characteristics:

1. Round at the inlet, with the widest transverse diameter only slightly greater than the anteroposterior diameter.
2. Sidewalls straight.
3. Ischial spines of average prominence.
4. Large sacrospinous notch.
5. Well-curved sacrum.
6. Spacious subpubic arch with an angle of approximately 90 degrees.

These features create a cylindrical shape that is spacious throughout. The fetal head generally rotates into the occipitoanterior position in this type of pelvis.

Android

The android pelvis is the typical male type of pelvis. It is found in less than 30% of women and has the following characteristics:

1. Triangular inlet with a flat posterior segment and the widest transverse diameter closer to the sacrum than in the gynecoid type.
2. Convergent sidewalls with prominent spines.
3. Shallow sacral curve.
4. Long and narrow (small) sacrospinous notch.
5. Narrow subpubic arch.

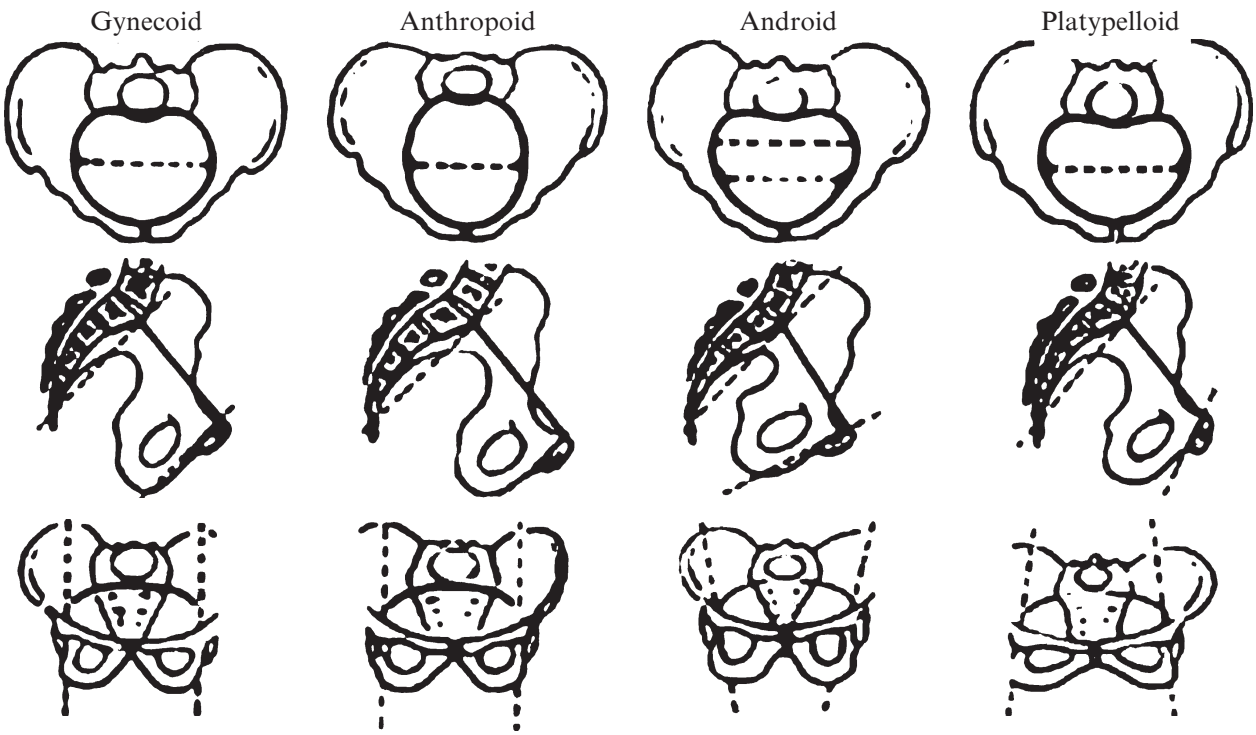


Fig. 3.10

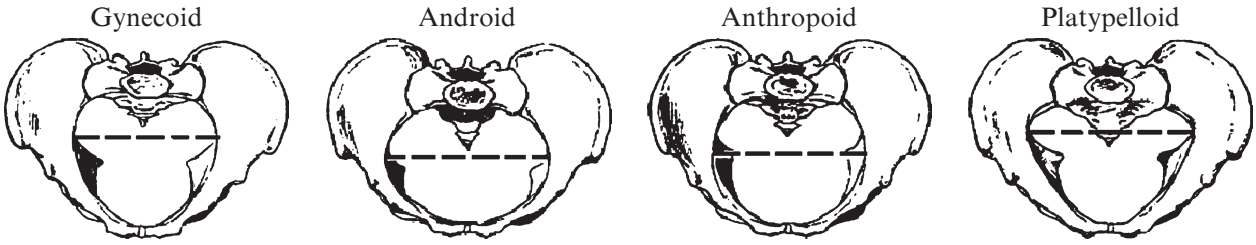


Fig. 3.11

This type of pelvis has limited space at the inlet and progressively less space as the fetus moves down the pelvis, because of the funnelling effect of the sidewalls, sacrum, and pubic rami. Thus, the amount of space is restricted at all levels. The fetal head is forced to be in the occipitoposterior position to conform to the narrow anterior pelvis. Arrest of descent is common at the midpelvis.

Anthropoid

The anthropoid pelvis resembles that of the anthropoid ape. It is found in approximately 20% of women and has the following characteristics:

1. A much larger anteroposterior than transverse diameter, creating a long, narrow oval at the inlet.
2. Sidewalls that do not converge.
3. Ischial spines that are not prominent but are close, because of the overall shape.
4. Variable, but usually posterior, inclination of the sacrum.
5. Small sacrospinous notch.
6. Narrow, outwardly shaped subpubic arch.

The fetal head can engage only in the anteroposterior diameter and usually does so in the occipitoposterior position because there is more space in the posterior pelvis.

Platypelloid

The platypelloid pelvis is best described as being a flattened gynecoid pelvis. It is found in only 3% of women, and it has the following characteristics:

1. A short anteroposterior and wide transverse diameter, creating an oval-shaped inlet.
2. Straight or divergent sidewalls
3. Posterior inclination of a flat sacrum.
4. A wide bispinous diameter.
5. Long but small sacrospinous notch.
6. A wide subpubic arch.

The overall shape is that of a gentle curve throughout. The fetal head has to engage in the transverse diameter.

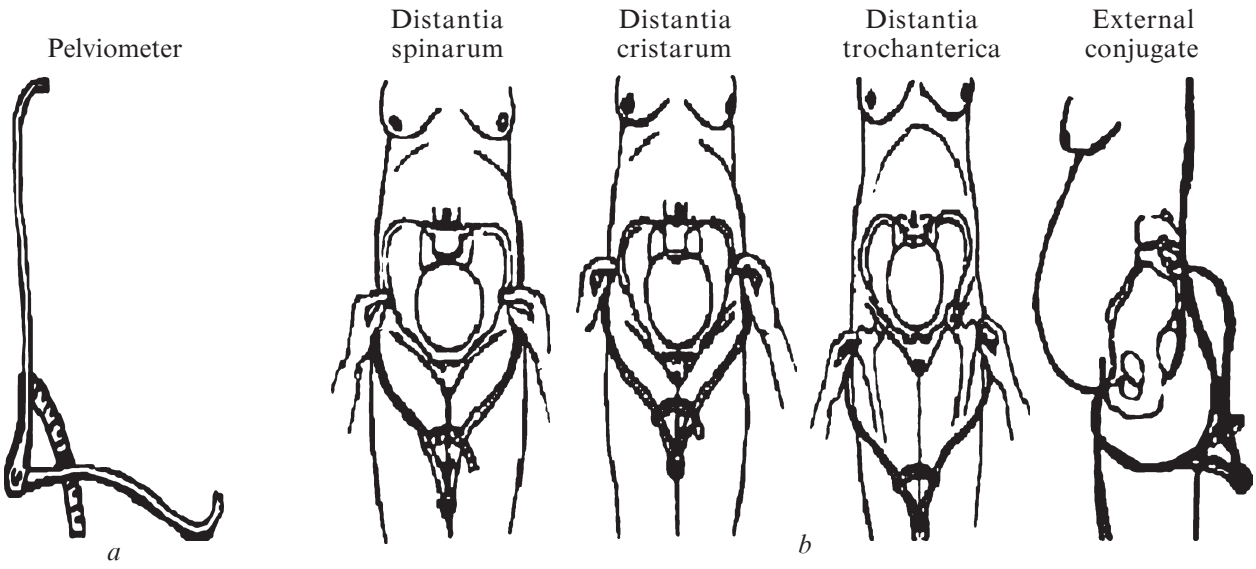


Fig. 3.12

CLINICAL PELVIMETRY

Pelvimetry: measurement of diameters of the pelvis. Clinical pelvimetry is the part of the pelvic exam that evaluates the "adequacy" of the pelvis — whether the dimensions are adequate for vaginal delivery.

Methods of pelvimetry:

• Clinical pelvimetry:

Manual pelvimetry: measurement of the essential diameters of the bony pelvis using the hands.

— External/indirect pelvimetry.

— Internal/direct pelvimetry.

• Radiographic pelvimetry procedure for measurement of the bony pelvis and fetal head using anteroposterior and lateral radiographs, with a device for the correction of magnification.

The main dimensions of the pelvis

Of all the methods of the pelvis examination the measurement is essential. Most of the internal pelvic sizes are available for measurement, so usually measured by its external dimensions and evaluate them by internal. Pelvic measurement is made by the pelvimeter. Usually measured four basic dimensions of the pelvis: three transverse and one direct (Fig. 3.12, *a*).

Distantia spinarum — the distance between the upper anterior iliac spine bones. This size is 26 cm.

Distantia cristarum — the distance between the most distant points of iliac bone wings. On average it is 28 cm.

Distantia trochanterica — the distance between the trochanter major of hip bones. This size is 31 cm (Fig. 3.12, *b*).

External conjugate — external size of pelvis. End of pelvimeter set on middle of the upper margin of symphysis, the other end is over the sacral fossa contained between fifth lumbar vertebra and the beginning of the first sacral vertebra. External conjugate is 20 cm. External conjugate exceeds the obstetric conjugate by 9 cm (Fig. 3.13, *a*).

Michael's rhomb has 4 angles. The upper angle is located in the suprasacral fossa. The lower angle is situ-

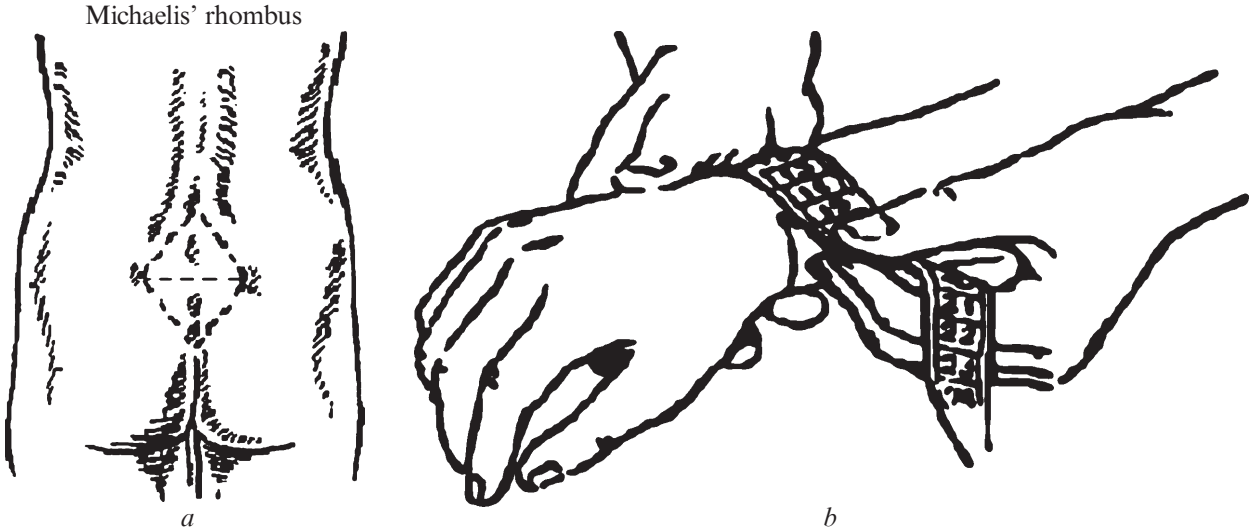


Fig. 3.13

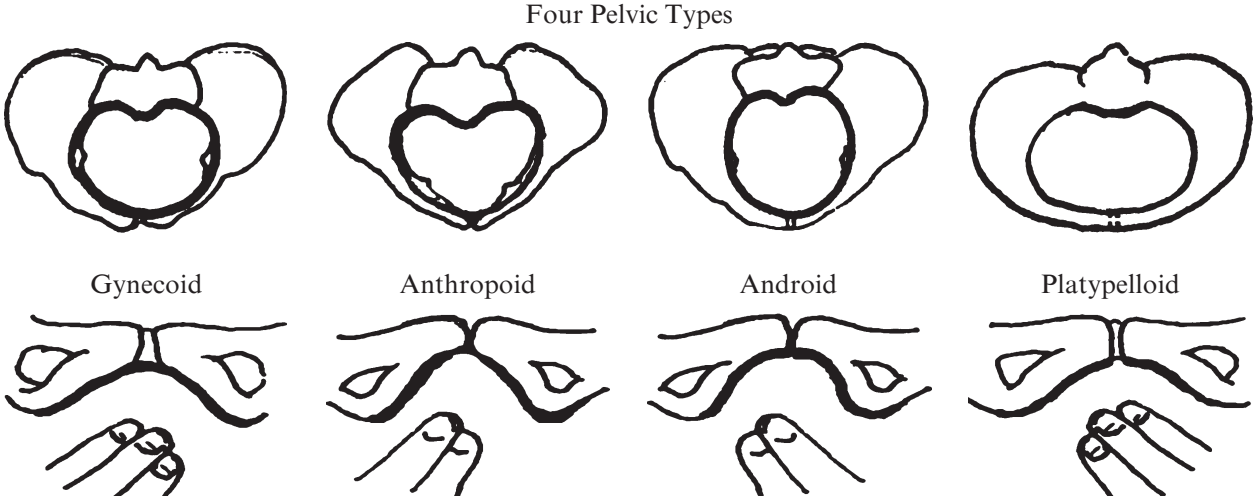


Fig. 3.14

ated in the apex of coccyx, and laterally angles are situated in the posterior superior iliac spines. In women with normal pelvis the rhomb has a regular form.

Its vertical diameter is 11 cm, and horizontal diameter is 10 cm. Vertical dimension of Michaels' rhomb equals the obstetric conjugate.

Soloviov's index. It is estimated by the circumference of radiocarpal joint. It is 14–16 cm and indicates pelvic thickness (Fig. 3.13, *b*).

The additional external pelvic sizes

Lateral conjugate — is a distance between the anterior superior iliac spine and posterior superior iliac spine of the same iliac bone. It is 14.5–16 cm.

Oblique conjugate — is a distance between the right anterior superior iliac spine to the left posterior superior iliac spine. It is 14.5–16 cm.

Anteroposterior diameter of the pelvic outlet is a distance between the lower part of symphysis pubis and apex of the coccyx. It is 9.5 cm.

Transverse diameter of the pelvic outlet is a distance between the posterior portions of the ishial tuberosities. It is 11.5 cm.

The main internal pelvic sizes

The diameters that can be clinically evaluated can be assessed at the time of the first prenatal visit to screen for obvious pelvic contractions, although some obstetricians believe that it is better to wait until later in pregnancy, when the soft tissues are more distensible and the examination is less uncomfortable and possibly more accurate. The clinical evaluation is started by assessing the pelvic inlet. The pelvic inlet can be evaluated clinically for its anteroposterior diameter. The obstetric conjugate can be estimated from the diagonal conjugate, which is obtained during clinical examination.

The diagonal conjugate is approximated by measuring from the lower border of the pubis to the sacral promontory, using the tip of the second finger and the point where the base of the index finger meets the pubis. The obstetric conjugate is then estimated by subtracting 1.5 to 2 cm, depending on the height and inclination of the pubis. Often the middle finger of the examining hand cannot reach the sacral promontory; thus, the obstetric conjugate is considered adequate. If the diagonal conjugate is greater than or equal to 11.5 cm, the anteroposterior diameter of the inlet is considered to be. The anterior surface of the sacrum is then palpated to assess its curvature. The usual shape is concave. A flat or convex shape may indicate anteroposterior constriction throughout the pelvis.

The midpelvis cannot be measured accurately clinically in either the anteroposterior or transverse diameter. The pelvic sidewalls can be assessed to determine if they are convergent rather than having the normal, almost parallel, configuration. The ischial spines are palpated carefully to assess their prominence, and several passes are made between the spines to approximate the bispinous diameter. The

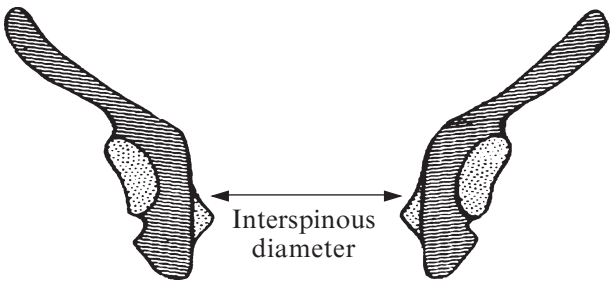


Fig. 3.15

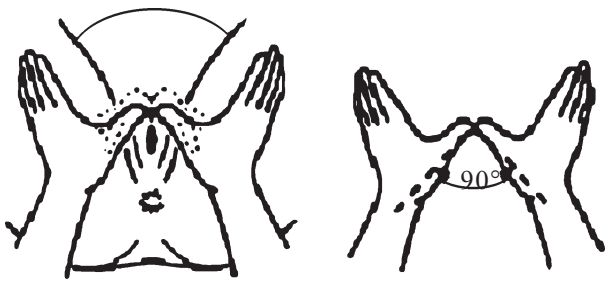


Fig. 3.16

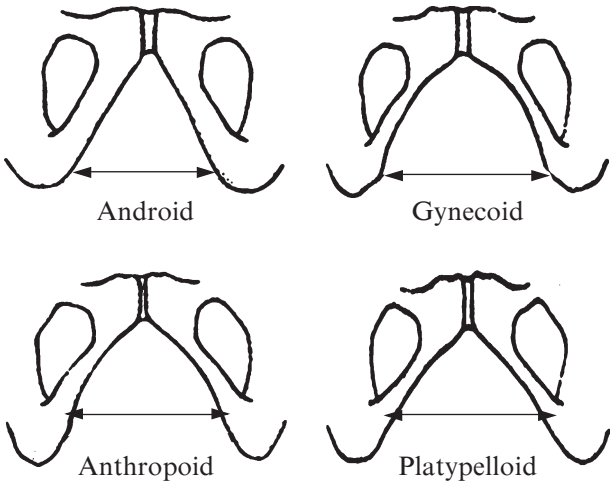


Fig. 3.17

length of the sacrospinous ligament is assessed by placing one finger on the ischial spine and one finger on the sacrum in the midline. The average length is three fingerbreadths. If the sacrospinous notch that is located lateral to the ligament can accommodate two-and-one-half fingers, the posterior midpelvis is most likely of adequate dimensions. A short ligament suggests a forward inclination of the sacrum and a narrowed sacrospinous notch (Fig. 3.14).

The *interspinous diameter* — the distance between the ischial spines — is an evaluation of the midpelvis.

This is the smallest dimension of the pelvis and needs to be at least 10 centimeters for the fetal head to be able to fit (Fig. 3.15).

Notice also whether the ischial spines are sharp, encroaching into the vagina, or otherwise reducing the diameter of the midpelvis.

Subpubic arch: normal = 90° pelvic outlet.

Intertuberous diameter: between the ischial tuberosities.

The subpubic arch — inferior to the symphysis pubis and created by the inferior pubic rami — is normally about 90 degrees. If you can fit two fingers side-by-side, that's about 90 degrees (Fig. 3.16).

The intertuberous diameter — the distance between the ischial tuberosities — can be palpated and compared to a measurement of your fist. Obviously you have to measure your hand ahead of time (Fig. 3.17).

The subpubic arch and the intertuberous diameter are evaluations of the pelvic outlet.

Chapter 4
ANATOMIC CHARACTERISTICS OF THE FETAL HEAD

The head is the largest and least compressible part of the fetus. Thus, from an obstetric viewpoint, it is the most important part, whether the presentation is cephalic or breech. The fetal skull consists of a base and a vault (the cranium). The base of the skull has large, ossified, firmly united, and non-compressible bones. This serves to protect the vital structures contained within the brain stem and its spinal connections. The cranium consists of the occipital bone posteriorly, two parietal bones bilaterally, and two frontal and temporal bones anteriorly. The cranial bones at birth are thin, weakly ossified, easily compressible, and interconnected only by membranes. These features allow them to overlap under pressure and to change shape to conform to the maternal pelvis, a process known as *moulding* (Fig. 4.1–4.3).

Sutures

The membrane-occupied spaces between the cranial bones are known as sutures. The sagittal suture lies between the parietal bones and extends in an anteroposterior direction between the fontanelles, dividing the head into right and left sides. The lamboid suture extends from the posterior fontanelle laterally and serves to separate the occipital from the parietal bones. The coronal suture extends from the anterior fontanelle laterally and serves to separate the parietal and frontal bones. The frontal suture lies between the frontal bones and extends from the anterior fontanelle to the glabella (the prominence between the eyebrows).

Clinical importance of sutures

Position of the fontanelle and sagittal suture can identify the attitude and position of vertex. By palpating the sagittal suture during labor, degree of internal rotation and moulding of the head can be noticed. In deep transverse arrest, this sagittal suture lies transversely at the level of the ischial spines.

Fontanelles

The membrane-filled spaces located at the point where the sutures intersect are known as fontanelles, the most important of which are the anterior and posterior fontanelles. Clinically, they are even more useful than the sutures for determining the fetal head position.

Anterior fontanelle: It is formed by joining of the four sutures in the midplane. The sutures are anteriorly frontal, posteriorly sagittal and on either side, coronal. The shape is like a diamond.

Its anteroposterior and transverse diameters measure approximately 3 cm each. The floor is formed by a membrane and it becomes ossified 18 months after birth. It becomes pathological, if it fails to ossify even after 24 months.

Posterior fontanelle: It is formed by junction of three suture lines — sagittal suture anteriorly and lambdoid suture on either side. It is triangular in shape

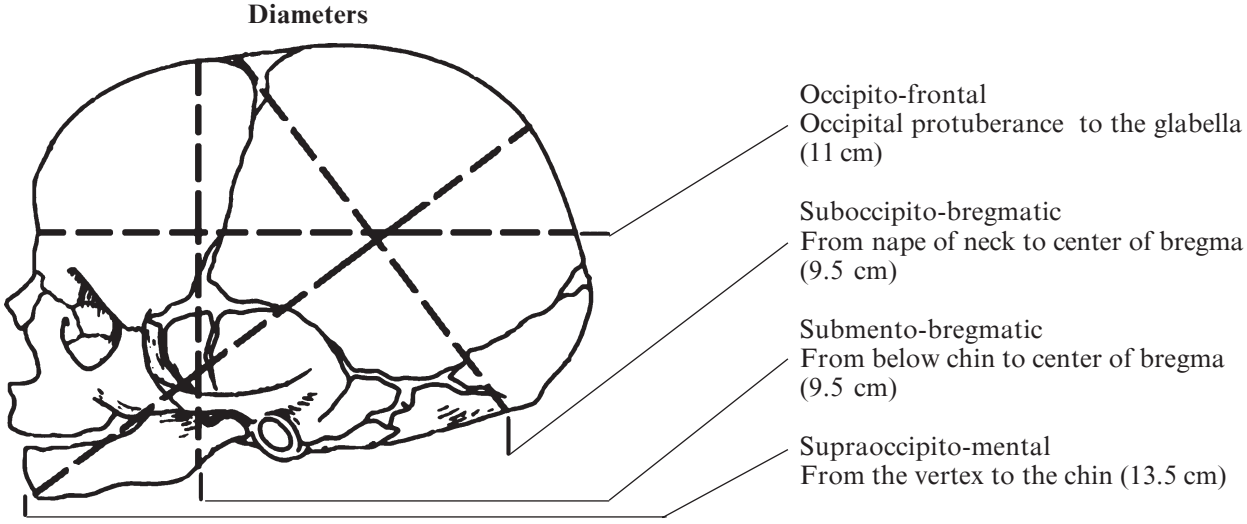


Fig. 4.1

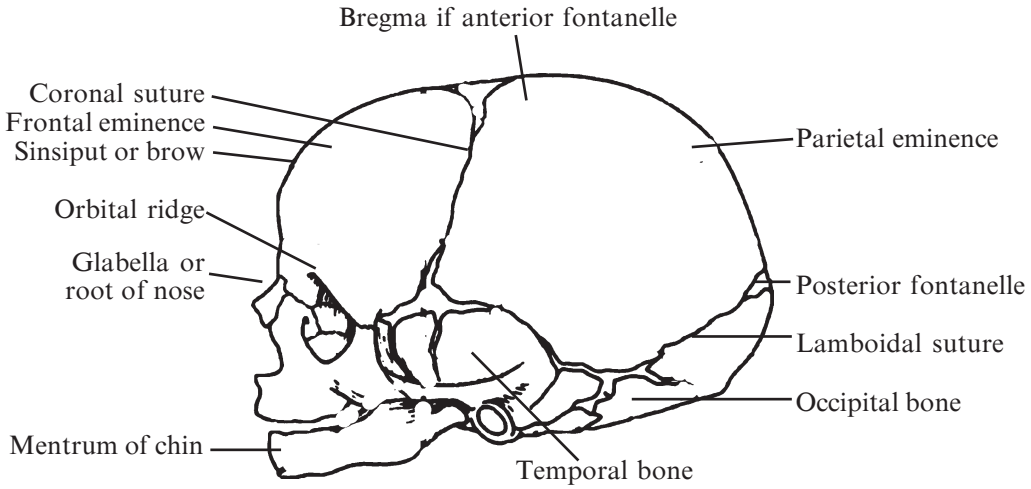
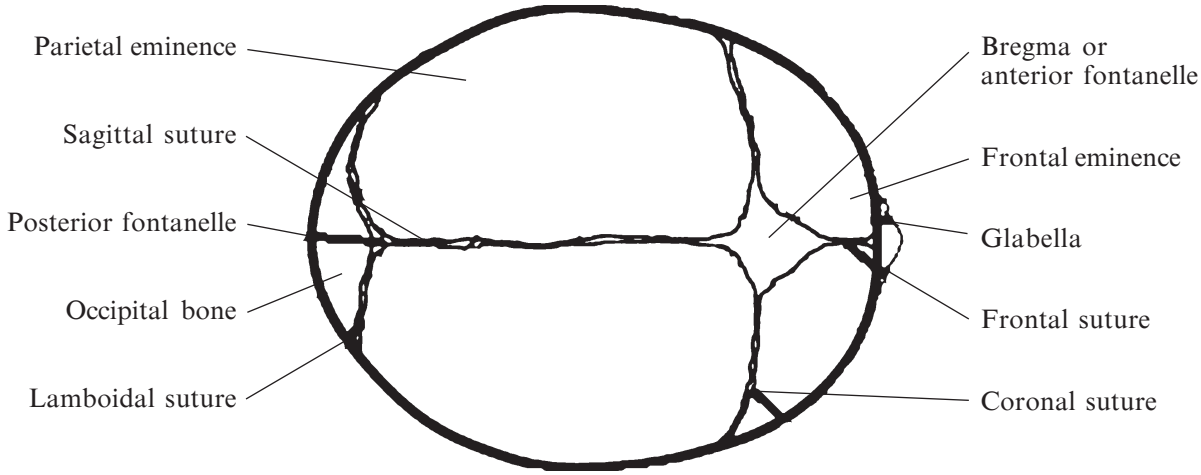
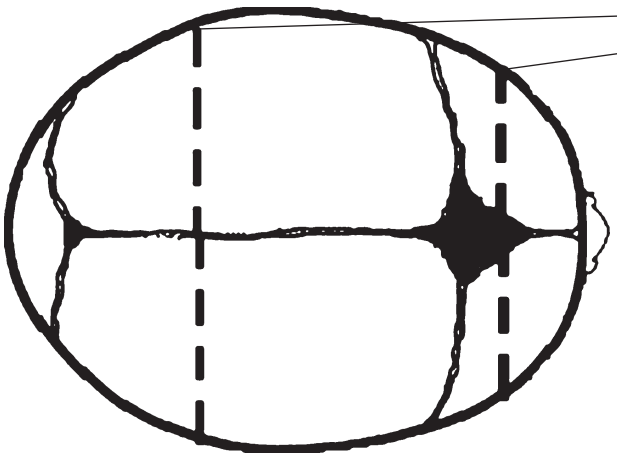


Fig. 4.2



a



Biparietal (9.5 cm) between two parietal eminences

Bitemporal (8.5 cm) greateat distance between two halves of coronal suture

Circumferences

Suboccipito-bregmatic biparietal (28 cm)
These are engaging diameters of well flexed vertex presentation

Occipito-frontal biparietal (33 cm)
There are engaging diameters of deflexed vertex presentation and found of occipito-posterior positions

Mento-vertical biparietal (35.5 cm)
This is the largest circumference of the head is found in brow presentation

b

Fig. 4.3

and measures about 1.2×1.2 cm. Its floor is membranous but becomes bony at term. Thus, truly its nomenclature as fontanelle is misnomer. It denotes the position of the head in relation to maternal pelvis.

Sagittal fontanelle: It is inconsistent in its presence. When present, it is situated on the sagittal suture at the junction of anterior two-third and posterior one-third. It has got no clinical importance.

Reshaping of the fetal skull

Obliteration of the sutures.

Overlapping of the bones of the vault:

— one parietal bone overlaps the other;

— both overlap the occipital bone.

It accounts for diminution of the biparietal diameter and suboccipitobregmatic diameters by 0.5–1 cm or even more (Fig. 4.4).

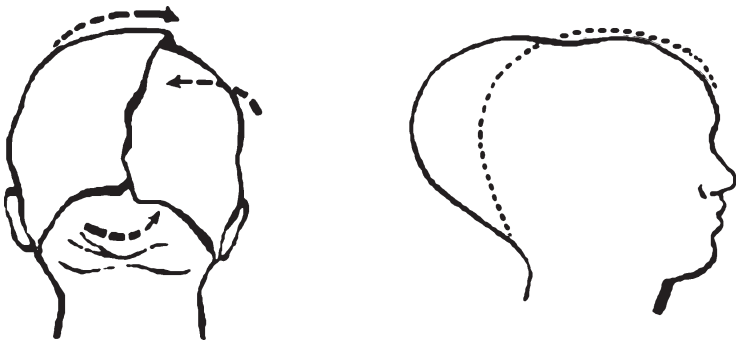


Fig. 4.4. Lateral and posterior view of moulding of the fetal skull

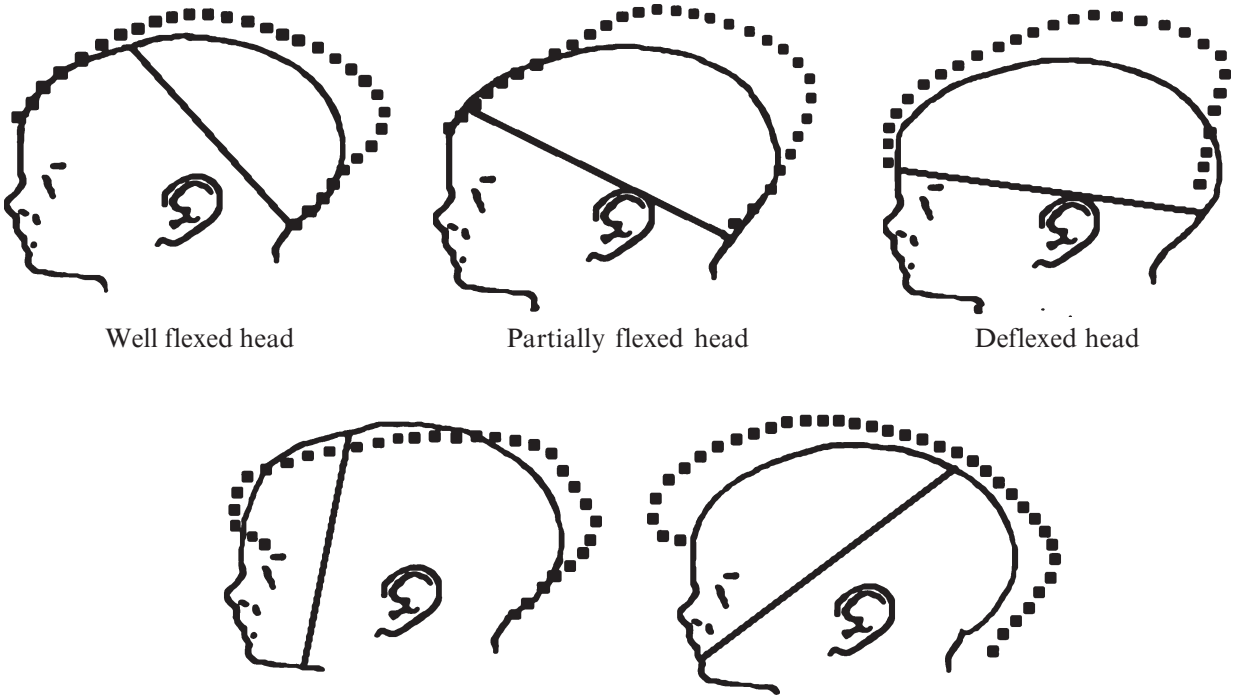


Fig. 4.5

Moulding of the fetal skull is the ability of the head to change its shape and so to adapt itself to the unyielding maternal pelvis during the progress of labor. This property is the greatest value in the progress of labor.

There is compression of the engaging diameter of the head with corresponding elongation of the diameter at right angle to it.

So, in the well flexed head of the anterior vertex presentation, the engaging suboccipito-bregmatic diameter is compressed with elongation of the head in mento-vertical diameter which is at right angle to suboccipito-bregmatic. During the process, the parietal bones tend to overlap the adjacent bones, viz. the occipital bone behind, the frontal bones in front and the temporal bones at the sides. In first vertex position, the right parietal bone tends to override the left one and this becomes reverse in second vertex position. Moulding disappears within a few hours after birth [18, 50] (Fig. 4.5).

— Slight moulding is inevitable and beneficial. It enables the head to pass more easily, through the birth canal.

— Extreme moulding as met in disproportion may produce severe intracranial disturbance in the form of tearing of tentorium cerebelli or subdural hemorrhage.

— Shape of the moulding can be useful information about the position of the head occupied in the pelvis.

Landmarks

The fetal skull is characterised by a number of landmarks. From front to back, they include the following:

1. Nasion: the root of the nose.
2. Glabella: the elevated area between the orbital ridges.
3. Sinciput (brow): the area between the anterior fontanelle and the glabella.
4. Anterior fontanelle (bregma): diamond-shaped.
5. Vertex: the area between the fontanelles and bounded laterally by the parietal eminences.
6. Posterior fontanelle (lambda): Y- or T-shaped.
7. Occiput: the area behind and inferior to the posterior fontanelle and lambdoid sutures.

Chapter 5
DIAGNOSIS OF PREGNANCY

FIRST TRIMESTER (FIRST 12 WEEKS)

The signs of pregnancy may be divided into *presumptive*, *probable*, and *positive*.

Positive or absolute signs:

- Palpation of fetal parts and perception of active fetal movements by the examiner at about the 20th week.
- Auscultation of fetal heart sounds.
- Ultrasound evidence of embryo as early as 6th week and later on the fetus.
- Radiological demonstration of the fetal skeleton at 16th week and onwards.

Presumptive symptoms and signs: (it includes the features mainly appreciated by the women):

- Amenorrhea.
- Frequency of micturition.
- Morning sickness.
- Fatigue.
- Breast changes.
- Skin changes.
- Quickening.

Probable signs:

- Abdominal enlargement.
- Braxton–Hicks contractions.
- External ballottement.
- Outlining the fetus.
- Changes in the size, shape and consistency of the uterus.
- Jacquemier's sign.
- Softening of the cervix.
- Osiander's sign.
- Internal ballottement.
- Immunological test.

SUBJECTIVE SYMPTOMS

The presumptive symptoms of early months of pregnancy:

Amenorrhoea

An overdue menstrual period remains, for most women with a regular menstrual cycle, the first suggestion of pregnancy. Pregnancy is the commonest cause of amenorrhoea but other causes such as disturbances in the hypothalamic-pituitary-ovarian axis or recent use of the contraceptive pill may be responsible.

Nausea or sickness

Many women suffer from some gastric upset in the early months of pregnancy, from nausea and anorexia to repeated vomiting, especially in the morning. The cause is unknown and raised levels of both estrogen and HCG in the circulation have been blamed. Gastric motility is reduced, and in early pregnancy, the lower esophageal sphincter is relaxed.

Bladder symptoms

Increased frequency of micturition in the second and third months is due to a combination of increased vascularity and pressure from the enlarging uterus. Near term, frequency may again appear due mainly to pressure of the fetal head on the bladder.

Breast discomfort

Breast discomfort in the form of feeling of fullness and 'pricking sensation' is evident as early as 6–8th week especially in primigravidae. Fatigue is a frequent symptom which may occur early in pregnancy.

OBJECTIVE SIGNS

Breast changes

The earliest symptoms and signs — increased vascularity and a sensation of heaviness, almost of pain — appear at 6 weeks. By 8 weeks the nipple and surrounding area — the primary areola — have become more pigmented. Montgomery's tubercles — sebaceous glands which become more prominent as raised pink-red nodules on the areola (Fig. 5.1).

By 16 weeks a clear fluid (colostrum) is secreted and may be expressed. By 20 weeks the secondary areola — a mottled effect due to further pigmentation — has become prominent.

Uterine changes

Uterine enlargement may be detected on bimanual examination at seven to eight weeks. At approx-

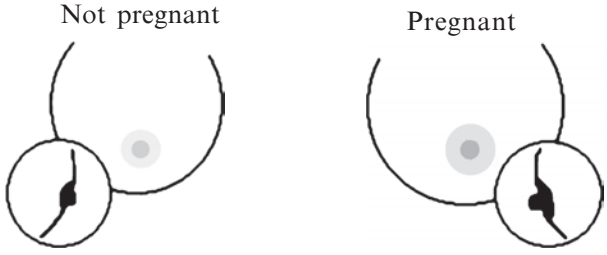


Fig. 5.1

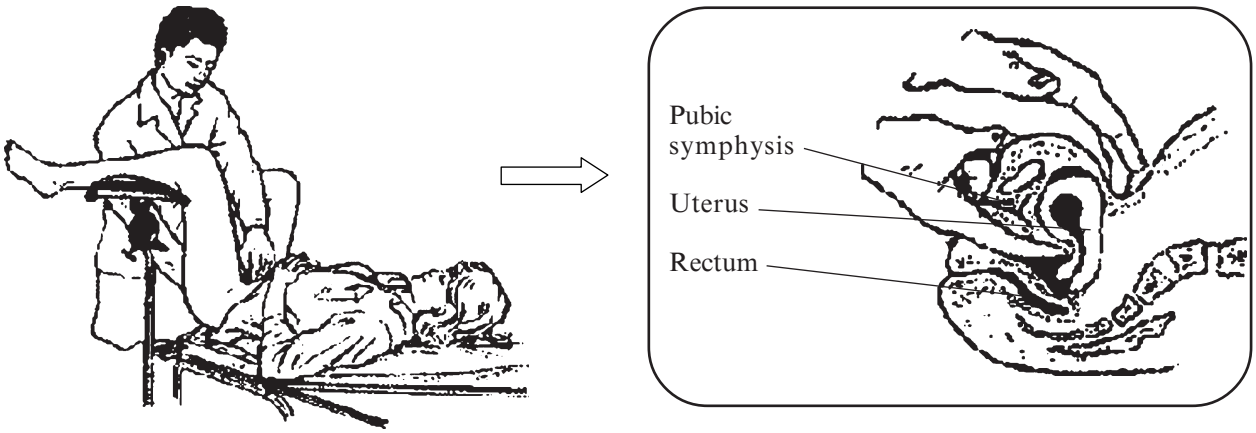


Fig. 5.2

imately 12 weeks from the onset of the last menstrual period, the uterus is generally enlarged sufficiently to be palpable in the lower abdomen (Fig. 5.2).

There may be asymmetrical enlargement of the uterus if there is lateral implantation. This is called *Piskacek's sign* where one half is more firm than the other half. As pregnancy advances, symmetry is restored. The pregnant uterus feels soft and elastic [21, 32].

Hegar's sign (it is present in two-thirds of cases).

It can be demonstrated between 6–10 weeks, a little earlier in multiparous women. This sign is based on the fact that: the upper part of the body of the uterus is enlarged by the growing fetus; the lower part of the body is empty and extremely soft and the cervix is comparatively firm. Because of variation in consistency, on bimanual examination (two fingers in the anterior fornix and the abdominal fingers behind the uterus), the abdominal and vaginal fingers seem to oppose below the body of the uterus. Examination must be gentle to avoid the risk of abortion (Fig. 5.3).

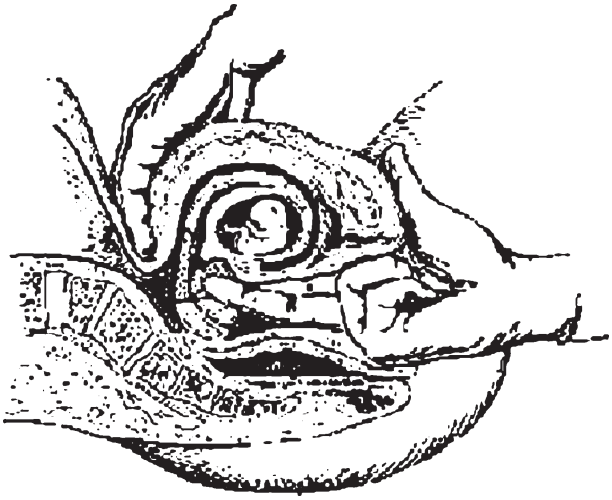


Fig. 5.3

IMMUNOLOGICAL TESTS FOR DIAGNOSIS OF PREGNANCY

Tests to detect pregnancy have revolutionised early diagnosis. Although they are considered a probable sign of pregnancy, the accuracy of these tests is very good. All commonly used methods depend on the detection of hCG or its β subunit in urine or serum.

Diagnosis of pregnancy by detecting hCG in maternal serum or urine can be made by 8 to 11 days after conception. The test is not reliable after 12 weeks. This hormone is a glycoprotein with high carbohydrate content. Serum hCG levels increase from the day of implantation and reach peak levels at 60 to 70 days. Thereafter, the concentration declines slowly until a plateau is reached at approximately 16 weeks (Fig. 5.4).

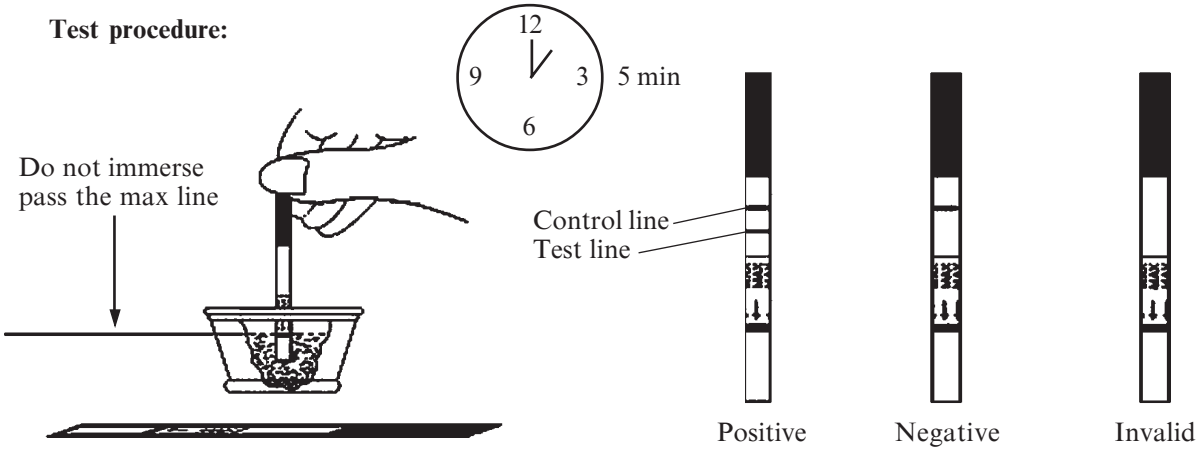


Fig. 5.4

The patient is advised to collect the first voided urine in the morning in a clean container (not to wash with soap). Kits to perform the test at home are also available.

ULTRASONOGRAPHY

Intradecidual gestational sac (GS) is identified as early as 29 to 35 days of gestation. Fetal viability and gestational age is determined by detecting the following structures by transvaginal ultrasonography. Gestational sac and yolk sac by 5 menstrual weeks; Fetal pole and cardiac activity — 6 weeks; Embryonic movements by 7 weeks. Fetal gestational age is best determined by measuring the CRL between 7 and 12 weeks (variation ± 5 days). The Doppler effect of ultrasound can pick up the fetal heart rate reliably by 10th week. The instrument is small, handy and cheap. The gestational sac (true) must be differentiated from pseudogestational sac.

SECOND TRIMESTER (13–28 WEEKS)

AWARENESS OF FETAL MOVEMENT

This may be felt by the mother at 18 weeks in parous women and two to three weeks later in a primigravida. It not only gives positive evidence of pregnancy but of a live fetus. The intensity varies from a faint flutter in early months to stronger movements in later months.

ABDOMINAL EXAMINATION

Inspection

1. Linear pigmented zone (linea nigra) extending from the symphysis pubis to ensiform cartilage may be visible as early as 20th week.
2. Striae (both pink and white) of varying degree are visible in the lower abdomen.

Palpation

Fundal height is increased with progressive enlargement of the uterus. Approximate duration of pregnancy can be ascertained by noting the height of the uterus in relation to different levels in the abdomen (Fig. 5.5).

The height of the uterus is midway between the symphysis pubis and umbilicus at 16th week; at the level of umbilicus at 24th week and at the junction of the lower third and upper two-third of the distance between the umbilicus and ensiform cartilage at 28th week.

The uterus feels soft and becomes ovoid in shape.

Contractions (Braxton–Hicks):

Uterine contraction in pregnancy has been named after Braxton–Hicks who first described its entity during pregnancy. From the very early weeks of pregnancy, the uterus undergoes spontaneous contraction. This can be felt during bimanual palpation in early weeks or during abdominal palpation when the uterus feels firmer at one moment and soft at another. Although spontaneous, the contractions may

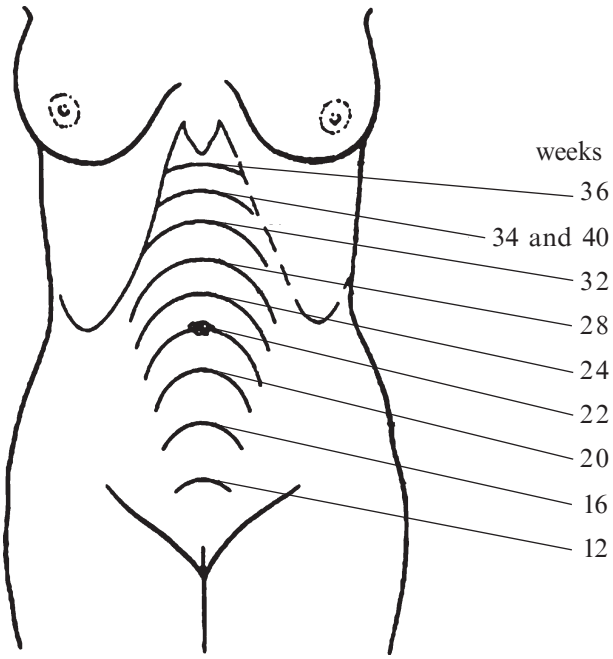


Fig. 5.5

be excited by rubbing the uterus. The contractions are irregular, infrequent, spasmodic and painless without any effect on dilatation of the cervix. The patient is not conscious about the contractions.

Palpation of fetal parts can be felt distinctly by the 20th week. The findings are of value not only to diagnose pregnancy but also to identify the presentation and position of the fetus in later weeks.

External ballottement is usually elicited as early as 20th week when the fetus is relatively smaller than the volume of the amniotic fluid.

AUSCULTATION OF THE FETAL HEART

The fetal heart may be heard with a fetal stethoscope pressed on the abdomen, over the back of the fetus, from about 24–26 weeks (Fig. 5.6).

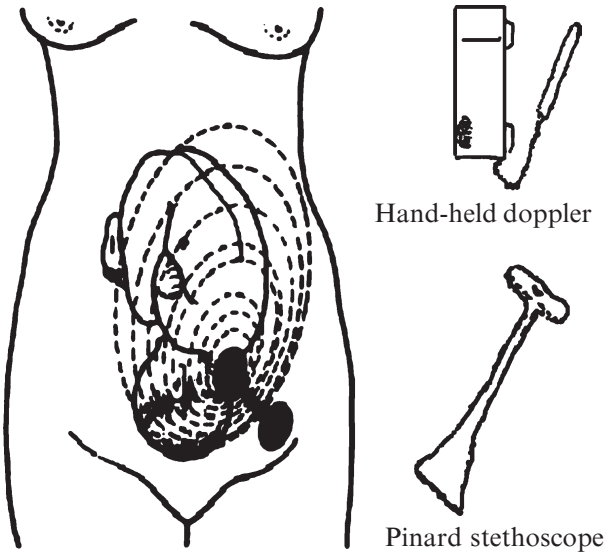


Fig. 5.6

The sounds resemble the tick of a watch under a pillow. Its location varies with the position of the fetus. The rate varies from 110–160 beats per minute.

SONOGRAPHY

Routine sonography at 18–20 weeks permits a detailed survey of fetal anatomy, placental localization and the integrity of the cervical canal. Gestational age is determined by measuring the biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC) and femur length (FL). It is most accurate when done between 12 and 20 weeks (variation ± 8 days). BPD is measured at the level of the thalami and cavum septum pellucidum. BPD is measured from the outer edge of the skull to the inner edge of the opposite side (Fig. 5.7).

Fetal organ anatomy is surveyed to detect any malformation. Fetal viability is determined by real-time ultrasound. Absence of fetal cardiac motion confirms fetal death.

Magnetic Resonance Imaging (MRI) can be used for fetal anatomy survey, biometry and evaluation of complex malformations. Radiologic evidence of fetal skeletal shadow may be visible as early as 16th week.

LAST TRIMESTER (29–40 WEEKS)

ABDOMINAL EXAMINATION

Palpation

Palpation of the fetal parts and their identification become much easier. Lie, presentation and position of the fetus are determined.

Fetal movements are easily felt.

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 32nd week 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 (Fig. 5.8).

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.

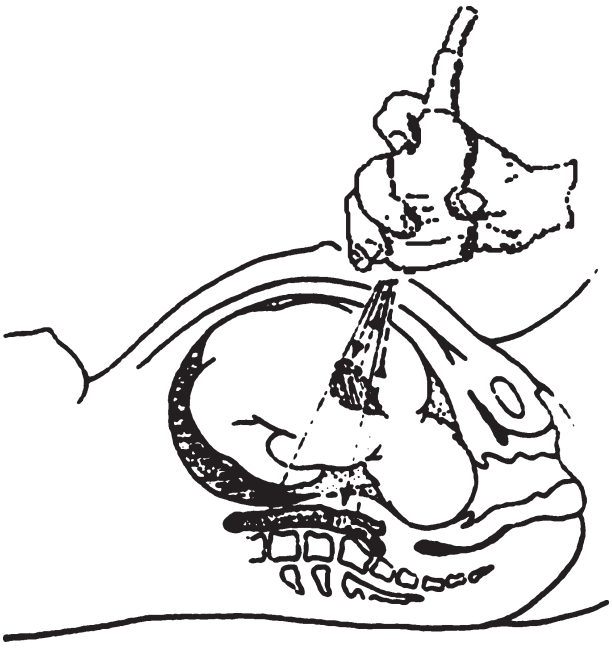


Fig. 5.7

FETAL HEART SOUNDS

FHS is heard distinctly in areas corresponding to the presentation and position of the fetus. FHS may not be audible in cases of maternal obesity, polyhydramnios (Fig. 5.9).

SONOGRAPHY

Sonography — gestational age estimation by BPD, HC, AC and FL is less accurate (variation ± 3 weeks). Fetal growth assessment can be made

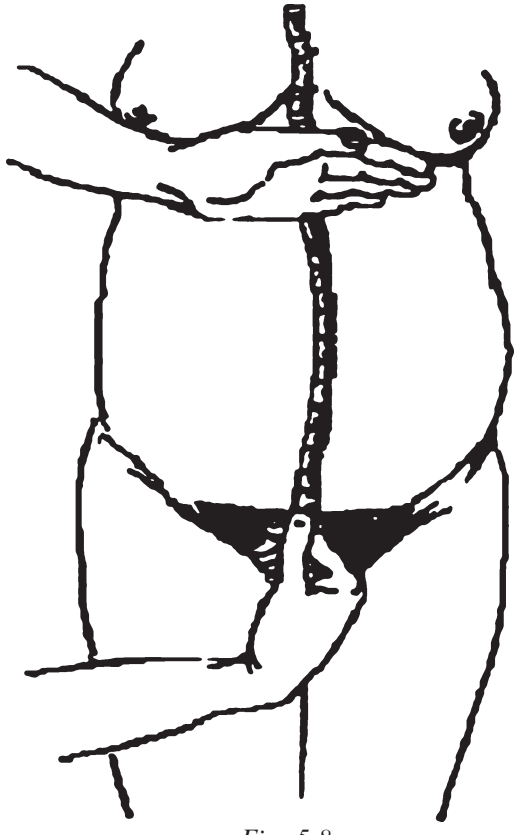
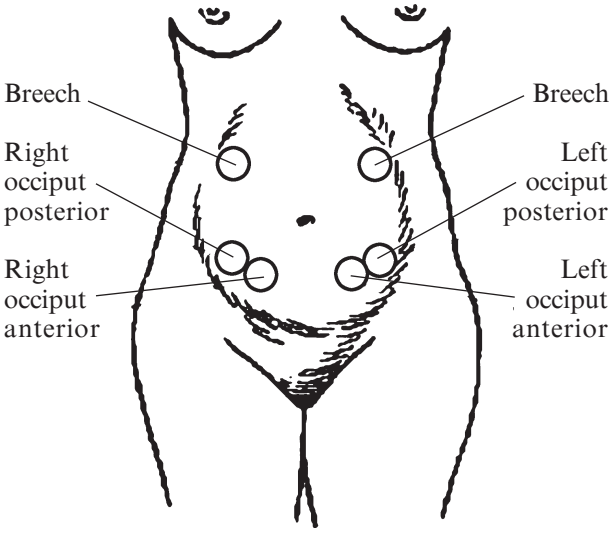


Fig. 5.8



Fetal heart tone positions

Fig. 5.9

provided an accurate dating scan has been done in the first or second trimester. Fetal AC at the level of the umbilical vein is used to assess gestational age and fetal growth profile (IUGR or macrosomia). Fetal weight estimation can be done using tables. Amniotic fluid volume assessment is done to detect oligohydramnios or polyhydramnios. Placental anatomy: Location (fundus or previa), thickness (placentomegaly in diabetes) or other abnormalities are noted. Other information: fetal presentation and organ anatomy as done in the first and second trimester are surveyed again.

ESTIMATION OF GESTATIONAL AGE AND PREDICTION OF EXPECTED DATE OF DELIVERY (EDD)

Estimation of gestational age and thereby forecasting the EDD is not only the concern of the individual but it is invaluable in the diagnosis of intrauterine growth retardation of the fetus or management of high risk pregnancy. Gestational age is about 280 days calculated from the first day of the last normal menstrual period (LMP). Accurate LMP is the most reliable parameter for estimation of gestational age. But in a significant number of cases (20–30%), the patients either fail to remember the LMP or report inaccurately. The matter becomes complicated when the conception occurs during lactation amenorrhea or soon following withdrawal of contraceptive pills (ovulation may be delayed for 4–6 weeks) or in cases with bleeding in early part of pregnancy (Fig. 5.10).

Date of fertile coitus:

If the patient can remember the date of the single fertile coitus with certainty, it is quite reliable to predict the expected date of delivery with accuracy of 50% within 7 days on either side. As previously men-

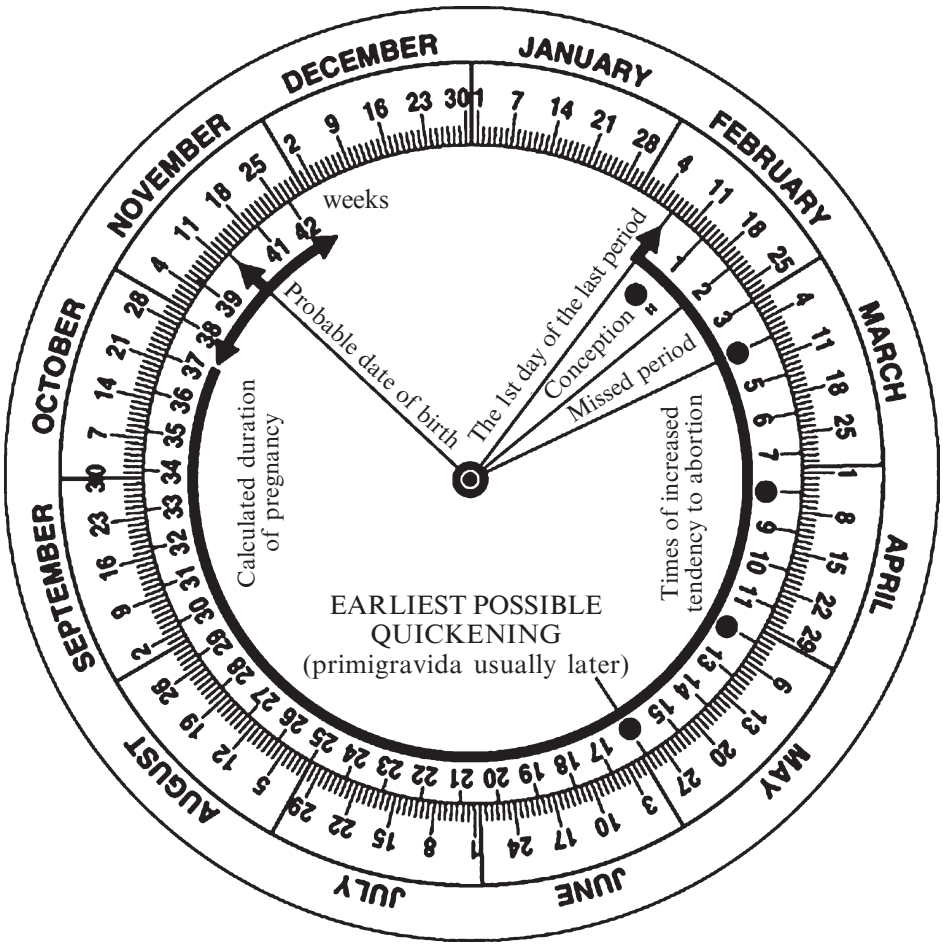


Fig. 5.10

tioned, 266 days are to be added to the date of the single fertile coitus to calculate the expected date.

The expected date of delivery (EDD) can be *calculated from LMP using Naegele's rule*: add 7 days to the LMP and subtract 3 months.

This rule is based on a menstrual cycle of 28 days and assumes ovulation in mid cycle. Where the cycle is regularly greater or less than 28 days the calculation should be adjusted accordingly, e.g. add a further 7 days for a 35 days cycle and subtract a further 7 for a 21 day cycle.

Most often done with an *obstetric calendar* ('wheel').

Fetal movement. Enquire about the first time the mother felt fetal movement: a supposable date of delivery can be calculated by the sensation by a pregnant woman of the fetal movements: in women who give birth for the first time 20 weeks are added to this date, who give birth at the second time — 18 weeks are added.

Objective signs:

1. Height of the uterus above the symphysis pubis in relation to the landmarks on the abdominal wall or SFH.

2. Size of the fetus, change in the uterine shape, volume of liquor amnii, hardening of the skull and girth of the abdomen are of value in assessing the maturity of the fetus specially if the examinations are done by the same person at intervals.

3. Vaginal examination: If the cervix becomes shorter and dilated, the labor is fairly not far off. But labor may start even with a long and closed cervix.

In addition, *obstetric ultrasound examination* is the most accurate measurement available in the determination of gestational age. Pelvic examination by an experienced examiner is accurate, within 1 to 2 weeks, in determining gestational age until about 14 to 16 weeks, at which time the lower uterine segment begins to form, thereby making clinical estimation of gestational age less accurate.

ESTIMATION OF FETAL WEIGHT

Approximate prediction of the fetal weight is more important than the mere estimation of the uterine size. This is more important prior to induction of labor or elective cesarean section (Fig. 5.11).

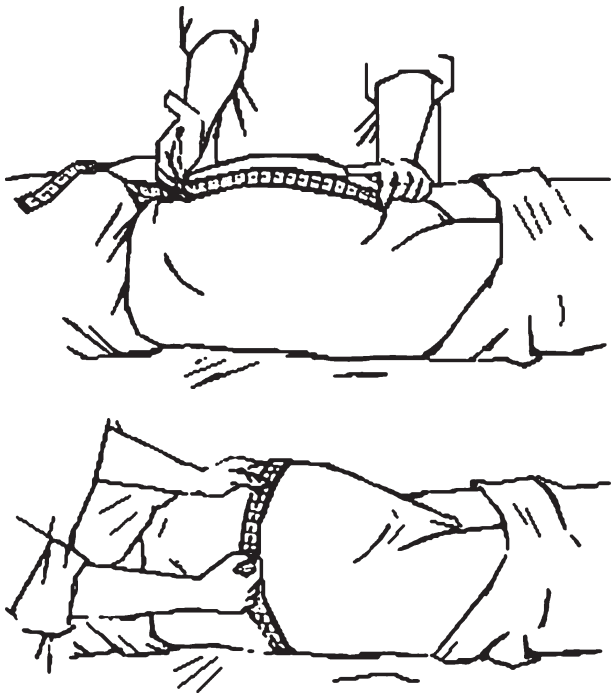


Fig. 5.11

Determine the estimated fetal weight (g).

The estimated fetal weight:

"the circumference of the abdomen" x "the height of the fundus of the uterus" (± 200 g).

The height of the fundus in centimeters should equal approximately the weeks of gestation.

Sonography

Fetal weight has been estimated by combining a number of biometric data, e.g. BPD, HC, AC and FL. Estimated fetal weight likely to be within 10 percent of actual weight.

Chapter 6
FETAL ORIENTATION

The fetus lies inside the uterus in a closed sac filled with liquor amnii. It has enough freedom of movement until the later months of pregnancy, when it becomes relatively fixed.

The fetal lie is the relationship between the sagittal plane of the fetus and the mother. Till then, periodic examination is essential to note its ***lie, presentation, attitude*** and ***position*** (Fig. 6.1).

***Lie*:** The lie refers to the relationship of the long axis of the fetus to the long axis of the centralised uterus or maternal spine, the commonest lie being longitudinal (99.5%). The lie may be transverse or oblique. Uterine or fetal anomalies may increase the rate of transverse or oblique lie. Sometimes the lie is unstable until labor sets in, when it becomes either longitudinal or transverse.

Fetal presentation refers to the part of the fetus that is closest to the pelvic inlet.

Only a cephalic presentation is normal after 32 weeks. At 30 weeks, however, up to 25% of babies present by the breech.

Accordingly, the presentation may be *head presentation* (97%) and *breech presentation* (3–4%) and *shoulder* (0.5%) [18, 26, 45, 50] (Fig. 6.2).

When more than one part of the fetus presents, it is called compound presentation.

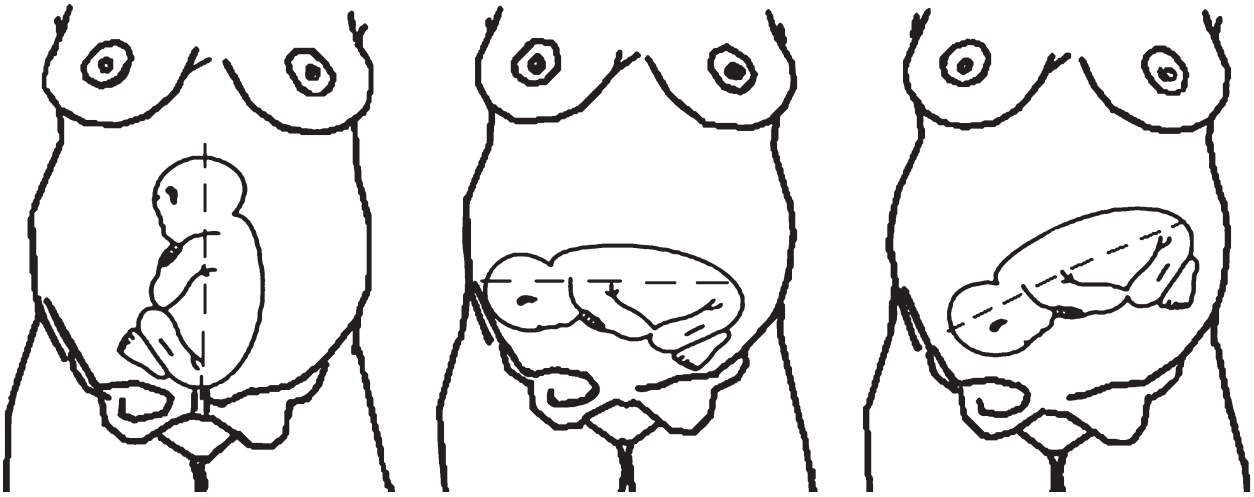
Presenting part: The presenting part is defined as the part of the presentation which overlies the internal os and is felt by the examining finger through the cervical opening. Thus, in cephalic presentation, the presenting part may be vertex (commonest), brow or face, depending upon the degree of flexion of the head. However, the term presentation and presenting part are often used synonymously and expressed more commonly in clinical practice according to the latter definition.

In full flexion, every fetal joint is flexed. This gives a *vertex* presentation, the only normal presentation. Sometimes the head may be extended. This gives a *face* presentation, which is highly abnormal (Fig. 6.3).

Breech presentation is classified into several subcategories: complete (hips and knees flexed), frank (hips flexed, knees extended), and incomplete or footling (one or both lower extremities presenting).

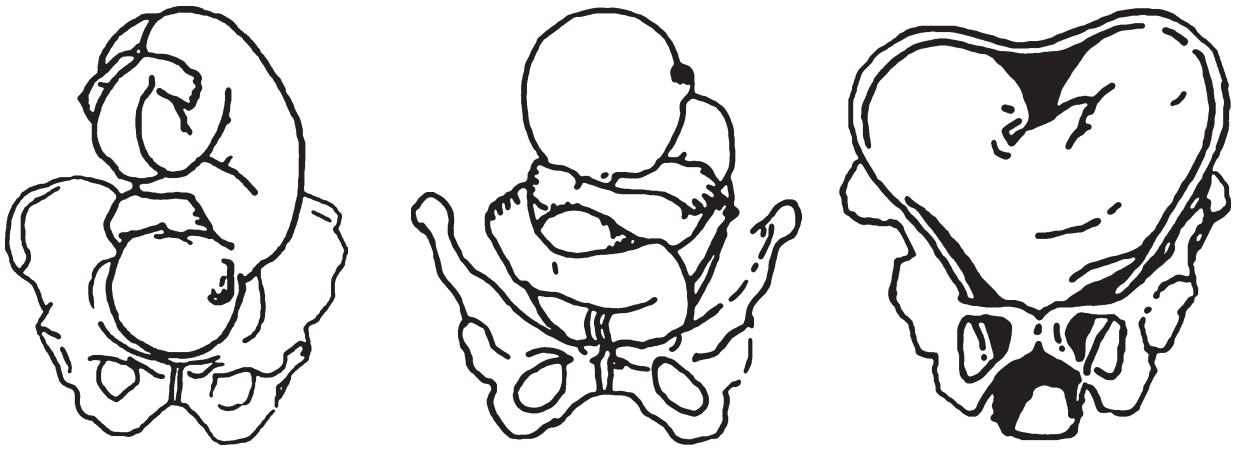
***Attitude*:**

The relation of the different parts of the fetus to one another is called the attitude of the fetus. The universal attitude is the posture of the fetus (flexion). There may be exceptions to this universal attitude and extension of the head may occur (deflexed vertex, brow or face presentation, according to the degree of extension), or the legs may become extended in breech (Fig. 6.4, Fig. 6.5).



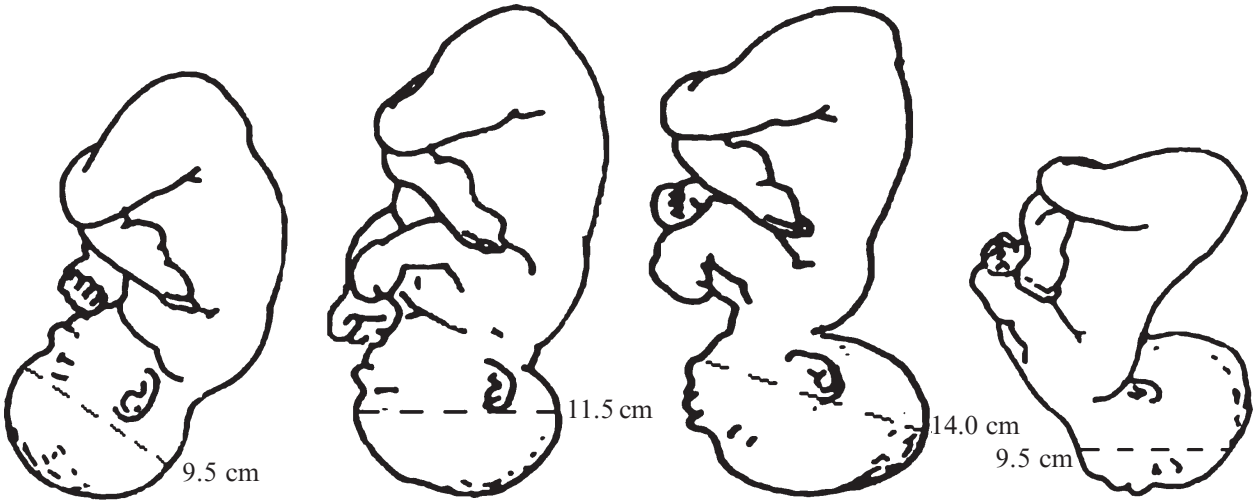
Longitudinal Transverse Oblique

Fig. 6.1



Vertex Breech Shoulder

Fig. 6.2



Vertex (weel-flexed head) Vertex (deflexed head) Brow Face

Fig. 6.3

Flexion Deflexion Extension

Fig. 6.4

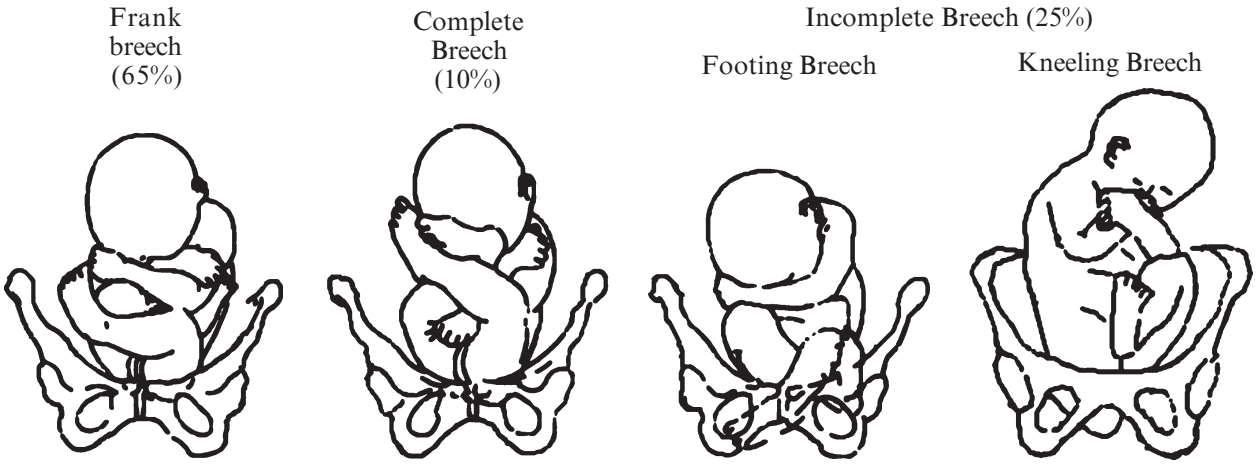


Fig. 6.5

Fetal attitude and dimensions of a term-size fetus:

1. Full flexion presents the smallest circumference of the fetal head to the narrower planes of the pelvis.
2. Military attitude usually changes to full flexion with descent into the pelvis.
3. Brow presentation usually converts to full flexion or a face presentation, as the occipitomental diameter is too large for all except the largest pelves to accommodate.
4. Face presentation shows dimensions that allow descent through the pelvis, unless the chin is posterior. Persistent mentum posterior must be delivered by cesarean section.

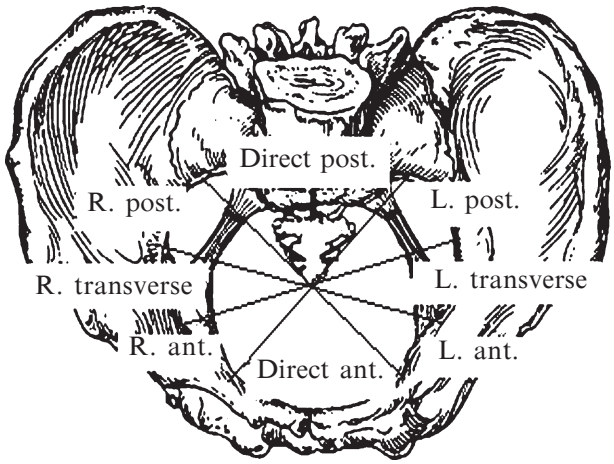


Fig. 6.6

THE POSITION OF THE BABY

The position of the baby 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 eight positions with each presenting part (Fig. 6.6).

Denominator is an arbitrary bony fixed point on the presenting part, which comes in relation with the various quadrants of the maternal pelvis.

The denominators of the different presentations: occiput in vertex, mentum (chin) in face, frontal eminence in brow, sacrum in breech and acromion in shoulder.

Anterior, posterior, right or left position is referred in relation to the maternal pelvis, with the mother in erect position (Fig. 6.7).

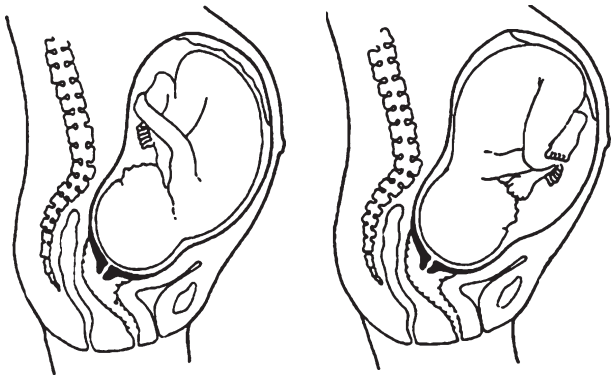


Fig. 6.7

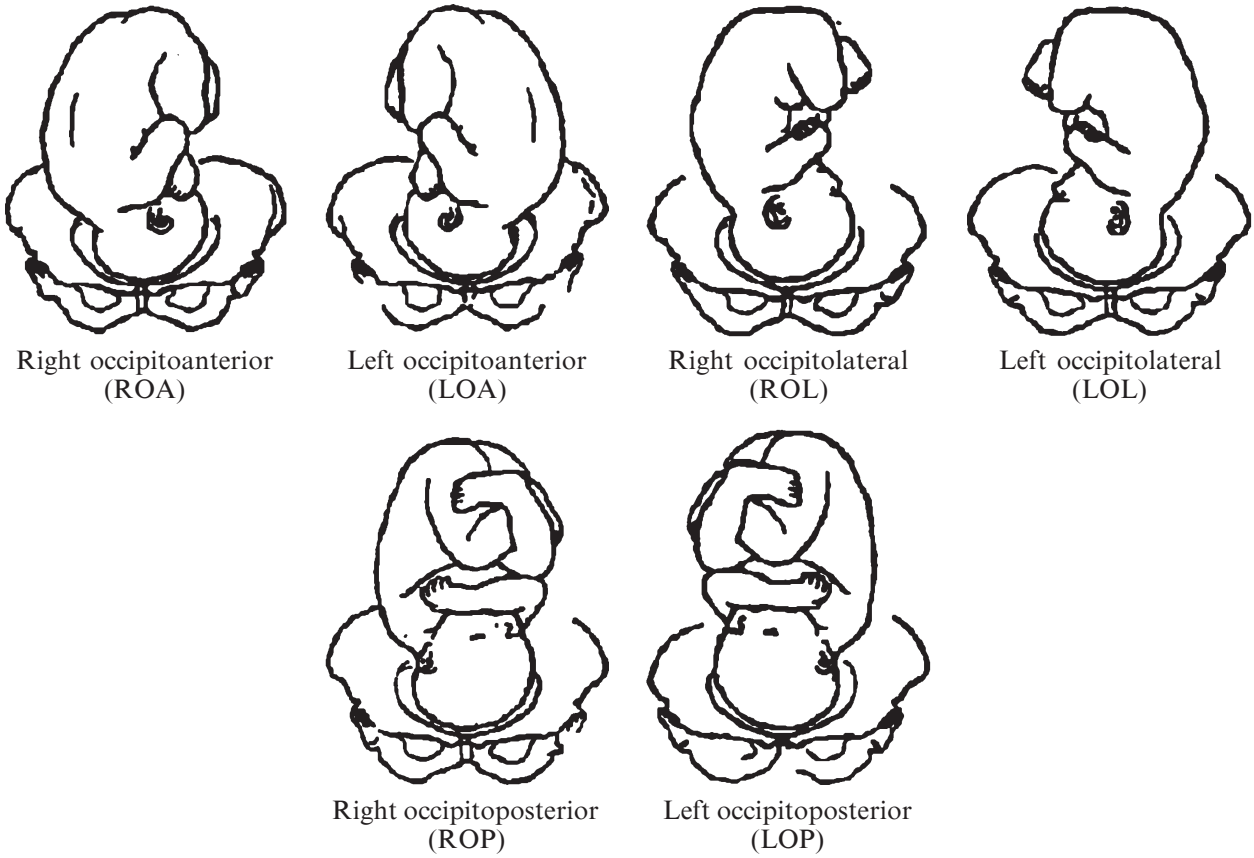


Fig. 6.8

However, some have retained the conventional description of the vertex positions. Vertex occupying the left anterior quadrant of the pelvis is the commonest one and is called left occipitoanterior (LOA). This is the first vertex position. Similarly, right occipitoanterior (ROA) is the second vertex; right occipitoposterior (ROP) third vertex and left occipitoposterior (LOP) is the fourth vertex position (Fig. 6.8).

The denominator of the vertex is the occiput and there are recognised positions.

The lateral and anterior positions are regarded as normal as the occiput will rotate forward in labor.

If the fetus is in a posterior position (i.e. the occiput rotates towards the sacrum and the fetus faces forwards), the labor may be longer and more difficult.

Fetal position has been traditionally evaluated with the use of *Leopold maneuvers.* These are initiated at midpregnancy, when fetal body parts are more clearly identified. The maneuvers consist of four parts; the first three are performed with the examiner standing to one side of the patient and facing her head and the last with the examiner facing the patient's feet.

The presentation of the fetus can be appreciated on clinical exam with the use of Leopold' maneuver:

1. The first maneuver permits identification of which fetal pole — that is, cephalic — occupies the uterine fundus. The breech gives the sensation of a large, nodular mass, whereas the head feels hard and round and is more mobile and ballottable (Fig. 6.9, *a*).

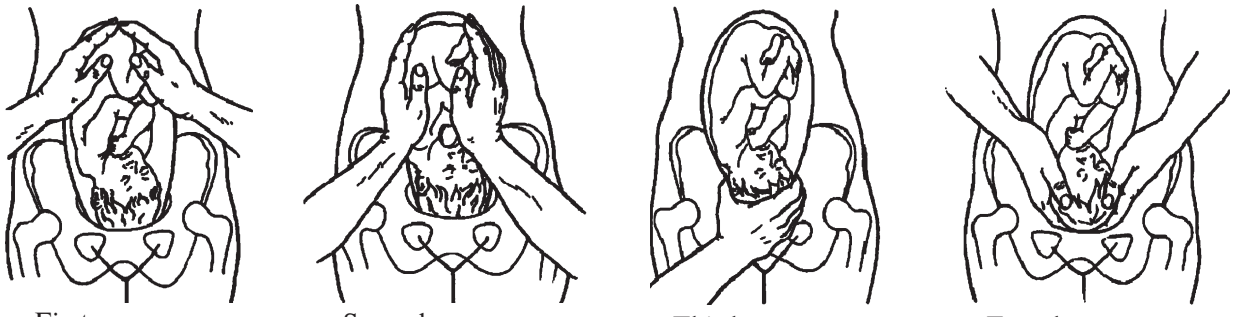


Fig. 6.9

2. Performed after determination of fetal lie, the second maneuver is accomplished as the palms are placed on either side of the maternal abdomen, and gentle but deep pressure is exerted. On one side, a hard, resistant structure is felt — the back. On the other, numerous small, irregular, mobile parts are felt — the fetal extremities. By noting whether the back is directed anteriorly, transversely, or posteriorly, the orientation of the fetus can be determined (Fig. 6.9, *b*).

3. The third maneuver is performed by grasping with the thumb and fingers of one hand the lower portion of the maternal abdomen just above the symphysis pubis. If the presenting part is not engaged, a movable mass will be felt, usually the head. The differentiation between head and breech is made as in the first maneuver. If the presenting part is deeply engaged, however, the findings from this maneuver are simply indicative that the lower fetal pole is in the pelvis, and details are then defined by the fourth maneuver (Fig. 6.9, *c*).

4. To perform the fourth maneuver, the examiner faces the mother's feet and, with the tips of the first three fingers of each hand, exerts deep pressure in the direction of the axis of the pelvic inlet. In many instances, when the head has descended into the pelvis, the anterior shoulder may be differentiated readily by the third maneuver (Fig. 6.9, *d*).

Chapter 7
ANTENATAL CARE

Proper antenatal care (ANC) begins before pregnancy. Even for an apparently normal, healthy couple may be advantages in discussing planning pregnancy with a professional adviser — a family doctor or a family-planning doctor. The woman can meet with both the psychological and medical problems within the context of health care delivery system, culture and religion in which the woman lives. ANC programs should address risk assessment, health promotion and care provision [25].

PURPOSE OF THE CARE IN PREGNANCY

The purpose are:

1. To screen the 'high risk' cases.
2. To prevent or to detect and treat any earliest complications.
3. To ensure continued risk assessment and to provide ongoing primary preventive health care.
4. To educate the mother about the physiology of pregnancy and labor by demonstrations, charts and diagrams (mother craft classes), so that fear is removed and psychology is improved.
5. To discuss with the couple questions about the place, time and mode of delivery, provisionally and care of the newborn.
6. To motivate the couple about the need of family planning and also an appropriate advice to couple seeking medical termination of pregnancy.

ANTENATAL VISITS

Regular monitoring of the mother and fetus is essential for identifying complications that may arise during pregnancy and to provide assurance and support for mother and family, especially for first pregnancies or when previous pregnancies have been complicated or had unfortunate outcomes. Ideally, the first visit should be at 8–10 weeks of gestation. For a patient with a normal pregnancy, periodic antepartum visits at 4-week intervals are usually scheduled until 28 weeks, at 2- to 3-week intervals between 28 and 36 weeks, and weekly thereafter. Patients with high-risk pregnancies or those with ongoing complications usually are seen more frequently, depending on the clinical circumstances. At each visit, patients are asked about how they are feeling and if they are having any problems, such as vaginal bleeding, nausea and vomiting, dysuria, or vaginal discharge. After quickening, patients are asked if they continue to feel fetal movement, and if it is the same or less since the last antepartum visit. Decreased fetal movement after the time of fetal viability is a warning sign requiring further evaluation of fetal well-being.

Use of a standardised record within a perinatal health-care system greatly aids antepartum and intrapartum management.

The new WHO ANC critical times: 8–12 weeks, 24–26 weeks, 32 weeks, 36–38 weeks.

The first visit: should occur in the first trimester, around or preferably before 12 weeks.

The second visit: no later than 2 weeks after the first visit.

The third visit: should be scheduled at 19–21 weeks after having the second ultrasound.

The fourth visit: should take place between 25–26 weeks of gestational age.

The sixth visit: ideally the booking visit should be at 34–35 weeks.

The seventh visit: 38 weeks.

The eighth visit: 40 weeks.

The ninth visit: 41 weeks.

DEFINITIONS

There are several definitions pertinent to establishment of an accurate prenatal record.

1. *Nulligravida* — a woman who currently is not pregnant nor has ever been pregnant.
2. *Gravida* — a woman who currently is pregnant or has been in the past, irrespective of the pregnancy outcome. With the establishment of the first pregnancy, she becomes a primigravida, and with successive pregnancies, a multigravida.
3. *Nullipara* — a woman who has never completed a pregnancy beyond 22 weeks' gestation. She may not have been pregnant or may have had a spontaneous or elective abortion(s) or an ectopic pregnancy.
4. *Primipara* — a woman who delivered only one fetus or fetuses alive or dead with an estimated length of gestation of 22 (500-g birth weight) or more weeks.
5. *Multipara* — a woman who has completed two or more pregnancies to 22 weeks' gestation or more. Parity is determined by the number of pregnancies

reaching 22 weeks. Moreover, stillbirth does not lower this number.

6. A *puerpera* is a woman who has just given birth.

Pregnancy (gestation) is the maternal condition of having a developing fetus in the body. The human conceptus from fertilisation through the eighth week of pregnancy is termed an embryo; from the eighth week until delivery, it is a fetus. For obstetric purposes, the duration of pregnancy is based on gestational age: the estimated age of the fetus is calculated from the first day of the last (normal) menstrual period (LMP), assuming a 28-day cycle. Gestational age is expressed in completed weeks. This is in contrast to developmental age (fetal age), which is the age of the offspring calculated from the time of implantation.

The term *gravid* means pregnant, and gravidity is the total number of pregnancies (normal or abnormal). Parity is the state of having given birth to an infant or infants weighing 500 g or more, alive or dead.

Live birth is the complete expulsion or extraction of a product of conception from the mother, regardless of the duration of pregnancy, which, after such separation, breathes or shows other evidence of life (e.g., beating of the heart, pulsation of the umbilical cord, or definite movements of the involuntary muscles) whether or not the cord has been cut or the placenta detached. An infant is a live-born individual from the moment of birth until the completion of 1 year of life.

Perinatology — a science which studies the perinatal period (from the Greek word "peri" — around, and Latin "natus" — birth).

The *perinatal period* includes the late fetal period (22 weeks of intrauterine development and till the beginning of birth), intranatal (during birth) and early neonatal (from birth to 6 days old). Diseases which occur during the perinatal period are called perinatal pathology. As a rule, the displays of these diseases last even in older babies, especially in the late neonatal period (till 28 days old).

A still-born baby is a baby that at the moment of birth had not respiratory movement and it could not be gained artificially. A heartbeat in such a baby can be heard for some time.

Still-born babies and the death rate of babies in the first 7 days after birth is called *perinatal death rate.* To determine the perinatal death rate, it is necessary to calculate in ppm (per mille) the difference between the number of still-born babies and those that died within the first 7 days after birth and the total number of births. The perinatal period and of course the pathology and death rate are divided into antenatal (before birth), intranatal (during birth) and postnatal (after birth), or neonatal.

Perinatal center — a treatment and preventive establishment which gives all kinds of qualified, highly technological and expensive medical hospital care in the field of obstetrics, gynecology, neonatology and neonatal surgeries and also gives out-patient, advisory-diagnostic and medical-rehabilitation care to women and children of early age.

Perinatal diagnostics (PD) is directed on revealing congenital and hereditary pathologies (CHP).

Perinatal care of the fetus should not begin at the moment the child is born and even at the moment the pregnancy occurred, but much earlier. It should begin at the moment a baby girl is born, who is considered as a potential mother.

The health of the future generation depends on the reproductive health of women and young women, who start the reproductive process today and who are the keepers of the gene pool of the nation. Thus, the condition of a newborn is closely connected to the condition of the mother's reproductive health. Oftentimes, girl teenagers enter the reproductive process being anatomically and physiologically immature, socially not adapted, which essentially reduces the health of the mother and newborn.

Questions about perinatal and newborn care are answered at the female consultations and also specialized obstetrical hospitals and branches of maternity hospitals, sanatoriums for pregnant woman, specialized therapeutic and pediatric hospitals, clinics, polyclinics for adults and children. With introduction of a family doctor into the system of public health services, they will play a big role in solving these problems.

THE FIRST EXAMINATION

All the women should be assessed early in pregnancy.

OBSTETRIC AND GYNECOLOGIC EXAMINATION

Personal history:

Name, age, marital status, planned or unplanned pregnancy, marital status, occupation.

History of present pregnancy:

Get information on the following points:

— Gravidity (i.e. number of pregnancies, including the current one).

— Parity (i.e. number of births beyond 22 weeks' gestation).

— Menstrual history: date of the LMP.

— Calculation of the expected date of delivery (EDD).

— Enquire about the first time the mother felt fetal movement.

— Presence of antenatal care elsewhere. Place and number of visits.

— Elaboration of chief complaints/

— Danger symptoms of pregnancy (vaginal bleeding, severe headache, blurring of vision, epigastric or severe abdominal pain, profuse vaginal discharge, absence or reduction of fetal movement, fever, persistent vomiting).

— Common complaints in pregnancy (like nausea and vomiting, weakness).

— Pregnancy — unplanned, unwanted and unsupported.

Past obstetric history:

Previous pregnancies: each prior pregnancy should be reviewed in chronological order and the following information recorded:

— Date of delivery (or pregnancy termination).

— Length of gestation: abortion, preterm, term, post term.

— Duration of gestation (recorded in weeks). When correlated with birth weight, this information allows an assessment of fetal growth patterns. The gestational age of any spontaneous abortion is of importance in any subsequent pregnancy.

— Duration of labor (recorded in hours). This may alert the physician to the possibility of an unusually long or short labor.

Type of delivery (or method of terminating pregnancy). This information is important for planning the method of delivery in the present pregnancy.

— Previous stillbirth or neonatal loss.

— History of three or more consecutive spontaneous abortion.

— Birth weight of last baby <2500 g.

— Birth weight of reproductive last baby >4000 g.

— Maternal complications during last pregnancy: urinary tract infections, vaginal bleeding, hypertension or preeclampsia/eclampsia, and postpartum complications may be repetitive; such knowledge is helpful in anticipating and preventing problems with the present pregnancy.

— Previous surgery (myomectomy, removal of septum, fistula repair, cone biopsy, CS, repaired ruptured uterus, ischemic-cervical insufficiency).

— Any unexpected event (pain, vaginal bleeding, others: specify).

— Periods of exclusive breast-feeding: When? How long?

Gynecologic history:

— Family planning methods: use, type, duration and side effects.

— Sexual history: assess risk of sexually transmitted infections and HIV/AIDS.

— Gynecology operations: female genital mutilation, laparatomy, dilatation and curettage, evacuation and curettage, manual vacuum aspiration.

— Menstrual history (age of menarche, interval of period 21–36 days, duration 1–8 days, regularity of cycle, hormonal contraception used three months prior to LMP).

Past medical and surgical history:

— Specific diseases and conditions: diabetes mellitus, renal disease, cardiac disease, chronic hypertension, tuberculosis, past history of HIV

— related illnesses and HAART, varicose veins, deep venous thrombosis, other specific conditions depending on prevalence in service area (for example, hepatitis, malaria), other diseases, past or chronic; allergy(-ies)

— Operations other than CS

— Blood transfusions. Rhesus (D) antibodies

— Current use of medicines — specify, any medications

— Social history: detail the woman's alcohol, tobacco and illicit drug intake? giving appropriate advice.

GENERAL PHYSICAL EXAMINATION

General physical examination (Fig. 7.1):

• General appearance.

• Measurement of blood pressure, pulse, body temperature, measurement of body weight (all pregnant women at every visit).

• The external examination; palpation of the thyroid gland; auscultation of the heart and lungs; inspection and palpation of the breast; palpation of the lymph nodes, measuring height, body-weight index (during the registration) [45].

Calculate body mass index:

$$\text{BMI} = \frac{\text{weight, kg}}{\text{height}^2, \text{m}^2}$$

Excessive weight gain or high pre-existing maternal weight is associated with increased risk factor for the infant in terms of birth trauma and delivery by Cesarean section.

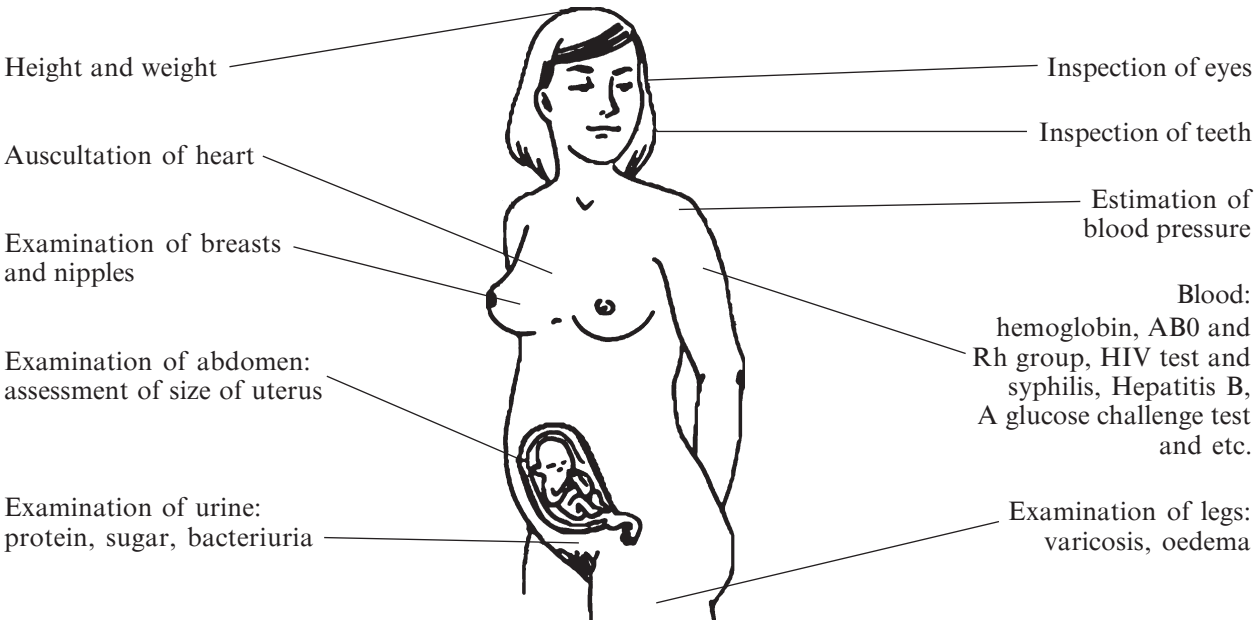


Fig. 7.1

Table 2

BMI Assessment and Recommended Weight Gain

BMI	Assessment of weight	Recommended weight gain
Less than 18.5	Underweight	12.5–18 kg
18.5–24.0	Normal weight	11.5–16 kg
24.0 and above	Overweight	7.0–11.5 kg

OBSTETRICAL EXAMINATION

Abdominal examination:

Palpation

Examination of the abdomen is critical in the evaluation of the gynecologic patient (Fig. 7.2).

The contour, whether flat, scaphoid, or protuberant should be noted. The protuberant appearance may suggest ascites. The presence and distribution of hair, especially in the area of the escutcheon, should be recorded, as should the presence of striae or operative scars. Abdominal tenderness must be determined by placing one hand flat against the abdomen in the non painful areas initially, then gently and gradually exerting pressure with the fingers of the other hand.

Pelvic examination

The initial pelvic examination should be done early in the prenatal period and should include the following:

• Inspection of the external genitalia, vagina, and cervix.

• Collection of cytologic specimens from the exocervix (or ectocervix) and superficial endocervical canal.

• Palpation of the cervix, uterus, and adnexa. The initial estimate of gestational age by uterine size becomes less accurate as pregnancy progresses.

• Rectal and rectovaginal examinations are also important aspects of this initial pelvic evaluation.

Vaginal examination in pregnancy

Steps of vaginal examination:

The patient must empty her bladder prior to examination and is placed in the dorsal position with

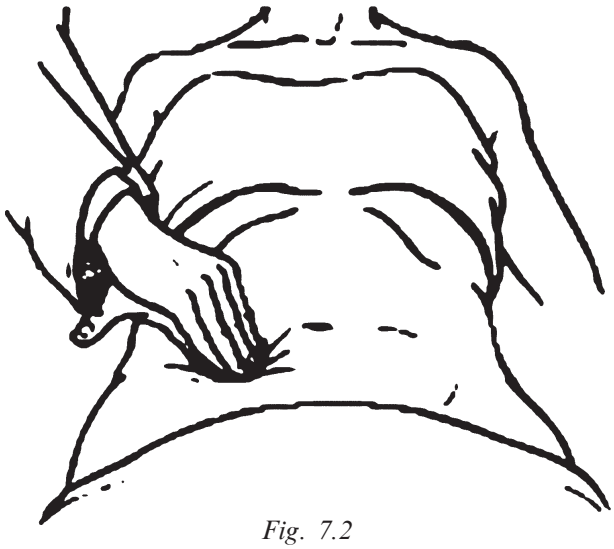


Fig. 7.2

the thighs flexed along with the buttocks placed on the foot end of the table.

Inspection: By separating the labia — using the left two fingers, the character of the vaginal discharge, if any, is noted. Presence of cystocele or uterine prolapse or rectocele is to be elicited.

The vulva is inspected for: swelling, inflammation, ulceration.

The urethral orifice is inspected for: urethritis, caruncle.

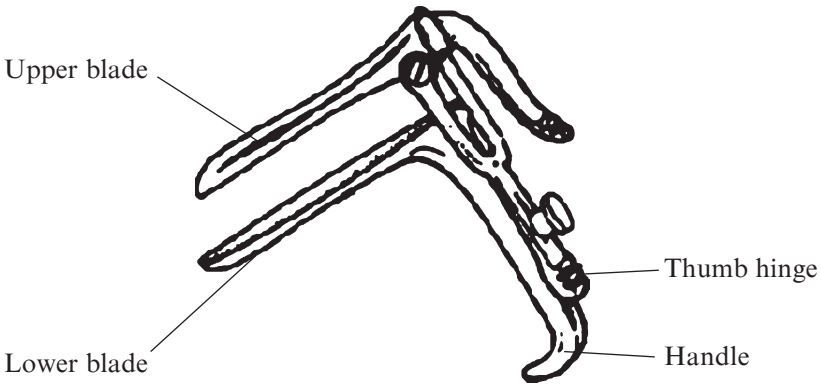
Examination with a speculum

This is an essential part of the gynaecological examination.

The bivalve speculum (Cusco's) consists of two limbs joined at the handle; it is made in various sizes and is useful for general cervical and upper vaginal examination (Fig. 7.3, *a*).

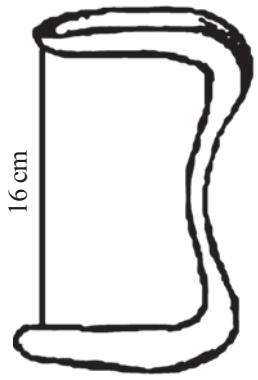
A Sim's speculum holding back the posterior wall gives a good view of the cervix and anterior vaginal wall (Fig. 7.3, *b*).

After gently spreading the labia to expose the introitus, the speculum should be inserted with the blades entering the introitus transversely, then directed posteriorly in the axis of the vagina with pressure exerted against the relatively insensitive perineum to avoid contacting the sensitive urethra. As the ante-



Speculum (Cusco's)

a



Sims Duckbill Speculum

b

Fig. 7.3

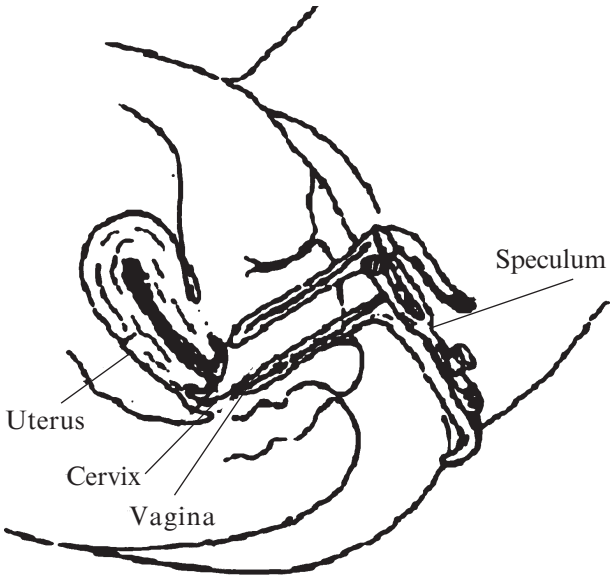


Fig. 7.4

rior blade reaches the cervix, the speculum is opened to bring the cervix into view. As the vaginal epithelium is inspected, it is important to rotate the speculum through 90 degrees, so that lesions on the anterior or posterior walls of the vagina ordinarily covered by the blades of the speculum are not overlooked (Fig. 7.4).

The cervix should be inspected to determine its size, shape, and colour. The nulliparous patient generally has a conical, unscarred cervix with a circular, centrally placed os; the multiparous cervix is generally bulbous and the os has a transverse configuration.

Any purulent cervical discharge should be cultured. A cervical cytologic smear (Papanicolaou, or Pap, smear), liquid-based sampling, or DNA probe for human papillomavirus (HPV) should be taken before the speculum is withdrawn. For the traditional Pap smear the exocervix (or ectocervix) is gently scraped with a wooden spatulum or plastic broom, and the endocervical tissue gently sampled with a cytobrush (Fig. 7.5).

Bacterial Smear

Bacteriological smear helps to assess the condition of the female reproductive system. Rod flora in smear of women may be normal or show pathogenic deviations. In the second case we are talking about gynecological diseases and even infections that infect exclusively sexually transmitted.

Taking bacterial smear on flora is carried out in all women as a preventive measure. However, its necessity can specify, and the patient's complaints, which she expresses to gynaecologist (abdominal pain, unpleasant odour and burning sensation). These symptoms are the first sign of bacterial vaginosis. If suspected, the doctor must confirm the analyses and make bacterioscopy — the study of the vaginal flora under the microscope.

Vaginal flora and pH

1. Normal vaginal flora is predominantly aerobic with an average of six different species of bacteria.
2. Normal vaginal discharge is clear to white, odourless.
3. Lactic acid helps to maintain a normal vaginal pH of 3.8 to 4.2.
4. Doderlein Bacillus. An anaerobic lactobacillus normally present in the vagina where it continually converts glycogen molecules to produce lactic acid which destroys or inhibits some potentially harmful bacteria entering the vaginal canal.

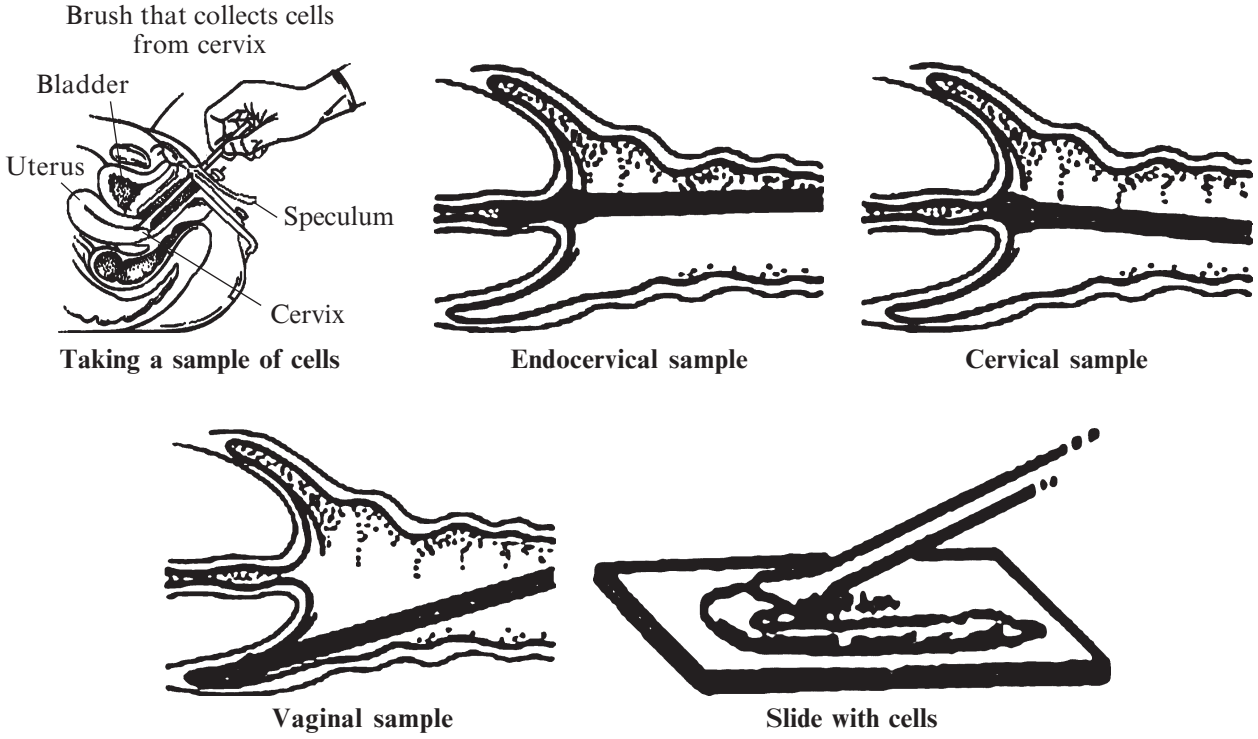


Fig. 7.5

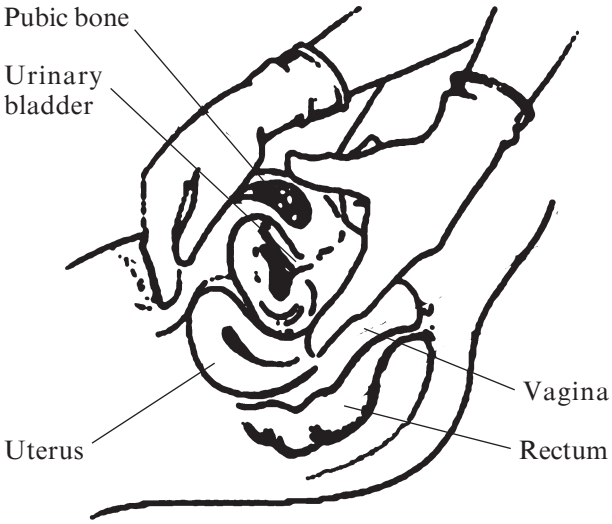


Fig. 7.6. Side view of bimanual exam

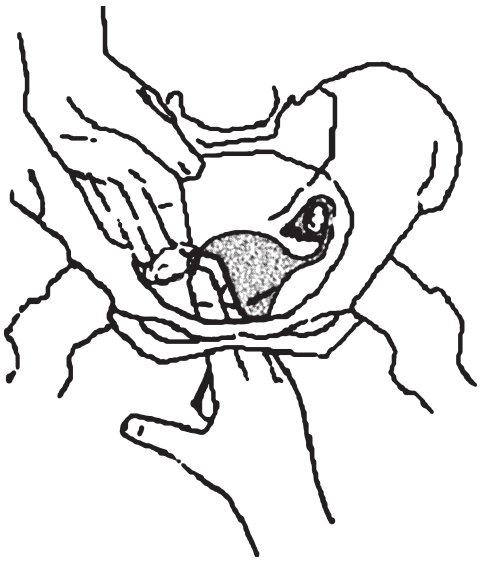


Fig. 7.7

5. Acidic environment and other host immune factors inhibit the overgrowth of bacteria.

Bimanual examination

Vaginal examination can usually be satisfactorily performed by using the index finger alone. This causes less discomfort and muscle spasm. If the vagina is long or voluminous or your fingers are small, a second finger may be needed. The finger(s) should be inserted slowly up to the level of the cervix. Assessment is by bimanual examination, the other hand being on the abdomen above the pubic symphysis. A three-dimensional image of the pelvis is built up from information obtained from both hands, not just the vaginal one (Fig. 7.6).

The cervix is palpated for consistency, contour, size, and tenderness to motion.

The uterus is evaluated by placing the abdominal hand flat on the abdomen with the fingers pressing gently just above the symphysis pubis. With the vaginal fingers supinated in either the anterior or the posterior vaginal fornix, the uterine corpus is pressed gently against the abdominal hand. As the uterus is felt between the examining fingers of both hands, the size, configuration, consistency, and mobility of the organ are appreciated (Fig. 7.7).

By shifting the abdominal hand to either side of the midline and gently elevating the lateral fornix up to the abdominal hand, it may be possible to outline a right (left) adnexal mass. The pouch of Douglas is also carefully assessed for nodularity or tenderness, as may occur with endometriosis, pelvic inflammatory disease, or metastatic carcinoma.

SIGNS OF PREVIOUS CHILDBIRTH

The following are the features which are to be considered in arriving at a diagnosis of having a previous birth.

Breasts become more flabby; nipples are prominent whoever breast-fed their infant; primary areolar pigmentation still remains and so also the white striae.

Abdominal wall is more lax and loose. There may be the presence of silvery white striae and linea alba.

Uterine wall is less rigid and the contour of the uterus is broad and round, rather than ovoid.

Perineum is lax and evidence of old scarring from previous perineal laceration or episiotomy may be found.

Introitus is gaping and there is presence of carunculae myrtiformes.

Vagina is more roomy.

Cervix: Nulliparous cervix is conical with a round external os. In parous women, it becomes cylindrical and the external os is a transverse patulous slit and may admit the tip of the finger (Fig. 7.8).

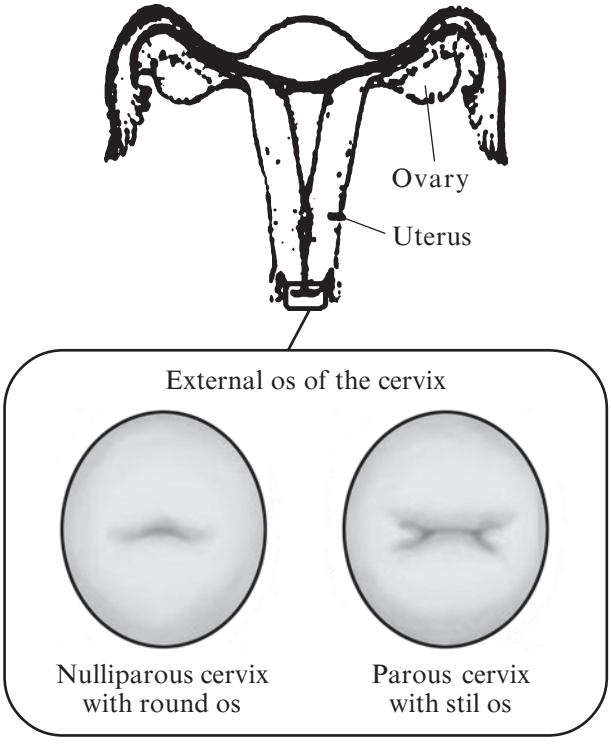


Fig. 7.8

However, as a result of operative manipulation even a nulliparous cervix may be torn and resembles a multiparous cervix [11, 8].

INVESTIGATIONS

Laboratory examination:

— Determination of the blood group and Rh affiliation (when registering).

— A blood test for antibodies with Rh negative blood (when registering, and 28 weeks).

— The general analysis of blood with determination of platelet count and hematocrit (when registering, at 29 weeks of pregnancy, if indicated).

— Serologic testing for syphilis (first when registering, the second — in 29 weeks of pregnancy).

— The test for the presence of HbsAg (when registering).

— The glucose challenge test (GCT) is a screening test performed for gestational diabetes in the third trimester (Two-hour glucose tolerance test for all pregnant women at 25–26 weeks), unless the pregnant patient is obese or at high risk for developing diabetes. In these cases, the test should be performed at the first visit. If the test result is abnormal, the glucose tolerance test (GTT) is performed to confirm diabetes.

— HIV test (the first when registering, the second at 22–23 weeks of pregnancy). If the test result is positive — the re-examination is not appointed. In the case of the first term in the treatment of a pregnant 23 weeks later — she immediately assigned a blood test for HIV, and in the case of a negative result, the re-examination is carried out not later than 32 weeks of pregnancy. In the case of the first treatment a pregnant after 30 weeks of pregnancy is immediately assigned a blood test for HIV, and in the case of a negative result, the re-examination is carried out by express method in childbirth.

— Serological tests for rubella, hepatitis B virus.

— Clinical urine analysis, including a test for the presence of protein (when registering and with each visit).

— Urine culture to detect asymptomatic bacteriuria (when registering).

— Cervical cytology study by Papanicolaou has become a routine in many clinics.

— Genetic screen.

In addition to the routine laboratory tests performed at the initial antepartum visit, additional tests are performed at specific intervals throughout the pregnancy to screen for birth defects and other conditions.

Biochemical screening in the I trimester of pregnancy from 11 weeks + 1 day — 13 weeks + 6 days of pregnancy (PAPP-A, free chorionic gonadotropin). The II trimester at 16–20 weeks (AFP + free chorionic gonadotropin (double-test), or (AFP + free chorionic gonadotropin + free estriol (triple-test) with the calculation of the individual risks of chromosomal and some congenital abnormalities of the fetus (with informed consent — all or indications). Pregnant women who underwent biochemical screening in the I trimester of pregnancy and were not included in the high risk group, it is recommended in II trimester to determine only the level of AFP.

— The first trimester scan of either transabdominal (TAS) or transvaginal (TVS) helps to detect early pregnancy accurate dating of number of fetuses, gross fetal anomalies, any uterine or adnexal pathology.

Ultrasound examination: the first — in the gestation of 11 weeks + 1 day — 13 weeks + 6 days of pregnancy; the second — in 18–21 weeks' gestation. Holding the third routine ultrasound determined individually (high-risk group of congenital heart disease and others).

— Prenatal risk calculation.

Ultrasound fetometry. Biparietal diameter (BPD) and frontooccipital (FOD) diameters of the fetal head, on the base of which it is possible to determine the circumference of the fetal head are detected in the profile of the fetal head in the place of the best imaging of the middle structures of the brain after the 12th week of pregnancy.

Indicators of ultrasound in the first trimester: crown-rump length + or nuchal fold thickness; in the second trimester, biparietal size.

PROCEDURE AT THE NEXT VISITS

As a rule check up takes place at intervals of 2–3 weeks up to 28 weeks; at intervals of 2 weeks up to 36 weeks and thereafter weekly till delivery. Ideally this should be more flexible depending on the need and the convenience of the patient.

The aim of assessment: fetal well being, lie, presentation, position and number of fetuses.

Palpation

After 20 weeks of gestation (when the fundus is palpable at or near the umbilicus in a woman of normal body habitus and a singleton pregnancy in the vertex presentation), the uterine size can be assessed with the use of a tape measure, which is the fundal height measurement (Fig. 7.9).

Circumference of the abdomen is measured on the level of the umbilicus by a centimeter strip (in a full-term pregnancy: 90–100 cm), height of the fundus uteri — by a centimeter strip or pelvimeter above the pubic symphysis.

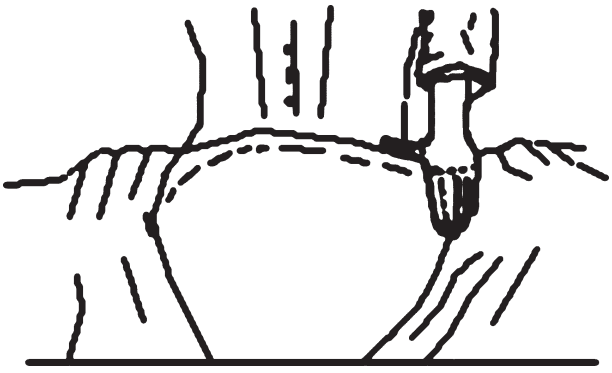


Fig. 7.9

The general condition of mother and child is recorded during the pregnancy in the gravidogram. True sign of "intrauterine growth restriction of the fetus" is delay of the indicators by 2 weeks and more from parameters, which accord to this gestational age (Fig. 7.10) [43, 45].

Birth weight is usually taken as the sole criterion to assess fetal growth and consequently fetuses with a birth weight of less than the 10th percentile of those born at the same gestational age or two standard deviation below the population mean, are considered growth restricted. The early detection of IUGR is therefore important to institute specific treatment wherever possible or plan appropriate timed delivery to reduce perinatal morbidity and mortality.

Auscultation of fetal heart rate (all pregnant women with 25–26 weeks of pregnancy).

Vaginal examination

Vaginal examination in the later months of pregnancy (beyond 37 weeks) with an idea to assess the pelvis is not informative. Pelvic assessment is best done with the onset of labor or just before induction of labor.

Late in pregnancy greater than 37 weeks:

— to diagnose contracted pelvis (refer chapter on);

— to assess Bishop score (refer to chapter on induction).

In labor cervical dilatation and effacement, status of the membranes and colour of liquor, presenting part, station of presenting part and position, moulding, caput, clinical pelvimetry are assessed.

During the vaginal examination it is determined:

— peculiarities of the inlet to the vagina (woman has given birth or has not), width of the vagina (wide, narrow), presence of the septums in it, perineal muscles condition (the pelvic floor); nature of the vaginal discharge — amount, color, smell (a few amount of white mucous discharge in the norm);

— cervical condition (form and length: preserved, short, flattened); its consistence: dense, soft; location in relation to the axis of the labor canal: centrated, positioned to the front, to the back; opening of the uterine fauces: passage of the cervical canal — for one, two and more fingers; the uterine os' edge condition: thick, thin, extends, does not extend; if the loops of the umbilical cord, tissue of the placenta and small parts of the fetus are detected in the limits of the uterine os;

— condition of the lower segment of the uterus (thinned, painful);

— condition of the fetal vesicle (if the finger passes in the cervical canal of the uterus): intact, absent, cut; its condition in labor pains: does it function during labor pains, if it is strained when they are absent; excessively strained, flat, does not function — in the case of oligoamnios;

— condition of the presenting part (head, buttocks, legs), place of its location in relation to the planes of the pelvis; fontanels, sutures, their location as for the sacrum or pubic symphysis. During the transverse or oblique location of the fetus during the vaginal examination a presenting part is not detected, the shoulder of the fetus is palpated above the plane of the pelvic inlet;

— condition of the pelvic walls, the presence of the bony prominences (exostoses), deformations, character of the internal surface of the pubic symphysis, height of the sacral cavity. Lowering the elbow, the doctor tries to reach the promontorium by the middle finger: diagonal conjugate is measured — a dis-

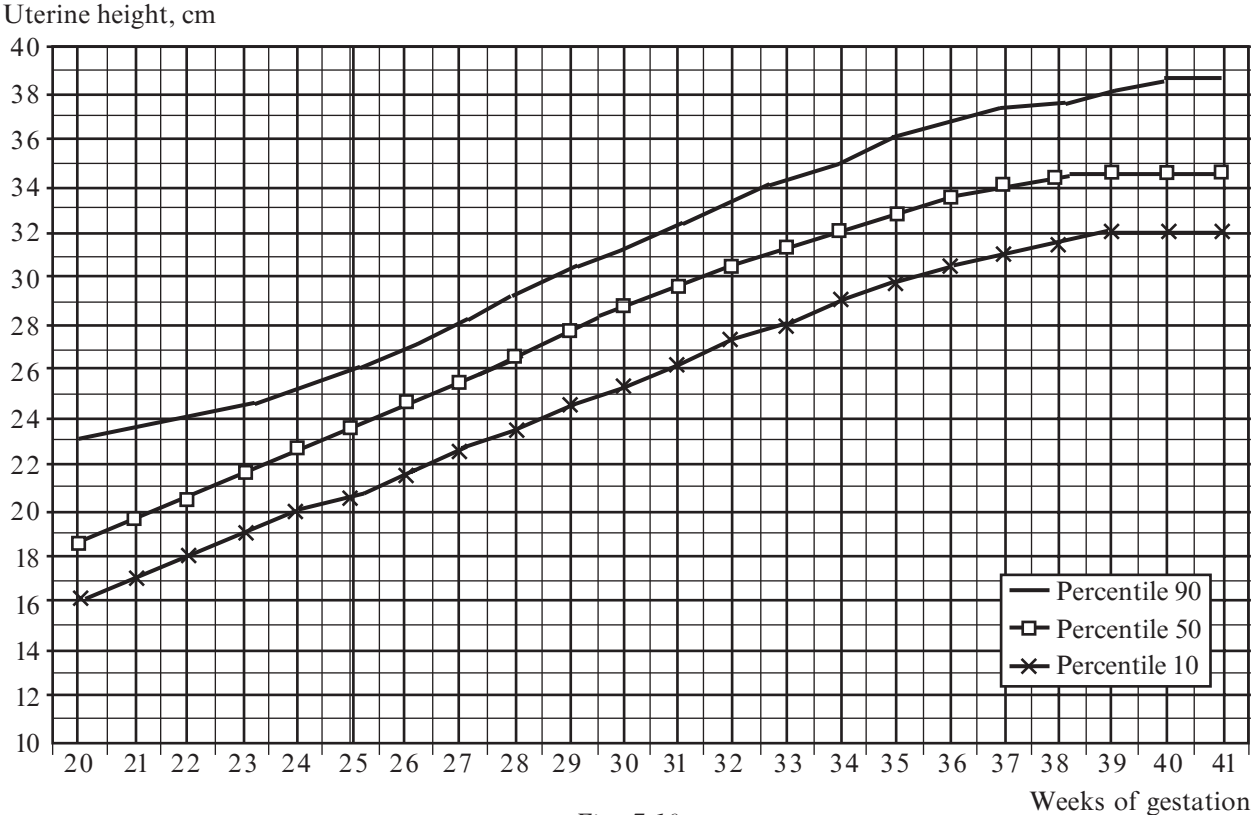


Fig. 7.10

tance between the inferior edge of the symphysis and the most outpouching points of the promontorium.

COMMON COMPLAINTS

Subjective complaints

Fatigue, somnolence, headache, 'blackouts', are often noticed in the early months and their cause is uncertain. Hypotension, secondary to peripheral vasodilatation, may be responsible for feelings of faintness.

Morning sickness

Nausea and vomiting are due probably to the effects of large amounts of circulating steroids, especially oestrogens or HCG and they seldom last beyond the 16th week. They can occur at any time of the day and are aggravated by cooking and fatigue.

Mild cases are treated by a light carbohydrate diet (biscuits and milk) in the morning and sometimes by anti-emetics. If the condition worsens it becomes hyperemesis gravidarum and is best treated in hospital.

Heartburn

The enlarging uterus encourages oesophageal reflux of gastric acid. Sleeping in a semi-recumbent position is helpful.

Urinary Frequency and Incontinence

Often during the first 3 months of pregnancy, the growing uterus places increased pressure on the bladder. Urinary frequency usually will improve as the uterus rises out of the pelvis by the second trimester. However, as the head engages near the time of delivery, urinary frequency may return as the head presses against the bladder. About 40–50% of women will experience urinary incontinence during the pregnancy. The risk of incontinence of urine is highest in the third trimester. The chances of experiencing incontinence are increased in multiparous women, especially those with a history of incontinence. Incontinence during pregnancy is a risk factor for persistent incontinence. If the patient experiences pain with urination or the new onset of incontinence, it is appropriate to check for infection.

Syncope

Compression of the veins in the legs from the advancing size of the uterus places patients at risk of venous pooling associated with prolonged standing. This may lead to syncope. Measures to avoid this possibility include wearing support stockings and exercising the calves to increase venous return. In later pregnancy, patients may have problems with supine hypotension, a distinct problem when undergoing a medical evaluation or an ultrasound examination. A left lateral tilt position with wedging below the right hip will help keep the weight of the pregnancy off the inferior vena cava (Fig. 7.11).

Pelvic pressure

The pressure of the fetal head in the pelvis can compress the iliac veins and obstruct venous outflow from the legs. As the baby grows, the uterus enlarges and applies pressure on important veins that return blood to the heart. This pressure can cause a slowing of the blood flow and valve damage, resulting in swelling, leg discomfort, and even varicose veins. Support tights may be helpful for leg varicosities.

Pelvic joint pain

This can occur because the ligaments of the pelvic joints are softened and relaxed during pregnancy by steroid induced fluid retention and the increased vascularity which occurs in all the soft tissues. The pelvis becomes less rigid which may be of some advantage in labor but movements can now take place in joints which are normally immovable and various symptoms may arise. Backache and sacro-iliac strain may occur because the softening of ligaments is aggravated by the postural change of pregnancy, a characteristic lordosis as the uterus grows.

Restless Legs Syndrome

About one in 5 to 10 women will develop restless legs syndrome (RLS) during the second half of pregnancy. RLS usually occurs as women fall asleep and is characterised by tingling or other uncomfortable sensations in the lower legs, resulting in the overwhelming urge to move the legs. Unfortunately, movement, walking around or other measures do not relieve RLS. Iron deficiency anemia has been associated with an increased risk for RLS, and in anemic women, iron supplementation may reduce leg restlessness. Avoidance of caffeine containing drinks like coffee, tea or sodas in the last half of the day should also be recommended, as caffeine may increase symptoms.

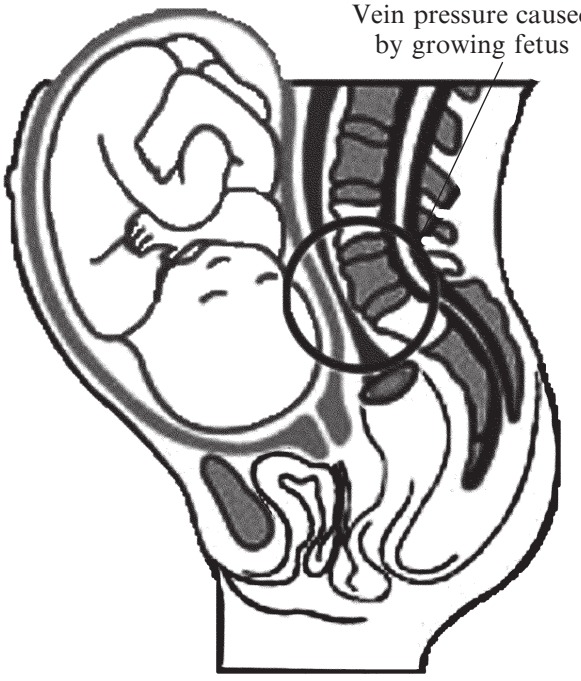


Fig. 7.11

Constipation

Constipation is physiologic during pregnancy with decreased bowel transit time, and the stool may be hardened. This is due principally to the relaxing effect of progesterone on smooth muscle. A bowel motion every second or third day is perfectly consistent with good health, but sometimes laxatives are required.

Hemorrhoids

Hemorrhoids are varicose veins of the rectum. Since straining during bowel movements contributes to their aggravation, avoidance of constipation is preventive. Prolonged sitting should also be avoided. Hemorrhoids will often regress after delivery but usually will not disappear completely.

Vaginal discharge

Increased secretion of cervical mucus and the vascularity of the vagina combine to produce a fairly copious discharge in pregnancy. It should not be offensive or itchy and ordinary hygiene should be the only treatment required.

Infection with *Candida albicans*, however, is a common complication. This is encouraged by the warmth and moisture of the vulva and vagina together with the increased vaginal glycogen which favours the fungus. The complaints are discharge and constant irritation. A swab should be taken and the characteristic plaques of yeast may be seen.

Trichomonads may also be seen.

Bacterial vaginosis is said to be commoner in pregnancy and may be associated with some cases of pre-term labor.

Treatment of candida and trichomonas infection is by the use of clotrimazole pessaries. It may be difficult to eradicate in pregnancy but treatment is desirable to relieve symptoms and reduce the chances of infecting the fetus during its passage down the vagina [31].

ANTENATAL ADVICE

The patient may continue her usual activities throughout pregnancy. However, excessive and strenuous work should be avoided especially in the first trimester and the last 4 weeks. Recreational exercise (prenatal exercise class) are permitted as long as she feels comfortable. There is individual variation of the amount of sleep required. However, on an average, the patient should be in bed for about 10 hours (8 hours at night and 2 hours at noon) especially in the last 6 weeks. In late pregnancy lateral posture is more comfortable.

One of the earliest purposes of prenatal care was to ensure that women received adequate nutrition for pregnancy.

The *diet* during pregnancy should be adequate to provide:

— good maternal health;
— optimum fetal growth;
— the strength and vitality required during labor;
— successful lactation.

During pregnancy, there is increased calorie requirement due to increased growth of the maternal tissues, fetus, placenta and increased basal metabolic rate.

Woman with normal BMI should eat adequately so as to gain the optimum weight (11 kg). Overweight women with BMI between 26–29 should limit weight gain to 7 kg and obese women (BMI >29) should gain less weight. Excessive weight gain increases antepartum and intrapartum complications including fetal macrosomia.

Nutritional requirements

Protein. Protein needs in the second half of pregnancy are 1 g/kg plus 20 g/d (approximately 80 g/d for the average woman). Protein intake is essential for embryonic development.

Calcium. Calcium intake should be increased to 1.5 g/d in the later months and during lactation. If calcium intake is inadequate, fetal needs will be met through demineralization of the maternal skeleton. Maternal calcium stores may be further drained during lactation.

Iron. To avoid iron-deficiency anemia, the IOM recommends supplementing the diet of every pregnant woman with 30 mg/d of elemental iron during the second and third trimester. If iron-deficiency anemia is diagnosed, the therapeutic doses of elemental iron prescribed range between 60 and 120 mg/d.

Vitamins and Minerals. As the essential vitamins are either lacking in the foods or are destroyed during cooking, supplementary vitamins are to be given daily from the 20th week onwards. Vitamin and mineral preparations are commonly given but should not be substituted for adequate food intake. Folic acid has been shown to effectively reduce the risk of neural tube defects (NTDs). A daily 4-mg dose is recommended for patients who have had a previous pregnancy affected by NTDs. It should begin more than 1 month prior to pregnancy (preferably 3 months) and continue through the first 6–12 weeks of pregnancy. Studies show this amount reduces the risk of recurrence by 70%. For all other women, a daily intake of at least 0.4 mg taken before conception and through the first 6 weeks of pregnancy is recommended. Patients with insulin-dependent diabetes mellitus and those with seizure disorders treated by valproic acid and carbamazepine are also at greater risk for neural tube defects and these women should ingest at least 1 mg/d of folic acid. Vitamin B_{12} supplements are also desirable for vegetarian patients and those with known megaloblastic anemia.

Chapter 8
INTRAPARTUM FETAL MONITORING

Majority (80%) of fetal deaths occur in the antepartum period. The important causes of deaths are: chronic fetal hypoxia (IUGR); maternal complications (diabetes, hypertension, infection); fetal congenital malformation and unexplained cause. There is progressive decline in maternal deaths all over the world. Currently more interest is focussed to evaluate the fetal health. The primary objective of antenatal fetal assessment is to avoid fetal death. As such simultaneously with good maternal care during pregnancy and labor, the fetal health in utero should be supervised with equal vigilance [3, 12, 25].

Aims of antenatal fetal monitoring:

— To provide satisfactory growth and well-being of the fetus throughout pregnancy.

— To screen out the high risk factors that affect the growth of the fetus.

— To detect those congenital abnormalities or metabolic disorders during early pregnancy.

Common indications for antepartum fetal monitoring:

— Routine antenatal testing.

— Pregnancy with obstetric complications: IUGR, multiple pregnancy, polyhydramnios or oligohydramnios, Rh isoimmunisation.

— Pregnancy with medical complications: diabetes mellitus, hypertension, epilepsy, renal or cardiac disease, infection (tuberculosis), collagen vascular disease, sickle cell disease, antiphospholipid syndrome, systemic lupus erythematosus.

— Others: advanced maternal age (>35 years), previous still birth or recurrent abortion, previous birth of a baby with structural (anencephaly, spina bifida) or chromosomal (autosomal trisomy) abnormalities (Fig. 8.1).

ASSESSMENT IN EARLY PREGNANCY

BIOCHEMICAL MONITORING

Maternal serum alpha fetoprotein (MSAFP)

AFP is an oncofetal protein. It is produced by yolk sac and fetal liver. The highest level of AFP in fetal serum and amniotic fluid is reached by the 13 weeks and thereafter it decreases. Maternal serum level reaches the peak by the 32nd week. Test it done between 15 to 20 weeks.

MSAFP level is elevated in a number of conditions:

— wrong gestational age;

— open neural tube defects (NTDs);

— multiple pregnancy;

— IUFD;

— anterior abdominal wall defects;

— renal anomalies.

Low levels are found in trisomies (Down's syndrome) and gestational trophoblastic disease.

Triple test

It is a combined biochemical test which includes MSAFP, HCG and UE3 (unconjugated oestriol). It is used for detection of Down's syndrome. In an affected pregnancy level of MSAFP and UE3 tend to be low while that of HCG is high. It is performed for 15–18 weeks.

Acetylcholine esterase (AChE):

Amniotic fluid AChE level is elevated in most cases of open neural tube defects. It has got better diagnostic value than AFP.

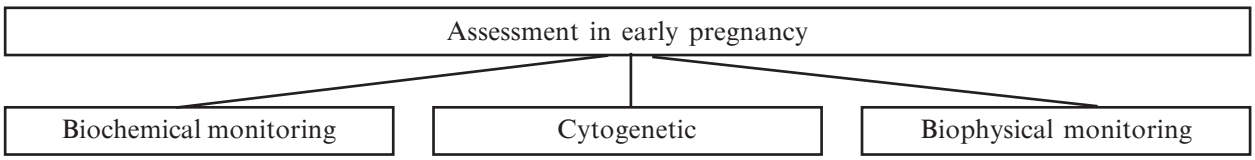


Fig. 8.1

Inhibin A is a dimeric glycoprotein. It is produced by the corpus luteum and the placenta. Serum level of inhibin A is raised in women carrying a fetus with Down syndrome.

PRENATAL GENETIC DIAGNOSIS

Amniocentesis

Aspiration of amniotic fluid from the pregnant uterus for examination. Typically scheduled between the 14th and 16th weeks of pregnancy. Ultrasound is done to determine the position of the fetus and the location of a pocket of amniotic fluid and the placenta (Fig. 8.2).

Diagnostic indications:

Early months (15–20 wks):

— sex related disorders;

— karyotyping;

— inborn errors of metabolism;

— neural tube defects.

Later months:

— fetal maturity;

— degree of fetal hemolysis in Rh sensitised mother;

— meconium staining of liquor.

Chromosome analysis: few fetal skin cells are always present in amniotic fluid. These cells may be cultured and stained for karyotyping for genetic analysis.

Fetal fibronectin. Fibronectin is a glycoprotein that plays a part in helping the placenta attach to the uterine decidua. Detection of fibronectin in either the amniotic fluid or in the mother's vagina can serve as an announcement that preterm labor may start.

Inborn errors of metabolism: can be detected by amniocentesis, for example: cystinosis and maple syrup urine disease (amino acid disorders).

Lecithin/sphingomyelin ratio: lecithin and sphingomyelin are the protein components of the lung enzyme surfactant that the alveoli begin to form at the 22nd to 24th weeks of pregnancy.

Chorionic villus sampling (CVS)

CVS is performed for prenatal diagnosis of genetic disorders.

It is carried out transcervically between 10–12 weeks and transabdominally from 10 weeks to term.

A few villi are collected from the chorion frondosum under ultrasonic guidance.

While it provides earlier diagnosis than amniotic fluid studies, complications like fetal loss (1–2%), mandibular limb deformities or vaginal bleeding are higher.

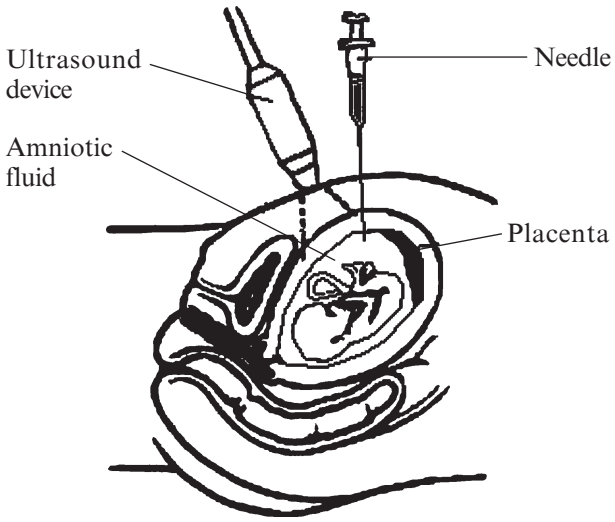
Cordocentesis

(Fetal blood sampling cordocentesis)

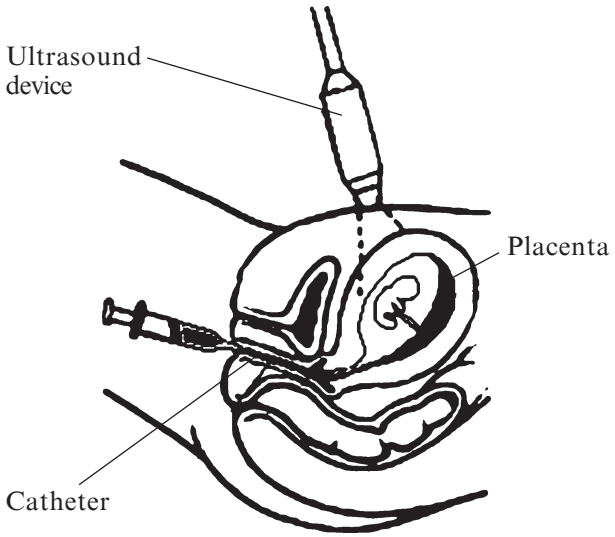
This technique is used to take a sample of fetal blood during pregnancy in order to screen for chromosomal abnormalities, hemoglobinopathies and other disorders affecting blood or cells.

It is performed under local anaesthetic usually after 18 weeks gestation.

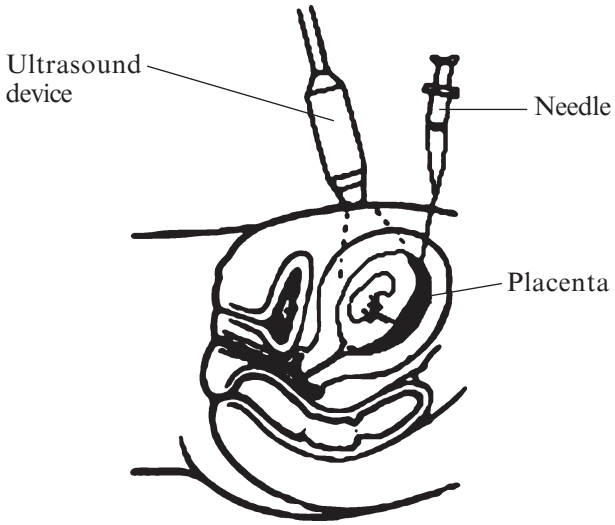
Risks: the invasive procedure may lead to abortion, preterm labor and intrauterine fetal death. These may be due to bleeding, cord haematoma



Amniocentesis



Chorionic Villus Sampling
(Transcervical method)



Chorionic Villus Sampling
(Transabdominal method)

Fig. 8.2

formation, infection or preterm rupture of membranes.

All the information as obtained in amniocentesis or chorionic villus sampling, could be gathered.

BIOPHYSICAL MONITORING

— Ultrasonographic examination of the fetus in the early (10–14 weeks) pregnancy can detect fetal anomalies [1, 2, 45].

— Crown-rump length (CRL) smaller than the gestational age is associated with the risk of chromosomal anomalies (trisomy or triploidy).

— Absence of nasal bone.

— Increased nuchal translucency (soft tissue marker) at 10–14 weeks is associated with many chromosomal abnormalities (trisomy, monosomy, triploidy).

ASSESSMENT OF FETAL WELLBEING IN LATE PREGNANCY

Clinical monitoring

• Maternal weight gain:

— In the second half of pregnancy: average weight gain is 1 kg/fortnight.

— Excess weight gain: could be the 1st sign of preeclampsia.

— If weight gain if less than normal, stationary or falling — IUGR.

• Blood pressure:

— Initial recording of BP prior to 12 weeks helps to differentiate pre-existing chronic hypertension from pregnancy induced hypertension.

— Hypertension, pre-existing or pregnancy induced may impair fetal growth.

• Assessment of size of uterus and height of fundus:

— After 24 weeks of pregnancy, distance measured in cm normally corresponds to the period of gestation in weeks.

• Clinical assessment of liquor.

• Edema of feet.

• Abdominal girth in the last trimester (Fig. 8.3).

Biophysical monitoring

The following biophysical tests are used:

• Antepartum tests:

1. Fetal movement count.
2. Cardiotocography (CTG).
3. The contraction stress test (CST).
4. The biophysical profile.
5. Doppler USG of fetal umbilical artery blood flow.

• Intrapartum assessment of fetal well-being:

1. Continuous electronic fetal monitoring:
   a. The fetal heart rate (FHR).
   b. Uterine activity.
2. Fetal scalp blood gas.

Fetal movement count

The simplest method of fetal assessment is daily fetal movement count (DFMC). Mothers perceive 88% of the fetal movements detected by Doppler Imaging. Normal fetal movement is a sign of functional integrity of fetal neuro-regulatory system. In the presence of mild hypoxemia, the fetus compensates by decreased frequency and strength of movements. The count should be performed daily starting at 28 weeks of pregnancy. Mother counts fetal movements starting at 9 am. Counting ends when 10 movements are perceived. Loss of fetal movements is commonly followed by disappearance of FHR within the next 24 hours.

The following three criteria are the most commonly used:

— Perception of at least 10 fetal movements during 12 hours of normal maternal activity.

— Perception of at least 10 fetal movements over two hours when the mother is at rest and focused on counting "Cardiff Count-to-Ten chart".

— Perception of at least 4 fetal movements in one hour when the mother is at rest and focused on counting.

Cardiotocography (CTG)

Monitoring of fetal heart activities is an indirect way for assessment of fetal oxygen status. Fetal hypoxia affects the cardiac control centers, and results in diminished heart activities "rate, variability and reactivates" through the autonomic nervous system.

Simultaneous recording of fetal heart rate (FHR) and uterine activity.

The non-stress test (NST)

The NST is the most commonly used method of antepartum fetal assessment. It is noninvasive (unlike the CST). It has no direct maternal or fetal risks, and virtually no contraindication.

In the non-stress test, a continuous electronic monitoring of the fetal heart rate along with the recording of fetal movements (cardiotocography) is undertaken. There is an observed association of FHR acceleration with fetal movements, which when present, indicates a healthy fetus (Fig. 8.4).

Non stress test Reactive (Reassuring)

— when two or more accelerations of more than 15 beats per minute above the baseline and longer than 15 seconds in duration are present in a 20 minutes observation in association with movement of the fetus.

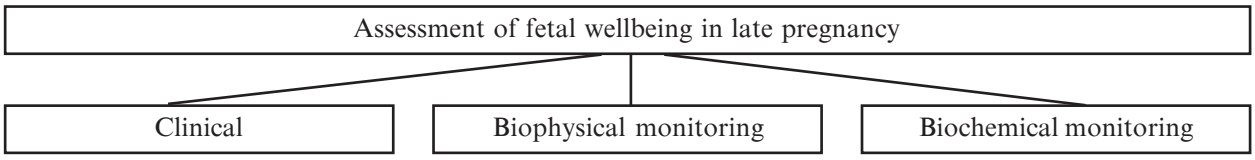


Fig. 8.3

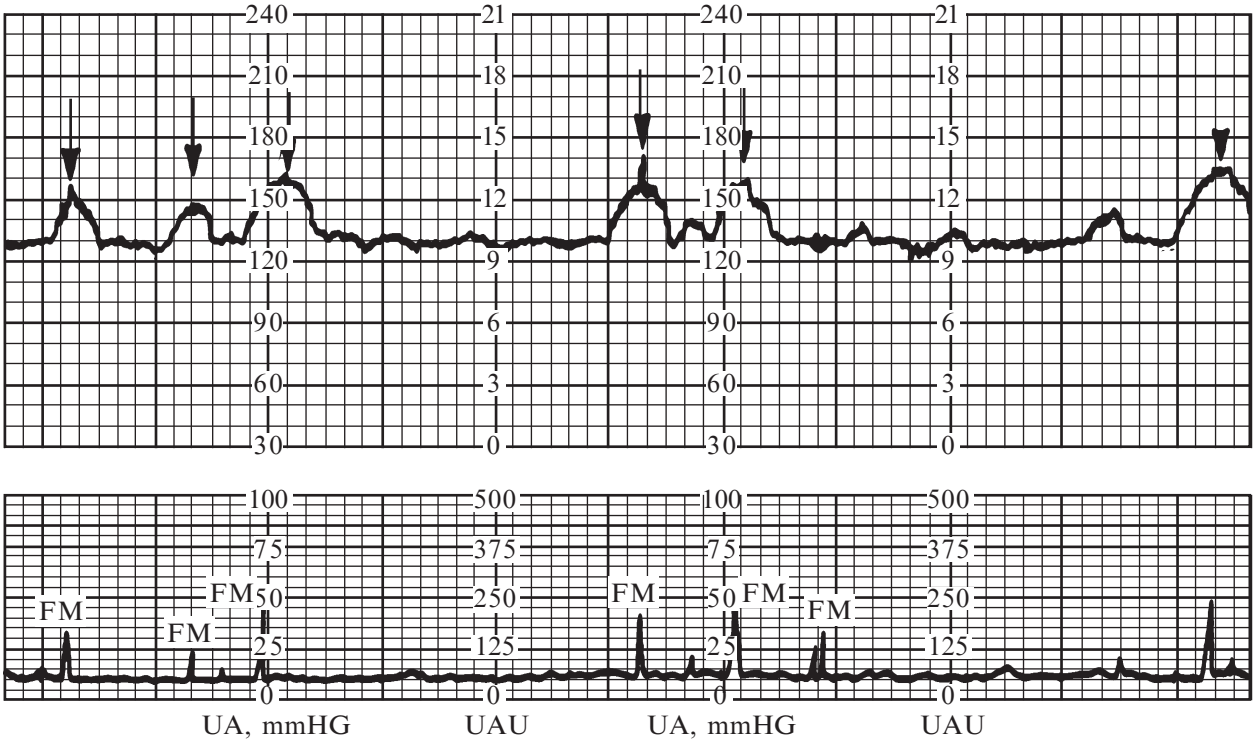


Fig. 8.4. Non stress test

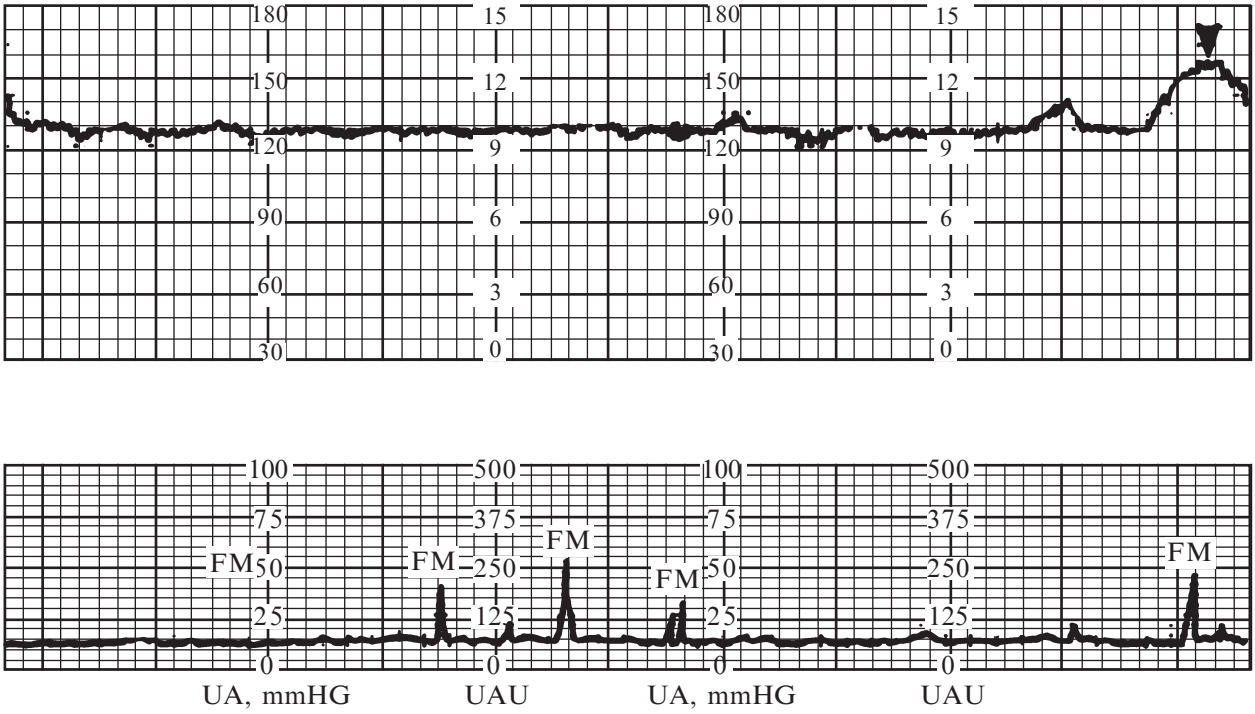


Fig. 8.5. Non-reactive stress test

Non-reactive (Nonreassuring) (Fig. 8.5)

— Absence of any fetal reactivity.

Important features to note while interpreting a CTG:

• Baseline FHR 110–160 bpm

— Moderate bradycardia 100–109 bpm

— Moderate tachycardia 161–180 bpm

— Abnormal bradycardia < 100 bpm

— Abnormal tachycardia > 180 bpm

• Accelerations and normal baseline variability (5–25 bpm) denote a healthy fetus.

• Absence of accelerations is the first feature to denote onset of gradual hypoxia.

• Absence of accelerations, reduced baseline variability may be due to fetal sleep, infection, hypoxia or due to maternal medications.

• Interpretation of the CTG should always be made in the context of a clinical situations.

• Baseline FHR is the mean level of FHR between the peaks and the depressions in beats per minute (bpm).

• Accelerations are increase in FHR by 15 bpm or more lasting for at least 15 seconds.

• Deceleration is decrease in FHR below the baseline by 15 bpm or more.

• Baseline variability is the oscillation of baseline FHR excluding the accelerations and decelerations. A baseline variability of 5–25 bpm is a sign of fetal wellbeing.

• Early Decelerations: uniform, repetitive decrease of FHR with slow onset early in the contraction and slow return to baseline by the end of the contraction.

• Late Decelerations: uniform, repetitive decreasing of FHR with, usually, slow onset mid to end of the contraction and nadir more than 20 seconds after the peak of the contraction and ending after the contraction. In the presence of a non-accelerative trace with baseline variability < 5 bpm, the definition would include decelerations < 15 bpm.

If test is nonreactive: test is repeated later the same day (or) perform another test of fetal well-being. Contraction stress test (CST) may be used as a confirmatory test when the NST is nonreactive or inadequate.

The contraction stress test.

Based on the idea that uterine contractions can compromise an unhealthy fetus. Pressure generated during contractions can briefly reduce or eliminate perfusion of intervillous space.

Indication is nonreactive non stress test.

• Healthy fetoplacental unit has sufficient reserve to tolerate this short reduction in oxygen supply.

• Under pathologic conditions, breathing reserve may be so compromised that reduction in oxygen results in fetal hypoxia.

• Similar to NST, CST monitors FHR and uterine contractions. If no spontaneous contractions occur (Gentle stimulation of the nipples releases oxytocin in the same way as happens with breastfeeding — oxytocin challenge test).

• A CST is considered completed if

— uterine contractions have spontaneously occurred within 30 minutes,

— lasted 40 to 60 seconds each, and

— occurred at a frequency of three within a 10-minute interval.

Interpretation:

• Positive: If late decelerations are consistently seen in association with contractions.

• Negative (is good): If at least three contractions of at least 40 seconds each occur within a 10-minute period without associated late decelerations.

• Suspicious: If there are occasional or inconsistent late decelerations.

Contraindications to CST:

• Patient with risk of preterm labor,

• PROM,

• Uterine surgery, classical cesarean section,

• Known placenta previa, multiple gestation, cervical incompetence, vasa previa.

The biophysical profile

The biophysical profile is a test used to evaluate the well-being of the fetus. The biophysical profile uses ultrasound and cardiotocography (CTG), also known as an electronic fetal heart rate monitoring, to examine the fetus. Biophysical profile is a screening test for the uteroplacental insufficiency. The examination is usually carried out over 30 minutes (Tables 3, 4).

Table 3

Biophysical profile

Component	Normal (2 points)	Abnormal (0 points)
Fetal Breathing Movements	One or more episodes of fetal breathing lasting at least 30 seconds within 30 minutes.	No episodes of fetal breathing movements lasting at least 30 seconds during a 30 minute period of observation.
Gross Body Movement	3 or more discrete body or limb movements within 30 minutes	Less than 3 body or limb movements in 30 minutes
Fetal Tone	At least one episode of motion of a limb from position of flexion to extension and rapid return to flexion (opening and closing of hand considered normal tone)	Position of semi or full limb extension with no return or slow return to flexion with movement; absence of fetal movement
Amniotic Fluid Volume*	A single deepest vertical pocket of amniotic fluid measures greater than 2 centimeters. is present	A single deepest vertical pocket of amniotic fluid measures 2 centimeters or less
Non-stress test**, ***	Reactive	Nonreactive

Notes: * — *Amniotic Fluid Volume* Measured as the vertical measurement, in centimeters, of the single deepest pocket of amniotic fluid with a transverse measurement of 1 cm or more wide without fetal small parts or umbilical cord; ** — *Reactive* two or more fetal heart rate accelerations that peak (but do not necessarily remain) at least 15 beats per minute above the baseline and last at least 15 seconds from baseline to baseline during 20 minutes of observation; *** — *Nonreactive* less than two accelerations of fetal heart rate as described above after 40 minutes of observation.

Table 4

Interpretation: A total score of 10

BPP score	Interpretation	Management
8–10	No fetal asphyxia	Repeat testing at weekly interval
6	Chronic asphyxia	If > 36 weeks — deliver
4	Chronic asphyxia	If = 36 weeks deliver, if 32 weeks repeat testing in 4–6 hours.
0–2	Certain asphyxia	Test for 120 min Persistent score ≤ 4 — deliver regardless of gestational age

A biophysical profile combines five parameters:
1. Fetal reactivity.
2. Fetal breathing movements.
3. Fetal body movement.
4. Fetal tone.
5. Amniotic fluid volume.

There are five components measured during the biophysical examination: a reactive NST, the presence of fetal breathing movements, the presence of fetal movement of the body or limbs, the finding of fetal tone, and an adequate amount of amniotic fluid volume. A score of 2 points is given for each component that meets criteria as listed in the table below. The test is continued until all criteria are met or 30 minutes have elapsed. The points are then added for a possible maximum score of 10.

Modified biophysical profile

— Use of only two assessments (amniotic fluid index and a nonstress test).

— A healthy fetus should show a reactive nonstress test and an amniotic fluid index range between 5 and 25 cm.

— Modified BPP is considered abnormal (nonreassuring) when the NST is non-reactive and/or an amniotic fluid index is < 5.

OBSTETRICAL ULTRASOUND

Introduced in the late 1950's ultrasonography is a safe, non-invasive, accurate and cost-effective means to investigate the fetus. Computer generated system that uses sound waves integrated through real time scanners placed in contact with a gel medium to the maternal abdomen. The information from different reflections are reconstructed to provide a continuous picture of the moving fetus on the monitor screen. Obstetric ultrasound examinations are performed with a transabdominal, transvaginal, or transperineal approach [2, 12, 43–45] (Fig. 8.6).

Indications:

— Unsure last menstrual period.

— Vaginal bleeding during pregnancy.

— Uterine size not equal to expected for dates.

— Use of ovulation-inducing drugs confirms early pregnancy.

— Obstetric complications in a prior pregnancy: ectopic, preterm delivery.

— Screen for fetal anomaly: abnormal serum screens, certain drug exposure in early pregnancy, maternal diabetes. Rh isoimmunisation.

— Postdate fetus.

— Twins.

— Intrauterine growth restriction (IUGR).

1st trimester (less than 12 weeks)

— Gestational sac location/size/shape

— Embryo

— Yolk sac

— Amnion

— Fetal cardiac activity

— Placental position/Umbilical cord

— Amnionitic fluid

— Fetal morphology >11 weeks

— Cranium

— Heart

— Stomach/Bladder/Cord insertion/presence of limbs, hands and feet.

— Pre and peri- ovulation (1–2 weeks): ovarian follicle matures and ovulation.

— Conceptus (3–5 weeks): Corpus luteum, fertilization, morula, blastocyst, bilaminar embryo — embryonic (6–10 weeks): Trilaminar C-shaped embryo.

— Fetal phase (11–12 weeks):

Gestational sac: seen at 4 weeks, fluid filled with echogenic border, grow at least 0.6 mm daily. An intrauterine gestational sac should be visualized by transvaginal ultrasound.

Yolk sac: 33 days (4.5 to 5 weeks). First sonographic sign of an intrauterine pregnancy.

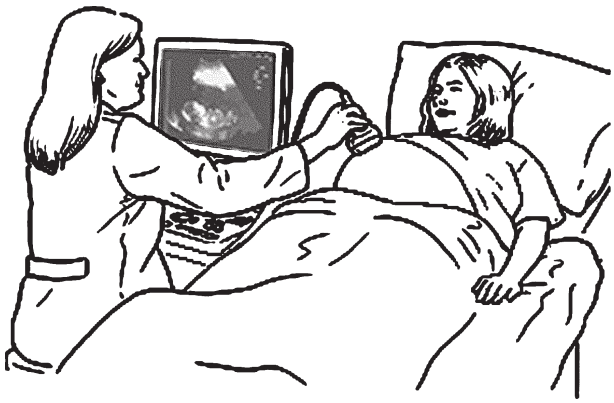
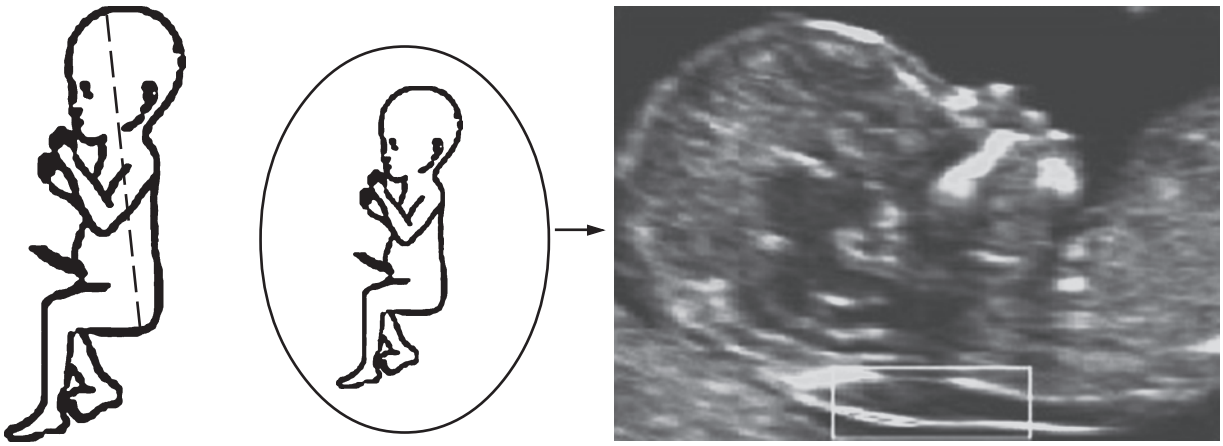


Fig. 8.6



Crown rump length
a

Measurement of the nuchal translucency
b

Fig. 8.7

Embryonic echoes: 38 days (5.4 weeks) with embryo at 6 weeks. In a normal pregnancy, the embryo should be visible if the gestational sac is 25 mm or larger in diameter.

Visible heart activity: 43 days (6.1 weeks). Normal heart rate at 6 weeks: 90–110 bpm. At 9 weeks: 140–170 bpm.

Gestational age and fetal maturity

— At 5–10 weeks: gestational sac.

— At 8–14 weeks: the crown-rump length.

— At 14–20 weeks: length of the femur.

— At 18–26 weeks: the biparietal diameter.

Crown Rump Length (CRL): longest length excluding limbs and yolk sac. The measurement of the length of human embryos and fetuses from the top of the head (crown) to the bottom of the buttocks (rump). Fetal CRL in centimeters plus 6.5 equals gestational age in weeks (Fig. 8.7, *a*).

CRL: 42–79 mm (performed between 11 and 13 weeks 6 days).

Nuchal translucency: translucent space between the back of the neck and the overlying skin in the first trimester of pregnancy. The scan is obtained with the fetus in the sagittal section and a neutral position. The fetal head (neither hyper flexed nor extended, either of which can influence the nuchal translucency thickness) (Fig. 8.7, *b*).

The maximum thickness is measured, from leading edge to leading edge (inner to inner measurement). Nuchal translucency > 6 mm is considered to be abnormal.

Ultrasound findings in a pregnancy destined to abort include:

— A poorly-defined, irregular gestational sac.

— A large yolk sac (6 mm or greater in size).

— Low site of sac location in the uterus.

— Empty gestational sac at 8 weeks gestational age (the blighted ovum).

2nd Trimester Ultrasound (13–24 weeks)

• Fetal survey:

— fetal number;

— viability;

— presentation;

— fetal biometry.

• Amniotic fluid

• Placenta

• Cervix

• Fetal anatomic screening

Cervical length

Endovaginal probe, examine in dorsal lithotomy position with empty bladder.

Normal cervix should have a length of 2.5 cm or more from 10 weeks gestation until 36 week (Fig. 8.8).

The width of the cervical canal at the level of the internal os should be less than 4 mm.

Optimal gestational age for cervical length assessment is after 16 to 20 weeks gestation.

Standard biometric parameters

Ultrasonography may be used to predict fetal maturity by measuring the biparietal diameter (the transverse distance between parietal bone protuberance) of the fetal head.

— *Biparietal diameter (BPD)* is 8.5 cm or greater, it can be predicted that the infant will weigh more than 2500 g or is at a fetal age of 40 weeks. Greatest accuracy between 12–28 weeks (better > 14 weeks) (Fig. 8.9, *a*)

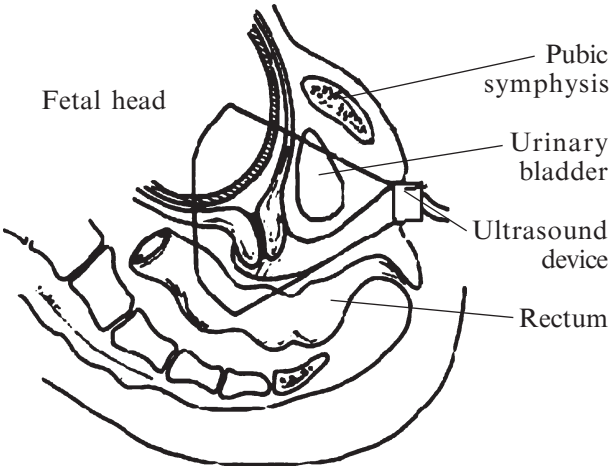


Fig. 8.8. Measurement of the cervix

The plane for measurement of *head circumference* (HC) and *biparietal diameter* (BPD) must include:

— Cavum septum pellucidum.

— Thalamus.

— Choroid plexus in the atrium of the lateral ventricles.

HC is measured from outer skull surface to outer skull surface at the same level as the biparietal diameter (34.5 cm indicates a 40-week fetus).

The average increase of biparietal diameter beyond 34 weeks is 1.7 mm per week. When the HC/AC ratio is elevated (> 1.0) after 34 weeks, IUGR is suspected.

— *Fetal abdominal circumference (AC)* (Fig. 8.9, *b*)

Determined on transverse view at the level of the junction of the umbilical vein, portal sinus, and fetal stomach.

Measured from the outer diameter to outer diameter. Assessing fetal weight/IUGR /macrosomia.

— *Femur length (FL):*

Can measure from 10 weeks. The femoral epiphyseal and proximal tibial ossification centers are well visualized by 32 and 35 weeks gestational age, respectively (Fig. 8.9, *c*).

The proximal humeral epiphysis also appears in the late third trimester and correlates with fetal lung maturity and gestational age.

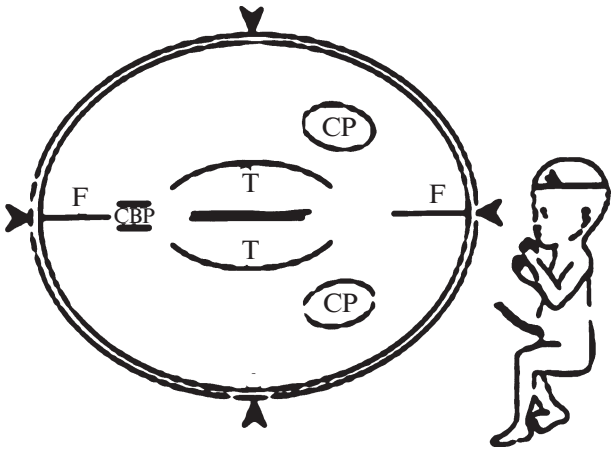
DOPPLER ULTRASOUND

The majority of blood flow to the uterus is supplied by the uterine arteries. Throughout gestation, uterine blood flow increases 10–12 fold due to trophoblastic invasion of the spiral arteries within the myometrium and decidua, and 50% increase in maternal blood volume. The shape of the uterine artery Doppler waveform is unique and changes as gestation advances. In early pregnancy, the uterine circulation is characterised by high vascular impedance and low flow, giving a waveform with persistent end-diastolic velocity and continuous forward blood flow throughout the diastole. As the trophoblastic invasion and spiral artery modification proceed, placental perfusion increases and the utero-placental circulation becomes a high-flow, low-resistance system giving a waveform with greater end-diastolic flow.

When the normal trophoblastic invasion and modification of spiral arteries is interrupted, there is increased impedance to flow within the uterine arteries and decreased placental perfusion. These pathological processes are key features common to the development of preeclampsia and IUGR.

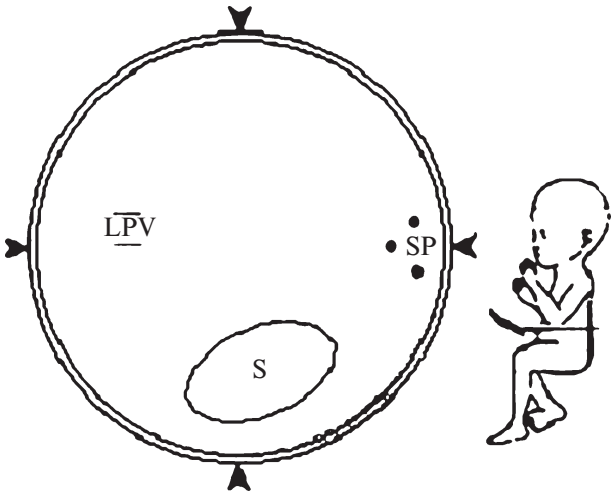
The umbilical artery (UA) was the first vessel to be studied by Doppler ultrasonography. By about 15 weeks of gestation, diastolic flow can be identified in the UA.

Doppler flow velocity waveforms are obtained from arterial and venous beds in the fetus. *Arterial Doppler* waveforms are helpful to assess the downstream vascular resistance. The arterial Doppler waveform is used to measure the peak systolic (S), peak diastolic (D) and mean (M) volumes. From these values S/D ratio, *pulsatility index* (PI) [PI = (S-D)/M] or *Resistance Index* (RI) [RI = (S-D)/S] are calculated.



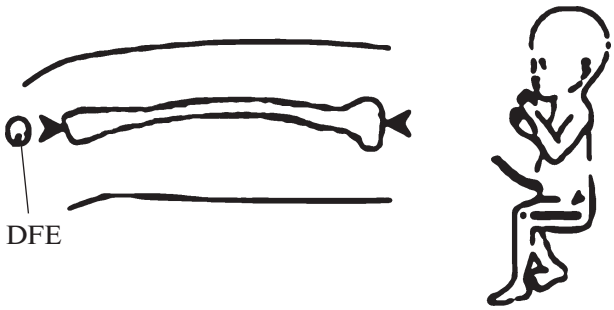
Fetal head measurement (BPD, HC)

a



Fetal abdominal circumference

b



Fetal femur length

c

Fig. 8.9

In a normal pregnancy the S/D ratio, PI and RI decreases as the gestational age advances. Higher values greater than 2 SDs above the gestational age mean indicates reduced diastolic velocities and increased placental vascular resistance. These features are at increased risk for adverse pregnancy outcome. Reversed umbilical artery diastolic flow (RUADF) "Fetus to placenta" an ominous sign, blood flow is reversed during diastole, fetuses need to be delivered promptly. At risk of neonatal death and significant morbidity.

Venous Doppler parameter provide information about cardiac forward function (cardiac compliance,

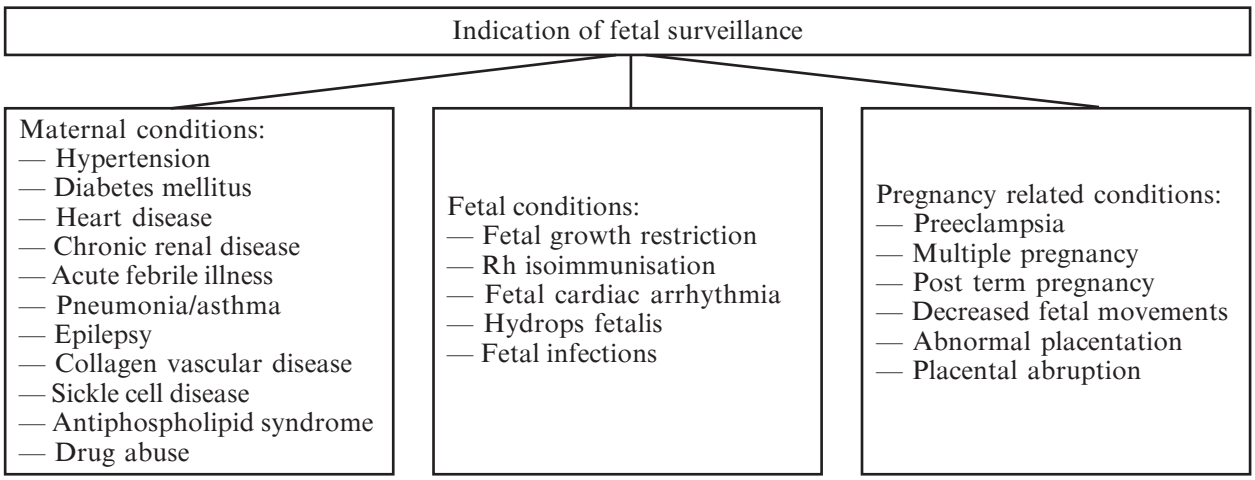


Fig. 8.10

contractility and after load). Fetuses with abnormal cardiac function show pulsatile flow in the umbilical vein (UV). Normal UV flow is monophasic.

Umbilical artery Doppler velocimetry measurements, in conjunction with other tests of fetal well-being, can reduce the perinatal mortality in IUGR by almost 40%. Doppler measurements of the middle cerebral artery can also be used in the assessment of the fetus at risk for either IUGR or anemia.

Middle cerebral artery (MCA) is another vessel well characterised by Doppler and has been shown to be affected by IUGR as well. MCA normally exhibits low amplitude of diastolic flow which increases in the presence of fetal hypoxia as a marker of cerebral vasodilation. This most commonly represents a later stage in the hypoxic process and typically occurs after changes in the uterine artery.

Amniotic fluid volume (AFV)

Amniotic fluid volume is primarily dependent upon the fetal urine, output, pulmonary fluid production and fetal swallowing. Decreasing AFV may be the result of fetal hypoxia and placental insufficiency. A vertical pocket of amniotic fluid > 2 cm is considered normal. *Amniotic fluid index (AFI)* is the sum of vertical pockets from four quadrants of uterine cavity. AFI < 5 is associated with increased risk of perinatal mortality and morbidity.

Placenta

Determining its upper and lower edges r/o placenta previa. With increasing gestational age, the placenta increases in echogenicity because of increased Grade 1 fibrosis and calcium content. This feature of placental maturation has led to a grading of placentas from immature (grade 0) to mature (grade 3).

Placentas can be graded by ultrasound as:
— 0 (12–24 weeks);
— 1 (30–32 weeks);
— 2 (36 weeks);
— 3 (38 weeks).

Three-Dimensional Ultrasonography

Three-dimensional ultrasonography represents an advance in imaging technology. With three-dimensional ultrasonography, the volume of a target anatomic region can be calculated. The defined volume then can be displayed in three orthogonal two-dimensional planes representing the sagittal, transverse, and coronal planes of a reference two-dimensional image within the volume. The volume also can be displayed in its rendered format, which depicts the topographic anatomy of the volume. The technical advantages of three-dimensional ultrasonography include its ability to acquire and manipulate a large number of planes and to display ultrasound planes traditionally inaccessible by two-dimensional ultrasonography. Despite these technical advantages, proof of a clinical advantage of three-dimensional ultrasonography in prenatal diagnosis in general still is lacking. Potential areas of promise include fetal facial anomalies, neural tube defects, fetal tumors, and skeletal malformations for which three-dimensional ultrasonography may be helpful in diagnosis as an adjunct to but not a replacement for two-dimensional ultrasonography [1, 2, 12, 43–45] (Fig. 8.10).

Chapter 9
OBSTETRICAL COMPLICATIONS

PLACENTAL INSUFFICIENCY

Placental insufficiency (or uteroplacental vascular insufficiency) is a complication of pregnancy when the placenta is unable to deliver an adequate supply of nutrients and oxygen to the fetus, and, thus, cannot fully support the developing baby. Placental insufficiency occurs when the placenta either does not develop properly or because it has been damaged. It is commonly defined as a reduction in the maternal blood supply (reduced uterine artery blood flow) (Fig. 9.1).

However, we define placental insufficiency to include reduction in maternal blood supply and/or the failure of the maternal blood supply to increase or adapt appropriately by mid-pregnancy. Placental insufficiency can result in pregnancy complications, including fetal growth restriction, preeclampsia and others, all of which are described below. The management of placental insufficiency is dependent upon additional tests and the unique characteristics of each patient. Factors considered during management of complicated pregnancies are maternal medical and obstetrical history, weight, ethnicity, and blood pressure [23, 49].

INTRAUTERINE GROWTH RESTRICTION

Intrauterine growth restriction by definition occurs when the birth weight of a newborn infant is below the 10th percentile for a given gestational age. About 3–10% (up to 15%) of all pregnancies are associated with IUGR. Perinatal mortality rate is 5–20 times higher for growth retarded fetuses.

The terms small for gestational age (SGA), low birth weight (< 2500 g), and IUGR should not be used synonymously. The term SGA merely indicates that a fetus or neonate is below a defined reference range of weight for a gestational age, whereas IUGR

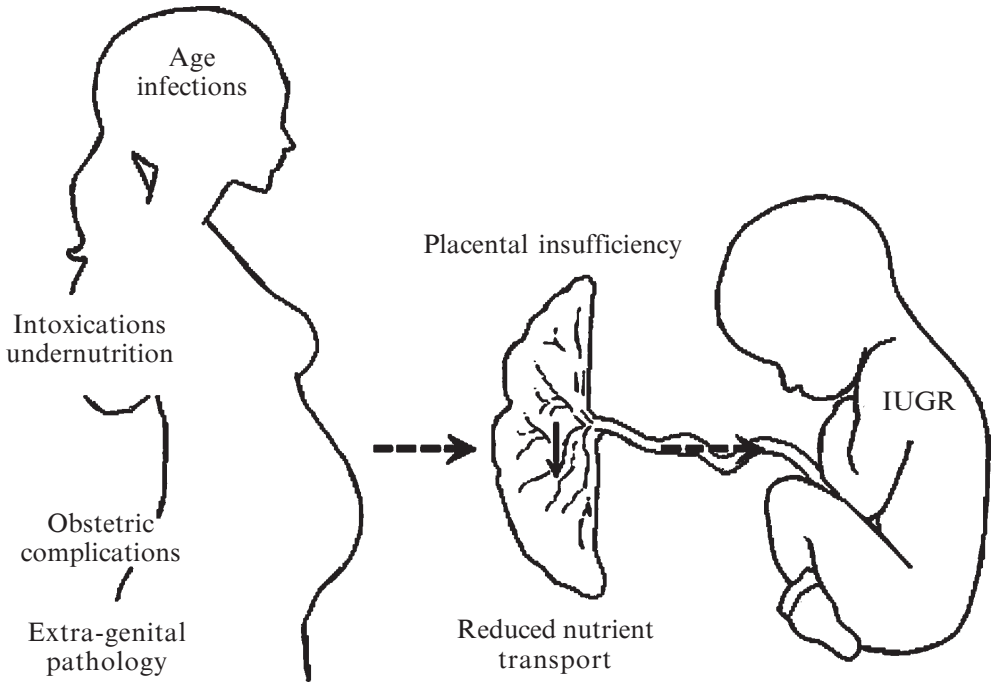


Fig. 9.1

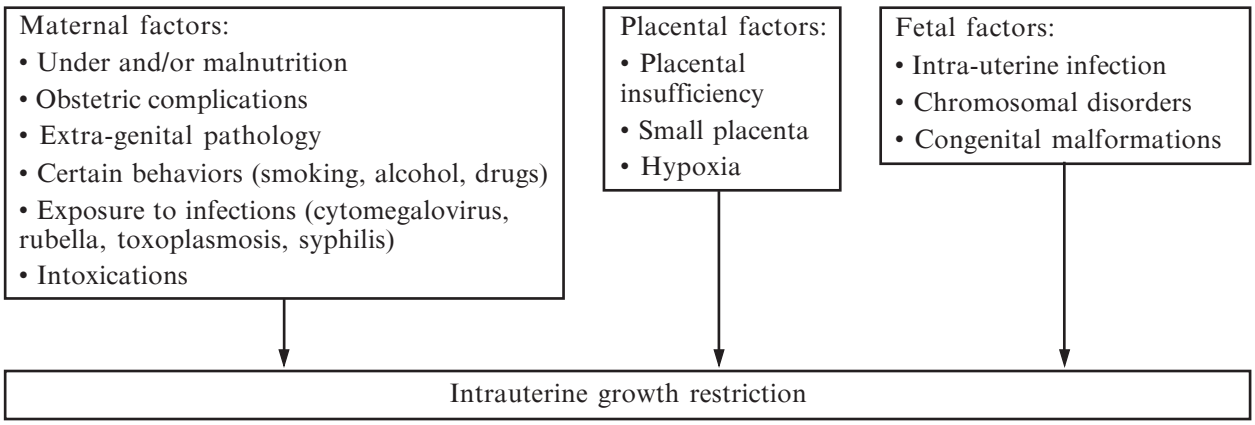


Fig. 9.2

(< 10th percentile) refers to a small group of fetuses or neonates whose growth potential has been limited by pathologic processes in utero, with resultant increased perinatal morbidity and mortality. Growth-restricted fetuses are particularly prone to problems such as meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation. They are at greater risk for developing adult onset conditions such as hypertension, diabetes, and atherosclerosis [37, 38].

The causes of IUGR

The causes of IUGR can be grouped into three main categories: maternal, placental, and fetal. Combinations of these are frequently found in pregnancies with IUGR (Fig. 9.2).

Risk factors of IUGR

— Multiple gestations.
— History of IUGR in previous pregnancies.
— Current heavy smokers.
— Current drug users.
— Pregnancies where SFH is less than expected.
— Women with underlying disorders (diabetes, cyanotic heart disease, antiphospholipid syndrome).

Classification of IUGR

Two types of fetal growth restriction have been described: symmetric and asymmetric (Table 5).

In fetuses with *symmetric growth restriction,* growth of both the head and the body is inadequate. The head-to-abdominal circumference ratio may be normal, but the absolute growth rate is decreased. Symmetric growth restriction is most commonly seen in association with intrauterine infections or congenital fetal anomalies.

When *asymmetric growth restriction* occurs, usually late in pregnancy, the brain is preferentially spared at the expense of abdominal viscera. As a result, the head size is proportionally larger than the abdominal size. The liver and fetal pancreas undergoes the most dramatic anatomical and biochemical changes. When there is insufficient nutrition to the fetus, caused by either poor maternal nutrition or poor uterine blood flow, the fetal liver fails to store glycogen because of inadequate glucose from the mother. Changes in the liver are now thought to play an important role in programming the fetus for a greater risk of obesity and diabetes later in life. The fetal phenotype (small size) is known as the thrifty phenotype, but when born into an environment of plenty, there is an increased risk of developing obesity, diabetes, and cardiovascular disease in later life.

Diagnosis

Growth restriction may go undiagnosed unless the obstetrician establishes the correct gestational age of the fetus, identifies high-risk factors from the

Table 5

Types of fetal growth restriction

Type	Symmetric/type 1 (20%)	Asymmetric/type 2 (80%)
Onset	Early in utero	Later onset
Etiology	Congenital infections, genetic disorders	Utero-placental insufficiency, maternal malnutrition, hypertension
Pathophysiology	— Impaired cell division — Decreased cell number — Irreversible	— Impaired cellular hypertrophy — Decreased cell size — Reversible
Clinical features	— Inadequate growth of head and body — Head: abdomen ratio may be normal	— Brain is spared, therefore head: abdomen ratio increased
Prognosis	Poor prognosis	More favorable prognosis

obstetric database, and serially assesses fetal growth by fundal height or ultrasonography. Fetal or neonatal IUGR is usually defined as weight at or below the 10th percentile for gestational age. Serial uterine fundal height measurements should serve as the primary screening tool for IUGR. A more thorough sonographic assessment should be undertaken when (1) the fundal height lags more than 3 cm behind expectations or (2) the mother has high-risk conditions such as preexisting hypertension, chronic renal disease, advanced diabetes with vascular involvement, preeclampsia, viral disease, addiction to nicotine, alcohol, or hard drugs, or the presence of serum lupus anticoagulant or antiphospholipid antibodies. Recently, interest has focused on the prediction of patients at risk for IUGR at mid-pregnancy. Patients with abnormal triple screens (AFP, hCG, and UE3) who do not have abnormal fetuses by ultrasonography and amniocentesis may be at increased risk for IUGR. In addition, elevations of umbilical artery and uterine artery Doppler assessments (increased resistance) as early as mid-pregnancy have been associated with a greater risk of IUGR as pregnancy progresses.

At present, a number of sonographic parameters are used to diagnose IUGR: biparietal diameter, head circumference, abdominal circumference, femoral length, amniotic fluid volume calculated fetal weight, and umbilical and uterine artery Doppler. Of these, the abdominal circumference is the single most effective parameter for predicting fetal weight because it is reduced in both symmetric and asymmetric IUGR. Most formulas for estimating fetal weight incorporate two or more parameters to reduce the variance of measurements.

Doppler-derived umbilical artery systolic-to-diastolic ratios are abnormal in IUGR fetuses. Fetuses with growth restriction tend to have increased vascular resistance and to demonstrate low, absent, or reversal of diastolic flow. This noninvasive technique can be used to evaluate high-risk patients, and may help in the timing of delivery when used in conjunction with the modified biophysical profile.

Complications of IUGR

Perinatal mortality and morbidity of IUGR infants is 3–20 times greater than normal infants. IUGR per se is not a contraindication to induction of labor, but there should be a low threshold to perform a cesarean delivery because of the poor capacity of the IUGR fetus to tolerate asphyxia. As a result, during labor, these high-risk patients must be electronically monitored to detect the earliest evidence of fetal distress.

A combined obstetric-neonatal team approach to delivery is mandatory because of the likelihood of neonatal asphyxia. After birth, the infant should be carefully examined to rule out the possibility of congenital anomalies and chronic infections.

The monitoring of blood glucose levels is important, because the fetuses do not have adequate hepatic glycogen stores, and hypoglycemia is a common finding. Furthermore, hypothermia is not uncommon in these infants. Respiratory distress syndrome is more common in the presence of fetal distress, because fetal acidosis reduces surfactant synthesis and release. Long term complications: increased risk of coronary heart disease, hypertension, type II diabetes mellitus and stroke.

PREMATURE RUPTURE OF THE MEMBRANES

Amniotic fluid is normally produced continuously, and after approximately 16 weeks' gestation is predominantly dependent on fetal urine production. However, passage of fluid across the fetal membranes, across the skin, and across the umbilical cord, as well as fetal saliva production and fetal pulmonary effluent, also contribute. Amniotic fluid protects against infection, fetal trauma, and umbilical cord compression. It also allows for fetal movement and fetal breathing, which, in turn, permits fetal lung, chest, and skeletal development. Decreased or absent amniotic fluid can lead to compression of the umbilical cord and decreased placental blood flow. Disruption (rupture) of the fetal membranes is associated with loss of protective effects and developmental roles of amniotic fluid [41, 45].

Spontaneous rupture of the membranes any time beyond 22 weeks of pregnancy but before the onset of labor is called ***prelabor rupture of the membranes*** (PROM). When rupture of membranes occur beyond 37th week but before the onset of labor it is called *term prelabor rupture of the membranes* and when it occurs before 37 completed weeks, it is called *preterm prelabor rupture of the membranes* (PPROM). Rupture of membranes for > 24 hours before delivery is called prolonged rupture of membranes.

Premature rupture of membranes occurs in approximately 12% of all pregnancies. PROM is associated with about 8% of term pregnancies (37 weeks or more of gestational age) and is generally followed by the onset of labor. Preterm PROM is a leading cause of neonatal morbidity and mortality, and is associated with approximately 30% of preterm deliveries. PROM leading to preterm delivery is associated with neonatal complications of prematurity such as respiratory distress syndrome, intraventricular hemorrhage, neonatal infection, necrotizing enterocolitis, neurologic and neuromuscular dysfunction, and sepsis. The major complication of PROM is intrauterine infection. The presence of lower genital tract infections with Neisseria gonorrhoeae and group B streptococcus as well as bacterial vaginosis increase the risk of intrauterine infection associated with PROM. Other complications include prolapsed umbilical cord and abruptio placentae. Consequences of preterm PROM depend on the gestational age at the time of occurrence. The cause is unknown, but is likely related to an inflammatory process.

Risk factors

— History of PROM or PPROM in a previous pregnancy
— Previous preterm labor
— Friability of the membranes
— Polyhydramnios

— Cervical incompetence
— Multiple pregnancy
— Genital tract infections
— Antepartum bleeding
— Low BMI (< 19 kg/m^2)
— Cigarette smoking

Confirming the diagnosis

Fluid passing through the vagina must be presumed to be amniotic fluid until proved otherwise.

— Speculum examination is done taking aseptic precautions to inspect the liquor escaping out through the cervix.

— Performing tests:

The nitrazine test uses pH to distinguish amniotic fluid from urine and vaginal secretions. Amniotic fluid is alkaline, having a pH above 7.1; vaginal secretions have a pH of 4.5 to 6.0, and urine has a pH of =6.0. To perform the nitrazine test, a sample of fluid obtained from the vagina during a speculum examination is placed on a strip of nitrazine paper. If the pH is 7.1 to 7.3, reflecting that of amniotic fluid, the paper turns dark blue. Cervical mucus, blood, and semen are possible causes of false-positive results (Fig. 9.3).

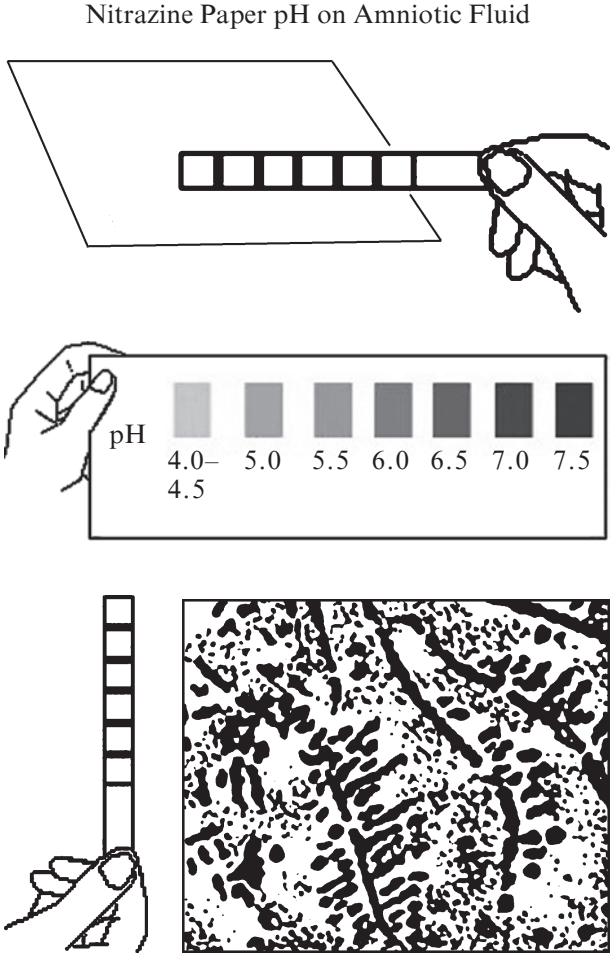


Fig. 9.3

The fern test is also used to distinguish amniotic fluid from other fluids. It is named for the pattern of arborization that occurs when amniotic fluid is placed on a slide and is allowed to dry in room air. The resultant pattern, which resembles the leaves of a fern plant, is caused by the sodium chloride content of the amniotic fluid. The ferning pattern from amniotic fluid is fine, with multiple branches; cervical mucus does not fern or, if it does, the pattern is thick with much less branching. This test is considered more indicative of ruptured membranes than the nitrazine test, but as with any test it is not 100% reliable.

Fluid from vagina placed on microscope slide, amniotic fluid dries to form a crystallization pattern called arborization (fern).

Ultrasonography is to be done not only to support the diagnosis but also to assess the fetal well being.

The differential diagnoses for PROM include

— urinary incontinence,

— increased vaginal secretions in pregnancy (physiologic),

— increased cervical discharge (pathologic, e.g., infection),

— exogenous fluids (such as semen or douche),

— vesicovaginal fistula.

Factors to be considered in the management of the patient with PROM include the gestational age at the time of rupture, assessment of fetal well-being, the presence of uterine contractions, the likelihood of chorioamnionitis, the amount of amniotic fluid around the fetus, and the degree of fetal maturity. These management factors, together with the patient's history, must be carefully evaluated for information relevant to the diagnosis and approach.

TERM PREMATURE RUPTURE OF MEMBRANES

If PROM occurs at term (≥ 37 weeks of gestation), spontaneous labor will ensue in 90% of women within about 24 hours [41, 45, 48].

• Expectant management and waiting for spontaneous labor may be considered in selected patients for the first 12–24 hours. The use of expectant management after the first 24 hours is questionable. Induction of labor after 24 hours.

• Antibiotic therapy (for the mother):

If the membranes have been ruptured for more than 18 hours, prescribe antibiotics (prophylactic infection). If there are no signs of infection after delivery, discontinue antibiotics.

• Assess the cervix:

— If the cervix is favourable (soft, thin, partly dilated), induce labor using oxytocin.

— If the cervix is unfavourable (firm, thick, closed), ripen the cervix using prostaglandins and infuse oxytocin.

• CTG (2 times a day). Daily fetal movement monitoring by the mother can also be helpful to assess fetal well-being.

• Control of temperature, pulse and blood pressure.

PRETERM PREMATURE RUPTURE OF MEMBRANES

Pregnancy ≥ 34 weeks of gestation:

— Wait for spontaneous onset of labor for 24–48 hours.

— Antibiotic therapy (for the mother):

— If the membranes have been ruptured for more than 18 hours, prescribe antibiotics (prophylactic infection). If there are no signs of infection after delivery, discontinue antibiotics.

— If the evaluation suggests intrauterine infection (fever, foul-smelling vaginal discharge), intravenous antibiotic therapy and delivery are indicated, regardless of gestational age.

— If there are uterine contractions: the risk of infection is greater than the risk of preterm birth: do not administer tocolytics.

— Induction of labor with oxytocin (CS for noncephalic presentation).

Pregnancy < 34 weeks of gestation:

— For ruptures occurring in the seventh and eighth month, transfer the mother, if possible, to a facility where the preterm infant can receive intensive care (NICU).

— If there are uterine contractions: tocolytic agent, except for signs of amniotic infection.

— Antibiotic therapy (for the mother):

— If the membranes have been ruptured for more than 18 hours, prescribe antibiotics (prophylactic infection). If there are no signs of infection after delivery, discontinue antibiotics.

— Prepare the fetus for preterm birth: After 26 weeks and before 34 weeks, help lung maturation with dexamethasone IM: 6 mg every 12 hours for 48 hours (Course dose: 24 mg). In case of severe maternal infection, start antibiotic therapy prior to dexamethasone.

PRETERM LABOR

Preterm labor is defined as regular contractions of the uterus resulting in changes in the cervix (effacement and dilation) that start before 37 weeks of pregnancy. When birth occurs between 22 weeks of pregnancy and 37 weeks of pregnancy, it is called *preterm birth*. Preterm birth is the significant cause of perinatal morbidity and mortality.

The prevalence widely varies and ranges between 5–10%. Preterm births may be classified into two general presentations: spontaneous and indicated. Approximately 40% to 50% of preterm births result from spontaneous preterm labor with intact membranes; 25% to 40% result from PROM. The remaining 20% to 30% occur following deliberate intervention for a variety of maternal or obstetric complications (e.g., eclampsia).

Risk factors of preterm labor

— Previous history of induced or spontaneous abortion or preterm delivery;

— Age < 18 or > 40 years;

— Poor nutrition/low pregnancy weight;

— Cervical injury or anomaly;

— Uterine anomaly or fibroid;

— Premature cervical dilatation (>) 2 cm or effacement (>) 80 percent;

— Over distended uterus (multiple pregnancy, polyhydramnios);

— Complications in present pregnancy: hypertension, bleeding, placenta previa or abruption;

— Pregnancy following assisted reproductive techniques (ART);

— Asymptomatic bacteriuria or recurrent urinary tract infection;

— Low socioeconomic and nutritional status;

— Maternal stress;

— Smoking habits.

Prediction of preterm labor (biomarkers for preterm labor)

Fetal fibronectin (fFN), after 20 weeks gestation fFN > 50 ng/ml, indicates possibility of preterm labor. Its sensitivity is up to 93%, specificity 82%.

Fibronectin is a protein produced by the fetal membranes that can leak into vaginal secretions if uterine activity, infection, or cervical effacement occurs.

Fetal fibronectin testing is sometimes done when preterm labor symptoms are present. When the fetal fibronectin test is negative, it is unlikely that you are having preterm labor. But even if the test is positive, it does not mean for sure that you are having preterm labor [31; 42] (Fig. 9.4).

Test:

For fetal fibronectin testing, a sample of fluid is collected from the vagina or the opening to the uterus (cervix). First, a speculum is used to spread the walls of the vagina to view the cervix. Next, a sterile swab is used to absorb fluid from the cervix or vagina. The speculum is removed.

A negative test result is quite accurate and shows that labor has not started. A positive test result may show that labor has started, but false-positive results are common.

Diagnosis:

— Regular uterine contractions with or without pain (at least one in every 10 minute) (Fig. 9.5);

— Dilatation (> 2 cm) and effacement (80%) of the cervix;

— Length of the cervix (measured by TVU) < 2.5 cm and funnelling of the internal os;

Positive Negative Invalid

Fig. 9.4

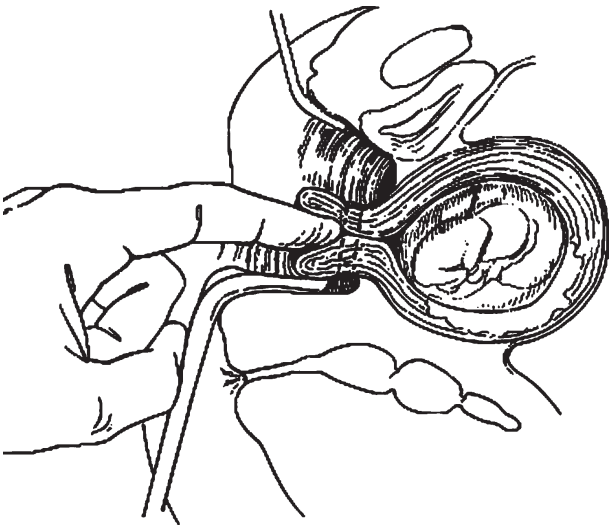


Fig. 9.5

— Pelvic pressure, backache and or vaginal discharge or bleeding.

Management

Management of preterm labor consists of tocolysis (trying to stop uterine contractions) or allowing labor to progress. Maternal problems are chiefly related to interventions carried out to stop contractions.

Confirm the diagnosis of preterm labor by documenting cervical effacement or dilatation over two hours.

Tocolytic therapy:

This intervention aims to delay delivery until the effect of corticosteroids has been achieved. Give a tocolytic drug and monitor maternal and fetal condition (pulse, blood pressure, signs of respiratory distress, uterine contractions, loss of amniotic fluid or blood, fetal heart rate, fluid balance, blood glucose, etc.). Duration of the treatment is 48 hours, regardless of which drug is used.

The main objective is to postpone delivery in order to administer corticosteroids for accelerated fetal lung maturation.

Nifedipine (short-acting capsule 10 mg). It is a calcium channel blocker of the dihydropyridine type. 10 mg to be repeated every 15 minutes if uterine contraction persists (maximum 4 doses or 40 mg), then 20 mg every 6 hours. Never administer sublingually (risk of placental hypoperfusion and fetal death); always use the oral route.

Beta-sympathomimetic drugs

Betamimetics such as Salbutamol and Ritodrine inhibit sympathetic control of myometrium and delay delivery for between 24 and 48 hours but do not alter perinatal mortality rates. They are of value in delaying delivery until transfer to a facility with adequate paediatric support has taken place, and in allowing the administration of corticosteroids to accelerate fetal lung maturity.

They have several side-effects including maternal tachycardia, hyperglycemia and hypokalemia. A pulse rate of between 130–140 is acceptable, but overdose may cause serious cardiac arrhythmias. There have been persistent reports of postpartum pulmonary oedema following the use of betamimetics in association with corticosteroids. Considerable caution should be exercised if these agents are used in combination.

Cardiac disease and diabetes are contraindications to the use of betamimetics.

Prostaglandin inhibitors

Drugs such as Indomethacin inhibit the production of prostaglandin synthetase and undoubtedly reduce uterine activity. Indomethacin appears to be a better tocolytic than betamimetics but there are risks of premature closure of the fetal ductus arteriosus and a reduction in fetal renal blood flow leading to renal failure if not adequately monitored. There have also been reports of increased rates of necrotising enterocolitis in the neonate if delivered within 48 hours of the last dose. Nevertheless, Indomethacin does appear to be an effective treatment for preterm labor and is being used increasingly. It may be given rectally as a 100 mg suppository twice daily for 48 hours.

Attempt tocolysis if:

— gestation is less than 37 weeks;

— the cervix is less than 3 cm dilated;

— there is no amnionitis, preeclampsia or active bleeding;

— there is no fetal distress.

Prepare the fetus for preterm birth:

After 26 weeks and before 34 weeks gestation, give corticosteroids to the mother to improve fetal lung maturity and chances of neonatal survival:

— dexamethasone 6 mg every 12 hours for 48 hours.

Note: Corticosteroids should not be used in the presence of frank infection.

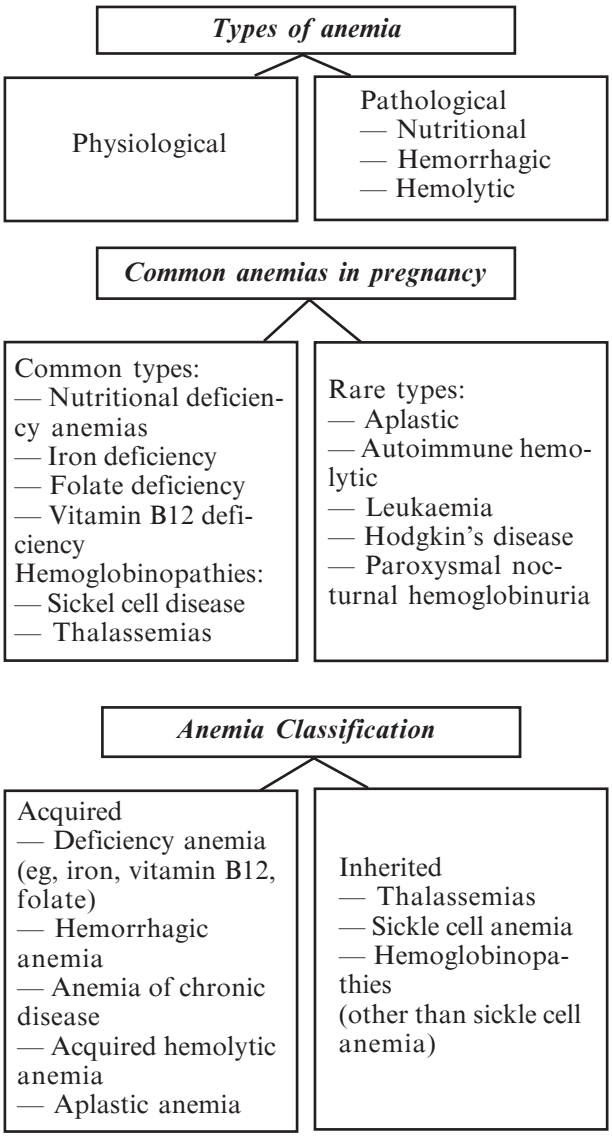
Monitor progress of labor using the partograph.

Chapter 10
PATHOLOGIES DURING PREGNANCY AND PREGNANCY-RELATED DISORDERS

ANEMIA

COMMONEST MEDICAL DISORDER IN PREGNANCY

Anemia is defined as reduction in circulating hemoglobin mass below the critical level. The normal hemoglobin (Hb) concentration in the body is between 12–14 g/percent. WHO has accepted up to 11 g/percent as the normal hemoglobin level in pregnancy.



Anemia ranges from mild, moderate to severe and the WHO pegs the hemoglobin level for each of these types of anemia in pregnancy at 9.0–10.9 g/dl (mild anemia); 7–8.9 g/dl (moderate anemia); < 7 g/dl (severe anemia) and very severe anemia < 4 g/dl.

According to WHO, in developing countries the prevalence of anemia in pregnant women averages 56%, ranging between 35–100% in different regions of the world. 38.2% of pregnant women suffer from anemia [14, 20, 35].

IRON DEFICIENCY ANEMIA (IDA)

During pregnancy there is increase in total blood volume (1500 ml = 30–40%), plasma volume (250 ml = = 40–50%) as well as the exhaled breath condensate (EBC) volume (350 ml = 20–30%) also, but increment in plasma volume is more than the increased total hemoglobin (15–20%). Hence there is dilution of blood, resulting in physiological anemia (upper limit for normal/100% Hb level in pregnancy is brought down to 11 gm%).

The gastrointestinal tract increases iron absorption when the body's iron stores are low, and it reduces the absorption when there are sufficient stores. Requirement for absorbed iron ranges from 0.8 mg/day in the first trimester to 7.5 mg/day in the second trimester, averaging approximately 4.4 mg/day in pregnancy. Iron requirements increase rapidly in the second and third trimester due to fetal growth, however iron absorption in the gut is not sufficient to meet this increased demand. Thus iron balance depends on maternal iron stores during this period.

Predisposing factors during pregnancy

— Increased demands of iron, folic acid, B12.

— Diminished intake (poor diet, morning sickness).

— Distributed metabolism (pregnancy induced depression of the bone marrow).

— Infection (malaria, asymptomatic bacteriuria, piles worm infestation).

— Pre pregnant state of iron reserves.

— Twin gestation.

Table 6

WHO classification of anemia,%

Degree	Hb	Haematocrit
Moderate	7–10.9	24–37%
Severe	4–6.9	13–23%
Very severe	< 4	< 13%

Major causes of anemia

— Low vegetable consumption and perhaps low B12 intake.
— Poor diet in absorbable iron.
— Chronic blood loss.
— Increased requirement of iron during pregnancy.
— Malabsorption.
— Poor pregnancy iron balance due to — untreated systemic diseases, closely spaced pregnancies and menstrual disorders.

Maternal risk factors (Fig. 10.1)

Fetal and neonatal risk factors

Prematurity
— Low birth weight
— Poor Apgar score
— Fetal distress
— Neonatal anemia

Clinical features of anemia

Symptoms

— Lethargy/Fatigue
— Shortness of breath
— Dyspnoea
— Palpitation
— Irritability
— Loss of appetite
— Dizziness
— Digestive upset

Signs

— Tachycardia
— Soft ejection systolic murmur
— Pallor of the conjunctiva, mucous membranes, palms
— Pale nails
— Pale tongue
— Koilonychias

Preventative

Regular iron-bearing foods in diet. If needed, iron tablet supplements.

Key points

1. All women should be offered screening for anemia:

— in first trimester (or at booking);
— with the next screening bloods (usually performed between 24–28 weeks);
— and at 36 weeks gestation.

2. IDA in most circumstances is diagnosed by a full blood examination and serum ferritin levels. Do not use serum iron, or serum ferritin alone to diagnose IDA.

Ferritin levels are elevated in active infection or inflammation and in these cases concurrent measurement of C-reactive protein (CRP) will support interpretation of ferritin levels.

Serum ferritin should be checked prior to starting iron with known hemoglobinopathy.

3. Oral iron if taken at the appropriate dose, and for a sufficient time, is an effective first-line treatment for most women in pregnancy.

A high iron diet should be recommended, including red meats (if possible), fortified cereals and drinks.

Pregnant women should be given 100 mg/day iron with 300 mg folic acid regardless of ID status in 2nd and 3rd trimester, prophylactically. Treatment of IDA should aim at replenishing body iron deficits.

4. Intravenous iron polymaltose therapy is an effective alternative to oral treatment during the second or third trimester only for treatment of IDA. Intravenous iron should only be used in women failing to respond to oral iron treatment with known IDA or in those whom a rapid repletion of iron stores is required.

5. At each antenatal visit all women taking iron supplements should be monitored for medication compliance and side-effects.

6. Women with a normal Hb and ferritin levels < 30μg/L should be commenced on oral iron supplements in pregnancy to prevent development of ane-

Fig. 10.1. Maternal risk factors

mia. A dosage of 65 mg elemental iron should be taken once daily.

7. Treating and preventing IDA can improve national productivity by 20% and reduce maternal mortality.

RHESUS (Rh) INCOMPATIBILITY

In 1909, Landsteiner classified the blood of human beings into the now well-known A, B, AB, and 0 groups and showed that transfusions between individuals of groups A or B do not result in the destruction of new blood cells and that this catastrophe occurs only when a person is transfused with the blood of a person belonging to a different group. (He had suggested that, because the characteristics which determine the blood groups are inherited, the blood groups may be used to decide instances of doubtful paternity.) (Fig. 10.2).

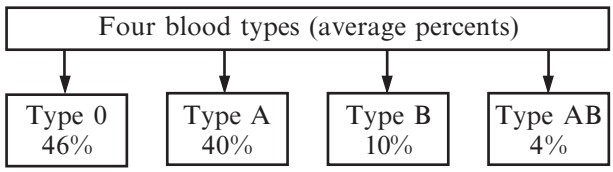


Fig. 10.2

Each blood type is additionally classified according to the presence or absence of the Rh factor.

The Rh genes are carried on a pair of chromosomes. There are six Rh antigens (C, D, E, c, d, e) of which D and d are the most important, for upon these depend whether a person is designated Rh-positive or Rh-negative.

The Rh factor, Rh+ and Rh- usually refers specifically to the presence or absence of antigen-D. There are two alleles, or genetic variants, of this antigen: D and d. A person who is Rh- has two recessive traits, dd. Anyone who has at least one D-DD or Dd is Rh+.

A person's Rh type is generally most relevant with respect to pregnancies (Fig. 10.3).

There is no problem if the woman is Rh-positive, even if her partner is a Rh-negative man; if homozygous, all her children will be Rh-positive; if heterozygous, she may have a Rh-negative child but that is no problem.

Should she be Rh-negative and her partner homozygous Rh-positive (35% of the male population), she will always have a Rh-positive child and there may be problems.

He may be heterozygous Rh-positive (65% of the male population) producing equal numbers of Rh-positive and Rh-negative gametes having equal chances of giving his Rh-negative partner a Rh-positive or a Rh-negative child.

Rh incompatibility is a condition which develops when there is a difference in Rh blood type between that of the pregnant mother (Rh-negative) and that of the fetus (Rh-positive). If the pregnant woman and her husband are Rh-negative, there is no reason to worry about Rh incompatibility.

Usually placenta acts as a barrier to fetal blood entering maternal circulation. However, sometimes during pregnancy or birth, fetomaternal hemorrhage (FMH) can occur. The woman's immune system reacts by producing anti-D antibodies that cause sensitization.

In most Rh-incompatible pregnancies no antibody is formed until after the first fetomaternal bleed most commonly in the third stage of labor and, consequently, the baby of the first pregnancy is unaffected. In subsequent pregnancies, if the fetus is Rh positive, small fetomaternal bleeds may evoke a major secondary antibody response. Large amounts of antibody (immunoglobulin G (IgG)) cross the placenta and can cause increasingly severe Rh disease in successive pregnancies if the fetus is Rh-positive. The antibody weakens the envelopes of the fetal red cells, which are then broken down in the spleen [17, 31].

Detect at-risk fetus

— Maternal Rh screening, anti-D antibody titres.

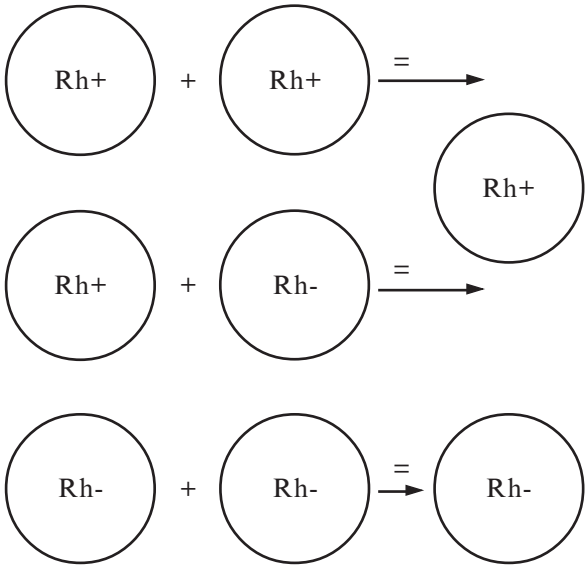
— Ultrasound scan to detect hydrops fetalis — oedema of the skin, pleural effusion, ascites, hepatosplenomegaly, cardiac enlargement.

— Amniocentesis or cordocentesis is performed under ultrasound guidance. Check for hemoglobin and bilirubin.

— Check cord blood immediately after birth.

Clinical picture

The fetus may die in utero if the anemia is severe enough. The infant may be born grossly anaemic and oedematous with hepato-splenomegaly-hydrops fetalis. There is a rapid rise in bilirubin following birth. Jaundice develops rapidly within the first 24 hours of life. The infant can be anaemic and continues to break down red blood cells after delivery as the maternal Rh antibodies are still circulating in his blood, and so can become more anaemic and jaundiced during the postnatal period.



Rh is inherited as a dominant allele, so an Rh- mather can still have an Rh+ child due to the father's genotype.

Fig. 10.3

Prevention

— Give anti-D immunoglobulin to all Rh-negative women at 26 and 34 weeks; or give anti-D immunoglobulin if she has a: therapeutic abortion, spontaneous abortion/ectopic pregnancy, amniocentesis, any bleeding in pregnancy/threatened miscarriage.

— If the baby is Rh-positive, another dose is administered within 72 hours after delivery. This will prevent her body from creating any future antibodies that could cause harm during a pregnancy.

Treatment

— Intrauterine transfusion.
— Elect time of delivery.
— Exchange transfusion after delivery.
— Phototherapy after delivery.

URINARY TRACT INFECTION IN PREGNANCY

Urinary tract infection (UTI) is the 2nd most common infectious presentation in community practices. Worldwide about 150 million people are diagnosed with UTI each year (Fig. 10.3).

Symptomatic

— Cystitis
— Pyelonephritis

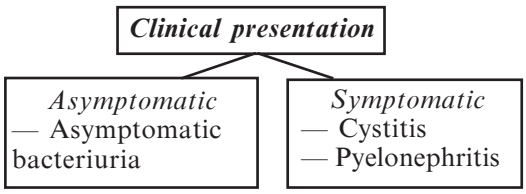
UTI is an inflammatory response of the urothelium to bacterial invasion and can occur in females and males, in all age groups. About 35% of healthy women suffer from symptoms of UTI at some time in their life. Pregnancy UTIs are detected in 2 to 8% of pregnant women [9, 31, 50].

During pregnancy the ureters are dilated and kinked because of:

— Increased progesterone levels which relax the smooth muscle.

— Mild obstruction of the lower ureters in late pregnancy.

This encourages:



— Stasis of urine.

— Reflux of infected urine to the kidney evokes pyelonephritis.

Risks

— Low birth weight baby

— Low gestational age (< 37 weeks) and prematurity

— IUGR

— Cesarean deliveries

— Increased neonatal mortality

ASYMPTOMATIC BACTERIURIA

The presence of more than 100.000 organisms/mL in 2 consecutive urine samples in the absence of symptoms. Pregnant: 2.5–11% Non-pregnant: 3–8%.

All pregnant women must be screened for asymptomatic bacteriuria (ASB) on their first prenatal visit between the 9th to 17th weeks, preferably on the 16th week of gestation.

Incidence

About 3% of pregnant women — increases with parity and age.

Significance

Asymptomatic bacteriuria is associated with a risk of:

— Acute pyelonephritis in pregnancy (30%).

— Structural abnormalities in the urinary tract (3–5%).

Stages of a urinary tract infection

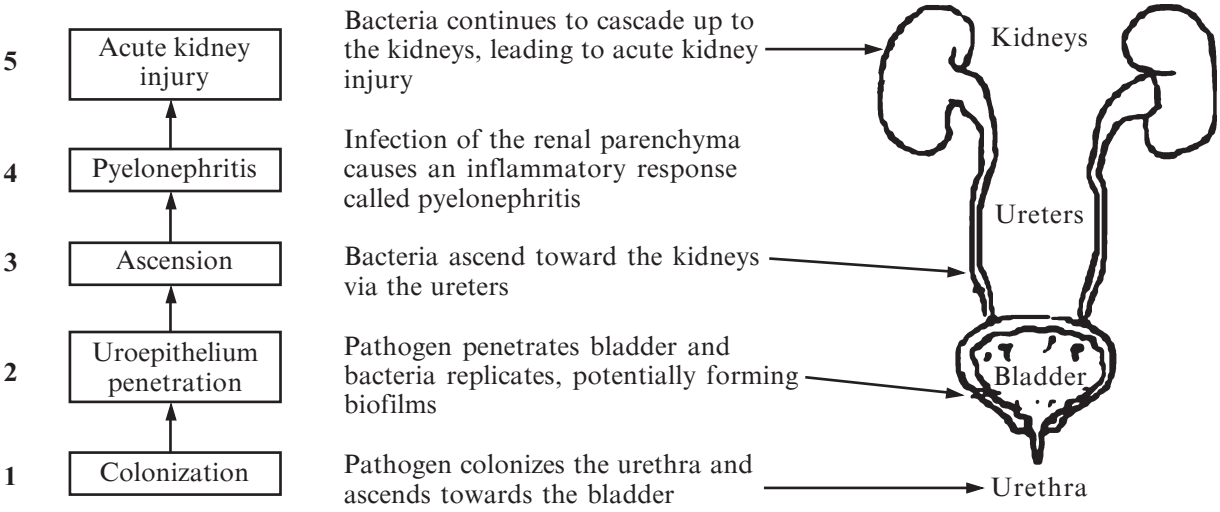


Fig. 10.3

Screening

In early pregnancy, all women should have urine tested for either:

— the presence of white cells and nitrites, or;

— cultured for bacteria.

Treatment

The most common organisms grown are:

— Escherichia coli.

— Proteus mirabilis.

A 5-day course of an antibiotic to which the organism is sensitive should be prescribed. This will result in a cure in more than 85% of women, but the urine should be recultured one week after treatment. A renal ultrasound and an intravenous urogram should be performed 3 months after delivery to exclude a structural urinary tract abnormality.

SYMPTOMATIC INFECTIONS

Incidence 1–2%; commoner in primigravidae. Increases with maternal age.

Symptoms

— Dysuria (due to urethritis).

— Increased frequency (due to trigonitis).

— Backache, loin pains, night sweats and rigors (due to pyelonephritis).

— Headache, vomiting and muscle aches (due to pyrexia).

Examination

— The woman is usually pyrexial if the infection has involved the kidneys. In many cases this may be at levels of up to 40.5° C.

— If the woman has pyelonephritis she will be tender in the renal angles.

Investigation

A mid-stream specimen of urine (MSU) should be sent for:

— Dipstick for nitrites and leucocytes.

— Microscopy for white cells.

— Culture to determine the organism responsible.

— Sensitivity of organisms to antibiotics.

Management

All women who have renal angle tenderness or a pyrexia must be admitted to hospital because of the threat of preterm labor.

Management consists of:

1. Bed rest.
2. Ample fluid intake, at least 3 liters a day.
3. Start a broad-spectrum antibiotic.

The antibiotics can be given orally once temperature is normal. A complete 5-day course at least should be given.

4. If the woman has pyelonephritis, do a renal ultrasound when she has recovered from the infection.
5. The urine should be recultured 5 days after the last dose of antibiotic has been given.

CHRONIC RENAL DISEASE

Renal changes in normal pregnancy.

— Renal blood flow increases.

— Glomerular filtration rate (GFR) increases.

— Plasma concentrations of urea and creatinine fall in normal pregnancy.

— There is an increase in total body water that exceeds the increase in total body sodium, resulting in a decrease in plasma osmolality.

— There is a 25% fall in serum uric acid concentrations during the first two trimesters but this returns to pre-pregnant levels by the third trimester. Watch if using urate to monitor preeclampsia.

Prognosis

The outcome of the pregnancy is worse if:

1. The woman was hypertensive before pregnancy.
2. The woman had proteinuria before pregnancy started.
3. There is active progression of renal disease or it is associated with other medical conditions. Pregnancy probably has no long-term adverse effects on renal disease.

Fetal prognosis

— Normotensive women with chronic renal disease have 2–3 times greater risk of developing preeclampsia. In the absence of preeclamptic toxaemia (PET) perinatal mortality is not increased. If PET develops, the risk of fetal death is directly related to the gestation at delivery.

— Women with more severe renal disease have a high incidence of both PET and impaired fetal growth. Among women with pre-existing hypertension and proteinuria, the perinatal mortality rates approach 30%. Cause of death is from preterm delivery and complications associated with small gestational age (SGA).

ACUTE RENAL FAILURE IN PREGNANCY

This may be:

1. Tubular necrosis: this is largely recoverable.
2. Cortical necrosis: this is usually irrecoverable and these patients go on to need long-term dialysis or transplantation.

Presentation

1. Oliguria: < 500 ml/day (20 ml/hour), the minimum volume to remove catabolites.
2. Anuria: the absence of urine.

Etiology in obstetrics

1. Hypovolaemia.

— Severe preeclampsia.

— Placental abruption.

— Postpartum hemorrhage (PPH).

— Hyperemesis gravidarum.

— Miscarriage.

2. Gram-negative shock. This may result from:

— Pyelonephritis.

— Chorioamnionitis.

— Puerperal infections.

— Septic miscarriage. The usual organism is E. coli, but it may be Clostridium.

3. Nephrotoxins. In modern obstetric practice these are rare. Illegal abortions may result in infection followed by hemolysis and renal failure.
4. Acute renal failure associated with acute fatty liver of pregnancy. Rare; usually fatal.

5. Vomiting in late pregnancy associated with jaundice. The disease occurs in many systems and renal failure, pancreatitis and colitis may occur.

Management

Three consecutive phases:

1. Oliguria: lasts from a few days to a few weeks. Complete anuria is rare in acute tubular necrosis and usually suggests acute cortical necrosis or obstruction.

2. Polyuria: markedly increased urine production that may last up to 2 weeks. The urine is dilute and metabolic waste products are poorly eliminated. Plasma urea and creatinine may continue to rise for several days following the increase in urinary output. Profound fluid and electrolyte losses can occur in this phase.

3. Recovery: urinary volumes decrease towards normal and renal function improves.

General management:

— Determine the cause.

— Insert a urinary catheter and maintain accurate fluid balance charts.

— Insert a central venous pressure line and measure the pressure.

— Send baseline investigations, including urea and electrolytes, liver function tests, serum amylase, plasma proteins, coagulation studies, and if required, perform an arterial blood sample for acid–base balance.

HYPERTENSIVE DISORDERS IN PREGNANCY (HDP)

Gestoses and hypertensive disorders are among the most common and yet serious conditions seen in obstetrics.

Hypertensive disorders of pregnancy affect about 10% of all pregnant women around the world. This group of diseases and conditions includes preeclampsia and eclampsia, gestational hypertension and chronic hypertension. Hypertensive disorders of pregnancy are an important cause of severe acute morbidity, long term disability and death among mothers and babies [6, 19, 24, 27, 45].

Normal blood pressure (BP) changes in pregnancy:

— Decreases during the first trimester

— Reaching its lowest point at 20 weeks

— Returns to pre-pregnancy levels during the third trimester

Accurate measurement of blood pressure is crucial to the diagnosis and management of hypertensive disorders in pregnancy. Blood pressure monitoring is the most important, and frequent, screening test in the antenatal period and is undertaken by healthcare assistants, midwives, general practitioners.

Blood pressure measurement is a key part of the assessment of hypertensive disorders in pregnancy, guiding diagnosis, admission, antihypertensive treatment and timing of delivery, as well as the assessment of hemodynamic shock in pregnancy, secondary to obstetric hemorrhage or sepsis. It is therefore important that all healthcare providers are aware of the issues surrounding accuracy of BP measurement in pregnancy.

Techniques for the measurement of blood pressure in pregnancy are described in 'Antenatal care' NICE clinical guideline 62 (National Institute for Health and Care Excellence) (Fig. 10.4).

Blood pressure measurement

— Sitting position

— Patient relaxed

— Arm well supported

— Measured in right arm

— Cuff at heart level

— Proper cuff size (80% of arm circumference)

Obstetricians use BP values to guide management in women with hypertensive disorders in pregnancy. If BP is underestimated or overestimated through inaccurate measurement, avoidable maternal and perinatal mortality and morbidity can result. Furthermore, obstetricians use BP thresholds recommended by national guidelines to aid in management decision making. If these thresholds are not supported by adequate evidence, maternal and perinatal mortality and morbidity can again result.

Definitions and stratification of hypertension in pregnancy

HDP are comprised of a spectrum of disorders typically classified into categories that include chronic (preexisting) hypertension, gestational hypertension, preeclampsia (including chronic (preexisting) hypertension with superimposed preeclampsia) and eclampsia. Hypertension and preeclampsia are stratified according to severity (Fig. 10.5).

HYPERTENSION

Hypertension during pregnancy is defined as a systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg. It should

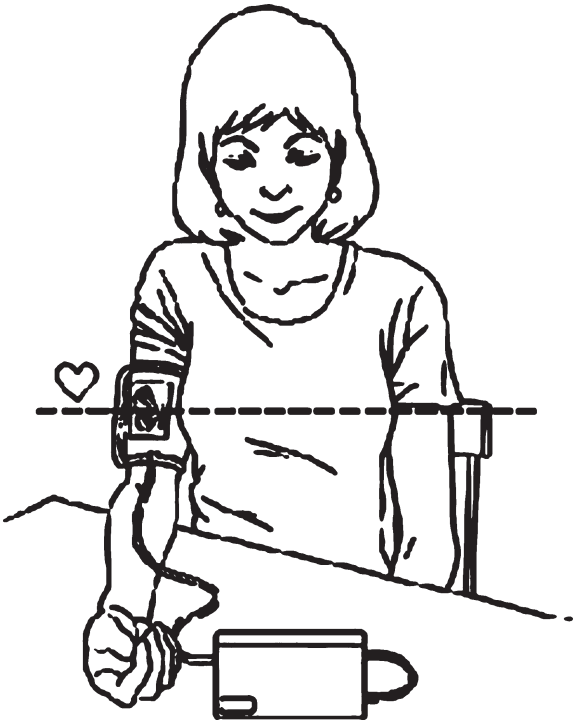


Fig. 10.4. Blood pressure measurement

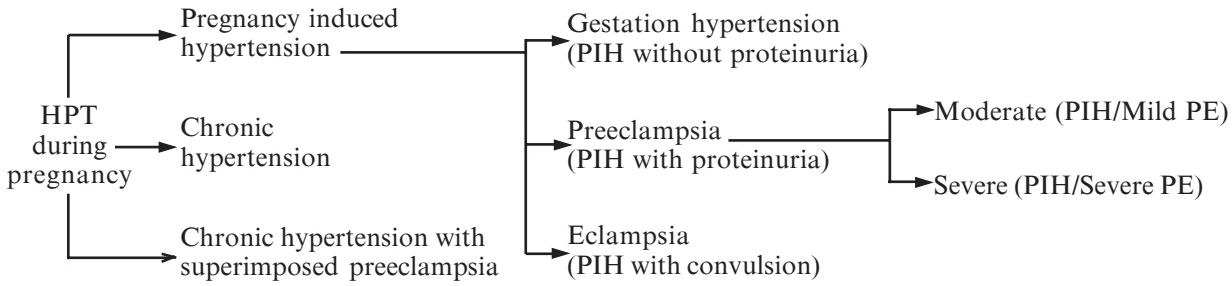


Fig. 10.5

be documented on at least 2 occasions measured 4 hrs apart. Readings should be confirmed using appropriate measurement technique, and should be re-measured after 10–15 minutes of rest.

The NICE guidelines on hypertension in pregnancy define:

Non-severe hypertension: SBP 140–159 mmHg or DBP 90–109 mmHg:

— mild: SBP 140–149 mmHg or DBP 90–99 mmHg;

— moderate: SBP 150–160 mmHg or DBP 100–110 mmHg.

Severe hypertension: SBP > 160 mm Hg or DBP >110 mmHg or both.

CHRONIC (PREEXISTING) HYPERTENSION

Chronic (preexisting) hypertension is defined as hypertension (systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg or both) that is present before 20 weeks of gestation or prior to pregnancy. Elevated readings should be documented on more than one occasion.

The diagnosis of chronic hypertension is relatively usual in women who take antihypertensive medications before conception. However, chronic hypertension can be difficult to distinguish from either gestational hypertension or preeclampsia in women who present for care with hypertension late in gestation. To establish the diagnosis of hypertension, it is optimal to measure blood pressure before 12 weeks of gestation because the normal decrease in blood pressure, which has its nadir at 16–18 weeks of gestation, may mask previously undiagnosed chronic hypertension. Thus, hypertension may only become apparent when blood pressure increases later in pregnancy. In this scenario, hypertension that persists longer than the postpartum period (12 weeks after delivery) is reclassified as chronic.

GESTATIONAL HYPERTENSION

Gestational hypertension (HTN) is new hypertension presenting after 20 weeks without significant proteinuria or other features of preeclampsia, followed by return of BP to normal within 12 weeks postpartum. This terminology replaces the term "Pregnancy Induced Hypertension" (PIH).

Fifty percent of women diagnosed with gestational hypertension between 24 and 35 weeks develop preeclampsia. The diagnosis of gestational hypertension mandates increased surveillance. Women who progress to severe gestational hypertension based on the degree of blood pressure elevation have worse perinatal outcomes than do women with mild preeclampsia, and require management similar to those with severe preeclampsia.

PREECLAMPSIA

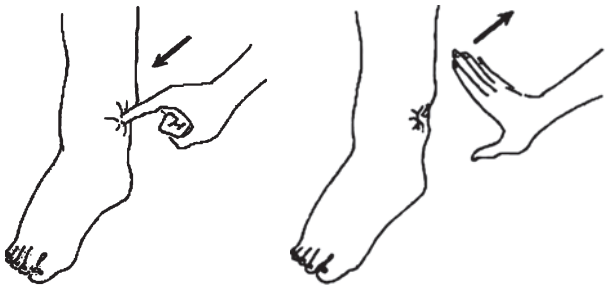
PREECLAMPSIA AND CHRONIC (PREEXISTING) HYPERTENSION WITH SUPERIMPOSED PREECLAMPSIA

Screening women at high risk and preventing recurrences are key issues in the management of preeclampsia.

Preeclampsia is defined as hypertension plus significant proteinuria (after 20 weeks of gestation), specifically gestational hypertension plus new onset proteinuria, or chronic (preexisting) hypertension with new or worsening proteinuria. When preeclampsia develops in women with chronic (preexisting) hypertension, the classification of disease is chronic (preexisting) hypertension with superimposed preeclampsia. Preeclampsia can also occur without proteinuria, with hepatic, hematopoietic, or other manifestations. Edema is no longer considered a specific diagnostic criterion for preeclampsia. Pregnant women with hypertension plus other adverse conditions but no proteinuria should have further evaluation for preeclampsia (Fig. 10.6) [39, 44, 45, 47].

SIGNIFICANT PROTEINURIA IN PREGNANCY

In the context of identification of preeclampsia, significant proteinuria is present when 24-hour urine protein is equal to or exceeds 300 mg/L of protein.



Press the ankle here with your finger and count to 10 before releasing

If your finger leaves a pit that stays for a whine it is "pitting oedema"

Fig. 10.6

The spot urine protein: creatinine ratio has also been used to define significant proteinuria in the identification of preeclampsia. NICE identify significant proteinuria as a protein: creatinine ratio of ≥ 30 mg protein/mmol creatinine.

SEVERE PREECLAMPSIA

American Congress of Obstetricians and Gynecologists (ACOG) parameters should be used to define severe preeclampsia; ACOG criteria for severe preeclampsia include the presence of any one of the following: severe hypertension (systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg, or both), cerebral or visual disturbance, epigastric or right upper quadrant pain, oliguria, pulmonary edema, cyanosis, impaired liver function, thrombocytopenia or intrauterine growth restriction (IUGR) (Fig. 10.7).

Danger sign of severe preeclampsia
- Headache
- Blindness
- Upper quadrant abdominal pain
- Visual disturbance
- Decreased urine output
- History of convulsion(s)
- Restlessness
- Nausea/vomiting
- Respiratory symptoms that might indicate pulmonary edema (dyspnea, chest pain, cough)

Fig. 10.7

ECLAMPSIA

Eclampsia is defined as new onset grand mal seizures in women with preeclampsia. Some women presenting with eclampsia do not have diagnosed preeclampsia, and some women may present with eclampsia in the post-partum period.

Eclampsia and HELLP syndrome (Hemolysis, Elevated Liver enzymes and Low Platelet count) are two severe conditions that can manifest in women with preeclampsia, and each triggers distinct management considerations. Eclampsia is defined as new onset grand mal seizures in women with preeclampsia. Eclampsia may manifest in women with preeclampsia postpartum, but ACOG guidelines note that other causes may be more likely if seizures occur beyond 48–72 hours postpartum (Fig. 10.8, Fig. 10.9).

HELLP syndrome

HELLP syndrome is a serious systemic disorder associated with preeclampsia and manifested by hemolysis, elevated liver enzymes and a low platelet count. HELLP syndrome can manifest with or without proteinuria. HELLP syndrome has been noted to occur in approximately 20% of women with severe preeclampsia, as noted in the ACOG preeclampsia practice bulletin.

Preeclampsia: pathophysiology

During normal pregnancy, the villous cytotrophoblast invades into the inner third of the myometrium, and spiral arteries lose their endothelium and most of their muscle fibers. These structural modifications are associated with functional alterations, such that spiral arteries become low-resistance vessels, and thus less sensitive, or even insensitive, to vasoconstrictive substances.

Preeclampsia has a complex pathophysiology, the primary cause being abnormal placentation. Defective invasion of the spiral arteries by cytotrophoblast cells is observed during preeclampsia. Recent studies have shown that cytotrophoblast invasion of the uterus is actually a unique differentiation pathway in which the fetal cells adopt certain attributes of the maternal endothelium they normally replace. In preeclampsia, this differentiation process goes awry. The abnormalities may be related to the nitric oxide pathway, which contributes substantially to the control of vascular tone. Moreover, inhibition of maternal synthesis of nitric oxide prevents embryo implantation. Increased uterine arterial resistance induces higher sensitivity to vasoconstriction and thus chronic placental ischemia and oxidative stress.

Table 7

Indicators of preeclampsia severity

Abnormalities	Mild	Severe
Blood pressure	≥ 140/90mmHg but < 160/110mmHg	≥ 160/110mmHg
Proteinuria	≤ 2+	≥ 3+
Oliguria	Absent	< 400 ml/day
Headache	Absent	Present
Visual disturbances	Absent	Present
Platelet count	Normal	Thrombocytopenia (< 100,000/mm^3)
HELLP syndrome	Absent	May be present ALT, AST > 70 IU/L LDH > 600 IU/L Bilirubin > 1.2 g/L
Serum transaminases (AST, ALT)	Normal (< 40 IU/L)	Elevated
Serum creatinine	Normal	Elevated

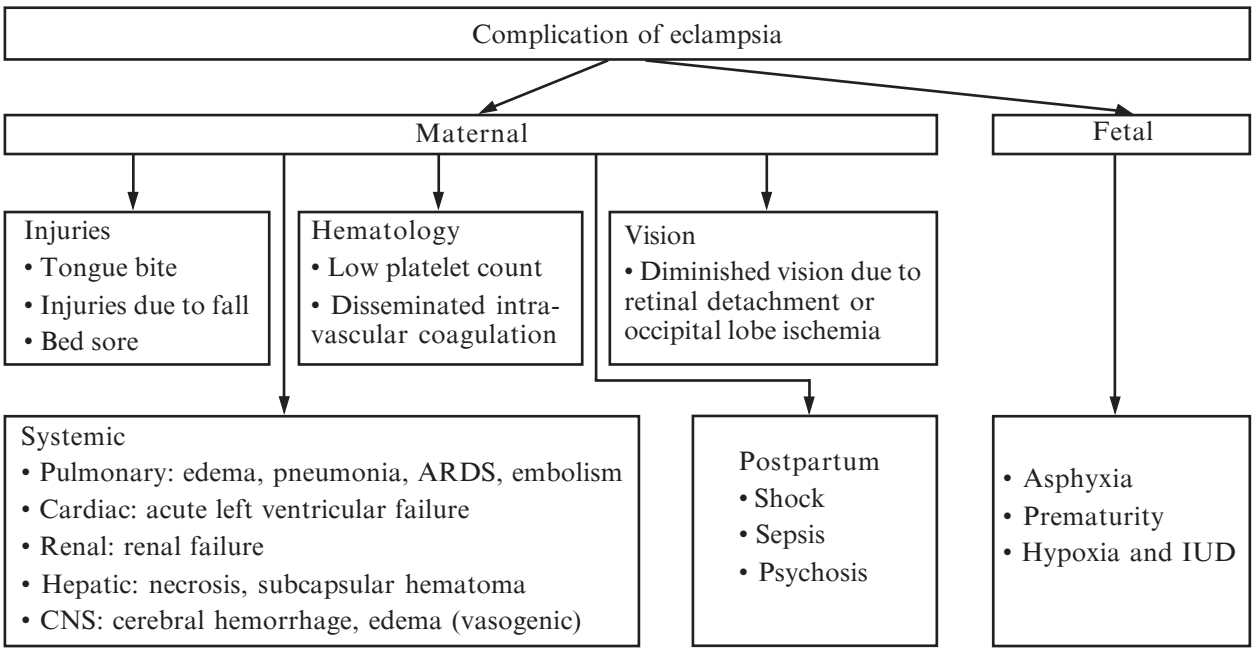


Fig. 10.8

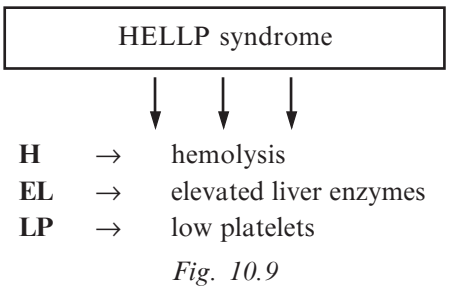


Fig. 10.9

This chronic placental ischemia causes fetal complications, including intrauterine growth retardation and intrauterine death. In parallel, oxidative stress induces release into the maternal circulation of substances such as free radicals, oxidized lipids, cytokines, and serum soluble vascular endothelial growth factor 1. These abnormalities are responsible for endothelial dysfunction with vascular hyperpermeability, thrombophilia, and hypertension, so as to compensate for the decreased flow in the uterine arteries due to peripheral vasoconstriction.

Endothelial dysfunction is responsible for the clinical signs observed in the mother, i.e., impairment of the hepatic endothelium contributing to onset of the HELLP syndrome, impairment of the cerebral endothelium inducing refractory neurological disorders, or even eclampsia. Depletion of vascular endothelial growth factor in the podocytes makes the endotheliosis more able to block the slit diaphragms in the basement membrane, adding to decreased glomerular filtration and causing proteinuria. Finally, endothelial dysfunction promotes microangiopathic hemolytic anemia, and vascular hyperpermeability associated with low serum albumin causes edema, particularly in the lower limbs or lungs.

The crucial issue to understand is that the prime mover of preeclampsia is abnormal placentation. Two common theories appear to be interlinked, i.e., a genetic theory and an immunological theory. Several susceptibility genes may exist for preeclampsia. These genes probably interact in the hemostatic and cardiovascular systems, as well as in the inflammatory response. Some have been identified, and in candidate gene studies they have provided evidence of linkage to several genes.

Preeclampsia can be perceived as an impairment of the maternal immune system that prevents it from recognizing the fetoplacental unit. Excessive production of immune cells causes secretion of tumor necrosis factor alpha which induces apoptosis of the extravillous cytotrophoblast. The human leukocyte antigen (HLA) system also appears to play a role in the defective invasion of the spiral arteries, in that women with preeclampsia show reduced levels of HLA-G and HLA-E. During normal pregnancies, the interaction between these cells and the trophoblast is due to secretion of vascular endothelial growth factor and placental growth factor by natural killer cells. High levels of soluble fms-like tyrosine kinase 1 (sFlt-1), an antagonist of vascular endothelial growth factor and placental growth factor, have been found in women with preeclampsia. Accordingly, assays of sFlt-1, placental growth factor, endoglin, and vascular endothelial growth factor, all of which increase 4–8 weeks before onset of the disease, may be useful predictors of preeclampsia.

Assessment of HDP

Assessment of HDP includes assessment of the risk for preeclampsia, the severity of preeclampsia, and the presence of additional relevant findings, including identifiable causes of hypertension or kidney disease.

Assessment of risk for preeclampsia

Various conditions predispose to preeclampsia, including chronic (preexisting) hypertension, previous

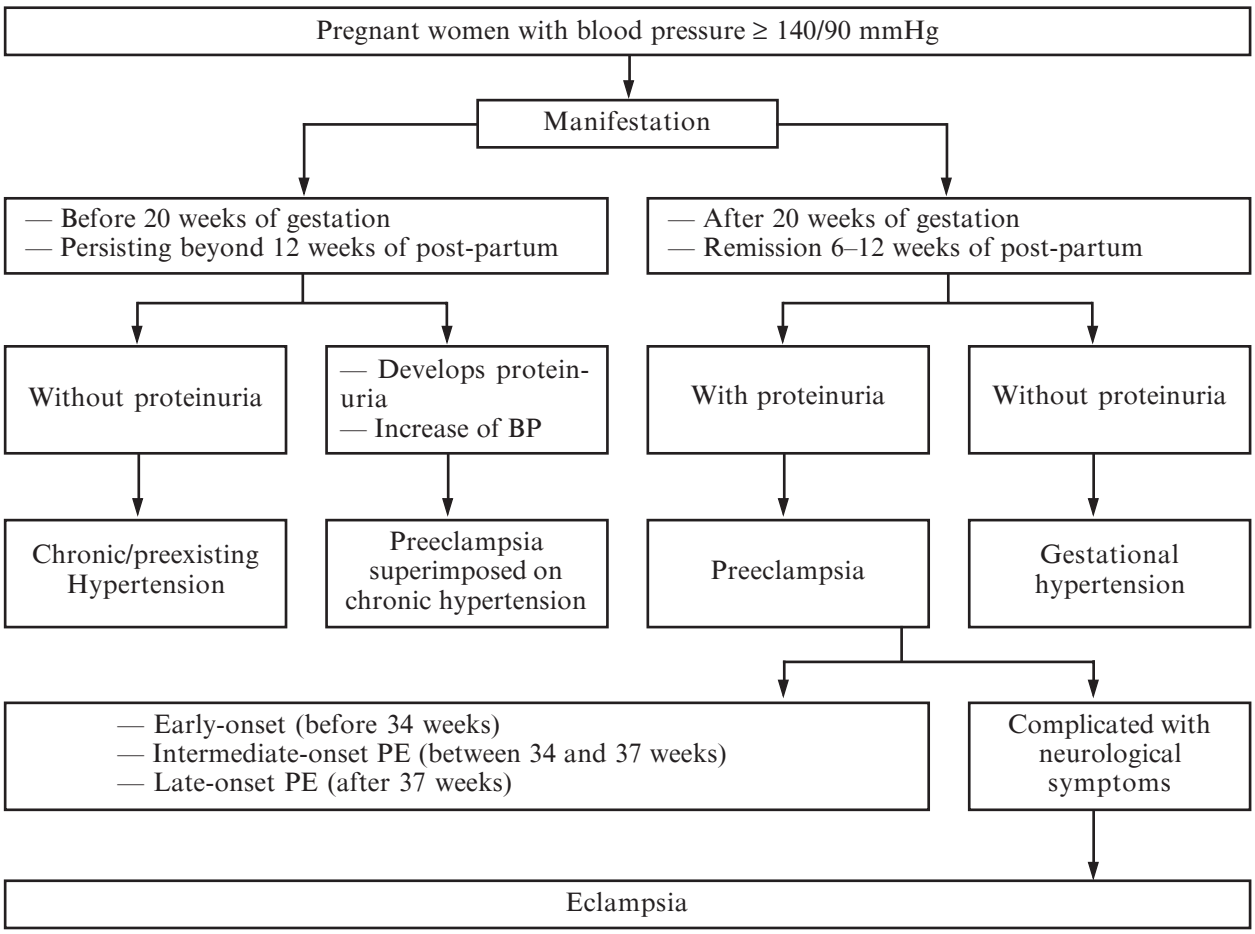


Fig. 10.10

preeclampsia, autoimmune disease/antiphospholipid antibodies, chronic kidney disease, and preexisting diabetes mellitus. Women with these conditions are considered to be at high risk for HDP. Other factors that increase the risk for HDP include, but are not limited to, multifetal pregnancy, elevated pre-pregnancy Body Mass Index (BMI), maternal age ≥ 40, nulliparity, vascular and connective tissue disease, family history of preeclampsia, thrombophilia and interpregnancy interval of greater than 10 years. Black race has also been associated with increased risk for preeclampsia. Consideration should be given to risk factors for preeclampsia when developing surveillance and monitoring strategies, including visit frequency.

Proteinuria testing is a priority area for the identification and management of HDP. All women should have standard dipstick screening for proteinuria at each prenatal visit. Women diagnosed with hypertension in pregnancy and other women at high risk for preeclampsia should have more definitive evaluation of proteinuria than women at low risk, with either 24-hour urine collection for protein or spot urinary protein: creatinine ratio to quantify the amount of proteinuria.

Baseline renal function assessment, including serum creatinine, blood urea nitrogen and 24-hour urinary protein or spot urine for protein: creatinine ratio, is recommended for all pregnant women with chronic (preexisting) hypertension. Most guidelines recommend additional laboratory testing for women at risk for preeclampsia, including but not limited to complete blood count, platelet count, aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), bilirubin and serum uric acid.

Diagnosis

— Determine the severity of hypertension
— Measure protein excretion
— 24-hour urine collection
— Evaluate for signs/symptoms of preeclampsia
— Perform laboratory evaluation
— +/- end-organ involvement

Severe preeclampsia is defined as any of the following:

— Markedly elevated blood pressure measurements (systolic ≥ 160 mm Hg or diastolic ≥ 110 mm Hg) taken at least 6 hours apart with the patient on bed rest.

— Proteinuria (≥ 5 g/24 hours or ≥ 3+ on two random samples 4 hours apart).

— Manifestations of end-organ disease: oliguria (< 500 mL in 24 hours), cerebral or visual disturbances, pulmonary edema, cyanosis, epigastric or right-upper quadrant pain, impaired liver function, thrombocytopenia, or fetal growth restriction.

— Hematologic changes include:

Thrombocytopenia — platelets are dramatically reduced, probably consumed by endothelial injury. Counts can be as low as 20 to 50 × 10^9/L.

Hemoconcentration — doctors used to follow preeclampsia with serial hematocrits.

Microangiopathic hemolysis — eventually, red cells are sheared through the microcirculation.

Hepatic changes are usually limited to hepatocellular necrosis, demonstrated by elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels.

Occasionally there is subcapsular hemorrhage and even hepatic rupture, which has a 60% maternal mortality rate.

— Neurologic changes are common and include headache, blurred vision, scotoma (seeing spots or "snow"), hyperreflexia, and rarely, cortical blindness, and the generalized seizures of eclampsia.

— Renal changes. Glomerular endotheliosis is the pathognomonic lesion of preeclampsia: the glomeruli are enlarged, distorted, and filled with occlusions, with hypertrophy of the intracapillary cells. Laboratory testing shows a decreased glomerular filtration rate, decreased renal blood flow (the former more than the latter), and nonselective proteinuria (i.e. all proteins including albumin; what a urine dip stick detects).

— Fetal changes. Intrauterine growth restriction is very common. Oligohydramnios also occurs, because the amniotic fluid is essentially fetal urine; with poor perfusion through the placenta, the fetus has diminished urine output. Intrauterine demise and placental abruption are not uncommon. Doppler waveforms are typically abnormal, and antenatal testing suggests that the fetus is in jeopardy. We use the ratio of forward flow of blood in the umbilical artery during systole to that during diastole (the "umbilical artery S/D ratio") to assess the degree of resistance to flow in the placenta. The higher the ratio, the less diastolic flow. The greater the resistance to flow, the greater the peril to the fetus.

HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets) used to be classified as a separate syndrome, but current thinking categorizes it as a manifestation of preeclampsia, occurring in about 20% of severe cases. It is associated with significant maternal and perinatal morbidity. A decreasing platelet count and an increasing l-lactate dehydrogenase level (indicative of both hemolysis and liver dysfunction) reflect disease severity.

Management of isolated hypertension

— Rest and monitoring: BP, weight; look for oedema and proteinuria.

— Daily fetal movement.

— Measure the fundal height (risk of fetal growth retardation).

— Fetal growth monitoring every 3–4 weeks.

— Antenatal steroids should be considered for women at risk for preterm delivery who present between 24 and 34 weeks gestation.

— In the event of proteinuria developing, treat as preeclampsia

— Markedly elevated blood pressure measurements taken at least 6 hours apart with the patient on bed rest.

If SBP is ≥ 160 mmHg or DBP is ≥110 mmHg, give an antihypertensive treatment (Table 8).

Management of mild preeclampsia

Preeclampsia places both mother and fetus at risk. It is, however, a maternal disorder. The mainstay of treatment is early detection and managed delivery to minimize both maternal and fetal risks.

Delivery is the only curative treatment for preeclampsia. Management decisions must balance the maternal risks of continued pregnancy against the fetal risks associated with induced preterm delivery. The criteria for delivery are based on two often interrelated factors, i.e., gestational age at diagnosis (estimated fetal weight) and severity of preeclampsia.

Regardless of the severity of preeclampsia, there is no advantage in continuing the pregnancy when preeclampsia is discovered after 36–37 weeks. Nor is expectant management justified for severe preeclampsia before 24 weeks, in view of the high risk of maternal complications and the poor neonatal prognosis. At 34–37 weeks, management depends on the severity of the preeclampsia. Expectant management is possible for mild preeclampsia to limit the risk of induced preterm delivery, but for severe preeclampsia, delivery remains the rule due to the increased risk of maternal and fetal complications.

Management of severe preeclampsia

Severe preeclampsia requires treatment with a dual aim, i.e., preventing the harmful effects of ele-

Table 8

Commonly used antihypertensive agents

Agent	Dosage range	Caution/comment
Labetalol	Standard dose: 200–800 mg orally per day in 2–3 divided doses Maximum dosage: 2.400 mg per day	Should be avoided in women with cardiac conduction abnormalities, systolic heart failure or asthma
Nifedipine (extended-release)	Standard dose: 30–60 mg orally per day Maximum dosage: 2.400 mg per day	Ensuring the correct form of nifedipine is not recommended due to the risk of hypotension. There is concern for severe hypotension if nifedipine is continued with intravenous magnesium
Methyldopa	Standard dose: 250–1000 mg orally per day in 2–3 divided doses Maximum dosage: 3000 mg per day	Associated with hepatitis, hemolytic anemia, depression and sedation

vated maternal blood pressure and preventing eclampsia.

Delivery is imperative within 24 hours, either vaginally or by cesarean section, depending on the state of the cervix, gestational age and condition of the fetus.

Antihypertensive treatment is useful only in severe preeclampsia because the sole proven benefit of such management is to diminish the risk of maternal complications (cerebral hemorrhage, eclampsia, or acute pulmonary edema).

There is no international consensus concerning antihypertensive treatment in preeclampsia. The four drugs authorized for the treatment of hypertension in severe preeclampsia are nicardipine, labetalol, clonidine, and dihydralazine. There is no ideal target blood pressure value, and too aggressive a reduction in blood pressure is harmful to the fetus. Therapy with a single agent is advised as first-line treatment, followed by combination treatment when appropriate.

Pulmonary maturation using corticosteroids must be considered, taking gestational age into account. Betamethasone remains the gold standard at a dosage of two injections of 12 mg 24 hours apart; this treatment reduces the risk of hyaline membrane disease, intraventricular hemorrhage, and neonatal mortality.

Magnesium sulfate ($MgSO_4$) may be part of the therapeutic armamentarium for severe preeclampsia. It is indicated in the treatment of eclamptic convulsions as well as for secondary prevention of eclampsia (Fig. 10.11).

The efficacy of $MgSO_4$ in the reduction of maternal and neonatal complications of eclampsia is well established. It is administered intravenously, first at a loading dose of 4 g over 15–20 minutes, which can be repeated at a half dose (2 g) if convulsion recurs, and then at a maintenance dose of 1 g/hour for 24 hours. $MgSO_4$ treatment must be monitored in the intensive care unit because organ failure may occur. This monitoring is based on repeated checking for a Glasgow score of 15, tendon reflexes, respiratory frequency > 12 per minute, and diuresis > 30 mL/hour. Any manifestation of overdose requires stopping the infusion, considering injection of *calcium gluconate,* and measuring blood magnesium levels.

Eclampsia is generally considered an indication for emergency cesarean section. Nonetheless, a decision to delay a cesarean, albeit rare, may be based on fetal status and justified if the mother's condition is stable and reassuring after treatment.

Management following delivery

Although delivery is the only effective treatment for preeclampsia, and despite the fact that clinical symptoms and laboratory abnormalities usually regress in the hours afterwards, the risk of complications persists for some time following delivery. Preeclampsia is associated with long-term morbidity and mortality. Approximately 20% of women with preeclampsia develop hypertension or microalbuminuria during long-term follow-up, and the risk of subsequent cardiovascular and cerebrovascular disease is doubled in women with preeclampsia and gestational hypertension compared with age-matched controls. Preeclampsia is a marker of increased mortality from cardiovascular disease.

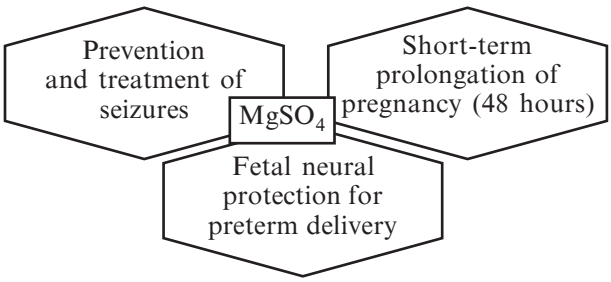


Fig. 10.11

Hemodynamic, neurological, and laboratory monitoring is necessary following delivery for patients with severe preeclampsia. Hemodynamic monitoring includes frequent blood pressure measurements to enable adjustment of antihypertensive treatment and frequent monitoring of diuresis and weight according to intake (oliguria should prompt progressive fluid resuscitation and sometimes diuretic use). Neurological monitoring consists of checking for signs of imminent eclampsia, including headaches, phosphene signals, tinnitus, and brisk tendon reflexes. Clinical monitoring must be done several times daily during the week after delivery, a period considered at high risk for complications. Monitoring must be performed in an intensive care unit.

Laboratory monitoring should be done several times daily in the first 72 hours after delivery and thereafter adapted according to progress of the indices. It must include a complete blood count, liver function tests, and measurement of lactate dehydrogenase. Discharge from hospital cannot be considered until all clinical and laboratory indices have returned to normal, and regular monitoring by the patient's general practitioner as necessary if treatment for hypertension is to be continued after discharge.

The risk of recurrence of preeclampsia during a subsequent pregnancy has to be considered. This risk is estimated to be less than 10% for all cases of preeclampsia, but is greater when preeclampsia is discovered before 28 weeks.

Three months after delivery, screening for underlying renal or hypertensive disease may be requested by the patient's primary physician. Such screening is intended to check for normalization of blood pressure values and disappearance of proteinuria, and if abnormalities persist, a referral should be made to a nephrologist or a hypertension expert to determine the cause. This examination is important because preeclampsia may unmask previously undiagnosed systemic or kidney disease or thrombophilia. It should include a specific set of questions, blood pressure measurement, a clinical examination looking for signs of autoimmune conditions, and a urinary dipstick test. Testing for antiphospholipid antibodies is recommended after severe preeclampsia.

Long-term monitoring of cardiovascular, renal, and metabolic risk factors is recommended after severe preeclampsia.

Prevention

Primary prevention of preeclampsia is based on the detection of modifiable risk factors. The literature is plentiful regarding the risk factors for preeclampsia, but should be interpreted with caution. Women at high risk are those with a personal history of severe preeclampsia, while those at low risk are defined as those who have never had preeclampsia but have at least one risk factor.

However, accurate prediction of preeclampsia would enable early and optimal management of women at high risk. Several predictive tests are being assessed currently. These include clinical tests, such as blood pressure measurement during the second trimester or 24-hour ambulatory blood pressure monitoring, but these lack sensitivity and specificity. Among the markers used to screen for trisomy 21 during the second trimester (beta human chorionic gonadotropin, alpha fetoprotein, and unconjugated estriol), elevated alpha fetoprotein is associated with a higher risk of preeclampsia (unless there are neural tube abnormalities, as when beta human chorionic gonadotropin is elevated). Frequent monitoring of women with elevated levels could be useful, but these tests may not be carried out for screening purposes due to their low negative predictive value.

Imaging tests have been evaluated, including uterine artery Doppler ultrasound. Uterine artery Doppler ultrasound is not advised during the first or second trimester in low-risk populations due to the excessive variability of likelihood ratios in this population, which allows for the prediction of only one-third of preeclampsia cases. In a high-risk population, the definition of which is often imprecise, uterine artery Doppler can be performed during the second trimester morphologic ultrasound examination and checked 1 month later in case of abnormal results (resistance index > 0.58 or 90–95th percentile, unilateral or bilateral notch). The combination of a uterine artery Doppler examination during the first trimester and a three-dimensional ultrasound assessing placental volume may predict the risk of preeclampsia as early as the first trimester.

In clinical practice, because no single marker effectively predicts the risk of preeclampsia, the current trend is to test a combination of markers. The most commonly used combination of markers assesses placental growth factor, endoglin, and vascular endothelial growth factor during the first or second trimester.

Secondary prevention is based on antiplatelet aspirin therapy, which reduces the risk of preeclampsia by 10% in women who have at least one risk factor. No study currently allows determination of the exact dosage or the best time for initiation of aspirin (75–80 mg/day). However, aspirin should be initiated as early as possible, i.e., before 12–14 weeks, which corresponds to the beginning of the first phase of trophoblast invasion. The efficacy of aspirin has been shown only in women with previous preeclampsia associated with intrauterine growth retardation and without thrombophilia. Low molecular weight heparin is indicated only in cases of complicated thrombophilia (history of thromboembolic complications or of preeclampsia). Calcium supplementation at a dosage of 1.5 g/day, beginning at 15 weeks and continued throughout the pregnancy, is recommended for prevention of pre-eclampsia in women with a daily calcium intake < 600 mg/day.

Chapter 11
BLEEDING DURING THE SECOND HALF OF PREGNANCY

Antepartum Hemorrhage (APH) or Bleeding in Late Pregnancy — is defined as bleeding from the genital tract after the 22 week of gestation till end of pregnancy. It occurs in approximately 2–5% of pregnancies. It is one of the leading causes of antepartum hospitalization, maternal morbidity and operative intervention (Fig. 11.1) [3, 40, 42, 44, 45, 50].

PLACENTA PREVIA (PP)

Abnormal location of the placenta over, or in close proximity to, the internal cervical os.

In general, the incidence of PP is 1 in 250 pregnancies. The frequency varies with parity, however. For nulliparas, the incidence is only 1 in 1000 to 1500, whereas in grand multiparas, it may be as high as 1 in 20. The most important risk factor for PP is a prior cesarean section.

The placenta may be located in the lower part of the uterus either covering or adjacent to the cervical outlet for a number of reasons. The placenta normally migrates away from the cervical opening as the pregnancy progresses, so women in the earlier stages of pregnancy are more likely to have PP than are women at term. Although up to 6% of women between 10 and 20 weeks' gestation will have some evidence of placenta previa on ultrasound examination, 90% of these cases resolve on their own as the pregnancy progresses.

Etiology

— Dropping down theory: The fertilized ovum drops down and is implanted in the lower segment. Poor decidual reaction in the upper uterine segment may be the cause. Failure of zona pellucida to disappear in time can be a hypothetical possibility. This explains the formation of central placenta previa.

— Persistence of chorionic activity in the decidua capsularis and its subsequent development into capsular placenta which comes in contact with decidua vera of the lower segment can explain the formation of lesser degrees of placenta previa.

— Defective decidua results in spreading of the chorionic villi over a wide area in the uterine wall to get nourishment. During this process, not only the placenta becomes membranous but encroaches onto the lower segment. Such a placenta previa may invade the underlying decidua or myometrium to cause placenta accreta, increta or percreta.

— Big surface area of the placenta as in twins may encroach onto the lower segment.

Pathological anatomy

Placenta. The placenta may be large and thin. There is often a tongue shaped extension from the main placental mass. Extensive areas of degeneration with infarction and calcification may be evident. The placenta may be morbidly adherent due to poor decidua formation in the lower segment.

Umbilical cord. The cord may be attached to the margin (battledore) or into the membranes (velamentous). The insertion of the cord may be close to the internal os or the fetal vessels may run across the internal os in velamentous insertion giving rise to vasa previa, which may rupture along with rupture of the membranes.

Lower uterine segment. Due to increased vascularity, the lower uterine segment and the cervix becomes soft and more friable.

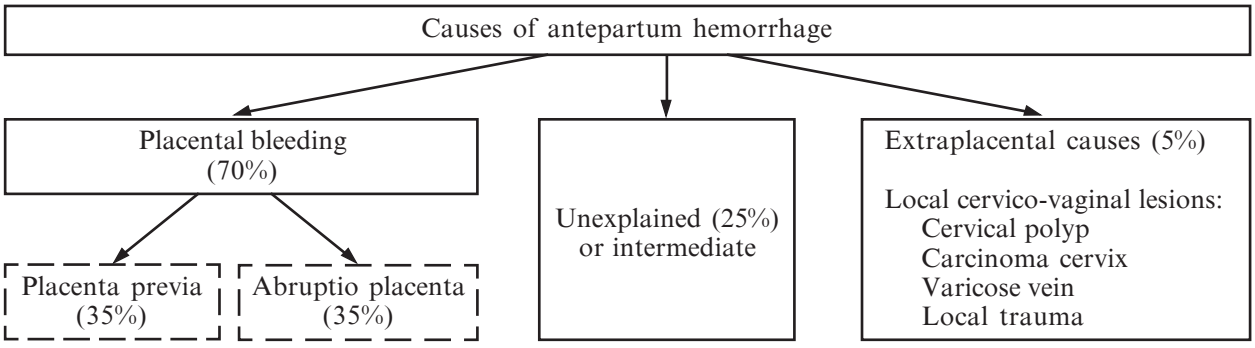


Fig. 11.1

Fig. 11.2. Degrees of placenta previa with findings on ultrasound examination

Types or degrees of placenta previa

There are four types of PP depending upon the degree of extension of placenta to the lower segment (Fig. 11.2).

— First degree (Type I = P.P. lateralis = low-lying placenta):

The lower edge of the placenta reaches the lower uterine segment but not the internal.

— Second degree (Type II = P.P. marginalis):

The lower edge of the placenta reaches the margin of the internal os but does not cover it.

— Third degree (Type III = P.P. incomplete or partial central):

The placenta covers the internal os when it is closed or partially dilated but not when it is fully dilated (Fig. 11.3).

— Fourth degree (Type IV = P.P. complete centralis or total):

The placenta covers the internal os completely whether the cervix is partially or fully dilated.

Terminology for placenta previa has been confusing. In a recent Fetal Imaging Workshop sponsored by the National Institutes of Health (2013), the following classification was recommended:

— ***Placenta previa*** — the internal os is covered partially or completely by placenta. In the past, these were further classified as either total or partial previa.

— ***Low-lying placenta*** — implantation in the lower uterine segment is such that the placental edge does not reach the internal os and remains outside a 2 cm wide perimeter around the os. A previously used term, marginal previa, described a placenta that was at the edge of the internal os but did not overlie it.

Clearly, the classification of some cases of previa will depend on cervical dilatation at the time of assessment. For example, a low-lying placenta previa at 2 cm dilatation may become a partial placenta previa at 4 cm dilatation because the cervix has dilated to expose the placental edge. Conversely, a placenta previa that appears to be total before cervical dilatation may become partial at 4 cm dilatation because the cervical opening now extends beyond the edge of the placenta. Digital palpation in an attempt to ascertain these changing relations between the placental edge and internal os as the cervix dilates usually causes severe hemorrhage.

With both total and partial placenta previa, a certain degree of spontaneous placental separation is an inevitable consequence of lower uterine segment remodelling and cervical dilatation. Although this frequently causes bleeding, and thus technically constitutes a placental abruption, this term is usually not applied in these instances. Somewhat but not always related is *vasa previa* (Fig. 11.4).

Mechanism of bleeding PP

Progressive stretching of the lower uterine segment normally occurs during the 3rd trimester and labor, but the inelastic placenta cannot stretch with it. This leads to inevitable separation of a part of the placenta with unavoidable bleeding. The closer to term, the greater the amount of bleeding. The separation of the placenta may be provoked by trauma including vaginal examination, coital act, external version or during high rupture of membranes.

Risk Factors

— History of previous cesarean section or any other scar in the uterus (myomectomy or hysterotomy), which changes the shape of the lower uterine segment.

Risks increase 1.5- to 5-fold with a history of cesarean delivery. Rate of placenta previa increases with a rate of 1% after 1 cesarean delivery, 2.8% after 3 cesarean deliveries, and as high as 3.7% after 5 cesarean deliveries.

— Multiparty
— Increased maternal age age (> 35 years)
— Previous placenta previa
— Multiple gestation
— Prior curettage.
— Uterine anomalies
— Maternal smoking (causes placental hypertrophy to compensate the carbon monoxide induced hypoxemia).

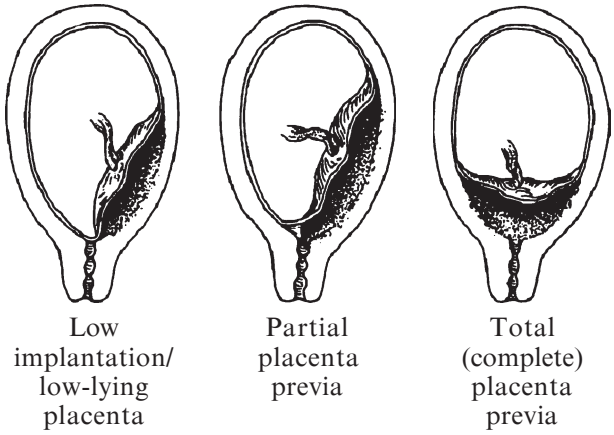


Fig. 11.3

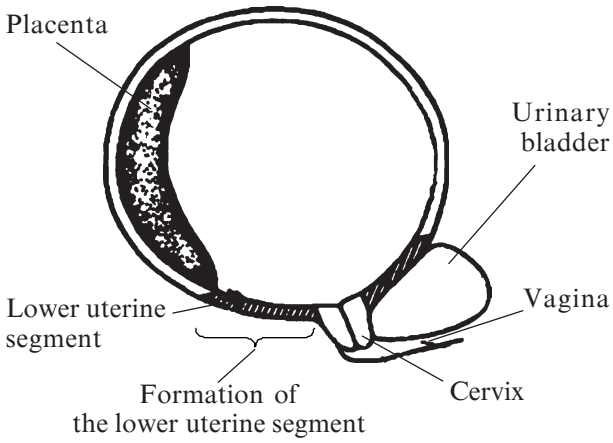


Fig. 11.4. Avulsion of villi, stretching of low uterine segment

Clinical manifestations

Painless bleeding is the most characteristic event with placenta previa. Bleeding usually does not appear until near the end of the second trimester or later, but it can begin even before mid-pregnancy. And undoubtedly, some late abortions are caused by an abnormally located placenta. Bleeding from a previa usually begins without warning and without pain or contractions in a woman who has had an uneventful prenatal course. This so-called *sentinel bleed* is rarely so profuse as to prove fatal. Usually it ceases, only to recur. In perhaps 10 percent of women, particularly those with a placenta implanted near but not over the cervical os, there is no bleeding until labor onset. Bleeding at this time varies from slight to profuse, and it may clinically mimic placental abruption.

Abnormally implanted placenta

Placenta previa is associated with a doubling of the rate of congenital malformations. These include malformations of the CNS, gastrointestinal tract, cardiovascular system, and respiratory system.

Placenta accreta syndromes arise from abnormal placental implantation and adherence and are classified according to the depth of placental ingrowth into the uterine wall. These include placenta accreta, increta, and percreta.

1. Placenta previa accreta. The placenta adheres to the uterine wall without the usual intervening decidua basalis. The incidence in patients with previa who have not had prior uterine surgery is approximately 4%. The risk is increased to 16–25% in patients who have had a prior cesarean section or uterine surgery.

2. Placenta previa increta. The placenta invades the myometrium.

3. Placenta previa percreta. The placenta penetrates the entire uterine wall, potentially growing into bladder or bowel.

Coagulation Defects

Placenta previa is rarely complicated by coagulopathy even when there is extensive implantation site separation. Placental thromboplastin, which incites the intravascular coagulation seen with placental abruption, is presumed to readily escape through the cervical canal rather than be forced into the maternal circulation. The paucity of large myometrial veins in this area may also be protective.

Diagnosis

Whenever there is uterine bleeding after mid pregnancy, placenta previa or abruption should always be considered. Whenever there is uterine bleeding after mid pregnancy, placenta previa or abruption should always be considered. Previa should not be excluded until sonographic evaluation has clearly proved its absence. Diagnosis by clinical examination is done using the double set-up technique because it requires that a finger be passed through the cervix and the placenta palpated. A digital examination should not be performed unless delivery is planned. A cervical digital examination is done with the woman in an operating room and with preparations for immediate cesarean delivery. Even the gentlest examination can cause torrential hemorrhage. Fortunately, double set-up examination is rarely necessary because placental location can almost always be ascertained sonographically.

Management of placenta previa

Women with a previa are managed depending on their individual clinical circumstances. The three factors that usually are considered include fetal age and thus maturity; labor; and bleeding and its severity.

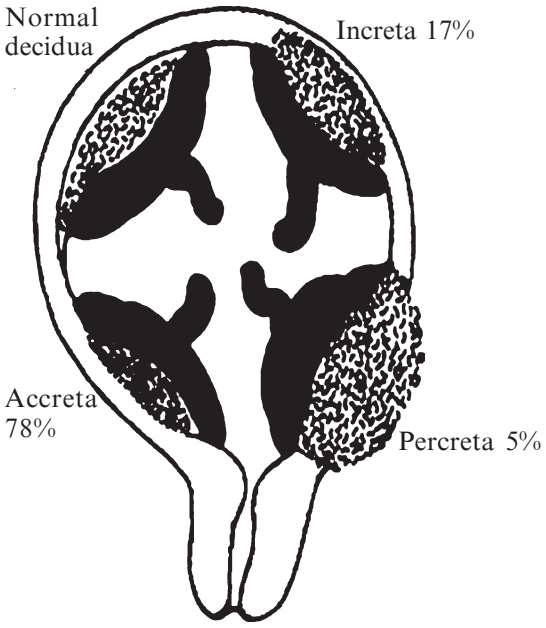
If the fetus is preterm and there is no persistent active bleeding, management favors close observation in an obstetrical unit. For women who are near term and who are not bleeding, plans are made for scheduled cesarean delivery. Timing is important to maximize fetal growth but to minimize the possibility of antepartum hemorrhage. women with a previa are best served by elective delivery at 36 to 37 completed weeks. With suspected placenta accrete syndromes — at 34 to 35 completed weeks.

Delivery

Practically all women with placenta previa undergo cesarean delivery. Cesarean delivery is emergently performed in more than half because of hemorrhage, for which about a fourth require blood transfusion. Although a low transverse hysterotomy is usually possible, this may cause fetal bleeding if there is an anterior placenta and the placenta is cut through. In such cases, fetal delivery should be expeditious. Thus, a vertical uterine incision may be preferable in some instances. That said, even when the incision extends through the placenta, maternal or fetal outcomes are rarely compromised. Following placental removal, there may be uncontrollable hemorrhage because of poorly contracted smooth muscle of the lower uterine segment. When hemostasis at the placental implantation site cannot be obtained by pressure, the implantation site can be oversewn with 0-chromic sutures. Other methods include bilateral uterine or internal iliac artery ligation. If these more conservative methods fail and bleeding is brisk, then hysterectomy is necessary.

PLACENTA ACCRETA SYNDROMES

These syndromes describe the abnormally implanted, invasive, or adhered placenta. Derivation of accrete comes from the Latin e ac- + crescere — to grow from adhesion or coalescence, to adhere, or to become attached to. Accrete syndromes thus include any placental implantation with abnormally firm adherence to myometrium because of partial or total absence of the decidua basalis and imperfect development of the fibrinoid layer. If the decidual spongy layer is lacking either partially or totally, then the physiological line of cleavage is absent, and some or all cotyledons are densely anchored. The surface area of the implantation site involved and the depth of trophoblastic tissue ingrowth are variable between women, but all affected placentas can potentially cause significant hemorrhage (Fig. 11.5) [3, 18, 40, 42, 44, 45].



Placenta accreta/increta/percreta

Fig. 11.5

Variants of placenta accrete syndrome are classified by the depth of trophoblastic growth. Placenta accreta indicates that villi are attached to the myometrium. With ***placenta increta,*** villi actually invade the myometrium, and ***placenta percreta*** defines villi that penetrate through the myometrium and to or through the serosa. In clinical practice, these three variants are encountered in an approximate ratio of 80 : 15 : 5, respectively. In all three varieties, abnormal adherence may involve all lobules — ***total placenta accreta***. If all or part of a single lobule is abnormally attached, it is described as a ***focal placenta accreta*** (Fig. 11.6, 11.7).

Risk Factors

These are similar in many aspects to those for placenta previa. That said, the two most important risk factors are an associated previa, a prior cesarean delivery, and more likely a combination of the two. In one study, an accrete placenta more likely followed an emergency compared with elective cesarean delivery.

Clinical presentation and diagnosis

In cases of first- and second-trimester accrete syndromes, there is usually hemorrhage that is the consequence of coexisting placenta previa. Such bleeding will usually prompt evaluation and management. In some women who do not have an associated previa, accreta may not be identified until third-stage labor when an adhered placenta is encountered. Ideally, abnormal placental ingrowth is identified antepartum, usually by sonography.

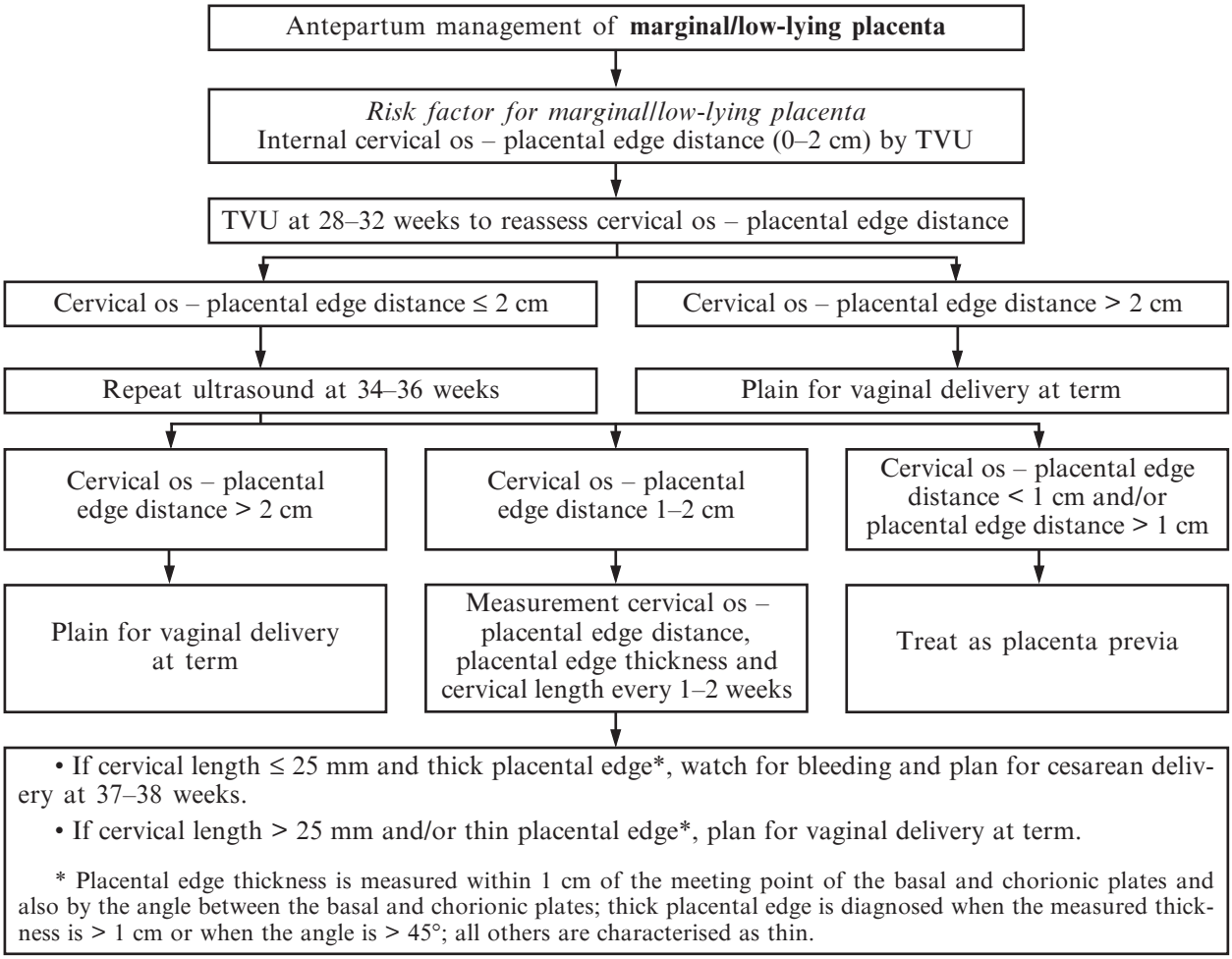


Fig. 11.6

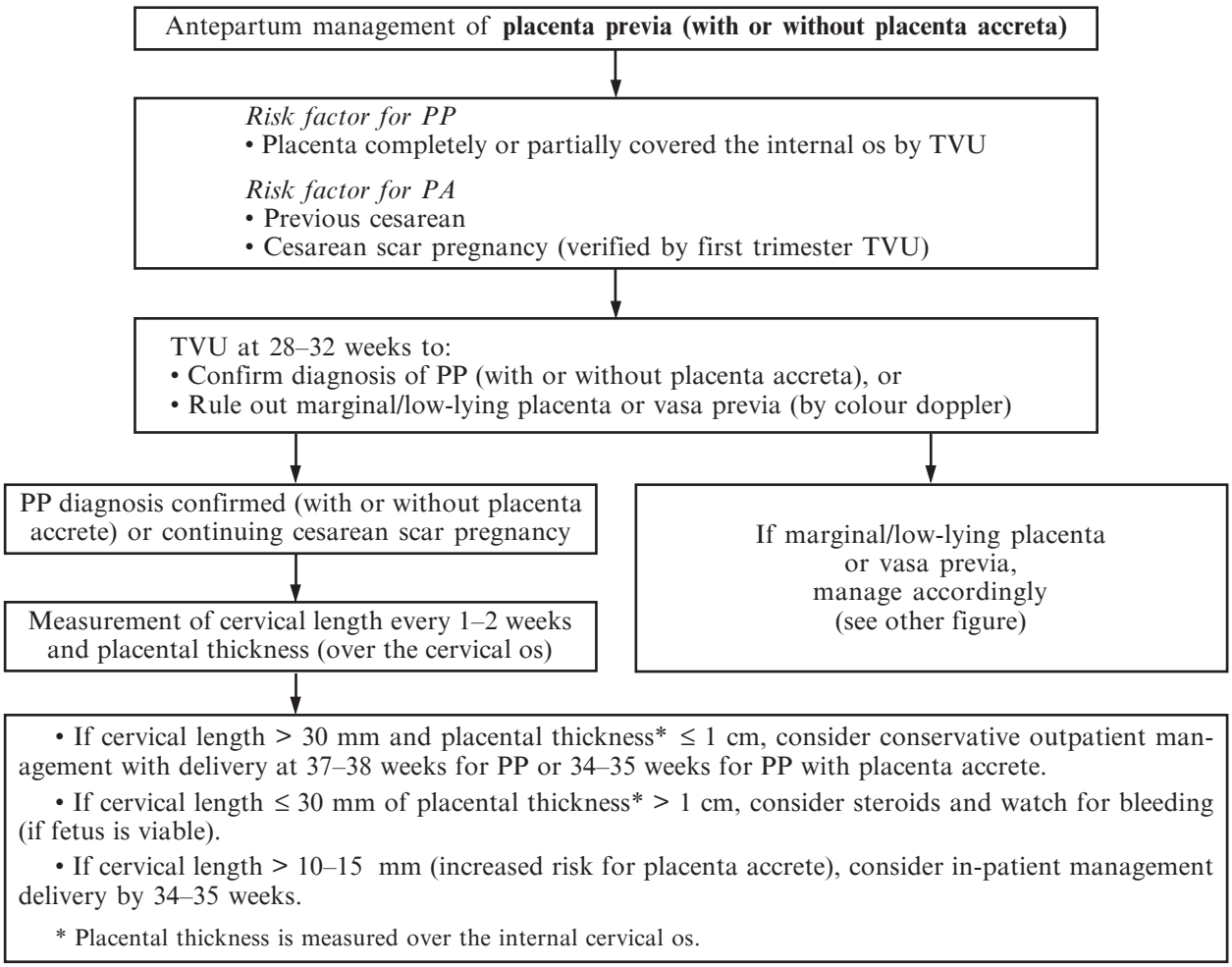


Fig. 11.7

Management

Preoperative assessment should begin at the time of recognition during prenatal care. A major decision concerns the ideal institution for delivery. Exigencies to be considered are appropriate surgical, anesthesia, and blood banking capabilities. Confirmation of a percreta or increta almost always mandates hysterectomy.

VASA PREVIA

This is a particularly dangerous variation of velamentous insertion in which the vessels within the membranes overlie the cervical os. The vessels can be interposed between the cervix and the presenting fetal part. Hence, they are vulnerable to compression and also to laceration or avulsion with rapid fetal exsanguination. Vasa previa (VP) is uncommon and identified in 1 in 5200 pregnancies (Fig. 11.8) [2, 3, 30, 40, 42, 44].

The *risk factors* for VP include:

• Twin and multiple pregnancies

• Velamentous, marginal, or furcate insertion of umbilical cord

• The presence of accessory placental lobe (succenturiate placenta)

• *In vitro* fertilization (IVF)

Types of VP

• ***type I*** (present in 90% of cases with vasa previa) abnormal fetal vessels connect a *velamentous cord insertion* with the main body of the placenta.

• ***type II***

— abnormal vessels connect portions of *bilobed placenta;*

— placenta with a *succenturiate lobe*: due to this association, vasa previa needs to be excluded in patients with *variant placental morphology*.

The signs and symptoms of VP may include:

— Painless vaginal bleeding

— Membrane rupture (also known as 'water breaking')

— Decreased fetal heart rate (fetal bradycardia)

A combination of the above signs and symptoms form the classic triad of VP.

Complications due to VP may include:

— Decreased blood flow to the developing fetus, which can result in intrauterine growth retardation (IUGR) and other congenital abnormalities in the developing fetus.

— Spontaneous abortions or miscarriages.

— Stillbirths due to rupture of blood vessels during early labor.

— Excessive hemorrhage/bleeding during childbirth can result in fetal mortality,

— Compression of the blood vessels can cause fetal distress.

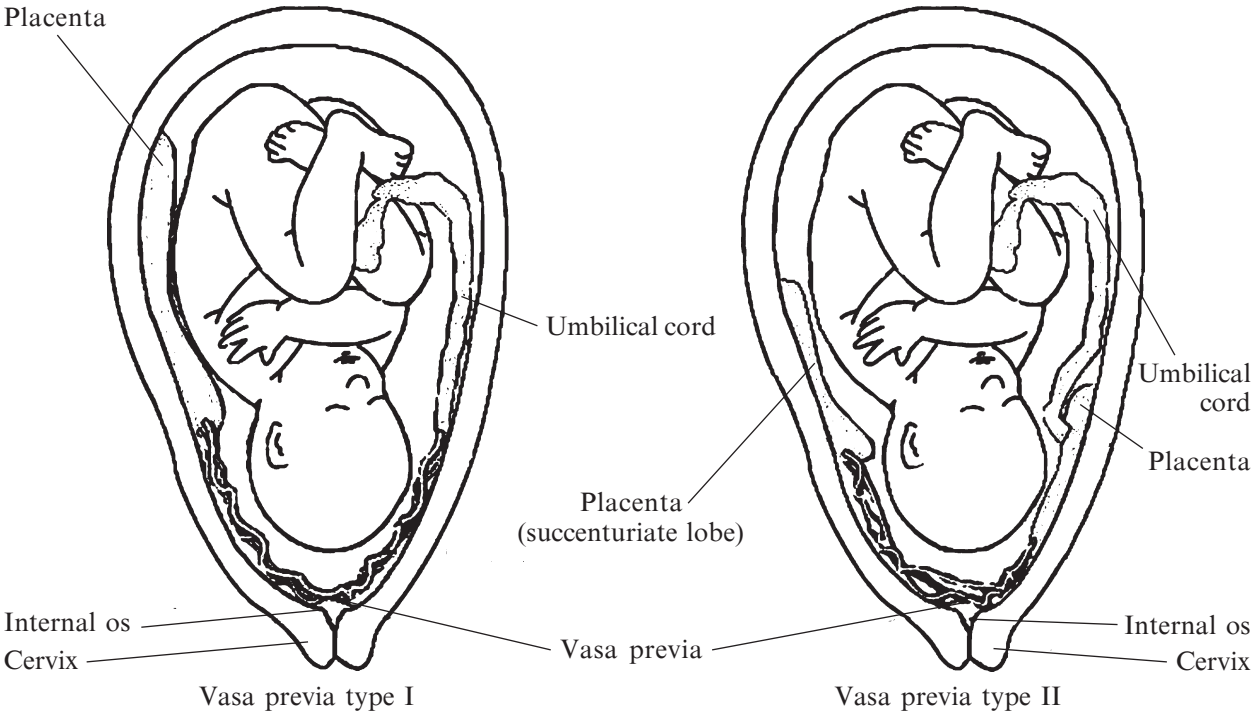


Fig. 11.8

Management

With transvaginal sonography, cord vessels may be seen inserting into the membranes — rather than directly into the placenta — with vessels running above the cervical internal os. Once vasa previa is identified, early scheduled cesarean delivery is planned.

PLACENTAL ABRUPTION (PA)

Separation of the placenta — either partially or totally — from its implantation site before delivery is described by the Latin term abruptio placentae. Literally translated, this refers to "rending asunder of the placenta," which denotes a sudden accident that is a clinical characteristic of most cases. In the purest sense, the cumbersome — and thus seldom used — term premature separation of the normally implanted placenta is most descriptive, because it excludes separation of a placenta previa implanted over the internal cervical os [42, 50].

Frequency

The reported incidence of placental abruption varies because of different criteria used. That said, its frequency averages 0.5 percent or 1 in 200 deliveries.

Etiopathogenesis

Placental abruption is initiated by hemorrhage into the decidua basalis. The decidua then splits, leaving a thin layer adhered to the myometrium. Consequently, the process begins as a decidual hematoma and expands to cause separation and compression of the adjacent placenta. The phenomenon of impaired trophoblastic invasion with subsequent atherosis is related in some cases of preeclampsia and abruption. Inflammation or infection may be contributory. Inflammation to be more common in prematurely separated placentas (Fig. 11.9).

Abruption likely begins with rupture of a decidual spiral artery to cause a retroplacental hematoma. This can expand to disrupt more vessels and extend placental separation. In the early stages of placental abruption, there may be no clinical symptoms. If there is no further separation, the abruption is discovered on examination of the freshly delivered placenta, as a circumscribed depression on the maternal surface. These usually measure a few centimeters in diameter and are covered by dark, clotted blood (Fig. 11.10).

Types

Based on blood lost from abruption

Even with continued bleeding and placental separation, placental abruption can still be either ***total*** or ***partial***. With either, bleeding typically insinuates itself between the membranes and uterus, ultimately escaping through the cervix to cause ***external hemorrhage***. Less often, the blood is retained between the detached placenta and the uterus, leading to ***concealed hemorrhage*** and e delayed diagnosis. The delay translates into much greater maternal and fetal hazards. With concealed hemorrhage, the likelihood of consumptive coagulopathy also is greater. This is because increased pressure within the intervillous space caused by the expanding retroplacental clot forces more placental thromboplastin into the maternal circulation.

Clinical classification (based on degree of abruption)

1. Mild separation
2. Moderate separation
3. Severe separation

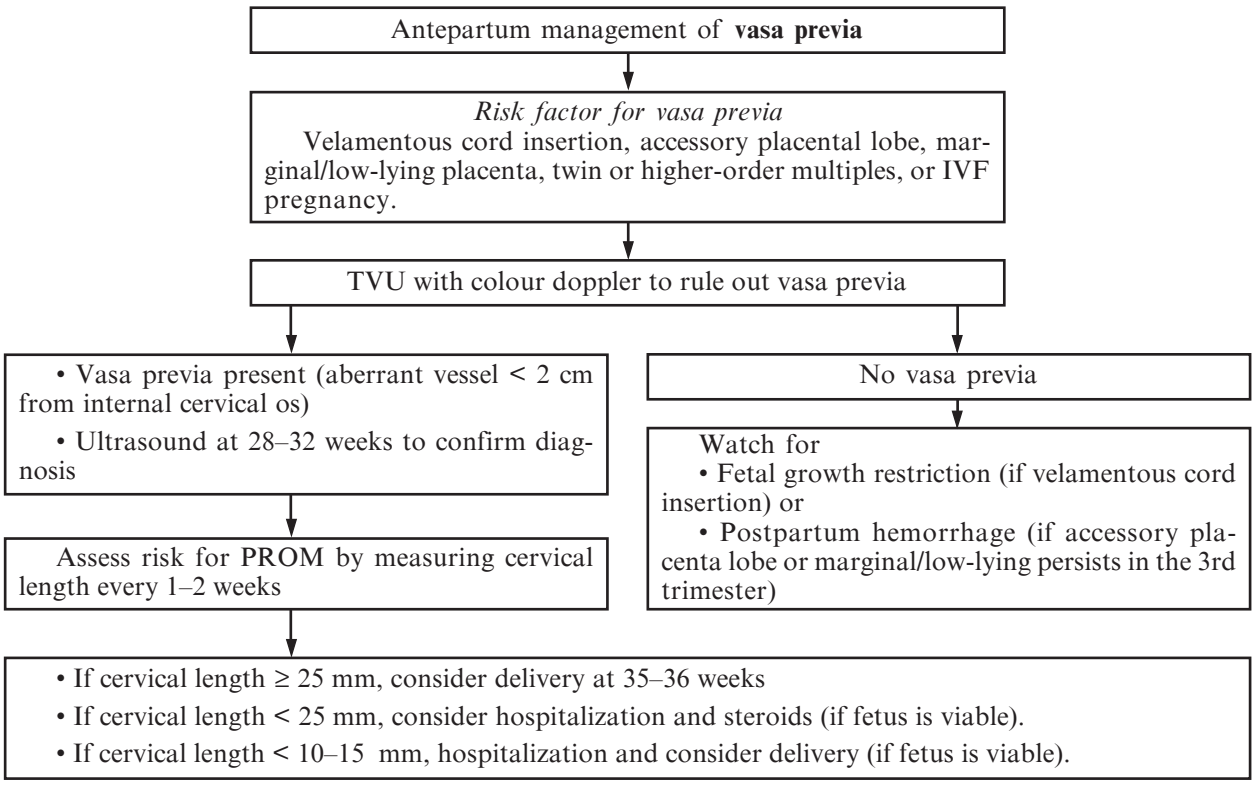


Fig. 11.9

Perinatal morbidity and mortality

Major fetal congenital anomalies have an increased association with placental abruption. Overall, perinatal outcomes are influenced by gestational age, and the frequency of placental abruption increases across the third trimester up to term.

Risk factors associated with developing of PA

— Maternal age: pregnant women who are younger than 20 or older than 35 are at greater risk
— Recurrent abruption
— Gestational hypertensive disease and preeclampsia.
— Preterm prematurely ruptured membranes
— Cigarette smoking
— Cocaine abuse
— Lupus anticoagulant and thrombophilias
— Multiple gestation
— Multiparity
— Prolonged PROM (24 hours or longer)
— Short umbilical cord
— Maternal trauma, such as motor vehicle accidents, assaults, falls.

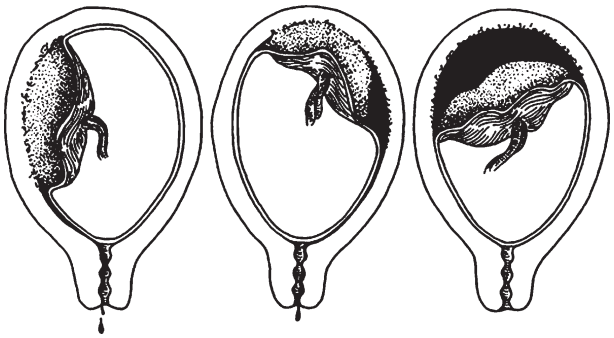


Fig. 11.10

Clinical findings and diagnosis

Most women with a placental abruption have sudden-onset abdominal pain, vaginal bleeding, and uterine tenderness. Importantly, the signs and symptoms of placental abruption can vary considerably. In some women, external bleeding can be profuse, yet placental separation may not be so extensive as to compromise the fetus. In others, there may be no external bleeding, but the placenta is sufficiently sheared off that the fetus is dead — a concealed abruption (Fig. 11.11).

Differential diagnosis

With severe placental abruption, the diagnosis generally is obvious. From the previous discussion, it follows that less severe, more common forms of abruption cannot always be recognized with certainty. Thus, the diagnosis is one of exclusion. Unfortunately, there are no laboratory tests or other diagnostic methods to accurately confirm lesser degrees of placental separation. Sonography has limited use because the placenta and fresh clots may have similar imaging characteristics.

In the woman with vaginal bleeding and a live fetus, it is often necessary to exclude placenta previa and other causes of bleeding by clinical and sonographic evaluation. It has long been taught — perhaps with some justification — that painful uterine bleeding signifies placental abruption, whereas pain-

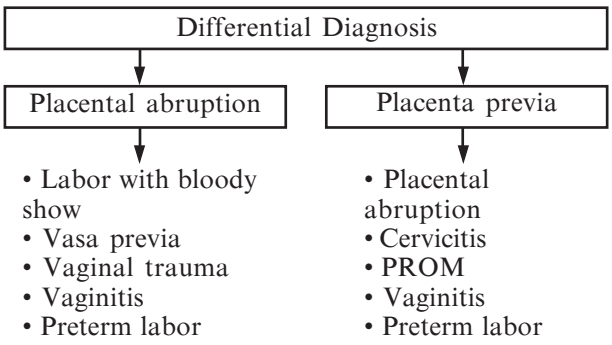


Fig. 11.11

less uterine bleeding is indicative of placenta previa.

The differential diagnosis is usually not this straightforward, and labor accompanying previa may cause pain suggestive of placental abruption. On the other hand, pain from abruption may mimic normal labor, or it may be painless, especially with a posterior placenta. At times, the cause of the vaginal bleeding remains obscure even after delivery (Table 9).

Complications

— Hypovolemic Shock.

Placental abruption is one of several notable obstetrical entities that may be complicated by massive and sometimes torrential hemorrhage. Hypovolemic shock is caused by maternal blood loss. Blood loss in these women often amounted to at least half of their pregnant blood volume. Importantly, massive blood loss and shock can develop with a concealed abruption. Prompt treatment of hypotension with crystalloid and blood infusion will restore vital signs to normal and reverse oliguria from inadequate renal perfusion.

— Consumptive Coagulopathy.

Obstetrical events — mainly placental abruption and amniotic-fluid embolism — led to the initial recognition of defibrination syndrome, which is currently referred to as consumptive coagulopathy or y disseminated intravascular coagulation. The major mechanism causing procoagulant consumption is intravascular activation of clotting.

Abruption is the most common cause of clinically significant consumptive coagulopathy in obstetrics — and indeed, probably in all of medicine. There are significant amounts of procoagulants in the retroplacental clots, but these cannot account for all missing fibrinogen. An important consequence of intravascular coagulation is the activation of plasminogen to plasmin, which lyses fibrin microemboli to maintain microcirculatory patency. With placental abruption severe enough to kill the fetus, there are always pathological levels of fibrinogen–fibrin degradation products and d-dimers in maternal serum. Most women with placental abruption will have some degree of intravascular coagulation.

— Couvelaire Uterus.

"Couvelaire uterus" is a rare complication of severe forms of placental abruption. It occurs when vascular damage within the placenta causes hemorrhage that progresses to and infiltrates the wall of the uterus, into connective tissue of the broad ligaments and even into the peritoneal cavity. It is a syndrome that can only be diagnosed by direct imaging or biopsy (or both). It is named after Couvelaire, who in the early 1900s termed it "Uteroplacental apoplexy".

— Acute Kidney Injury.

In obstetrics, it is most commonly seen in cases of severe placental abruption in which treatment of hypovolemia is delayed or incomplete.

— Sheehan Syndrome.

This is an especial problem in obstetrics if there is profound hypotension that remains uncorrected. During pregnancy the pituitary gland increases in size predisposing it to circulatory problems if there is blood loss. It has end arterial blood supply which means no collateral supply, and hypotension may result in an avascular pituitary gland. If this is not corrected quickly enough the pituitary gland will undergo avascular necrosis (Sheehan's syndrome). The consequences of this depend on which area of the pituitary gland is inactivated. If the anterior lobe is lost then no follicle-stimulating hormone, luteinizing hormone, thyroid-stimulating hormone, growth hormone, prolactin or adrenocorticotropic hormone will

Table 9

Differential characteristics between placenta previa and abruptio placentae

Characteristics	Placenta previa	Abruptio placenta
Magnitude of blood loss	Variable	Variable
Duration	Often ceases within 1–2 hours	Usually continues
Abdominal discomfort	None	Can be severe, pain
Fetal heart rate pattern on electronic monitoring	Absent	Tachycardia, then bradycardia; loss of variability; decelerations frequently present; intrauterine demise not rare
Coagulation defects	Rare	Associated, but infrequent; DIG often severe when when present cocaine use
Associated history	None	Abdominal trauma; maternal hypertension; multiple gestation; polyhydramnios

be produced resulting in secondary amenorrhoea, atrophy of breasts and genital organs, osteoporosis, hypothyroidism and Addisonian symptoms. The importance of adequate and urgent blood and fluid replacement in postpartum hemorrhage is thus obvious.

Management

Treatment of the woman with a placental abruption varies depending primarily on her clinical condition, the gestational age, and the amount of associated hemorrhage. With a living viable-size fetus and with vaginal delivery not imminent, emergency cesarean delivery is chosen by most. In some women, fetal compromise will be evident. When evaluating fetal status, sonographic confirmation of fetal heart activity may be necessary because sometimes an electrode applied directly to a dead fetus will provide misleading information by recording the maternal heart rate. If the fetus has died or if it is not considered mature enough to live outside the uterus, then vaginal delivery is preferable.

In either case, prompt and intensive resuscitation with blood plus crystalloid is begun to replace blood lost from retroplacental and external hemorrhage. These measures are lifesaving for the mother and hopefully for her fetus. If the diagnosis of abruption is uncertain and the fetus is alive and without evidence of compromise, then close observation may be warranted provided that immediate intervention is available.

Chapter 12
NORMAL LABOR

MATERNAL CHANGES BEFORE THE ONSET OF LABOR

As patients approach term, they experience uterine contractions of increasing strength and frequency. Spontaneous uterine contractions, which are not felt by the patient, occur throughout pregnancy. Late in pregnancy they become stronger and more frequent, resulting in the patient's perception of discomfort. These *Braxton Hicks contractions* (false labor) are not associated with dilation of the cervix, however, and do not fit the definition of labor. It is frequently difficult for the patient to distinguish these often uncomfortable contractions from those of true labor.

Braxton Hicks contractions are typically shorter in duration and less intense than true labor contractions, with the discomfort being characterized as over the lower abdomen and groin areas.

Parturition (true labor) is a continuous process in which progressive regular uterine contractions result in the expulsion of the products of conception from the uterus through the birth canal after progressive effacement and dilatation of the cervix.

These contractions become increasingly intense and frequent. Another event of late pregnancy is termed "*lightening*", in which the patient reports a change in the shape of her abdomen and the sensation that the baby is lighter, the result of the fetal head descending into the pelvis. The patient often notices that her lower abdomen is more prominent, and she may feel a need to urinate more frequently as the bladder is compressed by the fetal head. The patient may also notice that she is breathing more easily, because there is less pressure on the diaphragm as the uterus becomes smaller.

The patients often report the passage of blood-tinged mucus late in pregnancy. This "*bloody show*" results as the cervix begins thinning (effacement) with the concomitant extrusion of mucus from the endocervical glands and a small amount of bleeding from small vessels in the area. Cervical effacement is common before the onset of true labor, when the internal os is slowly drawn into the lower uterine segment. The cervix is often significantly effaced before the onset of labor, particularly in the nulliparous patient [7, 32, 34, 49] (Fig. 12.1).

Progressive development of the segments of the uterus at term:

In true labor, the woman is usually aware of her contractions during the first stage. The intensity of pain depends on the fetal/pelvic relationships, the quality and strength of uterine contractions, and the emotional and physical status of the patient. Few women experience no discomfort during the first stage of labor. Some women describe slight low back pain that radiates around to the lower abdomen. Each contraction starts with a gradual build-up of intensity, and dissipation of discomfort promptly follows the climax. Normally, the contraction will be at its height well before discomfort is reported. Dilatation of the lower birth canal and distention of the perineum during the second stage of labor will almost always cause discomfort.

The initial examination of the patient's abdomen may be accomplished using Leopold maneuvers, a series of four palpations of the fetus through the ab-

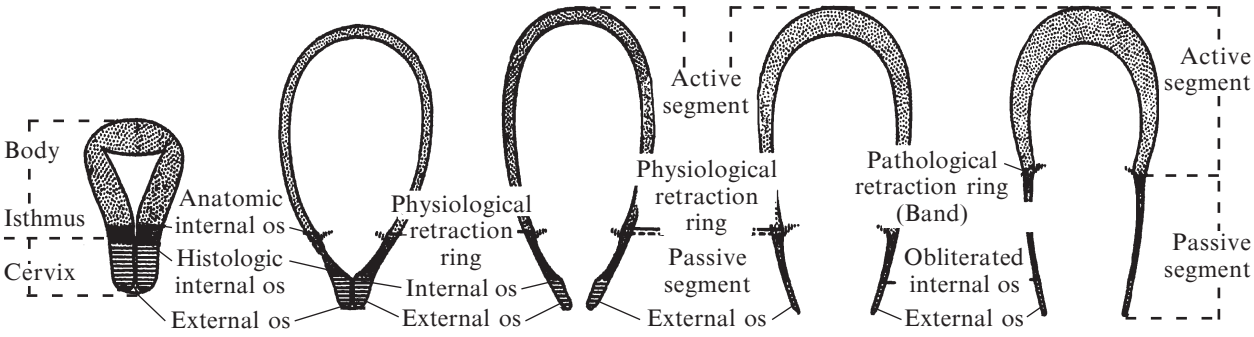


Fig. 12.1

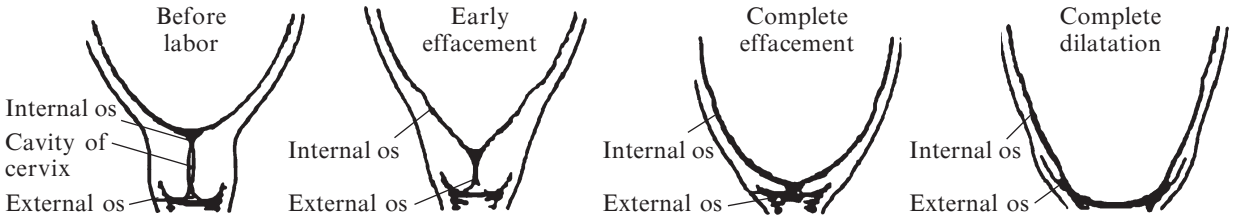


Fig. 12.2

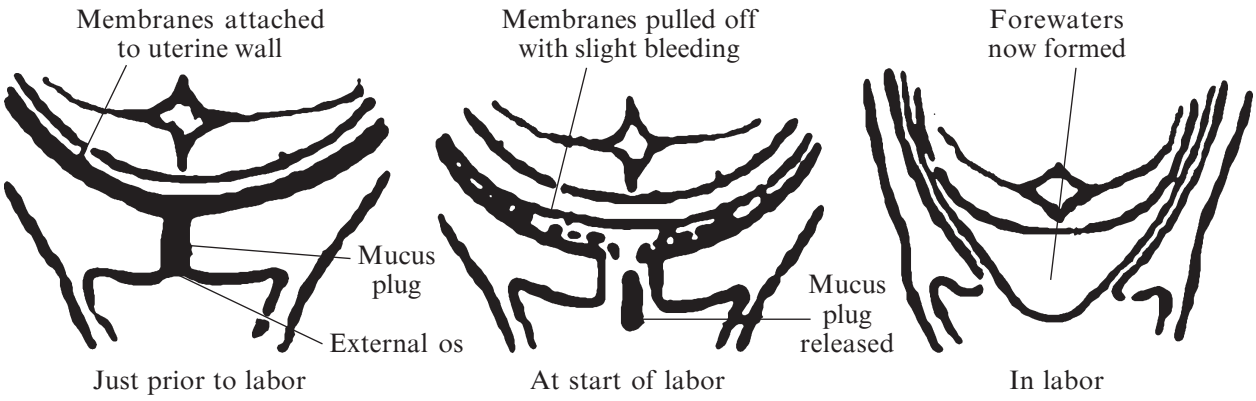


Fig. 12.3

dominal wall that helps accurately determine fetal lie, presentation, and position.

Palpation of the uterus during a contraction may also be helpful in determining the intensity of that particular contraction. The uterine wall is not easily indented with firm palpation during a true contraction, but may be indented during a Braxton Hicks "contraction."

A digital vaginal examination allows the examiner to determine the consistency and degree of effacement and degree of dilation of the cervix.

Effacement is the shortening of the cervical canal from a length of about 2 cm to a mere circular orifice with almost paper-thin edges. Effacement is expressed as a percent of thinning from a perceived uneffaced state (Fig. 12.2).

A cervix that is not effaced, but is softened, is more likely to change with contractions than one that is firm, as it is earlier in pregnancy. If the cervix is not significantly effaced, it may also be evaluated for its relative position, that is, anterior, midposition, or posterior in the vagina. A cervix that is palpable anterior in the vagina is more likely to undergo change in labor sooner than one found in the posterior portion of the vagina. This suggests that the presenting part has descended into the pelvis, creating more pressure on the cervix, thereby rotating it anteriorly. With more effective force on the lower uterine segment, contractions would cause a greater change in dilation and effacement of the cervix (Fig. 12.3).

The effect of the progressive retraction of the upper segment muscle is to stretch and thin the lower segment and cause effacement and dilatation of the cervix. The junction of the upper and lower segments is called the physiological retraction ring. Effacement is most striking in the primigravida. In the parous patient dilatation and effacement usually occur together.

'Show' and formation of forewaters. The effacement and dilatation of the cervix loosens the membranes from the region of the internal os with slight bleeding and sets free the mucus plug or operculum. This constitutes the 'show' and allows the formation of forewaters, the amniotic sac pushing against the cervix.

STAGES OF LABOR

Although labor is a continuous process, it is divided into four functional stages because each has differing physiological activities and requires differing management (Fig. 12.4).

— The first stage of labor is the interval between the onset of labor and full cervical dilation (10 cm).

The first stage is further divided into two phases:

The latent phase of labor encompasses cervical effacement and early dilation.

The active phase of labor, during which more rapid cervical dilation occurs, usually beginning at approximately 4 cm.

— The second stage of labor encompasses complete cervical dilation through the delivery of the infant.

— The third stage of labor begins immediately

Principle in the management of labor

↓

- Diagnosis of labor
- Monitoring the progress of labor
- Ensuring maternal well-being
- Ensuring fetal well-being

Fig. 12.4

after delivery of the infant and ends with the delivery of the placenta.

The immediate postpartum period of approximately 2 hours after delivery of the placenta, during which time the patient undergoes significant physiologic adjustment.

CHARACTERISTICS OF NORMAL LABOR

Normal labor is a continuous process that has been divided into three stages for purposes of study, with the first stage further subdivided into two phases, the latent phase and the active phase. The first stage of labor is the interval between the onset of labor and full cervical dilatation. The second stage is the interval between full cervical dilatation and delivery of the infant. The third stage of labor is the period between the delivery of the infant and the delivery of the placenta.

The first stage of labor in primiparous women is noted to range from 6–18 hours, while in multiparous patients the range is reported to be 2–10 hours. The lower limit of normal for the rate of cervical dilatation during the active phase is 1.2 cm per hour in first pregnancies and 1.5 cm per hour in subsequent pregnancies. The duration of the second stage in the primipara is 30 minutes to 3 hours, and is 5–30 minutes for multiparas. For both, the duration of the third stage was reported to be 0–30 minutes for all pregnancies.

ESSENTIAL FACTORS OF BIRTH CANAL

The fetus descends during the first and second stages of labor. The birth canal is formed by dilatation of the cervix and vagina and by stretching and displacement of the muscles of the pelvic floor and perineum. The bladder is pulled above the pubis because of its attachment to the uterus; the urethra is stretched and the bowel is compressed.

The change of the vagina and perineum:

A. The relative of the bladder, urethra and the genital organs at the beginning of labor.

B. Formation of the birth canal with the cervix fully dilated.

Note the forward displacement of the urethra and bladder neck behind the pubis.

C. Marked stretching with downward and backward displacement of the posterior wall of the canal as the head descends down (Fig. 12.5).

At the End of the Second stage the birth canal has been fully formed. The outlet of the canal is at right angles to the inlet. The angulation is called the Curve of Carus (Fig. 12.6).

DIAGNOSIS OF LABOR

Labor can be preterm (if it starts before 36 completed weeks) or *term* (if it starts between 37–42 weeks) or *post term* (if it starts after 42 weeks). It can be spontaneous or induced.

Labor is said to be normal only if the following are met. These are:

— It starts spontaneously

— It starts at term (37–42 completed weeks)

— The fetus presents by vertex

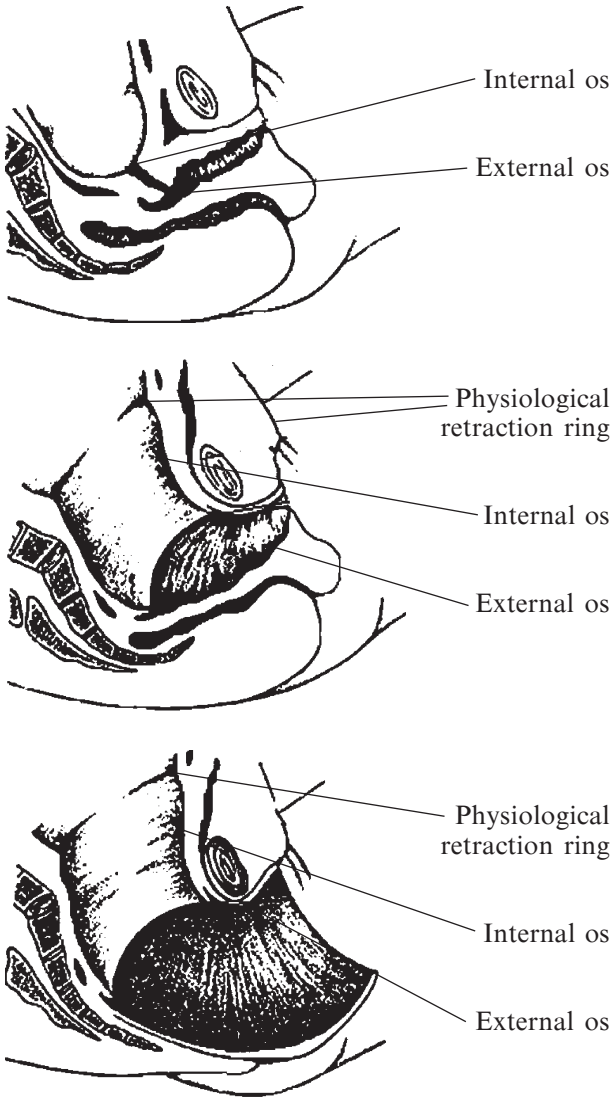


Fig. 12.5

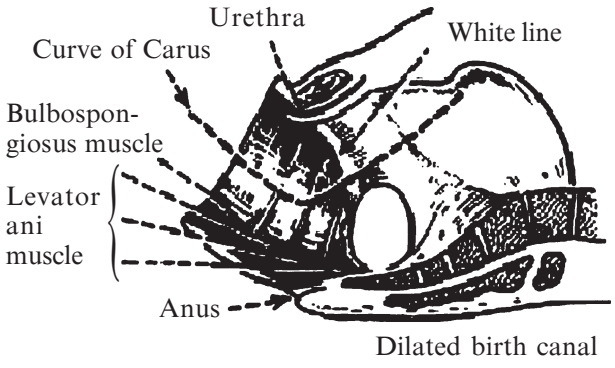


Fig. 12.6

— Delivery is effected vaginally spontaneously

— It ends in a birth of healthy newborn with minimal morbidity

False labor is a painless irregular uterine contraction occurring in the last 4–8 weeks of gestation and which doesn't result in cervical dilatation and effacement (Table 10).

Table 10

Differences between true labor and false labor

False labor	True labor
Contractions occur at irregular intervals	Contractions occur at regular intervals
With time contractions remain the same or decreases in intensity, frequency	With time contractions increase in intensity, frequency and duration
Contractions disappear with analgesics	Contractions persist despite analgesics
Lower abdominal and back pain present	Only lower abdominal discomfort present
Can occur in the last trimester	Occurs when labor commences
There is no cervical effacement and dilatation	There is progressive cervical dilatation and effacement
Occurs at night	Occurs at any time

SUPPORTIVE CARE DURING LABOR AND CHILDBIRTH

• The woman to have personal support from a person of her choice throughout labor and birth.

• The companion to give adequate support to the woman during labor and childbirth (rub her back, wipe her brow with a wet cloth, assist her to move about).

• Ensure good communication and support by staff:

— Explain all procedures, seek permission and discuss findings with the woman;

— History of allergies, use of medication, and time, amount, and content of last oral intake.

— Prenatal records with special attention to prenatal laboratory results that impact intrapartum and immediate postpartum management (eg, human immunodeficiency virus [HIV] and hepatitis B status). A blood group, Rh type, and antibody screen should also be done.

— Provide a supportive, encouraging atmosphere for birth that is respectful of the woman's wishes;

— Ensure privacy and confidentiality.

• Maintain cleanliness of the woman and her environment:

— Encourage the woman to wash herself or bathe or shower at the onset of labor;

— Wash the vulval and perineal areas before each examination.

• Ensure mobility:

— Encourage the woman to move about freely;

— Support the woman's choice of position during labor and birth

• Encourage the woman to empty her bladder regularly.

Note: Do not routinely give an enema to women in labor.

• Encourage the woman to eat and drink as she wishes.

• Teach breathing techniques for labor and delivery.

• Help the woman in labor who is anxious, fearful or in pain:

— Give her praise, encouragement and reassurance;

— Give her information on the process and progress of her labor;

— Listen to the woman and be sensitive to her feelings.

• If the woman is distressed by pain:

— Suggest changes of position;

— Encourage her companion to massage her back or hold her hand and sponge her face between contractions; and breathing techniques [3, 9, 45] (Fig. 12.7).

DIAGNOSIS AND CONFIRMATION OF LABOR

Palpable uterine contractions which are regular in frequency and intermittent in character. The interval between contractions is 10 minutes or less and each contraction may last half a minute or longer.

The uterus becomes firm and rises, altering the abdominal contour. This is due to the rising forwards of the uterus so that it approximates to the direction of the birth canal. This movement is easier if the patient is upright.

• *'Show'*. A little blood and mucus discharged from the vagina. This is from separation of the membranes at the lower pole causing bleeding which mixes with the operculum of the cervix.

• *Cervix.* Cervical effacement — the progressive shortening and thinning of the cervix during labor;

Cervical dilatation — the increase in diameter of the cervical opening measured in centimetres (Fig. 12.8).

Cervical dilatation is gauged by vaginal examination and is expressed in the diameter across the cervix (Fig. 12.9).

• *Factors affecting cervical dilatation:*

1. Contraction and retraction of the uterus.
2. The bag of fore-water.
3. Absence of membranes.
4. Fitting of the presenting part to the lower segment and the cervix.
5. Pre-labor changes in the cervix.

• *State of the liquor* if any (clear, meconium) (Table 11).

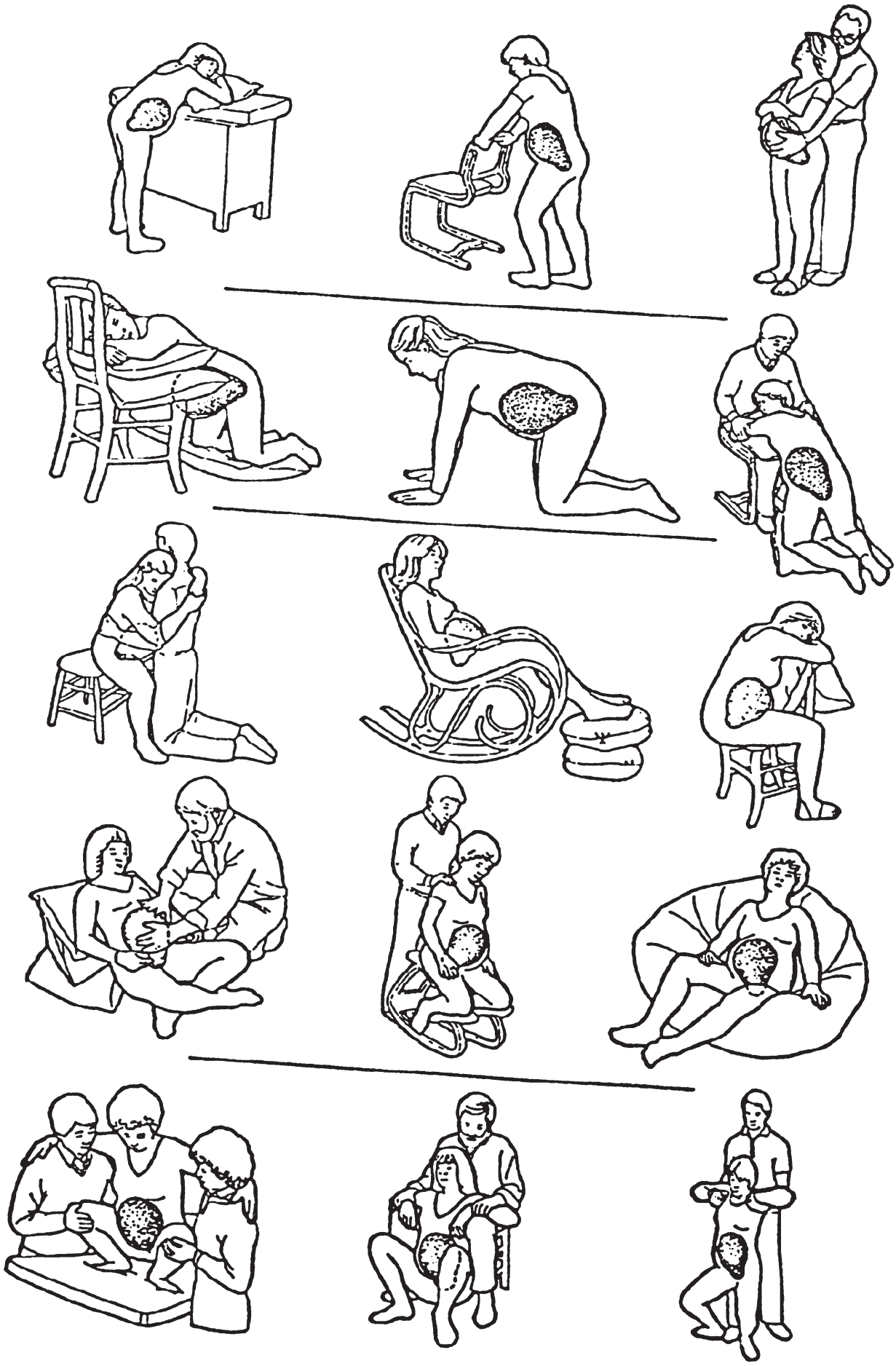


Fig. 12.7. Position of the laboring woman

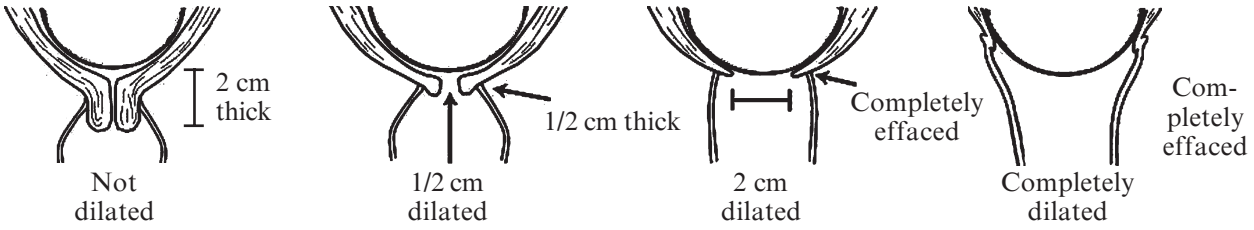


Fig. 12.8

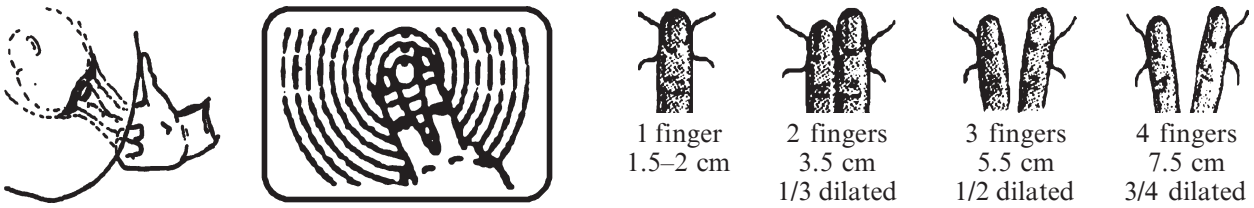


Fig. 12.9

Table 11

Diagnosis of stage and phase of labor

Symptoms and Signs	Stage	Phase
Cervix not dilated	False labor/ Not in labor	
Cervix dilated less than 4 cm	First	Latent
Cervix dilated 4–9 cm Rate of dilatation typically 1 cm per hour or more Fetal descent begins	First	Active
Cervix fully dilated (10 cm) Fetal descent continues No urge to push	Second	Early (non-expulsive)
Cervix fully dilated (10 cm) Presenting part of fetus reaches pelvic floor Woman has the urge to push	Second	Late (expulsive)
The third stage of labor begins with delivery of the baby and ends with the expulsion of the placenta.		

• *The presenting part* (Fig. 12.10).

The most common presenting part is the vertex of the fetal head. If the vertex is the presenting part, use *landmarks on the fetal skull* to determine the position of the fetal head in relation to the maternal pelvis.

• *Position of the presenting part.* This is determined by palpating the suture lines and fontanelles in relation to the pelvic diameters.

The fetal head normally engages in the maternal pelvis in an occiput transverse position, with the fetal occiput transverse in the maternal pelvis (Fig. 12.11).

With descent, the fetal head rotates so that the fetal occiput is anterior in the maternal pelvis (occiput anterior positions) (Fig. 12.12).

• *Level of the presenting part*. This is judged by its relationship to the brim or ischial spines (Fig. 12.13).

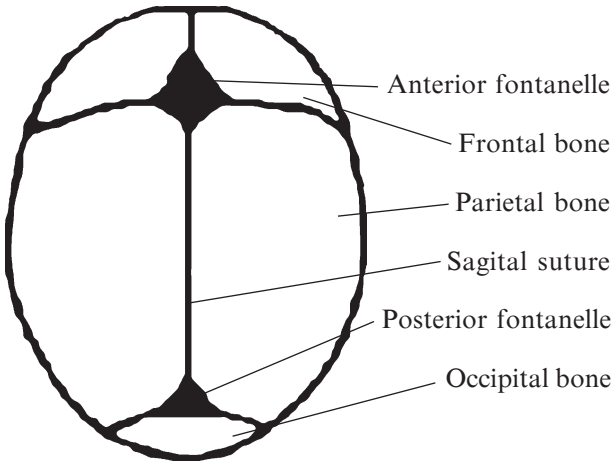


Fig. 12.10

An additional feature of a normal presentation is a *well-flexed vertex*, with the occiput lower in the vagina than the sinciput.

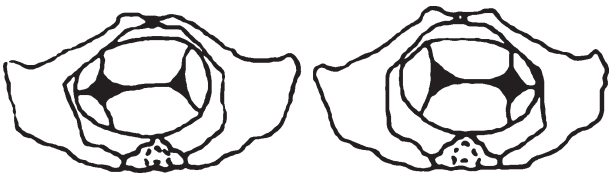


Fig. 12.11

Fig. 12.12

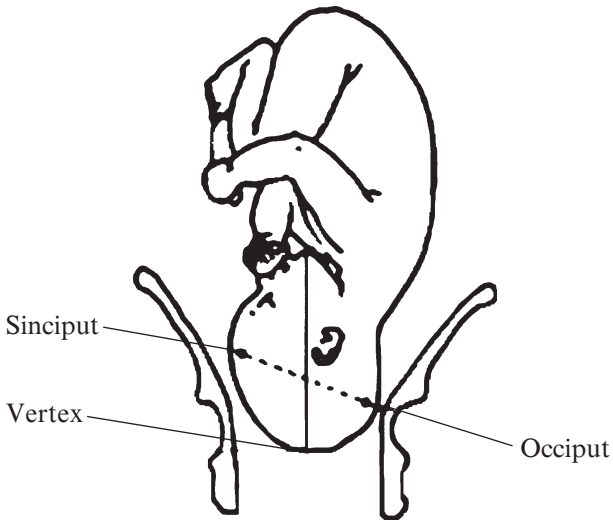


Fig. 12.13

FETAL STATION

Fetal station is determined by identifying the level of the fetal presenting part in the birth canal in relation to the ischial spines which are located approximately halfway between the pelvic inletand outlet (Fig. 12.14).

If the presenting part has reached the level of the ischial spines, it is termed zero station. The distance between the ischial spines to the pelvic inlet above and the distance from the spines to the pelvic outlet below are divided into fifths, and these measurements are used to further define the station. These divisions represent centimeters above and below the ischial spines. Thus, as the presenting fetal part descends from the pelvic inlet toward the ischial spines, the designation is -5, -4, -3, -2, -1, then 0 station. Below the ischial spines, the presenting fetal part passes +1, +2, +3, +4, with +5 station corresponding to the fetal head being visible at the introitus. The clinical significance of the fetal head presenting at zero station is that the biparietal diameter of the fetal head, the greatest transverse diameter of the fetal skull, is assumed to have negotiated the pelvic inlet.

The fetal head is said to be engaged at zero station, a crucial functional "*landmark*" in the labor path.

Engagement means the descent of the biparietal diameter through the pelvic brim.

Engagement occurs when the widest diameter of the fetal presenting part has passed through the pelvic inlet. In cephalic presentations, the widest diameter is biparietal; in breech presentations, it is intertrochanteric. The station of the presenting part in the pelvic canal is defined as its level above or below the plane of the ischial spines. The level of the ischial spines is assigned as "zero" station, and each centimeter above or below this level is given a minus or plus designation, respectively. In the majority of

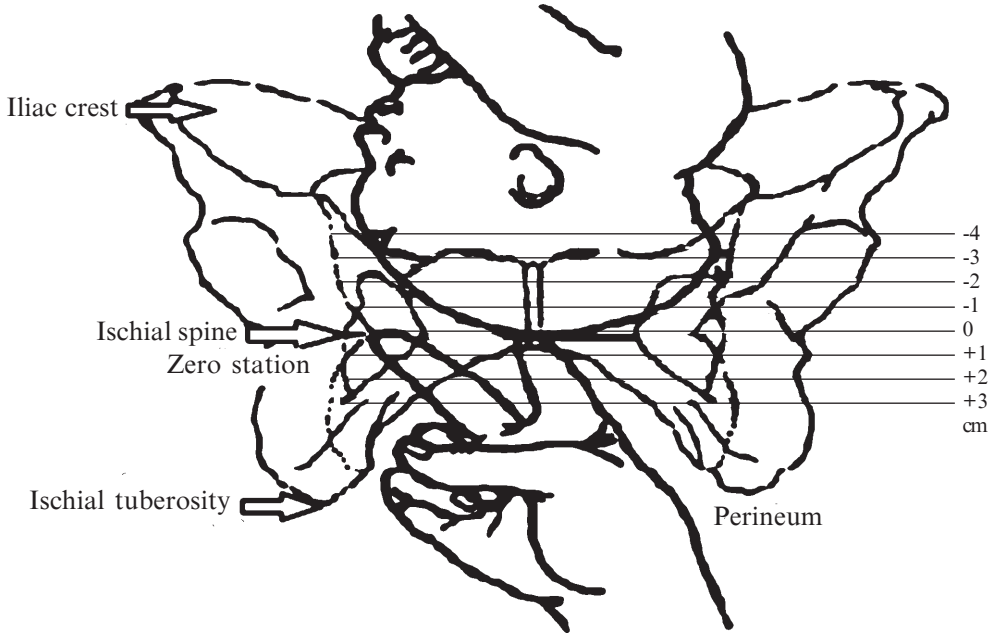
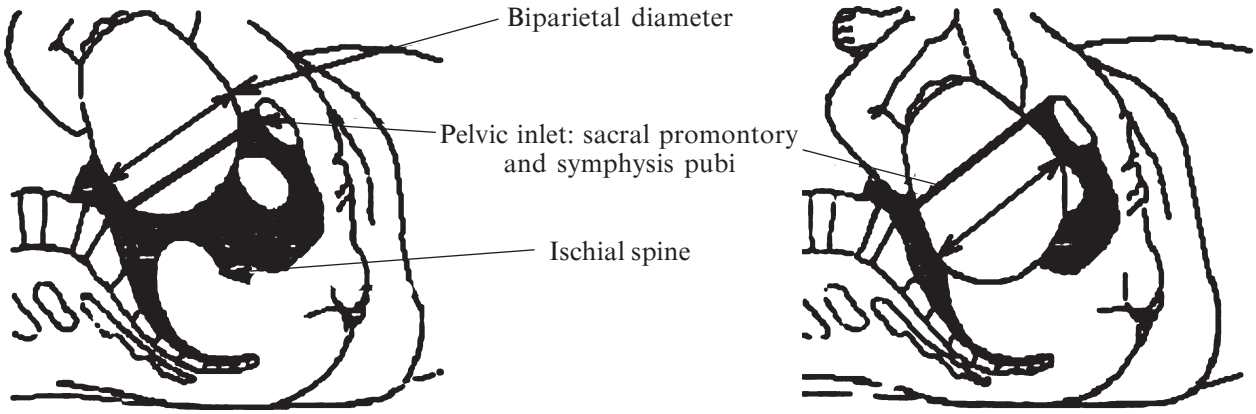


Fig. 12.14



Baby's head is not engaged as widest diameter is brim of pelvis

Baby's head is engaged as widest diameter is below of pelvis

Fig. 12.15

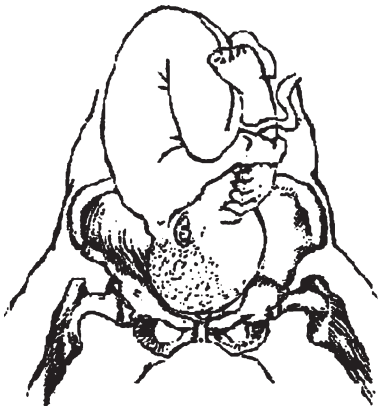
women, the bony presenting part is at the level of the ischial spines when the head has become engaged. The fetal head usually engages with its sagittal suture in the transverse diameter of the pelvis [13, 49] (Fig. 12.15).

ABDOMINAL PALPATION FOR DESCENT OF THE FETAL HEAD

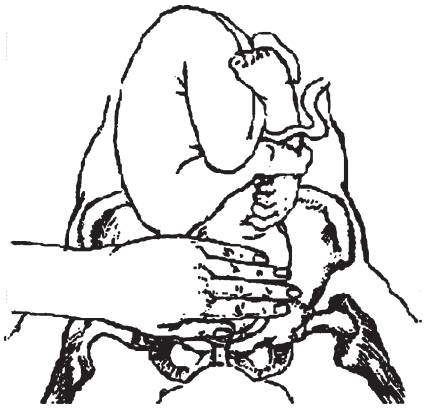
A convenient way to describe the amount of head above the brim is to identify the number of 'fifths' palpable. When the head is engaged 2/5 or less will be felt abdominally. Although it is discussed as a pre-labor phenomenon, engagement seldom occurs until after labor is established, and the term is often used to mean that the presenting part is 'fixed' — entering the pelvis — as opposed to 'free' or still mobile above the brim [3, 23, 48] (Fig. 12.16).

MECHANISM OF LABOR

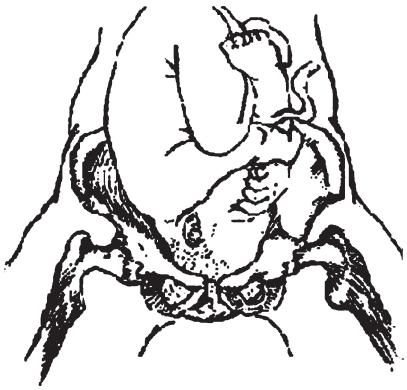
The mechanisms of labor (also known as the cardinal movements of labor) refer to the changes of the



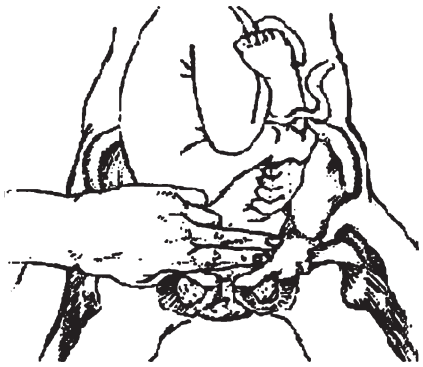
A. Head is mobile above the symphysis pubis = 5/5



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

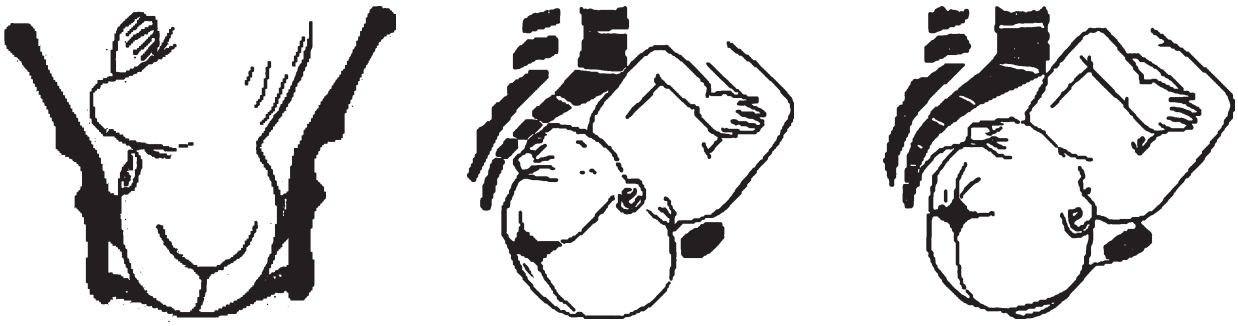


C. Head is 2/5 above symphysis pubis



D. Head accommodates two fingers above the symphysis pubis

Fig. 12.16



a *b* *c*

Fig. 12.17

position of the fetus as it passes through the birth canal. The fetus usually descends to where the occipital portion of the fetal head is the lowermost part in the pelvis, and it rotates toward the largest pelvic segment. Because vertex presentation occurs in 95% of term labors, the cardinal movements of labor are defined relative to this presentation. To accommodate the maternal bony pelvis, the fetal head must undergo several movements as it passes through the birth canal. These movements are accomplished by means of the forceful contractions of the uterus. These cardinal movements of labor do not occur as a distinct series of movements, but rather as a group of movements that overlap as the fetus accommodates and moves progressively through the birth canal.

The mechanism of labor in vertex presentation:

1. Engagement
2. Descent
3. Flexion
4. Internal rotation
5. Extension
6. Restitution
7. External rotation
8. Shoulder rotation
9. Delivery of the fetal body

Engagement is defined as descent of the biparietal diameter of the head below the plane of the pelvic inlet, suggested clinically by palpation of the presenting part below the level of ischial spines (zero station). Engagement commonly occurs days to weeks prior to labor in women who have not delivered a child, whereas in women who have had children it more commonly happens at the onset of active labor. In any event, the importance of this event is that it suggests that the bony pelvis is adequate to allow significant descent of the fetal head, although the extension of this to the idea that delivery through the pelvis will happen in the labor does not follow.

Descent of the presenting part is necessary for the successful completion of passage through the birth canal. The greatest rate of descent occurs during the latter portions of the first stage of labor and during the second stage of labor (Fig. 12.17, *a*).

Flexion of the fetal head allows for the smaller diameters of the fetal head to present to the maternal pelvis (Fig. 12.17, *b*).

Internal rotation, like flexion, facilitates presentation of the optimal diameters of the fetal head to the bony pelvis, most commonly from transverse to either anterior or posterior (Fig. 12.17, *c*).

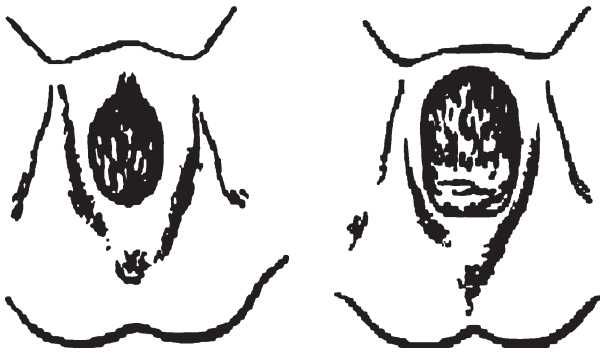
Extension of the fetal head occurs as it reaches the introitus. The flexed head now extends (Fig. 12.18, *a*).

Crowning: the *o*cciput is now below symphysis pubis (Fig. 12.18, *b*)

Further descent pushes the head forward with a movement of extension and the occiput is delivered.

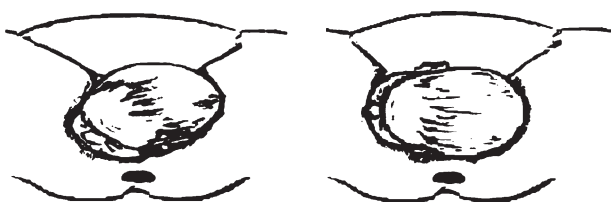
External rotation occurs after delivery of the head as the head rotates to "face forward" relative to its shoulders. This is known as *restitution*, followed rapidly by delivery of the body, expulsion (Fig. 12.19, *a, b*).

Further descent and rotation causes the head to rotate so that the occiput lies to the left maternal thigh. This is *external rotation.* The anterior shoulder now slips under the symphysis pubis and with lateral flexion of the fetal body, the posterior shoulder is born. The rest of the body follows easily (Fig. 12.20).



a *b*

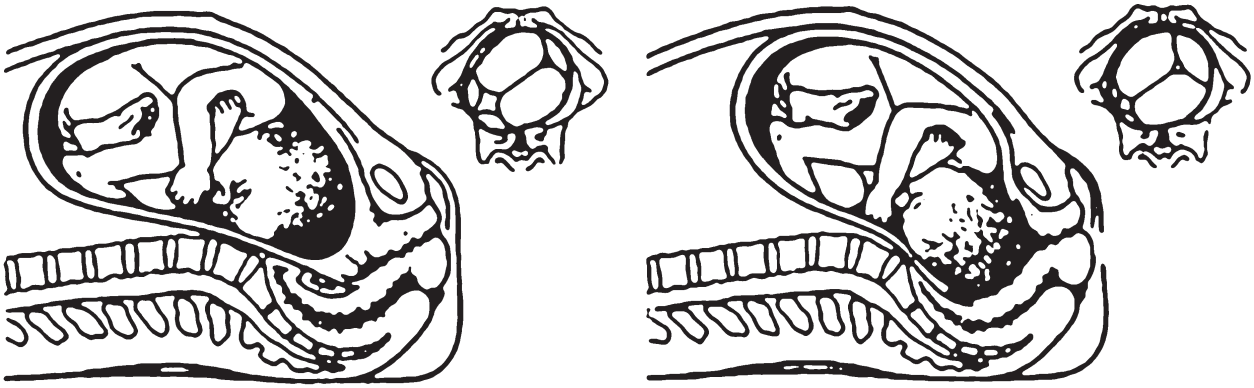
Fig. 12.18



a *b*

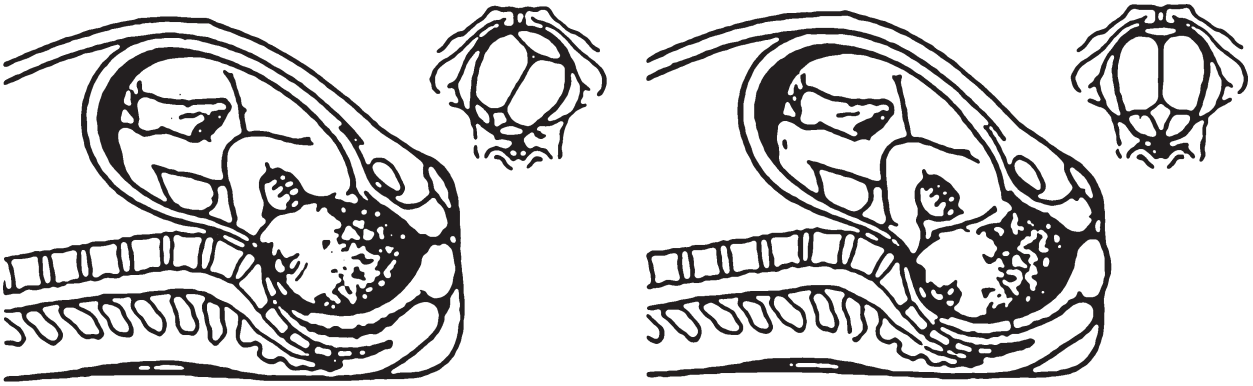
Fig. 12.19

The cardinal movements of labor



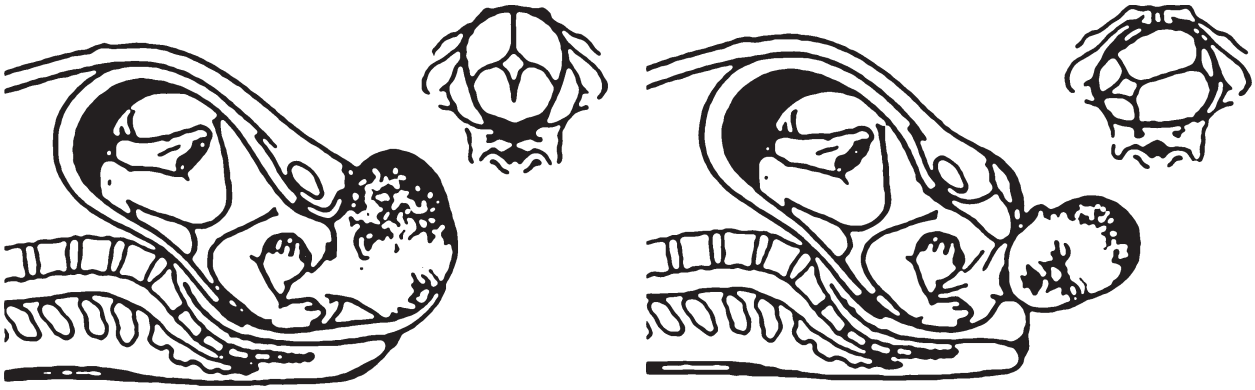
1. Head floating, before engagement

2. Engagement; descent, flexion



3. Further descent, internal rotation

4. Complete rotation, beginning extension



5. Complete extension

6. Restitution (external rotation)

7. Delivery of anterior shoulder

8. Delivery of posterior shoulder

Fig. 12.20

USING THE PARTOGRAPH

Partograph — a graphical representation of progress of labor. This record allows visual assessment of mother pulse rate and blood pressure, strength and frequency of uterine contraction. Cervical dilatation in centimeters against the time in hours plotted against expected norm so compared to an average curve for a normal primegravida or multigravida as may be appropriate in any of the given population [45, 48, 49].

Component of the partograph:

— Part I: fetal condition (at top).

— Part II: progress of labor (at middle).

— Part III: maternal condition (at bottom).

— Outcome.

On the partograph:

Patient information: Fill out name, gravida, para, hospital number, date and time of admission, and time of ruptured membranes or time elapsed since rupture of membranes (if rupture occurred before charting on the partograph began). Fetal heart rate: Record every half hour.

Fetal heart rate: Record every half hour (Fig. 12.21).

Amniotic fluid: Record the colour of amniotic fluid at every vaginal examination:

— I: membranes intact;

— R: membranes ruptured;

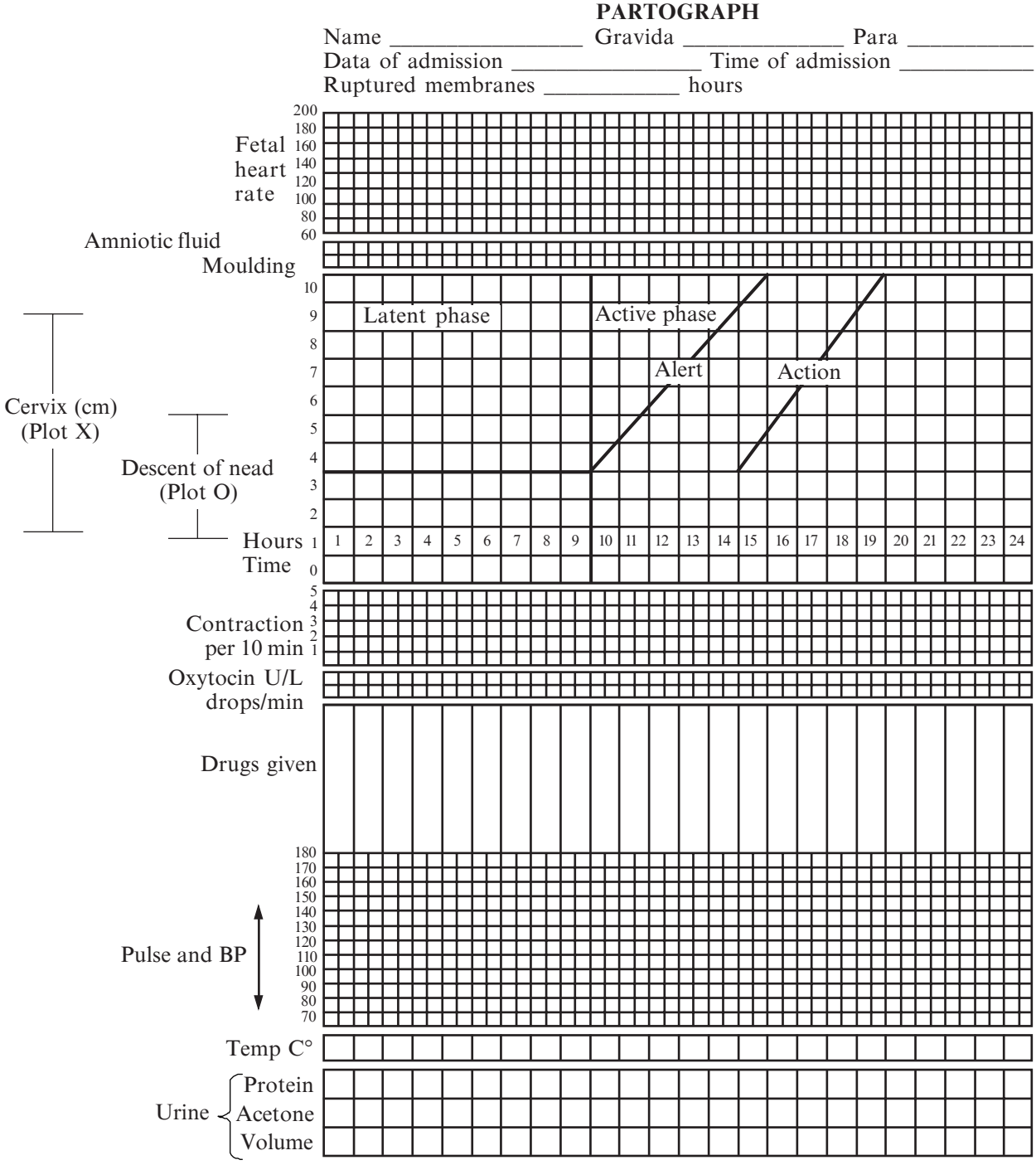


Fig. 12.21

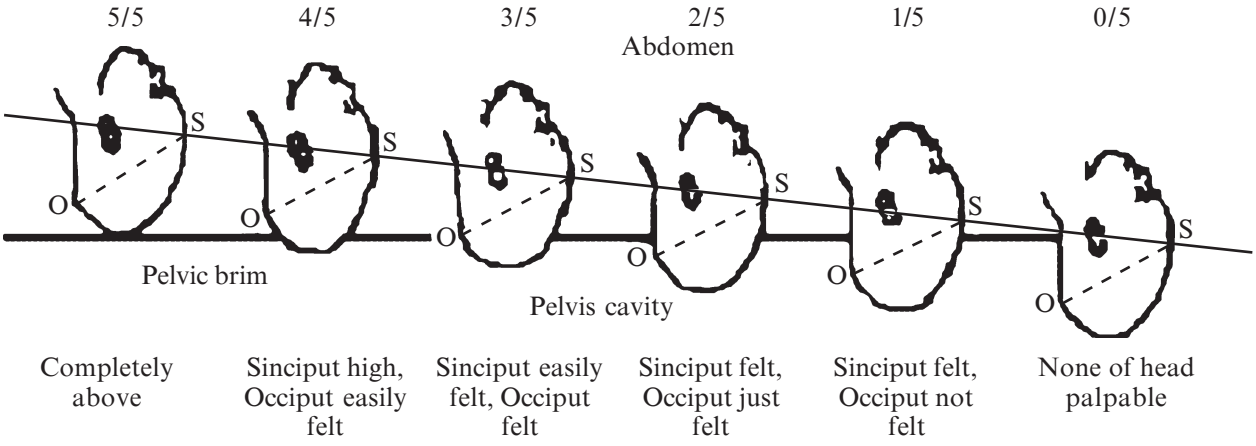


Fig. 12.22

— C: membranes ruptured, clear fluid;
— M: meconium-stained fluid;
— B: blood-stained fluid.

Moulding:

1: sutures apposed;
2: sutures overlapped but reducible;
3: sutures overlapped and not reducible.

Cervical dilatation:

Assessed at every vaginal examination and marked with a cross (X). Begin plotting on the partograph at 4 cm.

The cervical line does not cross the Alert line.

Alert line:

A line starts at 4 cm of cervical dilatation to the point of expected full dilatation at the rate of 1 cm per hour.

Action line:

Parallel and four hours to the right of the alert line.

The lines in the cervical dilated section are the expected patterns of cervical dilation in labor showing a slow latent phase and faster active phase. If dilation crosses the action line then the patient should be reviewed and/or an ARM (Fig. 12.22).

Descent assessed by abdominal palpation:

Refers to the part of the head (divided into five parts) palpable above the symphysis pubis; recorded as a circle (O) at every abdominal examination. At 0/5, the sinciput (S) is at the level of the symphysis pubis.

Hours: Refers to the time elapsed since onset of active phase of labor (observed or extrapolated).

Time: Record actual time.

Contractions: Chart every half hour; count the number of contractions in a 10-minute time period, and their duration in seconds.

— Less than 20 seconds:
— Between 20 and 40 seconds:
— More than 40 seconds:

Oxytocin: Record the amount of oxytocin per volume IV fluids in drops per minute every 30 minutes when used.

Drugs given: Record any additional drugs given.

Pulse: Record every 30 minutes and mark with a dot (•).

Blood pressure: Record every four hours and mark with arrows.

Temperature: Record every two hours.

Protein, acetone and volume: Record when urine is passed.

A sample partograph for normal labor (Fig. 12.23):

• A primigravida was admitted in the latent phase of labor at 5 AM:
— fetal head was 4/5 palpable;
— cervix dilated 2 cm;
— three contractions in 10 minutes, each lasting 20 seconds;
— normal maternal and fetal condition.

Note: Because the woman was in the latent phase of labor, this information is not plotted on the partograph.

• At 9 AM:
— fetal head 3/5 palpable;
— cervix dilated 5 cm;
— four contractions in 10 minutes, each lasting 35 seconds.

Note: The woman was in the active phase of labor and this information is plotted on the partograph. Cervical dilatation is plotted on the alert line.

• At 11 AM:
— fetal head 2/5 palpable;
— four contractions in 10 minutes, each lasting 45 seconds.

• At 1 PM:
— fetal head 0/5 palpable;
— cervical dilatation progressed at rate of more than 1 cm per hour and cervix fully dilated;
— five contractions in 10 minutes each lasting 45 seconds;
— spontaneous vaginal delivery at 1 : 20 PM.

A sample partograph showing arrest of dilatation and descent in the active phase of labor (Fig. 12.23).

Fetal distress and third degree moulding, together with arrest of dilatation and descent in the active phase of labor in the presence of adequate uterine contractions, indicates obstructed labor (Fig. 12.24).

• The woman was admitted in active labor at 10 AM:
— fetal head 3/5 palpable;
— cervix dilated 4 cm;
— three contractions in 10 minutes, each lasting 20–40 seconds;
— clear amniotic fluid draining;

PARTOGRAPH

Name _____ Cravida _3_ Para _2_ Ruptured membranes 1 h

Data of admission _12.05.2017_ Time of admission _5:00 A.M._

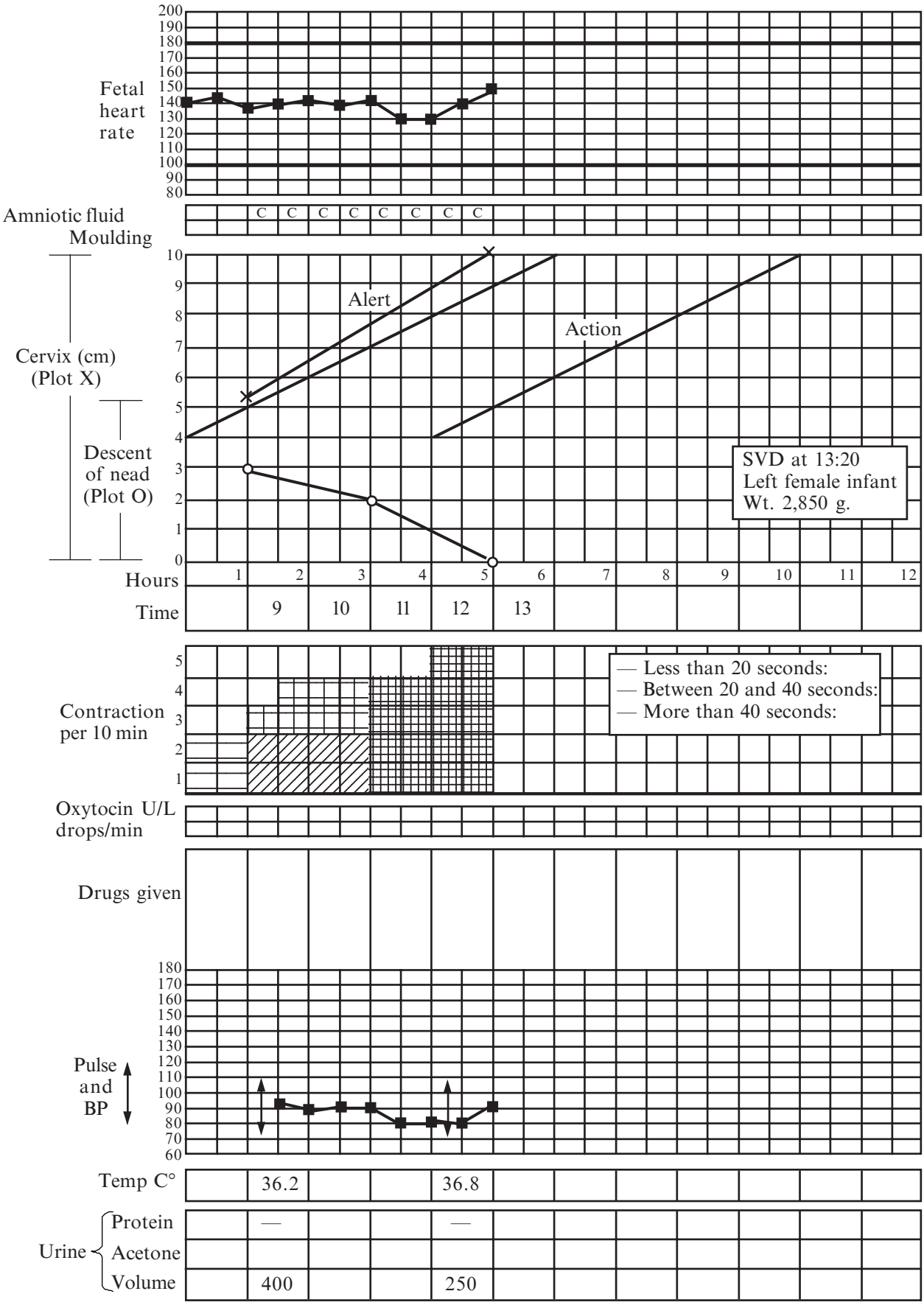


Fig. 12.23

PARTOGRAPH

Name _____ Cravida _4_ Para _3+0_ Ruptured membranes 1 h
Data of admission _12.05.2017_ Time of admission _10:00 A.M._

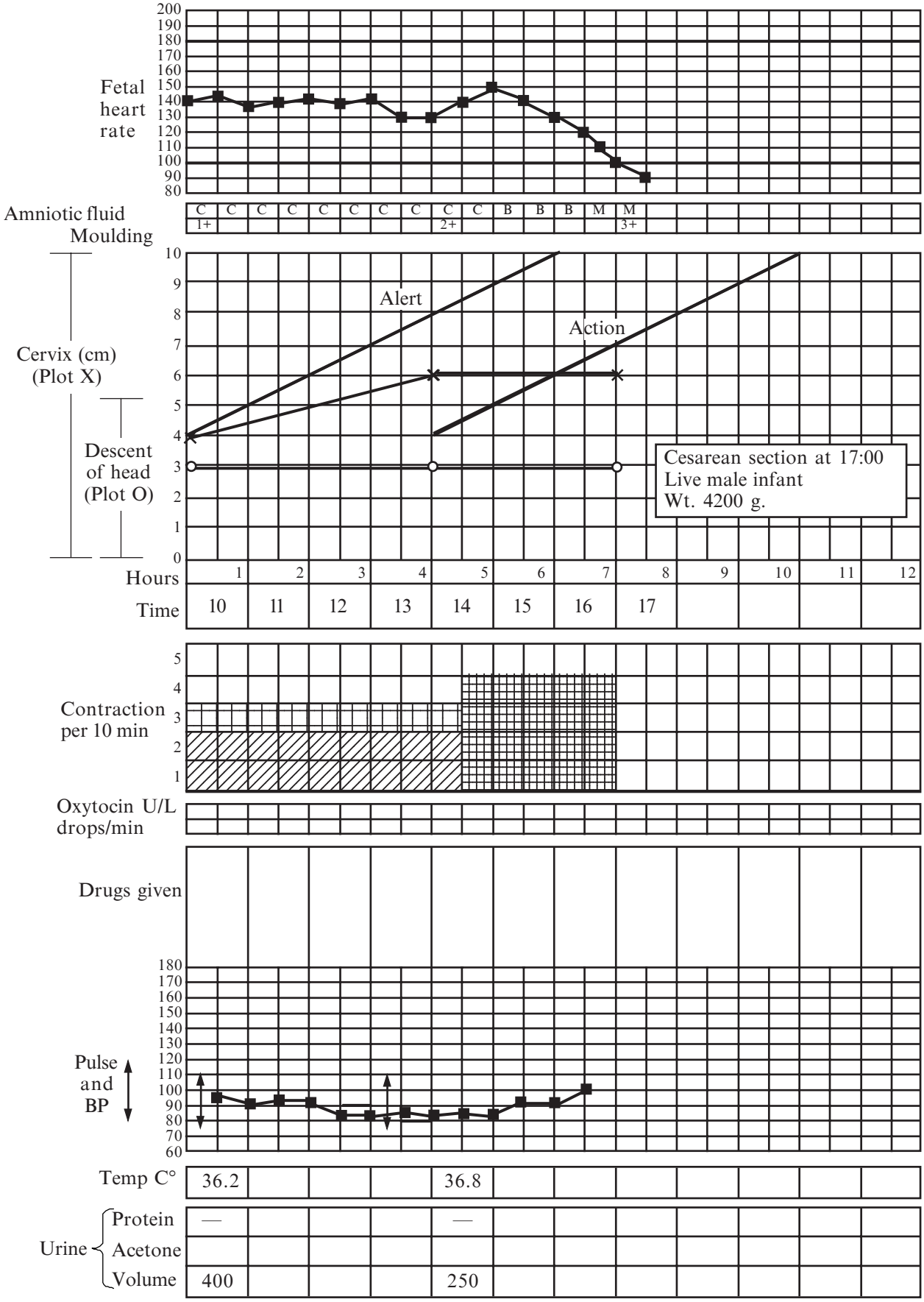


Fig. 12.24

— first degree moulding.

• At 2 PM:

— fetal head still 3/5 palpable;

— cervix dilated 6 cm and to the right of the alert line;

— slight improvement in contractions (three in 10 minutes, each lasting 45 seconds);

— second degree moulding.

• At 5 PM:

— fetal head still 3/5 palpable;

— cervix still dilated 6 cm;

— third degree moulding;

— fetal heart rate 92 per minute;

— amniotic fluid stained with meconium.

— Cesarean section performed at 5 : 30 pm due to fetal distress.

PROGRESS OF FIRST STAGE OF LABOR

• Findings suggestive of *satisfactory progress* in the first stage of labor are (Fig. 12.25):

— regular contractions of progressively increasing frequency and duration;

— rate of cervical dilatation at least 1 cm per hour during the active phase of labor (cervical dilatation on or to the left of alert line);

— cervix well applied to the presenting part.

• Findings suggestive of unsatisfactory progress in the first stage of labor are:

— irregular and infrequent contractions after the latent phase;

— OR rate of cervical dilatation slower than 1 cm per hour during the active phase of labor (cervical dilatation to the right of alert line);

— OR cervix poorly applied to the presenting part.

Unsatisfactory progress in labor can lead to prolonged labor.

PROGRESS OF SECOND STAGE OF LABOR

• Findings suggestive of satisfactory progress in the second stage of labor are:

— steady descent of fetus through birth canal;

— onset of the expulsive (pushing) phase.

The normal second stage of labor in a primigravida lasts about one hour and is much shorter in the parous woman. It is recognised by a change in the character of the contractions. They become more powerful and expulsive with a desire to bear down, and the secondary forces now come into action. The diaphragm is fixed, the patient holds her breath and the abdominal muscles contract. Sometimes the mother feels nauseated and may vomit. She may have the feeling that the bowel is about to move, due to pressure on the rectum, and this may have an inhibiting effect on her until the reason is explained. The head descends deeply in the pelvis and may be visible or palpable through the perineum (Fig. 12.26).

• Findings suggestive of unsatisfactory progress in second stage of labor are:

— lack of descent of fetus through birth canal;

— failure of expulsion during the late (expulsive) phase.

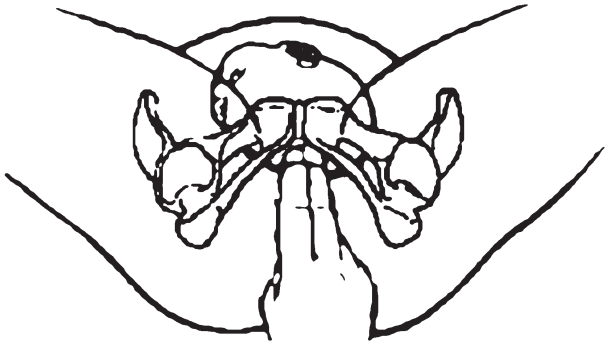


Fig. 12.25

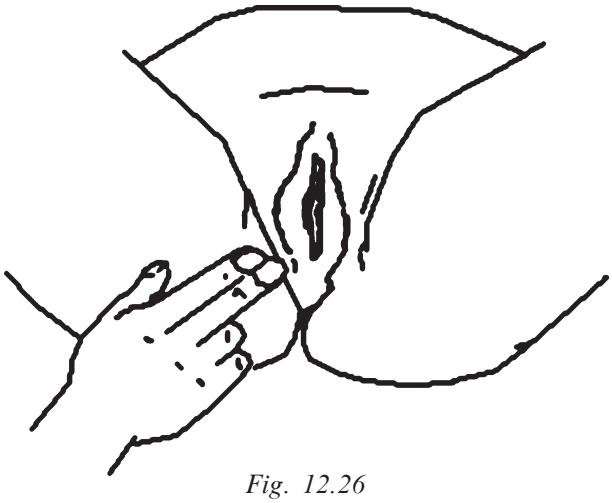


Fig. 12.26

PROGRESS OF FETAL CONDITION

• If there are fetal heart rate abnormalities (less than 110 or more than 170 beats per minute), suspect fetal distress.

• Positions or presentations in labor other than occiput anterior with a well-flexed vertex are considered malpositions or malpresentations.

• If unsatisfactory progress of labor or prolonged labor is suspected, manage the cause of slow progress.

MANAGEMENT OF NORMAL LABOR

Care of the mother (General):

Ideally the atmosphere created should be as near to a 'home within hospital' as much as is compatible with facilities and safety.

Minimal perineal shaving may be done in case an episiotomy is required. If the rectum is full a suppository is given to reduce the risk of faecal soiling in late labor. Access to a bath or shower should be available.

Analgesia is given as required. The method chosen depends on the mother's preference, her reaction to her contractions and the likely length of her labor [7, 21, 36, 45, 49].

Anesthesia for labor:

Types:

• General anesthesia

• Regional anesthesia (spinal, epidural or combined spinal and epidural anesthesia).

In urgent CS (such as cases with severe, rapid bleeding or other haemodynamic compromise), regional anesthesia may be appropriate and general anesthesia is considered.

Local anaesthetic (0.25% or 0.5% bupivacaine) is instilled at 3–4 hour intervals through a catheter inserted into the epidural space. This gives the patient complete removal from pain of labor. Most obstetricians consider epidural anaesthesia justified if the patient asks for it and it is the method of choice in some cases e.g. hypertension, premature labor.

The epidural space is about 4 mm wide and lies between the dura and the periosteum of the vertebral canal. It is limited above at the foramen magnum where dura and periosteum fuse, and below by the ligament covering the sacral hiatus.

Local anaesthetic can be injected into this space through a fine catheter introduced through a specially designed needle, which is traversed by the spinal nerves, and produces the same effect as a spinal block without the risk of headache, meningism or nerve root trauma (Fig. 12.27).

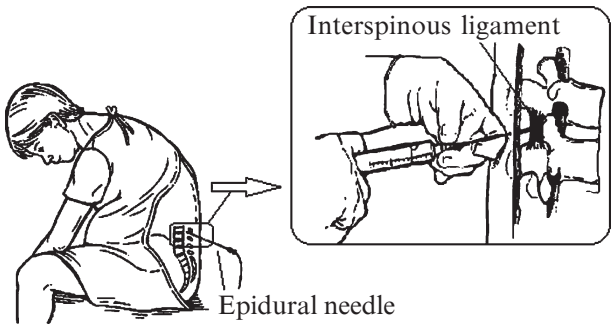


Fig. 12.27

Complications:

• Mild hypotension (about 20%).

• Sepsis (a bacterial filter should be attached to the syringe).

• Needle inserted into cerebrospinal space. (This is a failure of the technique.)

• Bladder atony and increased need for catheterisation (about 40%).

• Total spinal block. This occurs if the local anaesthetic is injected into the cerebrospinal space. Respiration is paralysed and there is hypotension. This is an acute anaesthetic emergency (Fig. 12.28).

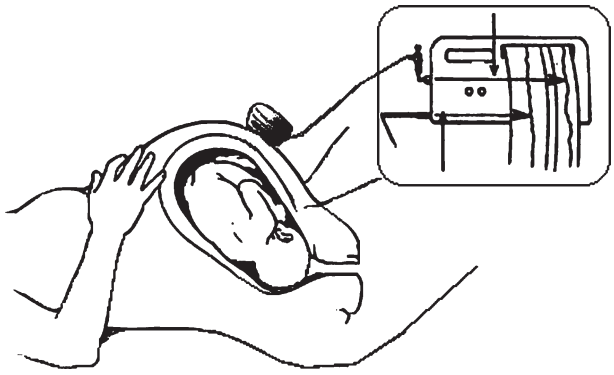


Fig. 12.28

Care of the baby

Assessment of the baby's condition in labor depends essentially on observations of the fetal heart rate. Initial 10- to 20-minute continuous FHR assessment on entry into labor/birth area. Intermittent auscultation every 30 minutes during the latent phase of labor and every 15 minutes during the active phase of labor.

When the fetal membranes rupture, spontaneously or artificially, assess the FHR and check the amniotic fluid for color, odor, and amount. The presence of meconium in the liquor should always be noted but is, at best, only a warning that there may be a problem. It is postulated that fetal hypoxia leads to an increased output by the vagus, stimulating the fetal gut and resulting in the passage of meconium (Fig. 12.29).

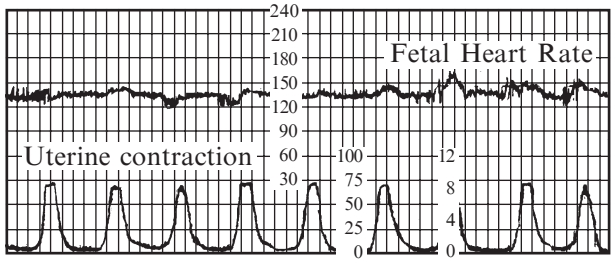


Fig. 12.29

INTERPRETATION OF THE FETAL HEART TRACING

The interpretation of the heart rate tracing should follow a systematic approach with a full qualitative and quantitative description of the following:

• Baseline FHR:

— rate;

— variability *(long-term and shortterm)*.

• Periodic changes in the rate:

— acceleration;

— deceleration.

• Frequency and intensity of uterine contractions (Table 12).

Interpretation of intrapartum FHR Tracings:

1. The *average baseline rate* should be between 110 and 170 beats per minute. Sustained tachycardia may be a warning of fetal distress and prolonged or severe bradycardia is ominous.

Fetal bradycardia occurs when the FHR is below 110 bpm and lasts 10 minutes or longer. It can be the initial response of a healthy fetus to asphyxia. Causes of fetal bradycardia might include fetal hypoxia, prolonged maternal hypoglycemia, fetal acidosis,

Table 12

Interpreting FHR Patterns

FHR Pattern	Signs
Reassuring FHR signs	— Normal baseline (110–160 bpm) — Moderate bradycardia (100–120 bpm); good variability — Good beat-to-beat variability and fetal accelerations
Nonreassuring signs	— Fetal tachycardia (> 160 bpm) — Moderate bradycardia (100–110 bpm); lost variability — Absent beat-to-beat variability — Marked bradycardia (90–100 bpm) — Moderate variable decelerations
Ominous signs	— Fetal tachycardia with loss of variability — Prolonged marked bradycardia (< 90 bpm) — Severe variable decelerations (< 70 bpm) — Persistent late decelerations

administration of drugs to the mother, hypothermia, maternal hypotension, prolonged umbilical cord compression, and fetal congenital heart block.

Fetal tachycardia is a baseline FHR greater than 160 bpm that lasts for 10 minutes or longer. It can represent an early compensatory response to asphyxia. Other causes of fetal tachycardia include fetal hypoxia, maternal fever, maternal dehydration, amnionitis, drugs (e.g., cocaine, amphetamines, nicotine), maternal hyperthyroidism, maternal anxiety, fetal anaemia, prematurity, fetal heart failure, and fetal arrhythmias. Fetal tachycardia is considered an ominous sign if it is accompanied by a decrease in variability and late decelerations.

2. *Baseline variability.* The normal FHR fluctuates by 10 beats/min every 5 seconds or so, evidence of fetal ability to react normally to the stress of labor. Loss of this variability, especially in association with tachycardia, indicates severe hypoxia.

Variability is one of the most important characteristics of the FHR. Two components of baseline variability are described: *short-term* and *long-term.* Short-term variability is the beat-to-beat change in FHR. It represents the variations or fluctuations of the baseline that, when seen on the fetal monitor tracing, produces the irregularity within the baseline. It can be measured by internal monitoring and is classified as either present or absent. The presence of short-term variability typically indicates a well-oxygenated, nonacidemic fetus. The most practical way to determine the presence or absence of short-term variability is visually. The fetal heart tracing line is evaluated for roughness or smoothness. If roughness is present in the baseline, short-term variability is present; if smoothness is present, it is absent.

Long-term variability is the waviness or rhythmic fluctuations, which are described as cycles per minute. The frequency of cycles is 3 to 6 per minute. It is classified as absent (< 3 bpm), decreased or minimal (3 to 5 bpm), average or moderate (6 to 25 bpm), and marked or salutatory (> 25 bpm).

FHR variability is an important clinical indicator that is predictive of fetal acid — base balance and cerebral tissue perfusion. As the central nervous system is desensitized by hypoxia and acidosis, FHR decreases until a smooth baseline pattern appears. Loss of variability may be associated with a poor outcome. Some causes of decreased variability include fetal hypoxia/acidosis, drugs that depress the central nervous system, congenital abnormalities, fetal sleep, prematurity (Fig. 12.30).

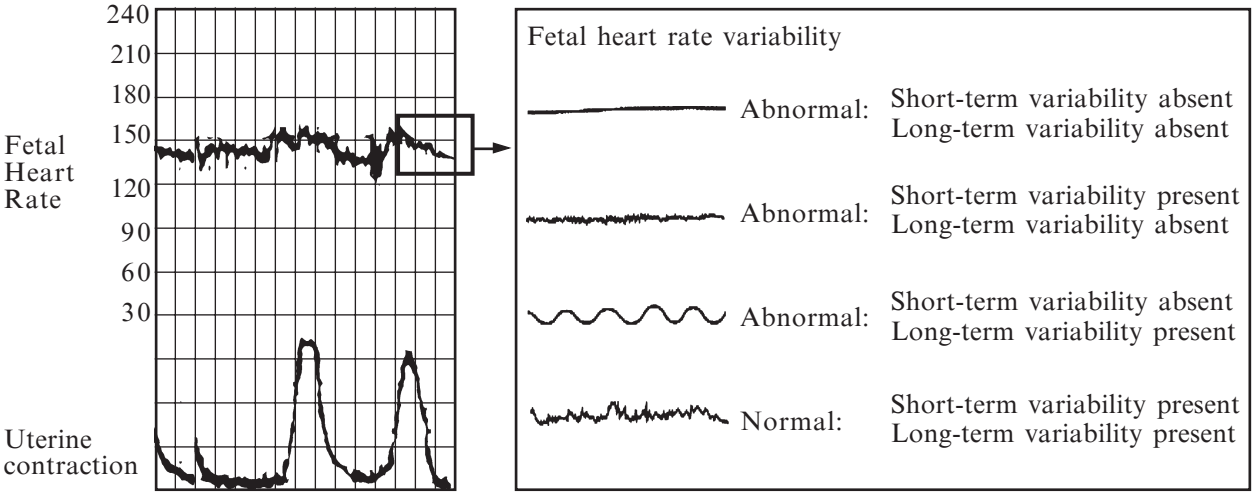


Fig. 12.30

3. *Periodic baseline changes* are temporary, recurrent changes made in response to a stimulus such as a contraction. The FHR can demonstrate patterns of acceleration or deceleration in response to most stimuli. *Fetal accelerations* are transitory increases in the FHR above the baseline associated with sympathetic nervous stimulation. They are visually apparent, with elevations of FHR of at least 15 bpm above the baseline, and usually last longer than 15 seconds but not for longer than 2 minutes. Their appearance provides evidence of fetal well-being and is generally considered reassuring and requires no interventions. Accelerations denote fetal movement and fetal well-being. *A deceleration* is a transient fall in FHR caused by stimulation of the parasympathetic nervous system. Decelerations are described by their shape and association to a uterine contraction. They are classified as *early*, *late*, *variable*, and *prolonged*.

Early, where the lowest rate of the FH coincides with the peak of contractions. (Fig. 12.31). These may be normal in late labor but should not be ignored if persistent or severe.

Late, where the lowest rate of the FH follows the peak of contraction. These may indicate hypoxia (Fig. 12.32).

Variable, where the pattern and timing of deceleration varies with contractions. These are thought to be due to cord compression. They are quite common and may be related to the mother's position. They should not however be ignored if persistent or associated with other adverse features (Fig. 12.33).

Prolonged decelerations are abrupt FHR declines of at least 15 bpm that last longer than 2 minutes but less than 10 minutes. The rate usually drops to less than 90 bpm. Many factors are associated with this pattern, including prolonged cord compression, abruptio placentae, cord prolapse, supine maternal position, vaginal examination, fetal blood sampling, maternal seizures, regional anesthesia, or uterine rupture. Prolonged decelerations can be remedied by

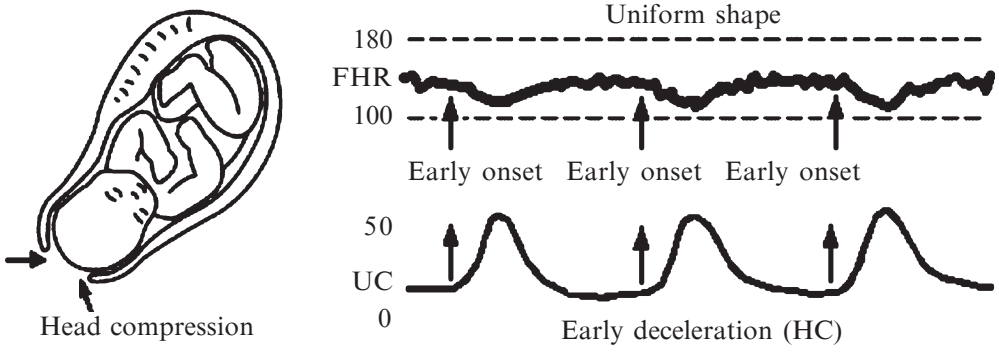


Fig. 12.31

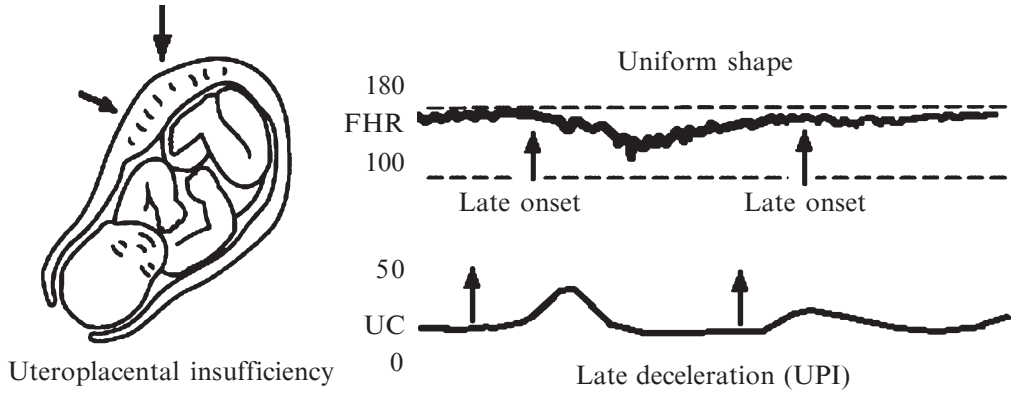


Fig. 12.32

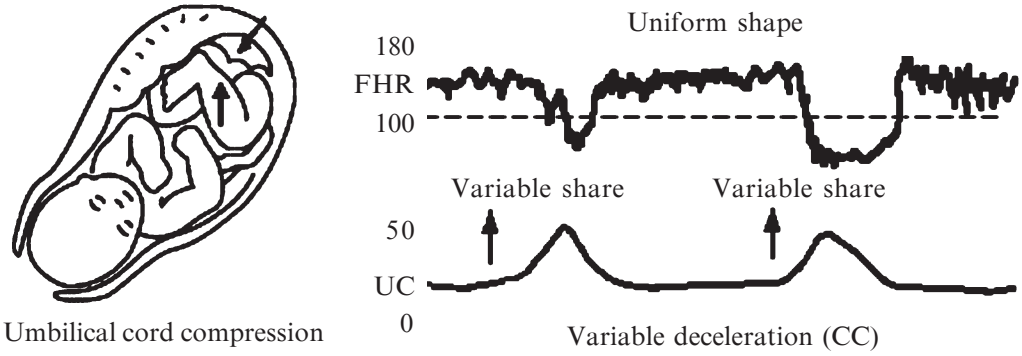


Fig. 12.33

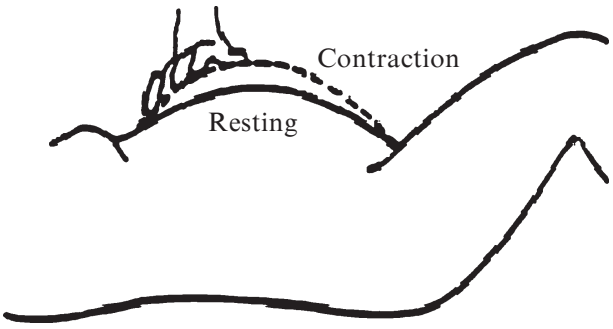


Fig. 12.34

identifying the underlying cause and correcting it [9, 23, 43].

Parameters of uterine contraction (Fig. 12.34):

Frequency — refers to the time between beginning of one contraction and the beginning of the next contraction.

Duration of each contraction is measured from the beginning of the contraction to the completion of the contraction.

— 20 second long contraction: early labor.

— 40 to 80 second long contraction: late labor.

Intensity refers to the strength of the uterine contraction during the peak of the contraction. Strength: mild, moderate, strong (Fig. 12.35).

Interval. Amount of time during which the uterus relaxes between contractions.

— 10 to 20 minutes between contractions: early labor.

— 3 to 5 minutes between contractions: late labor.

Once the cervix is fully dilated and the woman is in the expulsive phase of the second stage, encourage the woman to assume the position she prefers and encourage her to push (Fig. 12.36).

The mother is allowed to make involuntary expulsive efforts provided full dilatation of the cervix has been confirmed. Premature pushing can make the cervix oedematous and delay progress.

Organised pushing should not be started until the baby's head is visible. This is hard work and the mother will tire quickly.

MANAGEMENT OF DELIVERY

Delivery is a sterile and antiseptic procedure. Most women are delivered in the dorsal position. A sterile pad is placed over the anus.

Crowning of the head is when there is no recession between contractions and is due to the biparietal diameter having passed through the bony pelvis (Fig. 12.37).

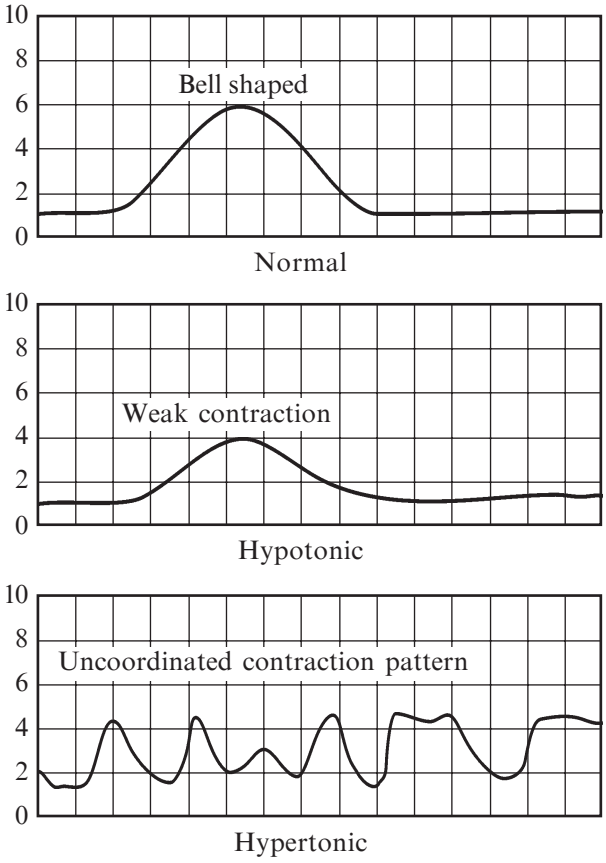


Fig. 12.35

Episiotomy is a surgical incision into the perineum between the vagina and anus, to enlarge the space at the outlet. Sometimes performed during the second stage of labor. Prior to instrumental delivery (forceps, vacuum) (Fig. 12.38).

Types of episiotomy:

Midline episiotomy:

— Cut the perineal body.

— Scar tissue replaces connective tissue.

— Considered to be "self-limiting".

Associated with a higher incidence of incontinence, pelvic prolapse, anovaginal fistula.

Mediolateral episiotomy:

— Cut the perineal body.

— Becoming more common.

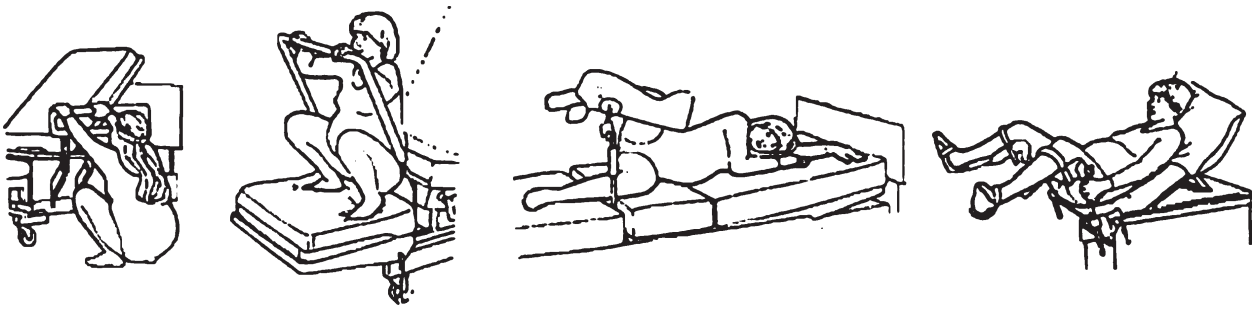


Fig. 12.36

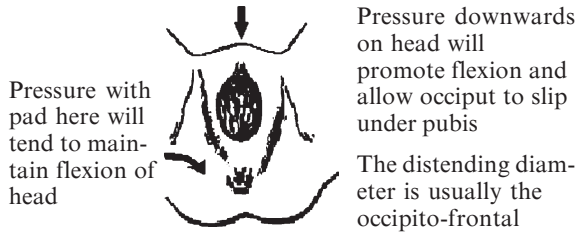


Fig. 12.37

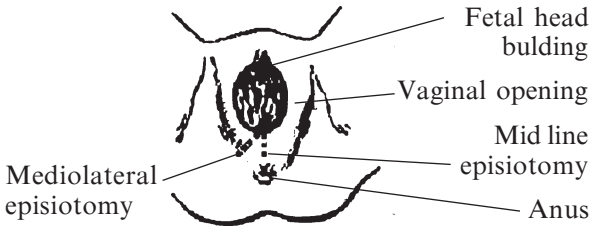


Fig. 12.38

— Appears to ameliorate some of the concerns of median episiotomy.

Episiotomy is no longer recommended as a routine procedure. There is no evidence that routine episiotomy decreases perineal damage, future vaginal prolapse or urinary incontinence. In fact, routine episiotomy is associated with an increase of third and fourth degree tears and subsequent anal sphincter muscle dysfunction.

General principles for procedure (Fig. 12.39):

— Gather and prepare all supplies.

— Explain the procedure and the need for it to the woman and obtain consent.

— Place the patient in a position appropriate for the procedure being performed. The most common position used for obstetric procedures (e.g. manual vacuum aspiration) is the *lithotomy position* (Fig. 12.40).

— Apply antiseptic solution to the perineal area.

— Make sure there are no known allergies to lidocaine or related drugs.

— Provide adequate pain medication according to the extent of the procedure planned.

Anaesthetize early to provide sufficient time for effect.

Use a pudendal block or local infiltration with lidocaine (infiltrate beneath the vaginal mucosa, beneath the skin of the perineum and deeply into the perineal muscle using about 10 mL 0.5% lignocaine solution) (Fig. 12.41).

Control the baby's head and shoulders as they deliver, ensuring that the shoulders have rotated to the midline to prevent an extension of the episiotomy.

Making the incision while inserting two fingers to protect the baby's head.

Episiotomy should be considered only in the case of:

— complicated vaginal delivery (breech, shoulder dystocia, forceps, vacuum extraction);

— scarring from female genital cutting or poorly healed third or fourth degree tears;

— fetal distress.

• To control the birth of the head, place the fingers of one hand against the baby's head to keep it flexed (bent) (Fig. 12.42).

• Continue to gently support the perineum as the baby's head delivers.

• Once the baby's head is delivered, ask the woman not to push.

• Suction the baby's mouth and nose.

• Feel around the baby's neck for the umbilical cord (Fig. 12.43):

— if the cord is around the neck but is loose, slip it over the baby's head;

— if the cord is tight around the neck, double clamp and cut it before unwinding it from around the neck.

• Allow the baby's head to turn spontaneously (Fig. 12.44).

• After the head turns, place a hand on each side of the baby's head. Tell the woman to push gently with the next contraction.

• Reduce tears by delivering one shoulder at a time. Move the baby's head posteriorly to deliver the shoulder that is anterior.

Note: If there is difficulty delivering the shoulders, suspect shoulder dystocia.

• Lift the baby's head anteriorly to deliver the shoulder that is posterior (Fig. 12.45).

• Support the rest of the baby's body with one hand as it slides out.

• Place the baby on the mother's abdomen. Thoroughly dry the baby, wipe the eyes and assess the baby's breathing:

Note: Most babies begin crying or breathing spontaneously within 30 seconds of birth:

— if the baby is crying or breathing (chest rising at least 30 times per minute) leave the baby with the mother;

— if baby does not start breathing within 30 seconds, take steps to resuscitate the baby.

— Clamp and cut the umbilical cord within one minute of delivery of the baby (Fig. 12.46, *a, b*).

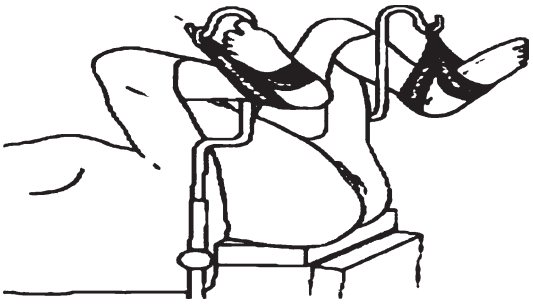


Fig. 12.39

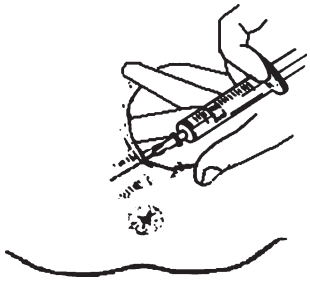


Fig. 12.40

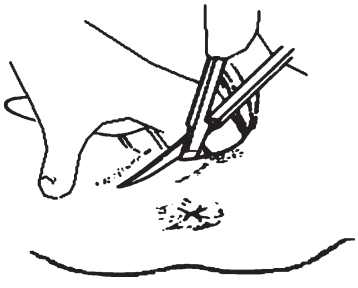


Fig. 12.41

Fig. 12.42

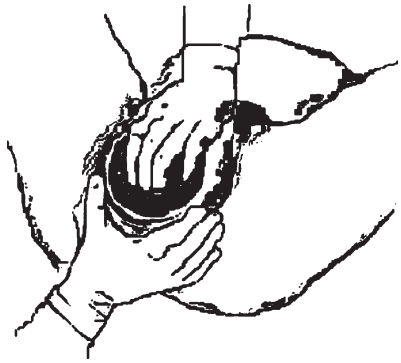


Fig. 12.43

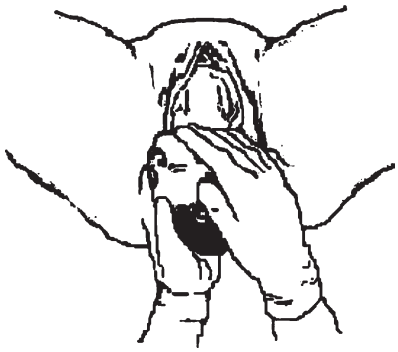


Fig. 12.44

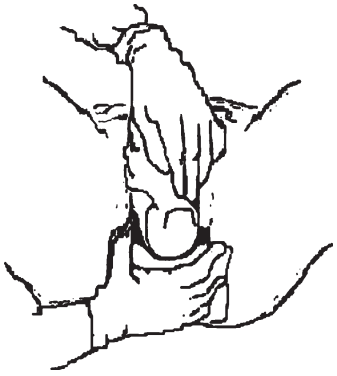
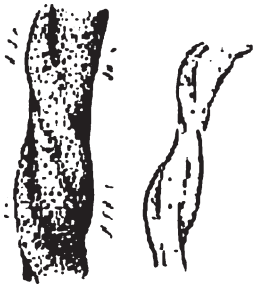
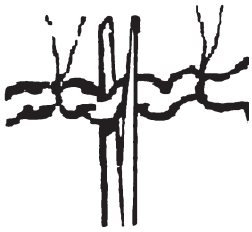


Fig. 12.45

a





b

Fig. 12.46

— Ensure that the baby is kept warm and in skin-to-skin contact on the mother's chest. Wrap the baby in a soft, dry cloth, cover with a blanket and ensure the head is covered to prevent heat loss (Fig. 12.47).

Palpate the abdomen to rule out the presence of an additional baby(s) and proceed with active management of the third stage [34, 45, 49].

Fig. 12.47

THE THIRD STAGE OF LABOR

The time from the birth of the baby to the expulsion of the placenta and its membrane.

Three hormones play important roles in the third stage. During this stage the woman experiences peak levels of *oxytocin* and *endorphins*, while the high *adrenaline* levels that occurred during the second stage of labor to aid with pushing begin falling. The hormone oxytocin causes uterine contractions and helps the woman to enact instinctive mothering behavior such as holding the newborn close to her body and cuddling the baby.

Skin-to-skin contact immediately after birth and the newborn's first attempt at breastfeeding further augment maternal oxytocin levels, strengthening the uterine contractions that will help the placenta to separate and the uterus to contract to prevent hemorrhage. Endorphins, the body's natural opiates, produce an altered state of consciousness and aid in blocking out pain. In addition, the drop in adrenaline level from the second stage, which had kept the mother and baby alert at first contact, causes most women to experience feelings of cold and shivering shortly after giving birth.

A crucial role for nurses during this time is to protect the natural hormonal process by ensuring unhurried and uninterrupted contact between mother and newborn after birth, providing warmed blankets to prevent shivering, and allowing skin-to-skin contact and breastfeeding.

— Duration is 5–20 minutes.

— 30 minutes have been suggested if there is no evidence of significant bleeding.

— The risk of complications continues for some period after delivery of the placenta.

During the third stage of labor, strong uterine contractions continue at regular intervals under the continuing influence of oxytocin. The uterine muscle fibers shorten, or retract, with each contrac-

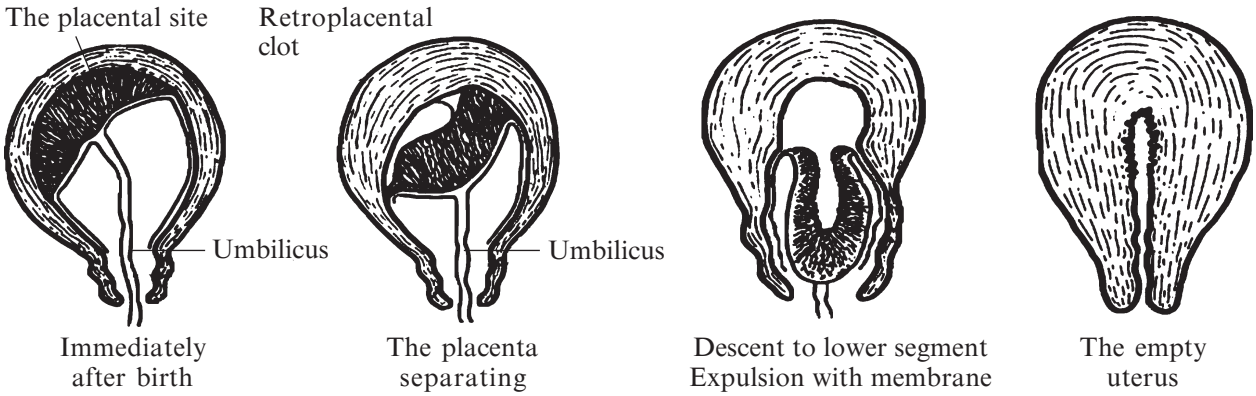


Fig. 12.48

tion, leading to a gradual decrease in the size of the uterus, which helps shear the placenta away from its attachment site. The third stage is complete when the placenta is delivered (Fig. 12.48) [45, 49, 50].

Placental separation methods:

There are two ways of separation.

— *Central (Schultze):* detachment starts from the centre, uterine sinuses are opened, retro placental collection of blood occurs resulting in further separation.

— *Marginal (Duncan):* separation starts at the margin, more area get separated with progressive uterine contractions. This occurs more frequently (Fig. 12.49).

The placenta now descends into the lower segment and gives it form. The fundus thus rises above the umbilicus, is hard and, no longer containing the placenta, is narrower and often displaced laterally (Fig. 12.50).

The placenta is then expelled or removed, and as the lower segment is again empty it collapses and the fundus is now narrow, hard and found below the umbilicus.

Two methods of the third stage management:

• Physiologic ("expectant") management.

Oxytocin is not used. Placenta is delivered by gravity and maternal efforts spontaneously.

Signs of placental separation:

— fresh blood show from vagina;

— the umbilical cord lengthens outside the vagina;

— the fundus of the uterus rises up;

— the fundus becomes firm and globular.

• Active management.

Traditionally it was considered prudent to await signs of placental separation then expelling it by pressure on the fundus which is associated with risk of postpartum hemorrhage.

Modern management is the active management of the third stage which involves a procedure called

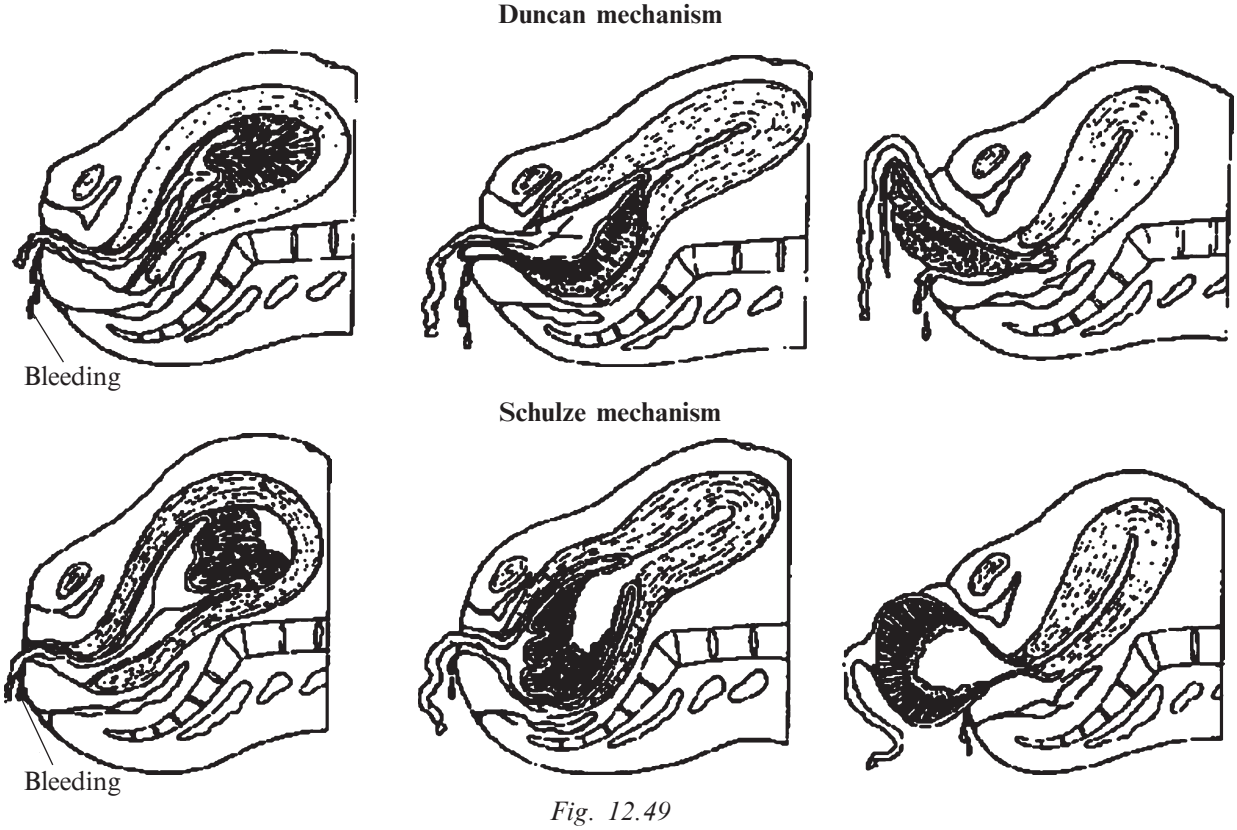


Fig. 12.49

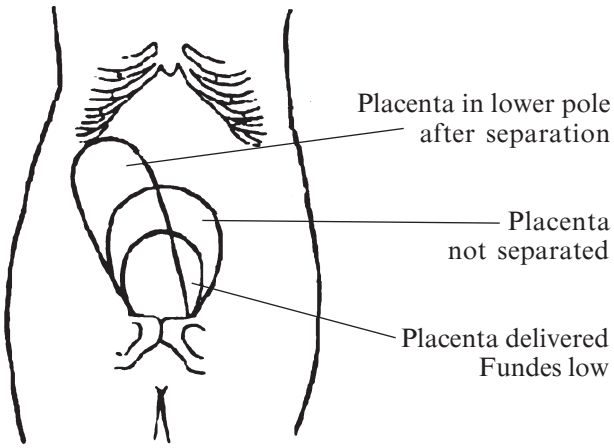


Fig. 12.50

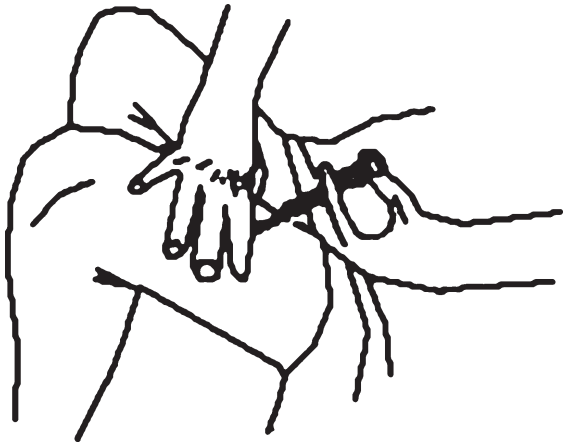


Fig. 12.51

controlled cord traction (Brandt — Andrews' method).

ACTIVE MANAGEMENT OF THE THIRD STAGE

Active management of the third stage (active delivery of the placenta) helps prevent postpartum haemorrhage.

Active management of the third stage of labor includes:

— immediate oxytocin;
— controlled cord traction; and
— uterine massage.

Anticipate the need for resuscitation and have a plan to get assistance for every baby but especially if the mother has a history of eclampsia, bleeding, prolonged or obstructed labor, preterm birth or infection.

Oxytocin

— Within one minute of delivery of the baby, palpate the abdomen to rule out the presence of an additional baby(s) and give oxytocin 10 units IM (Fig. 12.51).

— Oxytocin is preferred because it is effective two to three minutes after injection, has minimal side effects and can be used in all women. If oxytocin is not available, ergometrine 0.2 mg i/m or prostaglandins are used.

Note: Do not give ergometrine to women with preeclampsia, eclampsia or high blood pressure because it increases the risk of convulsions and cerebrovascular accidents.

Controlled cord traction

• Clamp the cord close to the perineum using sponge forceps within one minute of delivery. Hold the clamped cord and the end of forceps with one hand (Fig. 12.52).

• Place the other hand just above the woman's pubic bone and stabilize the uterus by applying counter traction during controlled cord traction. This helps prevent inversion of the uterus.

• Keep slight tension on the cord and await a strong uterine contraction (two to three minutes).

• When the uterus becomes rounded or the cord lengthens, very gently pull downward on the cord to deliver the placenta. Do not wait for a gush of blood before applying traction on the cord. Continue to apply counter traction to the uterus with the other hand (Fig. 12.53).

• If the placenta does not descend during 30 to 40 seconds of controlled cord traction (i.e. there are no signs of placental separation), do not continue to pull on the cord:

— gently hold the cord and wait until the uterus is well contracted again. If necessary, use a sponge forceps to clamp the cord closer to the perineum as it lengthens (Fig. 12.54, *a*);

— with the next contraction, repeat controlled cord traction with counter traction.

• As the placenta is delivered, the thin membranes can tear off. Hold the placenta in two hands and gently turn it until the membranes are twisted.

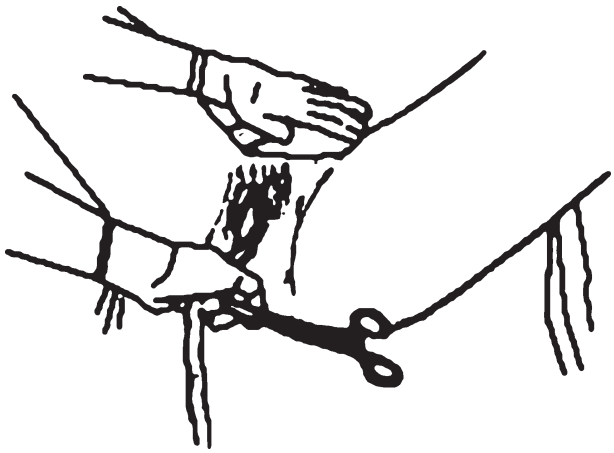


Fig. 12.52

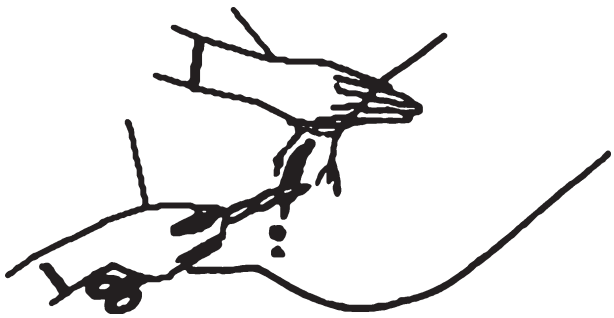


Fig. 12.53

a *b*

Fig. 12.54

• Slowly pull to complete the delivery.

• If the membranes tear, gently examine the upper vagina and cervix wearing high-level disinfected or sterile gloves and use a sponge forceps to remove any pieces of membrane that are present.

• Look carefully at the placenta to be sure none of it is missing. If a portion of the maternal surface is missing or there are torn membranes with vessels, suspect retained placental fragments.

• If uterine inversion occurs, reposition the uterus.

• If the cord is pulled off, manual removal of the placenta may be necessary.

Uterine massage

• Immediately massage the fundus of the uterus through the woman's abdomen until the uterus is contracted (Fig. 12.54, *b*).

• Repeat uterine massage every 15 minutes for the first two hours.

• Ensure that the uterus does not become relaxed (soft) after you stop uterine massage.

The blood loss should be estimated.

Bleeding in the delivery, composed till 0,5% of the puerpera's body weights is physiological (if more than 0.5% of the puerpera's body weights is pathological bleeding).

The uterus can only retract property if it is empty. Sooner or later, retained debris will lead to haemorrhage or infection.

Examination of the placenta

• A one-minute examination of the placenta performed in the delivery room provides information that may be important to care of both mother and infant.

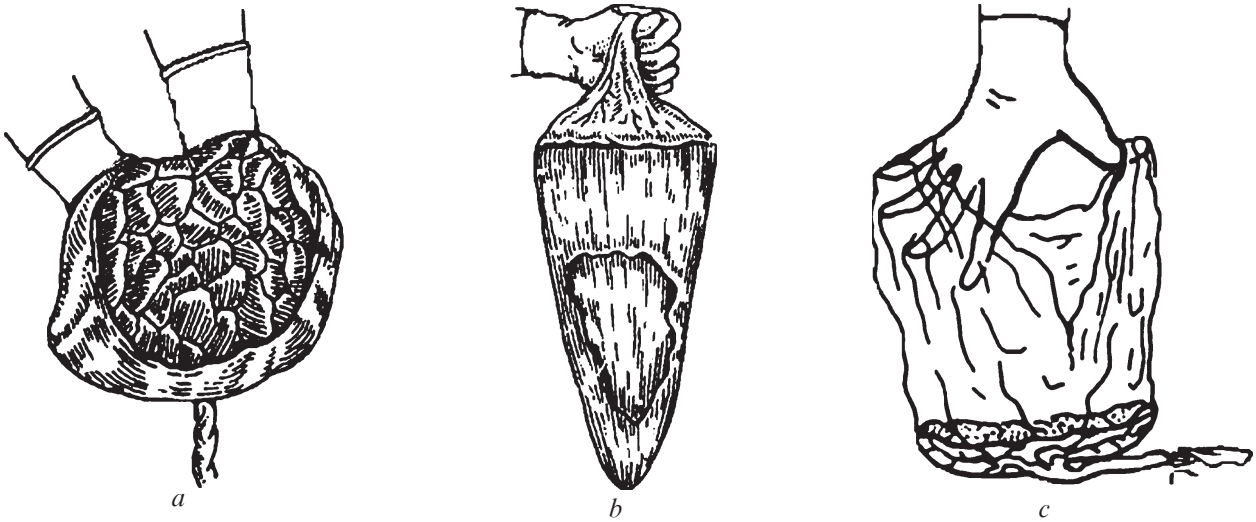
• The findings of this assessment should be documented in the delivery records (Fig. 12.55).

• During the examination, the size, shape, consistency and completeness of the placenta should be determined, and the presence of accessory lobes, hemorrhage, tumors and nodules should be noted.

Various abnormalities of placental development are seen and may have clinical significance.

Bipartite placenta: two complete and separate lobes, each with a cord leaving it (Fig. 12.56).

Succenturiate lobe of placenta: small extra lobe, separate from the main part and joined by membrane which harbors blood vessels. It has a risk of being retained post delivery with further complications of hemorrhage and infection. Upon examination, the placenta looks torn or the blood vessels run beyond the edge of the placenta (Fig. 12.57).



a *b* *c*

Fig. 12.55

Fig. 12.56

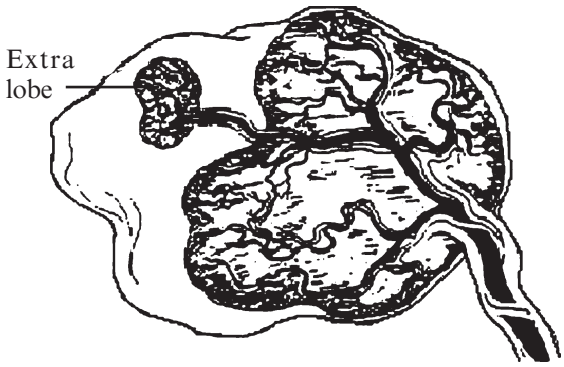


Fig. 12.57

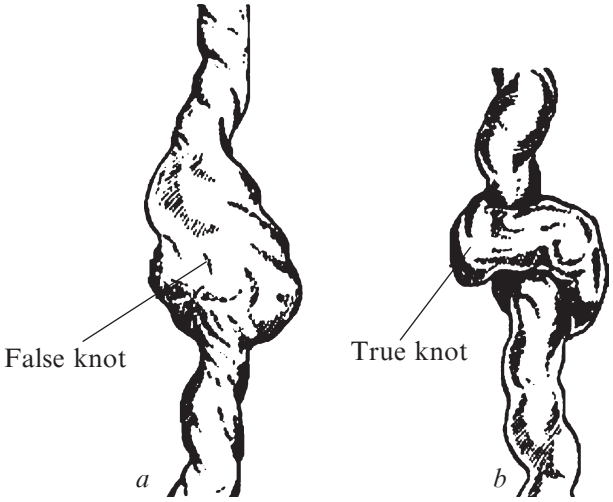


Fig. 12.58

Abnormalities of the umbilical cord

Very short cord — 20–30 cm. It brings problems during delivery.

Very long cord — 80–90 cm. It tends to entwine around the neck or extremity of the fetus.

True knots — occur in about 1% of pregnancies, they form during labor as a result of the fetus passing through a loop of the cord. It causes fetal anoxia (Fig. 12.58).

False knot — localized collection of Wharton's jelly containing a loop of umbilical vessels.

Perineal lacerations or tears can occur during the second stage when the fetal head emerges through the vaginal introitus.

The vagina, labia and perineum are inspected for tears or other injuries.

REPAIR OF EPISIOTOMY

The incision is generally repaired after delivery of the placenta is completed.

It is important that absorbable sutures be used for closure. Polyglycolic sutures are preferred over chromic catgut for their tensile strength, non-allergenic properties and lower probability of infectious complications and episiotomy breakdown. Chromic catgut is an acceptable alternative, but is not ideal (Fig. 12.59, *a*).

Step 1 — *suturing the vaginal wall*:

— identify the apex of the vaginal trauma;

— insert the first stitch 5 mm to 10 mm above the apex to secure any bleeding points that may not be visible;

— using a surgeon's square knot, secure the first stitch (cut off the short end of the suture material, leaving about 1 cm to 2 cm);

— sutures should be placed approximately 5 mm to 10 mm from wound edges;

— each stitch should reach the trough of the wound to close any dead space;

— match each stitch on either side of the wound for depth as well as width;

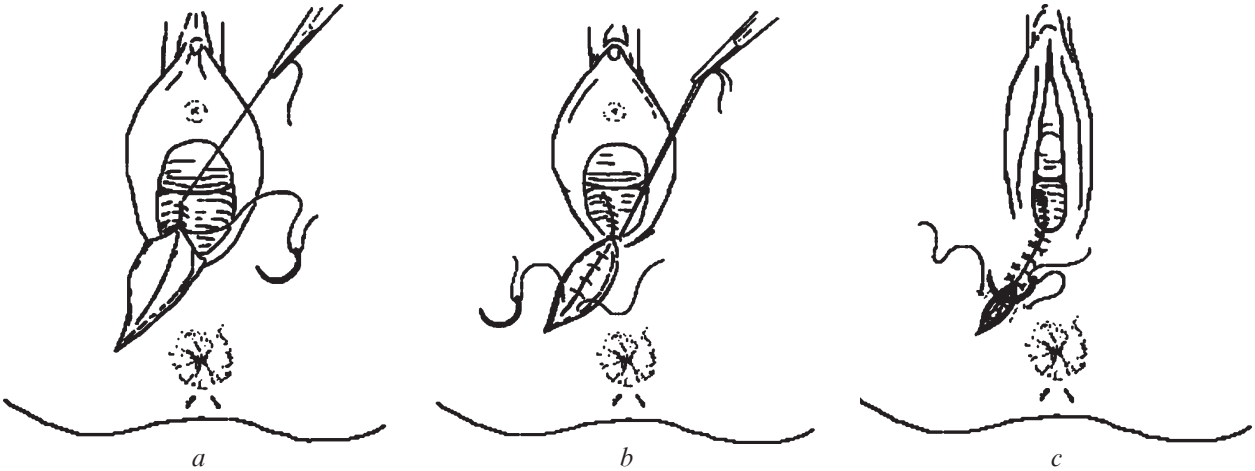


Fig. 12.59

— suture the posterior vaginal trauma using a loose continuous non-locking stitch (usually about three to four stitches) until hymenal remnants are reached (Fig. 12.59, *b*);

— insert one more suture to close the hymenal ring.

Step 2 — *suturing the perineal muscle layer*:

— insert the needle near to the hymenal ring to emerge deep in the centre of the muscle layer;

— check the depth of the trauma;

— using a continuous non-locking suture;

— close the perineal muscles in one layer, or if the trauma is very deep use two layers, ending with the needle at the inferior aspect of the trauma.

Step 3 — *suturing the skin layer* (Fig. 12.59, *c*):

— each stitch should be placed opposite each other, not pulled too tight and approximately 5 to 10 mm apart.

Once suturing is complete, the doctor should inspect the repaired perineal trauma to ensure it has been anatomically aligned correctly and that haemostasis has been achieved. A vaginal examination should be performed ensuring that two fingers can easily be inserted into the vagina. Next, a rectal examination should be carried out (with consent) to confirm that no sutures have penetrated the rectal mucosa.

Complication of the third stage:

— Post partum haemorrhage.

— Hematoma formation.

— Cervical, vaginal and perineal tears.

— Retained placenta.

— Inversion of uterus.

— Shock.

Chapter 13
ABNORMALITIES OF THE THIRD STAGE OF LABOR

The third stage of labor, from the delivery of the child until the expulsion of the placenta, remains the most unpredictable and dangerous stage of labor from the mother's point of view. The incidence of postpartum haemorrhage is approximately 7%.

POSTPARTUM HEMORRHAGE

Postpartum hemorrhage (PPH) is defined as blood loss in excess of physiologic blood loss at the time of vaginal delivery — 0.5% from a woman's body weight. Hemorrhage may occur before, during, or after delivery of the placenta. Actual measured blood loss during uncomplicated vaginal deliveries averages 700 mL, and blood loss often may be underestimated. Nevertheless, the criterion of a 500-mL loss is acceptable on historical grounds.

Most severe haemorrhages occur within the first two hours of delivery. Blood lost during the first 24 hours after delivery is *early postpartum hemorrhage*; blood lost between 24 hours and 6 weeks after delivery is *late postpartum hemorrhage*. Most cases (99%) of postpartum hemorrhage are primary [18, 40, 49] (Fig. 13.1; 13.2; Tables 13, 14).

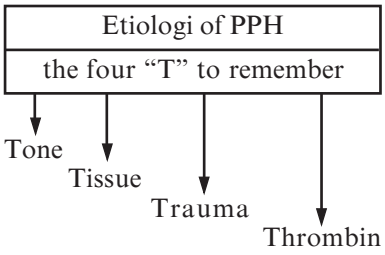


Fig. 13.1

Causes of postpartum hemorrhage
- Uterine atony
- Retained placental tissue
- Genital tract trauma (cervical, vaginal laceration)
- Low placental implantation
- Uterine inversion
- Coagulation disorders
- Amniotic fluid embolism
- Retained dead fetus
- Inherited coagulopathy
- Abruptio placentae (usually ante- or intrapartum)

Diagnosis
- Vaginal bleeding may be revealed or concealed
- Alteration in pulse, blood pressure and pulse pressure
- Flabby uterus in atonic uterus

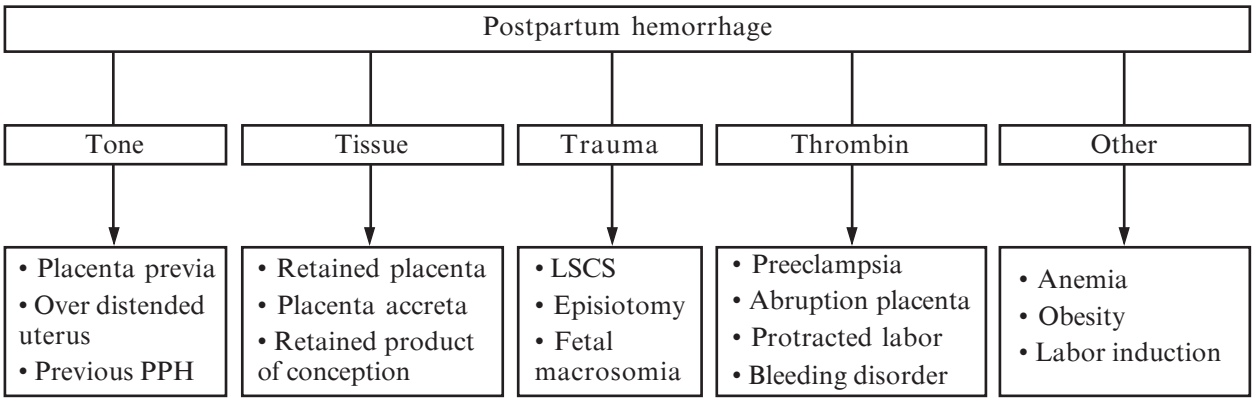


Fig. 13.2

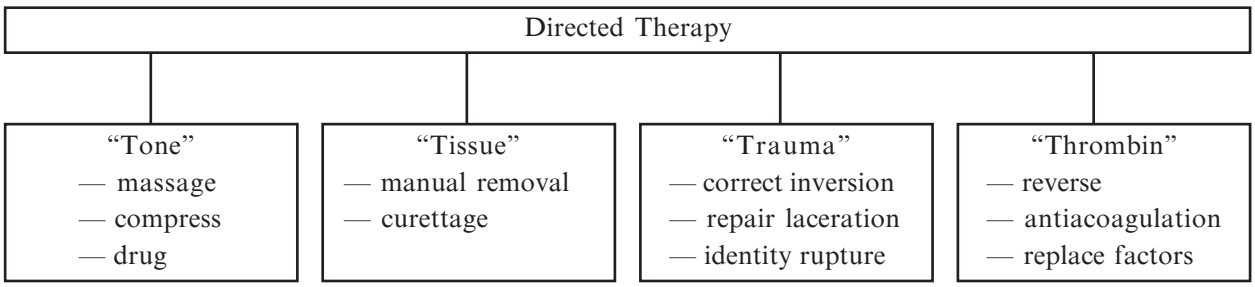


Fig. 13.3. Management of PPH

Table 13

Clinical findings in PPH

Degree of Shock				
Parameter	Compensation	Mild	Moderate	Severe
Blood loss	500–1000 ml 10–15%	1000–1500 ml 15–25%	1500–2000 ml 25–35%	2000–3000 ml 35–45%
Blood pressure change (systolic pressure)	none	slight fall (80–100 mmHg)	marked fall (70–80 mmHg)	profound fall (50–70 mmHg)
Symptoms and signs	palpitations dizziness tachycardia	weakness sweating tachycardia	restlessness pallor oliguria	collapse air hunger anuria

Table 14

Postpartum obstetric hemorrhage

Stage 0 All women in labor or giving birth	Assess for Risk Factors 1. Active management: IV infusion of oxytocin after delivery of the fetus and fundal uterine massage after delivery of the placenta. 2. Complete evaluation for missing placental cotyledons and examination of vagina and cervix for lacerations with repair when needed to control bleeding. 3. If high risk (history of postpartum hemorrhage plus 1 or more risk factors), consider typing and crossmatching 2 U of PRBCs
Stage 1 Blood loss > 500 mL (vaginal delivery) > 1000 mL (cesarean delivery)	Begin Hemorrhage Protocol 1. Alert charge nurse and anesthesia staff. 2. Type and crossmatch 2 U of PRBCs (if not already done). 3. Increase infusion rate of oxytocin, give methergine and repeat fundal massage. 4. Measure blood loss
Stage 2 Total blood loss between 1000 and 1500 mL	Call for Help (Rapid Response Team) 1. Consider complete reexamination of vagina, cervix, and uterine cavity for source of bleeding; if the patient is in the postpartum unit, consider moving her to labor and delivery or the operating room. 2. Consider the next level of drugs (carboprost 250 μg IM or misoprostol 800 to 1000 μg per rectum) and request additional laboratory testing (e.g., a coagulation panel). 3. Consider blood transfusion (have 2 U of PRBCs plus 2 U of fresh frozen plasma at the bedside). 4. Consider placement of intrauterine balloon or involve interventional radiology when available for embolization
Stage 3	Mobilize Surgical Team 1. Consider repeat laboratory tests, including coagulation studies and acid-base gas assessment. 2. Transfuse appropriately with PRBCs and platelets. 3. Consider B-Lynch suture, uterine artery ligation, or hysterectomy

UTERINE ATONY

Postpartum bleeding is physiologically controlled by constriction of interlacing myometrial fibers that surround the blood vessels supplying the placental implantation site. Uterine atony exists when the myometrium cannot contract.

The majority of PPH cases (75–80%) are due to uterine atony.

Factors predisposing to postpartum uterine atony

- History of postpartum hemorrhage,
- Prolonged labor,
- Grand multiparity (a parity of 5 or more),
- Overdistention of the uterus,
  - Multiple gestations
  - Polyhydramnios
  - Fetal macrosomia
- Oxytocic augmentation of labor,
- Precipitous labor (one lasting < 3 hr),
- Magnesium sulphate treatment of preeclampsia,
- Chorioamnionitis,
- Genetic and epigenetic factors (maternal, environmental, and fetal).

Prevention of uterine atony by considering the following steps:

1. Active management of the third stage of labor reduces blood loss by 50% — although it leads to a slightly increased risk of retained placenta.
2. The placenta should be carefully assessed at delivery to make certain there are no missing cotyledons (lobules of placenta).
3. In some high-risk conditions an intravenous infusion of oxytocin is used in addition to the bolus dose. High-risk conditions would include prolonged labor, known placenta previa, polyhydramnios, twin pregnancy, high parity, uterine fibroids, abruptio placentae or previous PPH.
4. The vagina and perineum should be inspected to rule out any lacerations that could cause excessive bleeding.
5. The uterus should be evaluated by abdominal palpation during the first 1 to 2 hours before transfer to the postpartum unit. The nurses on the postpartum unit should frequently assess the status of uterine contractility, instructing the patient on how to assess uterine firmness and reporting any excessive bleeding. For high-risk patients, continuation of the oxytocin infusion during the early postpartum hours should be considered.

General management

Treatment is always the same, and performed immediately to avoid massive haemorrhage.

- Ask for help. Urgently mobilize all available personnel.
- Perform a rapid evaluation of the general condition of the woman, including vital signs (pulse, blood pressure, respiration, temperature) (Fig. 13.4).

If shock is suspected, immediately begin treatment. Even if signs of shock are not present, keep shock in mind as you evaluate the woman further because her status may worsen rapidly. If shock develops, it is important to begin treatment immediately.

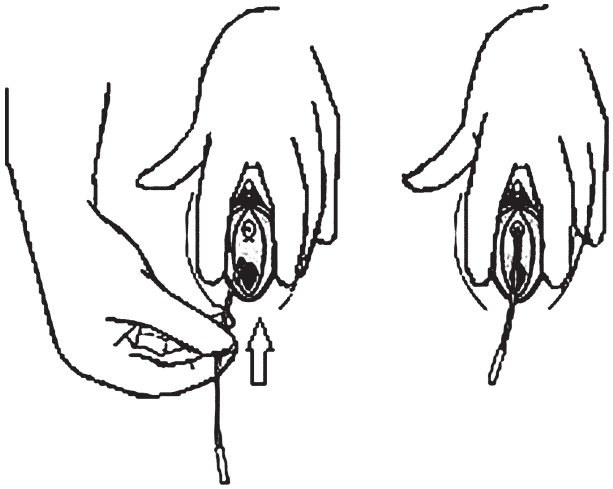


Fig. 13.4

- Give oxytocin: 5 to 10 IU.
- Catheterize the bladder: keeping bladder empty facilitates uterine retraction.
- Massage the uterus to expel blood and blood clots. Blood clots trapped in the uterus will inhibit effective uterine contractions (Fig. 13.5).
- Start an IV infusion and infuse IV fluids (catheter 16–18 G), rapid fluid resuscitation.
- Combine with uterine massage; maintain bimanual compression if bleeding is severe.

If the effect within 15 minutes (Fig. 13.6) misoprostol sublingually (800 μg) and/or methylergometrine IM: 0.2 mg.

Fig. 13.5

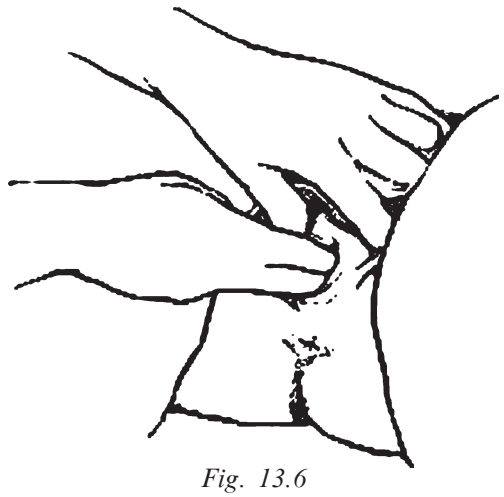


Fig. 13.6

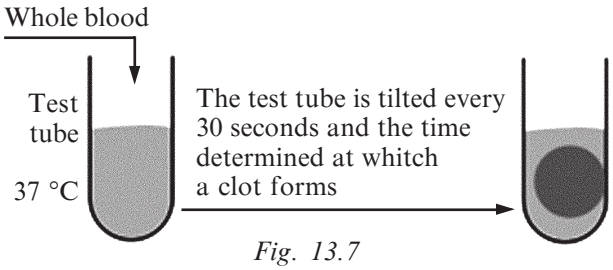


Fig. 13.7

• Check to see if the placenta has been expelled, and examine the placenta to be certain it is complete.

— If there are signs of retained placental fragments (absence of a portion of maternal surface or torn membranes with vessels), remove remaining placental tissue.

— Assess clotting status using a bedside *clotting test.*

Bedside clotting test:

— Take 2 mL of venous blood into a small, dry, clean, plain glass test tube (approximately 10 mm x x 75 mm) (Fig. 13.7);

— Hold the tube in a closed fist to keep it warm (±37°C);

— After four minutes, tip the tube slowly to see if a clot is forming. Then tip it again every minute until the blood clots and the tube can be turned upside down;

— Failure of a clot to form after seven minutes or a soft clot that breaks down easily suggests coagulopathy.

• Examine the cervix, vagina and perineum for tears.

• Check for anaemia after bleeding has been stopped for 24 hours.

• *If bleeding continues in spite of management above* (Fig. 13.8):

— Perform bimanual compression of the uterus:

— Wearing high-level disinfected or sterile gloves, insert a hand into the vagina and remove any blood clots from the lower part of the uterus or cervix;

— Form a fist;

— Place the fist into the anterior fornix and apply pressure against the anterior wall of the uterus;

— With the other hand, press deeply into the abdomen behind the uterus, applying pressure against the posterior wall of the uterus;

— Maintain compression until bleeding is controlled and the uterus contracts.

Operative Management

Alternatively, compress the aorta:

— Apply downward pressure with a closed fist over the abdominal aorta directly through the abdominal wall:

— the point of compression is just above the umbilicus and slightly to the left;

— aortic pulsations can be felt easily through the anterior abdominal wall in the immediate postpartum period (Fig. 13.9).

— With the other hand, palpate the femoral pulse to check the adequacy of compression:

— if the pulse is palpable during compression, the pressure exerted by the fist is inadequate;

— if the femoral pulse is not palpable, the pressure exerted is adequate;

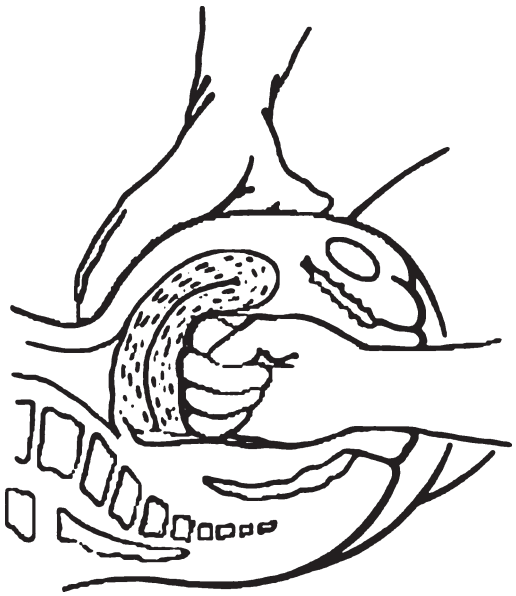


Fig. 13.8

— maintain compression until bleeding is controlled.

• If bleeding continues in spite of compression:

— perform uterine and utero-ovarian artery ligation;

— if life-threatening bleeding continues after ligation, perform a subtotal hysterectomy.

Uterine Artery Ligation

During pregnancy, 90% of the blood flow to the uterus is supplied by the uterine arteries. Direct ligation of these easily accessible vessels can successful-

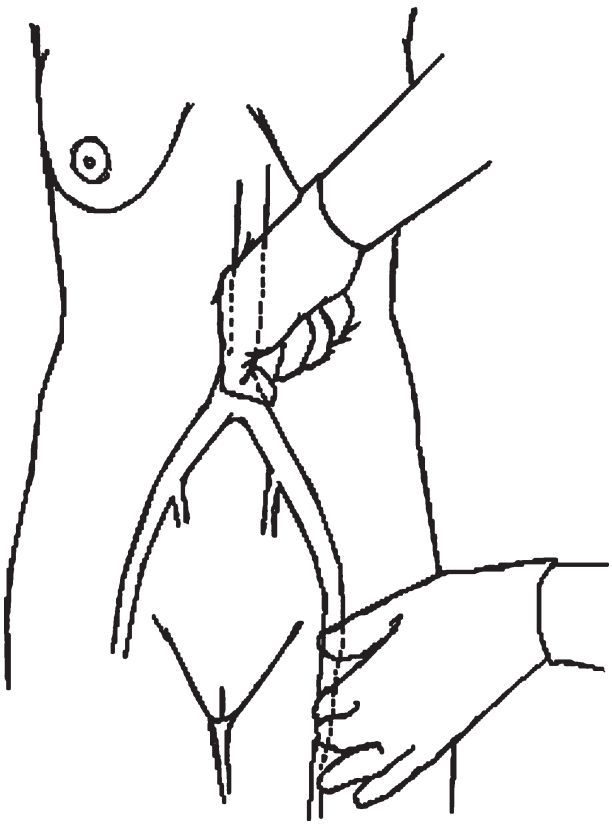


Fig. 13.9

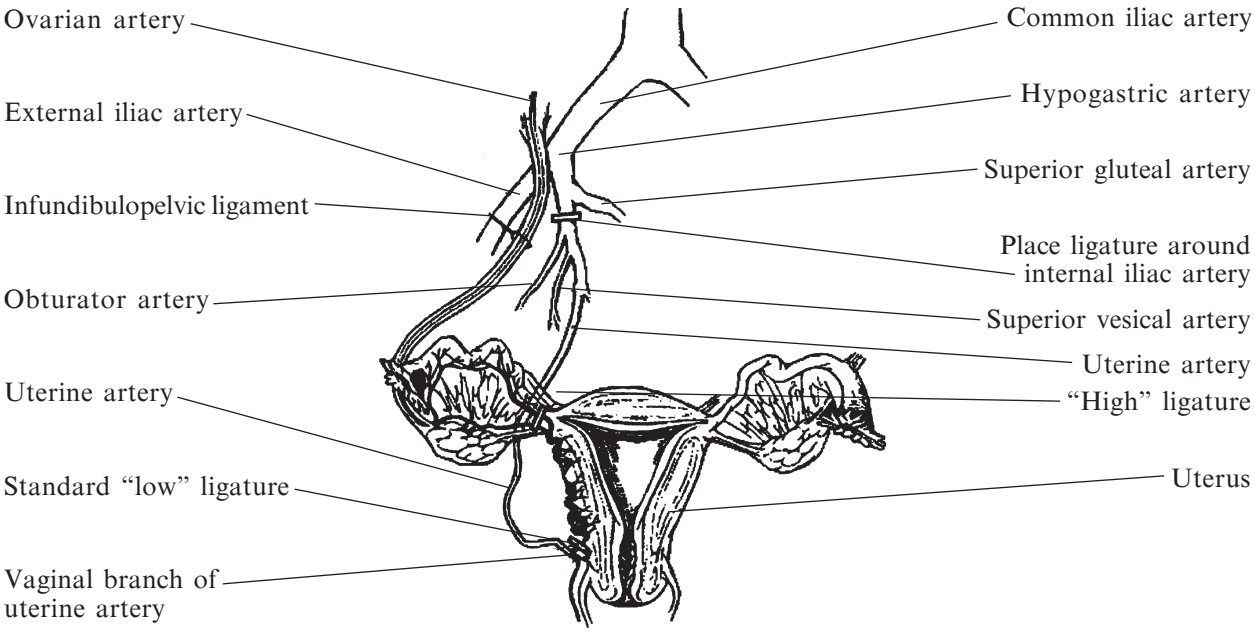


Fig. 13.10

ly control hemorrhage in 75–90% of cases, particularly when the bleeding is uterine in origin (Fig. 13.10).

Technique:

— The uterus is lifted upward and away from the side to be ligated.

— Absorbable suture on a large needle is placed around the ascending uterine artery and vein on 1 side of the uterus, passing through the myometrium 2–4 cm medial to the vessels and through the avascular area of the broad ligament. The suture includes the myometrium to fix the suture and to avoid tearing the vessels.

— The same procedure is then performed on the opposite side.

— If the ligation is performed during cesarean section, the sutures can be placed just below the uterine incision under the bladder flap. It is not necessary to mobilize the bladder otherwise. Bilateral utero-ovarian artery ligation can also be performed in an attempt to reduce blood flow to the uterus.

— This technique should be performed with absorbable suture near the point of anastomoses between the ovarian artery and the ascending uterine artery at the utero-ovarian ligament.

Internal Iliac Artery Ligation

Bilateral internal iliac (hypogastric) artery ligation is the surgical method most often used to control severe postpartum bleeding. Exposure can be difficult, particularly in the presence of a large boggy uterus or hematoma. Failure rates of this technique can be as high as 57% but may be related to the skill of the operator, the cause of the hemorrhage, and the patient's condition before ligation is attempted (Fig. 13.11) [49, 50].

Technique:

— The peritoneum lateral to the infundibulopelvic ligament is incised parallel with the ligament, or the round ligament is transected. In either case, the peritoneum to which the ureter will adhere is dissected medially, which removes the ureter from the operative field.

— The pararectal space is then enlarged by blunt dissection.

— The internal iliac artery on the lateral side of the space is isolated and doubly ligated (but not cut) with silk ligatures at its origin from the common iliac artery.

— The operator must be careful not to tear the adjacent thin veins. Blood flow distally to the uterus, cervix, and upper vagina is not occluded, but the pulse pressure is sufficiently diminished to allow hemostasis to occur by in situ thrombosis.

— Fertility is preserved, and subsequent pregnancies are not compromised.

B-Lynch Brace Suture

An alternative to the vessel ligation techniques is placement of a brace suture to compress the uterus in cases of diffuse bleeding from atony or percreta.

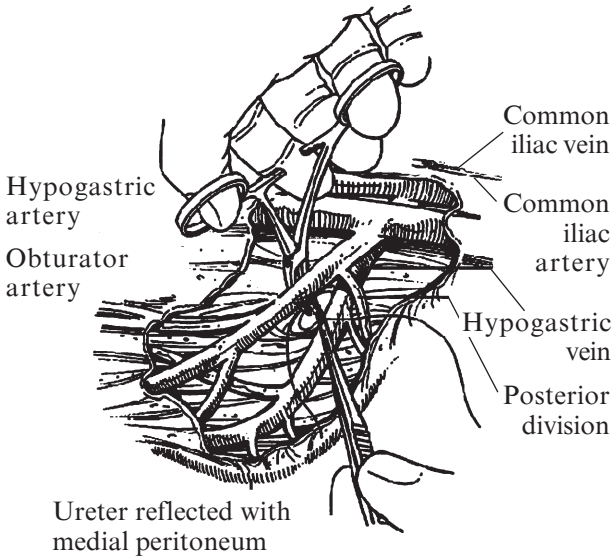


Fig. 13.11

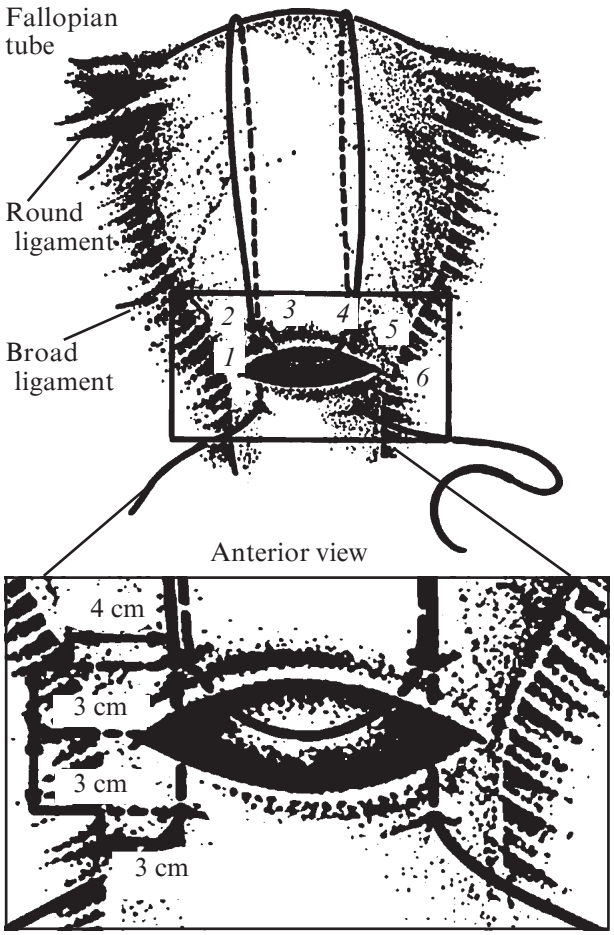


Fig. 13.12

A case series shows success and avoidance of hysterectomy using this novel approach [10, 36, 49] (Fig. 13.12).

Technique:

— Laparotomy is made in the standard way for cesarean section, and a low-transverse uterine incision is made after the bladder is taken down. The uterus is exteriorized.

— Using no. 2 catgut, the uterus is punctured 3 cm from the right lower incision and 3 cm from the right lateral border. The suture is threaded to emerge 3 cm above the upper incision margin and 4 cm from the lateral border.

— The catgut is now visible anteriorly as it is passed over to compress the uterine fundus approximately 34 cm from the right cornual border.

— The suture is fed posteriorly and vertically to enter the posterior wall of the uterine cavity at the same level as the previous entry point.

— After manual compression, the suture is tightened and then passed posteriorly on the left side and passed around the uterine fundus again, this time on the left.

— The suture is brought anteriorly to puncture the uterus at the upper part of the left uterine incision and then reemerge below the lower incision in a symmetric fashion. With 1 operator providing compression, the other throws the knot.

— The hysterotomy is closed in the standard fashion for a cesarean section.

RETAINED PLACENTA

The placenta is usually separated from the uterus during the process of uterine retraction and maternal effort or controlled cord traction used to expel the placenta and membranes from the uterus.

If the placenta does not separate or only partially separates and there is bleeding, removal needs to be facilitated. Controlled cord traction may encourage delivery of the placenta. If this measure fails, manual removal of the placenta will be required.

Causes of retained placenta:

• Uterine atony: full bladder, twins, prolonged labor, precipitate labor.

• Constriction ring: hourglass constriction of uterus, prolonged labor.

Complication of retained placenta:

• Shock.

• Postpartum haemorrhage.

• Puerperal sepsis.

• Subinvolution.

• Retained parts with subsequent haemorrhage, infection, placental polyp formation or choriocarcinoma.

• Complications of the methods used for its separation.

Management

• Immediate manual removal if the placenta has not been yet delivered and / or routine uterine exploration to remove any clots or placental debris and to verify that there was no uterine rupture.

Steps of manual removal of placenta:

— The operation is done under general anesthesia. The patient is placed in a lithotomy position. With all aseptic measures, the bladder is catheterized (Fig. 13.13).

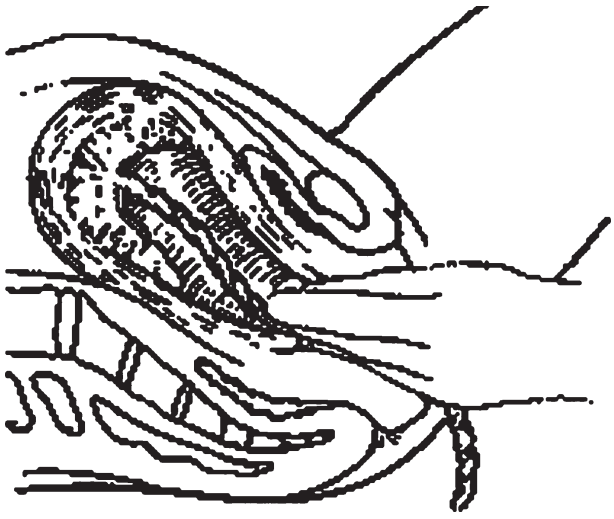


Fig. 13.13

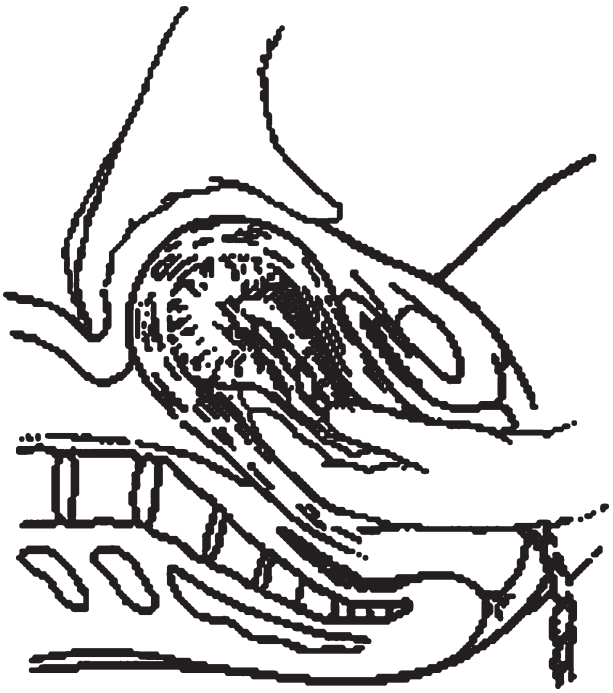


Fig. 13.14

— One hand is introduced into the uterus after smearing with the antiseptic solution in a cone shaped manner following the cord, which is made taut by the other hand. The fingers of the uterine hand should locate the margin of the placenta.

— Counter pressure on the uterine fundus is applied by the other hand placed over the abdomen. The abdominal hand should steady the fundus and guide the movements of the fingers inside the uterine cavity till the placenta is completely separated.

— As soon as the placental margin is reached, the fingers are insinuated between the placenta and the uterine wall with the back of the hand in contact with the uterine wall. The placenta is gradually separated with a sideways slicing movement of the fingers, until the whole of the placenta is separated (Fig. 13.14).

— When the placenta is completely separated, it is extracted by traction of the cord by the other hand. The uterine hand is still inside the uterus for exploration of the cavity to be sure that nothing is left behind.

— The uterine hand is gradually removed while massaging the uterus by the external hand to make it hard. After the completion of manual removal, inspection of the cervicovaginal canal is to be made to exclude any injury.

— The placenta and membranes are inspected for completeness and to be sure that the uterus remains hard and contracted.

• Give routine antibiotic prophylaxis.

• Give oxytocin 5 units in 200 ml fluids (normal saline or Ringer's lactate).

• Ask an assistant to massage the fundus of the uterus to encourage a tonic uterine contraction.

• If there is continued heavy bleeding, give ergometrine 0.2 mg IM or prostaglandins

• Examine the uterine surface of the placenta to ensure that it is complete. If any placental lobe or tissue is missing, explore the uterine cavity to remove it.

• Examine the woman carefully and repair any tears to the cervix or vagina, or repair episiotomy.

RETAINED PLACENTAL FRAGMENTS

When a portion of the placenta — one or more lobes — is retained, it prevents the uterus from contracting effectively.

— Feel inside the uterus for placental fragments. Manual exploration of the uterus is similar to the technique described for removal of the retained placenta.

— Remove placental fragments by hand, ovum forceps or wide curette.

Note: Very adherent tissue may be placenta accreta. Efforts to extract fragments that do not separate easily may result in heavy bleeding or uterine perforation which usually requires hysterectomy.

INJURIES TO THE BIRTH CANAL

Tears of the birth canal are the second most frequent cause of PPH. Tears may coexist with the atonic uterus. Postpartum bleeding with a contracted uterus is usually due to a cervical or vaginal tear.

Examine the woman carefully and repair tears to the cervix or vagina and perineum.

CERVICAL LACERATIONS

• Cervical tears are most presented in the lateral cervical sides.

• Rupture of the cervix is characterized by persistent bleeding of a contracted firm uterus.

Causes of cervical lacerations:

• Precipitate labor

• Application of forceps with the cervix incompletely dilated

• A previous cervical operation

• Rapid delivery of the head in breech presentation

The cervix must be examined and this may be difficult because of the bleeding and friability of the tissues (Fig. 13.15).

There are 3 degrees of cervical lacerations:

• First degree lacerations:

— length of cervical rupture not over 2 cm.

• Second degree lacerations:

— length of rupture > 2 cm but does not extend to vaginal fornices.

• Third degree lacerations:

— ruptured area extends to the vaginal fornices,

If it extends to vaginal fornices: very dangerous.

Treatment of cervical lacerations

— Use vaginal retractors as necessary to expose the cervix (Fig. 13.16).

— Gently grasp the cervix with ring or sponge forceps. Apply the forceps on both sides of the tear and

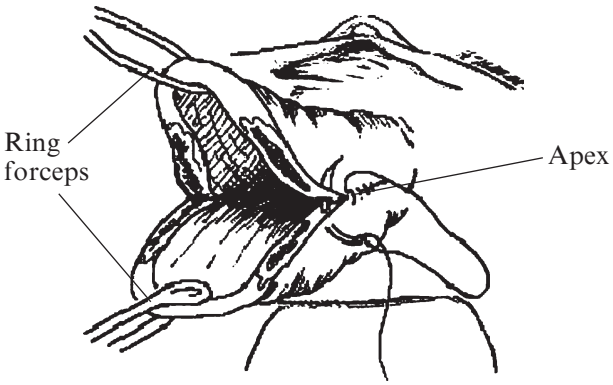


Fig. 13.15

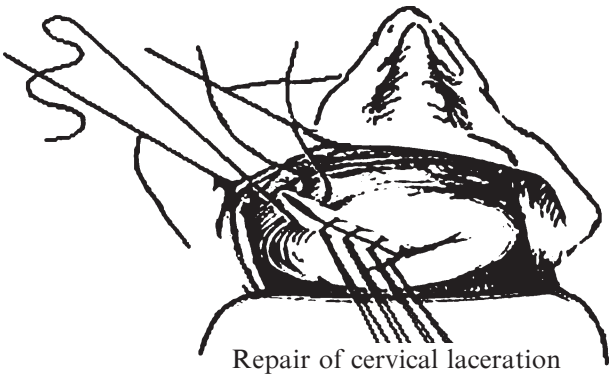
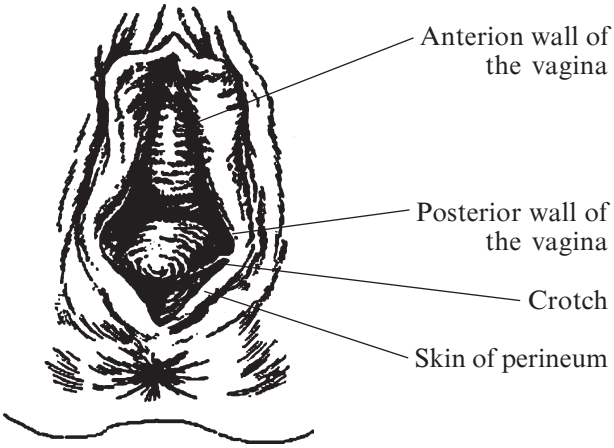
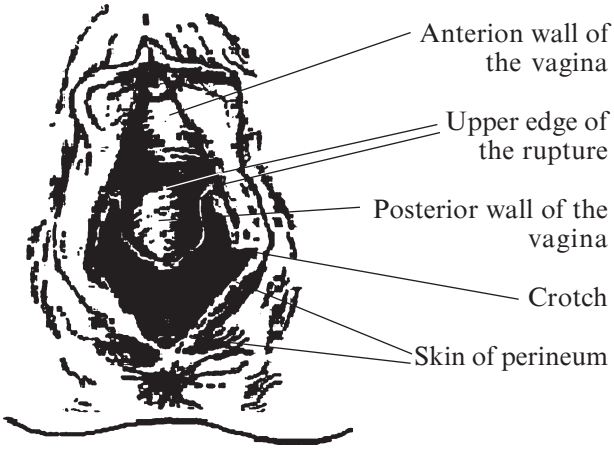


Fig. 13.16



Rupture of perineum of the 1st degree

Fig. 13.17



Rupture of perineum of the 2nd degree

Fig. 13.18

gently pull in various directions to see the entire cervix. There may be several tears.

— Close the cervical tears with continuous 0 chromic catgut suture starting at the apex (upper edge of tear), which is often the source of bleeding.

LACERATION OF THE PERINEIUM AND VAGINA

There are four degrees of tears that can occur during delivery (Fig. 13.17):

• First degree — injury to skin only.

• Second degree — injury to the perineal muscles but not the anal sphincter (Fig. 13.18).

• Third degree — injury to the perineum involving the anal sphincter complex (Fig. 13.19):

— less than 50% of external anal sphincter thickness torn;

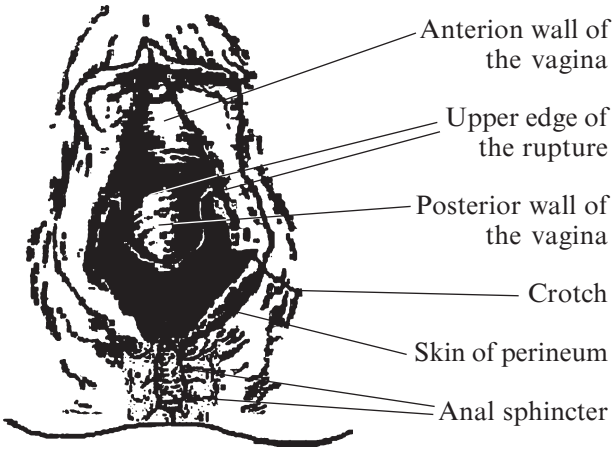
— more than 50% of external anal sphincter thickness torn;

— internal anal sphincter torn.

• Fourth degree — injury to the perineum involving the anal sphincter complex (external and internal anal sphincter) and anal epithelium.

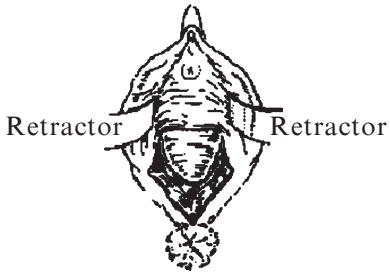
Repair of first and second degree laceration (Fig. 13.20)

• Carefully examine the vagina, perineum and cervix.



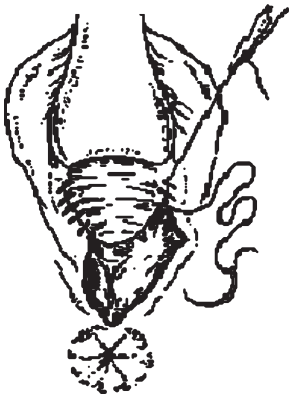
Rupture of perineum of the 3rd degree

Fig. 13.19



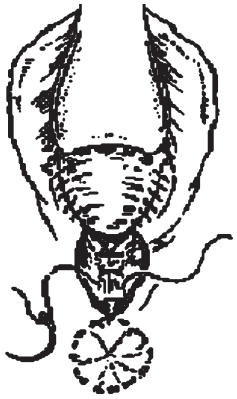
Expressing a perineal tear

Fig. 13.20



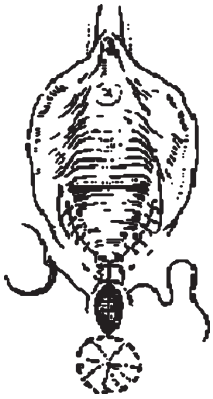
Repairing
the vaginal mucosa

Fig. 13.21



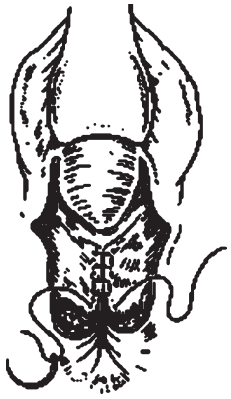
Repairing
the perineal muscles

Fig. 13.22



Repairing
the skin

Fig. 13.23



Closing the muscle wall
of the rectum

Fig. 13.24

• If the tear is long and deep through the perineum, inspect to be sure there is no third or fourth degree tear:

— place a gloved finger in the anus;

— gently lift the finger and identify the sphincter;

— feel for the tone or tightness of the sphincter.

• Change to clean, high-level disinfected or sterile gloves (Fig. 13.21).

• If the sphincter is injured (repair of third and fourth degree tears).

• If the sphincter is not injured, proceed with repair.

• Apply antiseptic solution to the area around the tear.

• Repair the vaginal mucosa using a continuous 2-0 suture (Fig. 13.22):

— start the repair about 1 cm above the apex (top) of the vaginal tear. Continue the suture to the level of the vaginal opening;

— at the opening of the vagina, bring together the cut edges of the vaginal opening;

— bring the needle under the vaginal opening and out through the perineal tear and tie.

• Repair the perineal muscles using interrupted 2–0 suture. If the tear is deep, place a second layer of the same stitch to close the space (Fig. 13.23).

• Repair the skin using interrupted (or subcuticular) 2–0 sutures starting at the vaginal opening.

• If the tear was deep, perform a rectal examination. Make sure no stitches are in the rectum.

Repair of third and fourth degree perineal tears:

The woman may suffer loss of control over bowel movements and gas if a torn anal sphincter is not repaired correctly. If a tear in the rectum is not repaired, the woman can suffer from infection and rectovaginal fistula (passage of stool through the vagina).

• To see if the anal sphincter is torn:

— place a gloved finger in the anus and lift slightly;

— identify the sphincter, or lack of it;

— feel the surface of the rectum and look carefully for a tear (Fig. 13.24).

• Change to clean, high-level disinfected or sterile gloves.

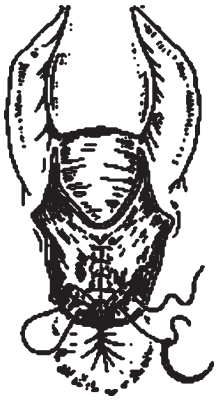
• Apply antiseptic solution to the tear and remove any faecal material, if present.

• Repair the rectum using interrupted 3–0 or 4–0 sutures 0.5 cm apart to bring together the mucosa:

— place the suture through the muscularis (not all the way through the mucosa);

— cover the muscularis layer by bringing together the fascial layer with interrupted sutures (Fig. 13.25);

— apply antiseptic solution to the area frequently.



Suturing
the anal sphincter

Fig. 13.25

• If the sphincter is torn:

— grasp each end of the sphincter with a clamp (the sphincter retracts when torn). The fascial sheath around the sphincter is strong and will not tear when pulling with the clamp;

— repair the sphincter with two or three interrupted stitches of 2–0 suture.

• Apply antiseptic solution to the area again.

• Examine the anus with a gloved finger to ensure the correct repair of the rectum and sphincter. Then change to clean, high-level disinfected or sterile gloves.

• Repair the vaginal mucosa, perineal muscles and skin

PUERPERAL HEMATOMAS

Hematomas — bleeding into loose connective tissue as the vulva or vagina. Vaginal or paravaginal hematomas arise from damage to the descending branch of the uterine artery. Pelvic hematoma blood loss is not always visible but can be diagnosed by vaginal examination. Occasionally, traumatic laceration of blood vessels can lead to pelvic hematoma formation. Puerperal hematomas occur in 1 : 300 to 1 : 1500 deliveries [17, 45].

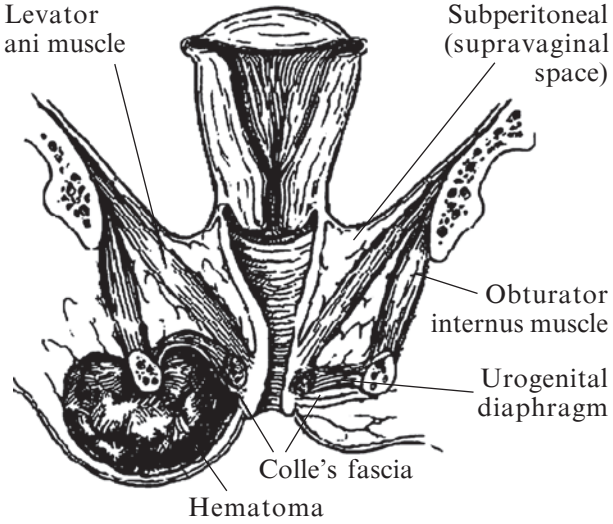
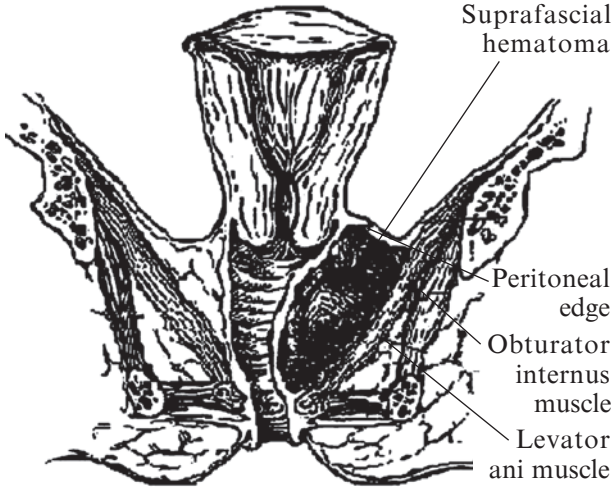


Fig. 13.26



Paravaginal haemotoma: Supralevator

Fig. 13.27

Pelvic hematoma 3 types:
— vulvar;
— vaginal / paravaginal area;
— retroperitoneum.

Risk factors

Women at increased risk of developing puerperal hematomas include those who are nulliparous or who have an infant over 4000 gr, preeclampsia, prolonged second stage of labor, multifetal pregnancy, vulvar varicosities, or clotting disorders.

Assessment: location, size, vital signs.

Genital tract hematomas may not be recognized until hours after the delivery. Sometimes occur in the absence of vaginal or perineal lacerations. The main symptoms are pelvic or rectal pressure and pain.

VULVAL AND PARAVAGINAL HEMATOMAS

Greater than 4 cm in diameter it occurs in 1/1000 deliveries. Injury occurs with episiotomy. In 20% of cases occur with intact perineum. Half of women with genital hematoma have spontaneous delivery (Fig. 13.26, Fig. 13.27).

Divided into two types:

— Infralevator hematoma: includes vulva, perineum, paravaginal, ischiorectal fossa.

— Supralevator hematoma: it spreads upwards and outwards beneath the broad ligament or partly downwards to bulge into the wall of the upper vagina and can track backwards into the retroperitoneal space.

Usual signs: subacute volume loss and vulvar pain.

Management

The management of vulvar hematomas in labor needs to be tailored to the individual patient.

< 3 cm: observation

≥ 3 cm: surgical evacuation with suture closure and dressing compression.

SUBPERITONEAL HEMATOMA

Broad ligament hematoma, less common than genital hematoma. It occurs in 1 in deliveries. They follow spontaneous vaginal or c/s or forceps. 50% discovered immediately, the other half 24 hours later presentation abdominal pain.

Management is conservative. With unstable hemodynamic state the surgical exam may need hysterectomy.

Chapter 14
THE PUERPERIUM

NORMAL PUERPERIUM

Is the period of adjustment following pregnancy and delivery when anatomic and physiologic changes of pregnancy are reversed and the body returns to non-pregnant state.

Physiologic changes of puerperium

Involution is a process by which the reproductive organs return to the pre-gravid state.

The uterus from a size of 20 weeks (at or just below the umbilicus) just after delivery reduces in size at a rate of one finger per day. By the end of the first week it is 12 weeks, by 10–14 days it becomes impalpable per abdomen and reaches non gravid state by 6 weeks. Its weight reduces from 1000 grams at the end of delivery to 50–100 grams by 6 weeks.

In the first 2–3 days after delivery the uterus contracts strongly causing lower abdominal discomfort and pain. This is called the after pain and is commonly seen in multiparas. It is worse after suckling.

The endometrium, besides the placental site, differentiates into superficial and basal layers in 2–3 days. The superficial layer gets necrotic and is cast off as lochia. Regeneration of the basal layer is completed in 10–16 days. The placental site is reduced by 50% following delivery. Regeneration starts by day 7 and is completed between 3–6 weeks.

Lochia is an alkaline discharge of variable amount from the uterus during puerperium. Depending on the color, it is classified as:

• Lochia rubra reddish discharge from day 1–4 which rapidly becomes reddish brown and mainly contains blood.

• Lochia serosa pink colored discharge from day 5–9.

• Lochia Alba thick yellowish whitish discharge starting from the 10th day and extends for an indefinite period. It mainly contains white blood cells and degenerated decidual cells.

The cervix becomes a little more than one centimeter dilated at the end of the first week, and then closes slowly. For those that have delivered vaginally, the external os changes to transverse slit. Complete healing and re epithelization of laceration takes 6–12 weeks. Vagina, perineum and abdominal wall regain their tone but some degree of laxity remains. Mensuration resumes in 6 weeks in 30% and in 12 weeks in 70% of non-lactating women. In lactating women, the range for resumption of menstruation is 2–18 months.

Body weight

On average a woman will lose 6 kg through labor and parturition (water loss and products of conception). Body weight stabilizes by the 10th week post-delivery. A diuresis commences within the first 3 to 4 days postnatally. The haemoglobin level is lowest on day 4 to 5 postdelivery and then rises slowly until 8 weeks postpartum. Changes in platelet levels and other coagulation factors produce a relative hypercoagulability, persisting for approximately 8 weeks.

Endocrine

Serum progesterone and oestradiol fall to non-pregnant levels by 72 hours. Human placental lactogen (HPL) levels fall rapidly in the first 48 hours but are still detectable at the end of the first week. Thyroxine and thyroid-binding globulin fall slowly to normal over 6 weeks. Fasting plasma, insulin and the insulin response curve are normal 2 days postpartum (Fig. 14.1).

Milk

The suckling stimulus releases prolactin and oxytocin — the former stimulates lactogenesis, the latter controls milk ejection. Initially, milk rich in colostrum is released. Milk production commences by day 3.

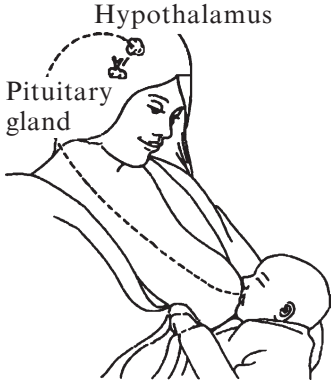


Fig. 14.1

Advantages	Contraindications
• Balanced nutritionally • Passive immunity • Enhanced infections of: — middle ear; — respiratory system; — urinary tract; — gastrointestinal tract. • Reduction in childhood insulin-dependent diabetes mellitus (by 50%) • Reduction problems of prematurity — necrotizing enterocolitins — suboptimal neurological development • Cheap and readily available • Reduced incidence in the mother of premenopausal breast cancer • Mother more likely to lose weight naturally	• Breast implant (breast augmentation) • Previous surgery for breast abscess (relative contraindication) • Maternal phenylketonuria • Drugs taken by mother • Very poor maternal health • Puerperal psychosis • HIV positive status

Fig. 14.2. Advantages and contraindications for breastfeeding

Breastfeeding

Most women have made the decision to breast feed prior to delivery. The correct positioning of the baby on the breast is vital to prevent chewing of the nipples, causing sore or cracked nipples which can predispose to infection and discomfort. Milk production requires a good fluid intake. Many mothers feed their babies 'on demand', others introduce a 3- to 4-hourly feeding regime. The best management is demand feeding. All babies will initially lose weight until lactation is fully established. Human milk delivered at a rate of 750–800 ml a day (in a healthy, well-nourished mother) contains calcium at a concentration of around 34 mg/dl. The loss of calcium from the mother is substantially more during lactation than during pregnancy. Bone density studies indicate a loss of bone mineral density over 6 months, but this is recovered after feeding ceases (Fig. 14.2).

Postpartum contraception

The spacing of pregnancies is essential for the health of the mother and child. Severe anaemia may result if pregnancies follow each other too closely. For breast feeding alone to be effective contraception, lactation must be complete. Progesterone-based contraception does not suppress lactation and may be used by breastfeeding women. For the bottle-feeding mother, the combined oral contraceptive pill is the most effective method of contraception. Hypertension in pregnancy is not a contraindication to the combined oral contraceptive pill as long as the blood pressure has returned to normal. Women who intended to breast feed but stopped will need to be reminded to revise their contraception. The coil is traditionally fitted at the 6-week postnatal visit. Risk of uterine perforation is slightly higher during lactation and following cesarean section. If the previously used contraception was the diaphragm, it will need to be re-fitted 6 weeks postpartum.

INITIAL CARE OF THE NEWBORN

A healthy infant born at term (between 38–42 weeks) should have an average birth weight (usually exceeds 2500 g), cries immediately following birth, establishes independent rhythmic respiration and quickly adapts to the changed environment. The length (crown to foot) is 50–52 cm. The length is a more reliable criterion of gestational age than the weight. Occipitofrontal circumference measures about 32–37 cm and the biparietal diameter measures about 9.5 cm (Fig. 14.3) [17, 23, 48].

But at present, according to the parameters denoted by Apgar (Dr Virginia Apgar, 1953), a scor-

Fig. 14.3

Table 15

Apgar scoring

Signs	Score		
	0	1	2
Respiratory effort	Apneic	Slow, irregular	Good, crying
Heart rate	Absent	Low (below 100)	Over 100
Muscle tone	Flaccid	Flexion of extremities	Active body movements
Reflex irritability	No response	Grimace	Cough or sneeze
Color	Blue, pale	Body pink, extremities blue	Complete pink
Total score = 10. No depression = 7–10. Mild depression = 4–6. Severe depression = 0–3			

ing procedure has been designed for better understanding of the clinical state. This also helps in comparison of results and standardization of methods of management. The Apgar score is related to the status of oxygenation of the fetus at or immediately after birth. This scoring is done in a newborn baby at 1 minute, 5 minutes (Table 15).

• Check the baby's breathing and colour every five minutes.

• If the baby becomes cyanotic (bluish) or is having difficulty breathing (less than 30 or more than 60 breaths per minute), give oxygen by nasal catheter or prongs.

• Check warmth by feeling the baby's feet every 15 minutes:

— If the baby's feet feel cold, check axillary temperature;

— If the baby's temperature is less than 36.5°C, rewarm the baby.

• Check the cord for bleeding.

• Apply antimicrobial drops (1% silver nitrate solution or 2.5% povidone iodine solution) or ointment (1% tetracycline ointment) to the baby's eyes.

Note: Povidone-iodine should not be confused with tincture of iodine, which could cause blindness if used.

• Wipe off any meconium or blood from skin.

• Encourage breastfeeding when the baby appears ready.

IMMEDIATE NEWBORN CONDITIONS OR PROBLEMS

• The newborn has serious conditions or problems:

— gasping or not breathing;

— breathing with difficulty (less than 30 or more than 60 breaths per minute, indrawing of the chest or grunting);

— central cyanosis (blueness);

— preterm or very low birth weight (less than 32 weeks gestation or less than 1500 g);

— hypothermia (axillary temperature less than 36.5°C);

— convulsions.

• The newborn has other conditions or problems that require attention in the delivery room:

— low birth weight (1500–2500 g);

— possible bacterial infection in an apparently normal newborn whose mother had prelabor or prolonged rupture of membranes or amnionitis;

— possible congenital syphilis (mother has positive serologic test or is symptomatic).

GASPING OR NOT BREATHING

General management

• Dry the baby, remove the wet cloth and wrap the baby in a dry, warm cloth.

• Clamp and cut the cord immediately if not already done.

• Move the baby to a firm, warm surface under a radiant heater for resuscitation.

• Observe standard infection prevention practices when caring for and resuscitating a newborn.

Opening the airway:

• Position the newborn:

— place the baby on its back;

— position the head in a slightly extended position to open the airway;

— keep the baby wrapped or covered, except for the face and upper chest (Fig. 14.4).

• Clear the airway by suctioning first the mouth and then the nostrils. If blood or meconium is in the baby's mouth or nose, suction immediately to prevent aspiration.

Note: Do not suction deep in the throat as this may cause the baby's heart to slow or the baby may stop breathing.

Fig. 14.4. Correct position of the head for ventilation

• Reassess the baby:

— If the newborn starts crying or breathing, no further immediate action is needed. Proceed with initial care of the newborn;

— if the baby is still not breathing, start ventilating.

Ventilating the newborn

• Recheck the newborn's position. The neck should be slightly extended.

• Position the mask and check the seal (Fig. 14.5):

— place the mask on the newborn's face. It should cover the chin, mouth and nose;

— form a seal between the mask and the face;

— squeeze the bag with two fingers only or with the whole hand, depending on the size of the bag;

— check the seal by ventilating twice and observing the rise of the chest.

• Once a seal is ensured and chest movement is present, ventilate the newborn. Maintain the correct rate (approximately 40 breaths per minute) and pressure (observe the chest for an easy rise and fall):

— If the baby's chest is rising, ventilation pressure is probably adequate;

— If the baby's chest is not rising:

a) repeat suction of mouth and nose to remove mucus, blood or meconium from the airway;

b) recheck and correct, if necessary, the position of the newborn.

• Ventilate for 1 minute and then stop and quickly assess if the newborn is breathing spontaneously:

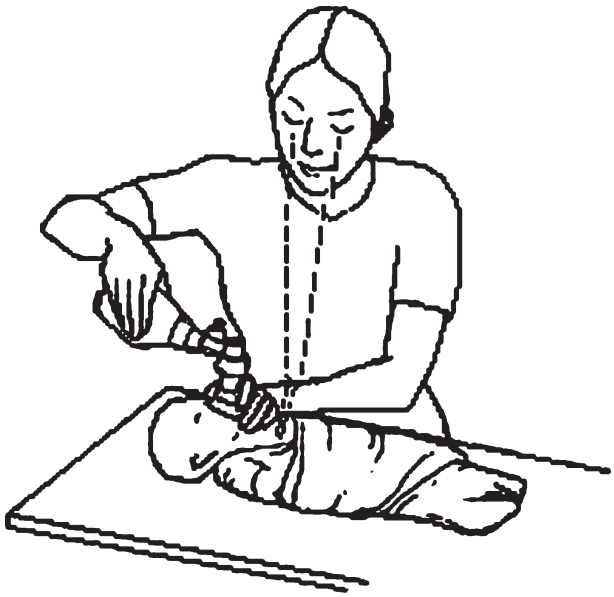


Fig. 14.5. Ventilation with bag and mask

— If breathing is normal (30–60 breaths per minute) and there is no indrawing of the chest and no grunting for 1 minute, no further resuscitation is needed. Proceed with initial care of the newborn;

— If the newborn is not breathing, or the breathing is weak, continue ventilating until spontaneous breathing begins.

Chapter 15
ABNORMAL LABOR

INDUCTION AND AUGMENTATION OF LABOR

• Induction of labor and augmentation of labor are performed for different indications but the methods are the same.

— Induction of labor: stimulating the uterus to begin labor.

Termination of a pregnancy by inducing labor may be indicated because of a suspected or confirmed risk to the mother or baby, or both [28, 29, 46].

Such indications are:

— Hypertensive disorders.
— Prolonged pregnancy.
— Compromised fetus e.g. growth restriction.
— Fetal abnormality or death.
— Maternal diabetes.
— Rhesus sensitisation.

• Augmentation of labor: stimulating the uterus during labor to increase the frequency, duration and strength of contractions.

A good labor pattern is established when there are three contractions in 10 minutes, each lasting more than 40 seconds.

If the membranes are intact, for induction and augmentation of labor is recommended to first perform artificial rupture of membranes (ARM). In some cases, this is all that is needed to induce labor. Membrane rupture, whether spontaneous or artificial, often sets off the following chain of events:

— Amniotic fluid is expelled;
— Uterine volume is decreased;
— Prostaglandins are produced, stimulating labor;
— Uterine contractions begin (if the woman is not in labor) or become stronger (if she is already in labor) (Fig. 15.1).

As labor approaches, the cervix normally shows changes known as 'ripening' so that it becomes 'inducible' and is then called a 'favourable' cervix. The condition of the cervix is the most important factor in successful induction and, where ripening has not occurred, there is a greater chance of a long labor, fetal hypoxia and operative delivery. An unripe cervix is hard, long, closed and not effaced. A ripe cervix is soft, effaced or becoming effaced and admits the finger [50].

Assessment of the cervix

The success of induction of labor is related to the condition of the cervix at the start of induction. To assess the condition of the cervix, a cervical exam is performed and a score is assigned based on the criteria (Bishop's score).

— If the cervix is favorable (has a score of 6 or more), labor is usually successfully induced with oxytocin alone.

— If the cervix is unfavorable (has a score of 5 or less), ripen the cervix using prostaglandins before induction.

Bishop score, also Bishop's score (1964), also known as cervix score is a pre-labor scoring system to assist in predicting whether induction of labor will be required.

The degree of favorable of the cervix is determined by the following features: the length of the cervix, its position relative to the axis of the pelvis, its consistency and thickness, the degree of opening (for cross-finger) of the outer and inner mouth, the degree of softening of the internal fauces. The parameters of these indicators vary depending on the gestational age or concomitant obstetric pathology.

There are 3 grades of cervical state: immature, not fully mature and mature cervix (Table 16).

ARTIFICIAL RUPTURE OF MEMBRANES (AMNIOTOMY)

— Review for indications.

Note: It is prudent to leave the membranes intact for as long as possible to reduce perinatal transmission of HIV.

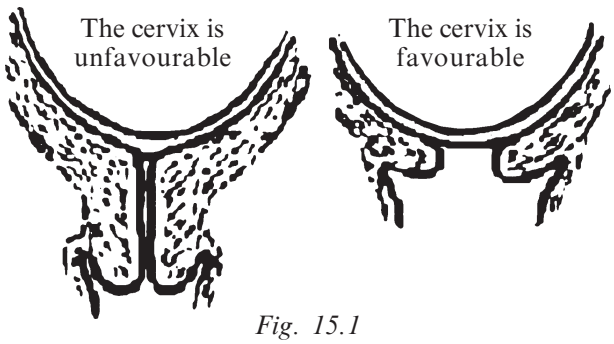


Fig. 15.1

Table 16

Bishop score

Examination	Points		
	0	1	2
1. Position of cervix	Directed toward the symphysis	Middle	The pelvic axis
2. Length of cervix (cm)	> 2	1–2	< 1
3. Consistency of cervix	Dense	Mederate	Soft
4. Cervical dilatation (cm)	Close	1–2	> 2
5. Station of presenting part	Above the pelvic inlet plane	Between the superior and posterior margin of the symphysis	Posterior margin of the symphysis and below

Note. 0–2 points — the cervix is unfavourable; 3–5 points — "not fully mature" cervix; > 6 points — the cervix is favourable.

— Listen to and note the fetal heart rate.

— Ask the woman to lie on her back with her legs bent, feet together and knees apart.

— Wearing high-level disinfected or sterile gloves, use one hand to examine the cervix and note the consistency, position, effacement and dilatation (Fig. 15.2).

— Use the other hand to insert an amniotic hook or a Kocher clamp into the vagina.

— Guide the clamp or hook towards the membranes along the fingers in the vagina.

— Place two fingers against the membranes and gently rupture the membranes with the instrument in the other hand. Allow the amniotic fluid to drain slowly around the fingers.

Note the colour of the fluid (clear, greenish, bloody). If thick meconium is present, suspect fetal distress.

— After ARM, listen to the fetal heart rate during and after a contraction. If the fetal heart rate is abnormal (less than 100 or more than 180 beats per minute), suspect fetal distress.

— If membranes have been ruptured for 18 hours, give prophylactic antibiotics to help reduce Group B streptococcus infection in the neonate.

— If good labor is not established after ARM, begin oxytocin infusion.

Complications of Amniotomy:

Failure to induce effective contractions.

Labor may not become established after amniotomy alone and it is usual to stimulate the uterus further by intravenous oxytocin after an interval of 3 hours or so if contractions are inadequate.

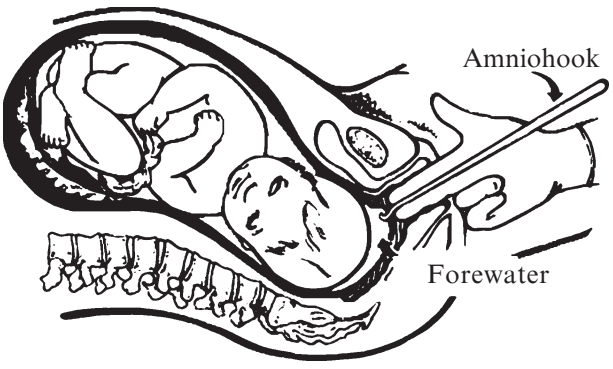


Fig. 15.2

Placental separation (Abruption)

This may be caused by the sudden reduction in the volume of liquor where there has been polyhydramnios.

Prolapse of the cord

This will only happen with an ill-fitting presenting part. Cord prolapse, occult or frank, should give warning signs on the Fetal Heart Rate monitor.

Bleeding

This is not uncommon. The usual source is maternal blood from an element of forced dilatation of the cervix by the examining fingers. Occasionally it may come from fetal vessels running in the membranes (velamentous insertion of the cord).

Infection

The uterus may become infected if the interval from amniotomy to delivery is excessive, and both mother and child are at risk. Infection may perhaps be delayed by observing careful antiseptic techniques, and by exhibiting antibiotics whenever delay is anticipated.

Pulmonary embolism of amniotic fluid

This rare condition presents as severe shock of rapid onset, with intense dyspnoea and often bleeding. It is associated with amniotomy and strong uterine contractions, and must be distinguished from eclampsia, abruption, ruptured uterus, and acid aspiration. Treatment must include positive pressure ventilation, and correction of the inevitable coagulation defect.

OXYTOCIN

Indication

— Induction of labor.

— Correction of a dynamic dystocia: delayed dilation in a woman in labor, with arrest for more than 2 hours, due to inadequate uterine contraction. The cervix must be dilated more than 3 to 4 cm, and effacement in progress. The membranes must have been ruptured.

— Contractions fail to resume 15 minutes after the birth of a first twin.

Risks of using oxytocin during labor

Use oxytocin with great caution, as fetal distress can occur from hyperstimulation and, rarely, uterine rupture can occur. Multiparous women are at higher risk for uterine rupture.

The effective dose of oxytocin varies greatly between women. Cautiously administer oxytocin, gradually increasing the rate of infusion until good labor is established (three contractions in 10 minutes, each lasting more than 40 seconds). Maintain this rate until delivery. The uterus should relax between contractions. When oxytocin infusion results in a good labor pattern, maintain the same rate until delivery.

Contraindications to the use of oxytocin during labor

— Obvious fetopelvic disproportion, including malpresentation (brow, transverse, etc.).
— Complete placenta previa.
— Spontaneous uterine hypertonia.
— Fetal distress.
— Two or more prior cesarean section.
— Prior classical cesarean section (vertical uterine incision).
— Absence of medical indication.

Management

• Monitor the woman's pulse, blood pressure and contractions, and check the fetal heart rate.
• Be sure induction is indicated, as failed induction is usually followed by cesarean section.
• Record the following on a partograph every 30 minutes:
— rate of infusion of oxytocin;
— duration and frequency of contractions;
— fetal heart rate. Listen every 30 minutes, always immediately after a contraction. If the fetal heart rate is less than 100 beats per minute, stop the infusion and manage fetal distress.

Infuse oxytocin 2.5 units in 500 mL of normal saline at 10 drops per minute. This is approximately 2.5 mIU per minute. Increase the infusion rate every 30 minutes until a good contraction pattern is established (three contractions in 10 minutes, each lasting more than 40 seconds). Maintain this rate until delivery is completed. If good contractions are not established at the maximum dose, deliver by cesarean section (Fig. 15.3) [28,29, 48].

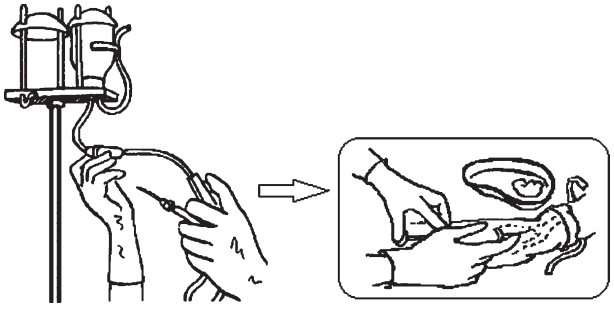


Fig. 15.3

Complications of oxytocin:

Poor uterine action.

This may occur where amniotomy has been carried out in spite of an unfavourable cervix. Ripening of the cervix with prostaglandin should be used first in these circumstances. Sometimes, in spite of apparently satisfactory uterine action, little dilatation of the cervix occurs and labor has to be terminated by cesarean section. This is due to incoordinate uterine action resulting in dysfunctional labor.

Abnormal fetal heart rate patterns

Prolonged or excessive oxytocin administration can cause fetal hypoxia by over-stimulation of the uterus. Continuous fetal heart rate monitoring is required for all patients undergoing oxytocin stimulation.

Hyperstimulation

Overdose can cause excessive, painful contractions and even a prolonged spasm (tetanic contraction). If hyperstimulation becomes evident the infusion should be stopped to allow the uterus to relax.

Rupture of the uterus

The possibility of rupture must be borne in mind when using oxytocin. It is unlikely in a primigravida but has been reported. It is more to be expected in the parous woman or in the patient who has had a previous cesarean section or hysterotomy. The use of an intrauterine pressure transducer may be advisable in such patients. Epidural anaesthesia does not mask the pain of uterine rupture but it should be used with caution.

Water intoxication

This may result from the prolonged administration of high doses of oxytocin in large volumes of electrolyte-free fluid. This should not be an issue in labor using normal dosage of oxytocin.

PROSTAGLANDINS

Prostaglandins are highly effective in ripening the cervix during induction of labor. Prostaglandin E2 (PGE2) is available in several forms (3 mg pessary or 2–3 mg gel). The prostaglandin is placed high in the posterior fornix of the vagina and may be repeated after six hours if required.

Monitor uterine contractions and fetal heart rate of all women undergoing induction of labor with prostaglandins.

Discontinue use of prostaglandins and begin oxytocin infusion if:
— membranes rupture;
— cervical ripening has been achieved;
— good labor has been established;
— or 12 hours have passed.

MISOPROSTOL

Use misoprostol (Fig. 15.4) to ripen the cervix only in highly selected situations such as: fetal death in-utero if the woman has not gone into spontaneous labor after four weeks and platelets are decreasing.

— Place misoprostol 25 mcg in the posterior fornix of the vagina. Repeat after six hours, if required;

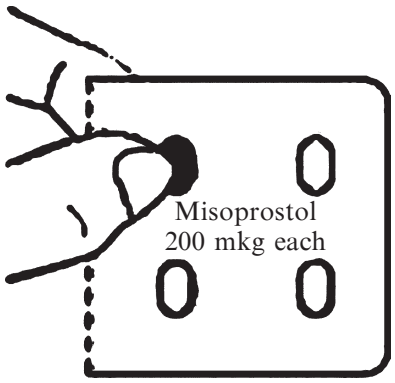


Fig. 15.4

— If there is no response after two doses of 25 mcg, increase to 50 mcg every 6 hours;

— Do not use more than 50 mcg at a time and do not exceed four doses (200 mcg).

Unsatisfactory progress of labor

Without timely intervention, abnormal labor usually leads to prolonged labor. Maternal and neonatal complications are increased with increasing duration of labor. The diagnostic criteria of abnormal progress of labor depend on the stage and phase of labor. The clinical parameters used include cervical effacement and dilatation, descent (station) and rotation of the head, and molding.

Problems

— Cervix not dilated beyond 4 cm after 8 hours of regular contractions.

— Cervical dilatation is to the right of the alert line on the partograph.

— The woman has been experiencing labor pains for 12 hours or more without delivery (prolonged labor).

PROLONGED LATENT PHASE

The diagnosis of prolonged latent phase is made retrospectively. When contractions cease, the woman is said to have had false labor. When contractions become regular and dilatation progresses beyond 4 cm, the woman is said to have been in the latent phase.

The causes include:

— unripe cervix;

— malposition and malpresentation;

— cephalopelvic disproportion and;

— premature rupture of the membranes.

Prolonged latent phase may be worrisome to the patient but does not endanger the mother or fetus.

Management

If a woman has been in the latent phase for more than eight hours and there is little sign of progress, reassess the situation by assessing the cervix:

• If there has been no change in cervical effacement or dilatation and there is no fetal distress, review the diagnosis. The woman may not be in labor.

• If there has been a change in cervical effacement or dilatation, rupture the membranes and induce labor using oxytocin.

— Reassess every four hours;

— If the woman has not entered the active phase after eight hours of oxytocin infusion, deliver by cesarean section.

• If membranes have been ruptured for 18 hours, give prophylactic antibiotics.

— If the woman delivers vaginally, discontinue antibiotics postpartum;

— If the woman has a cesarean section, continue antibiotics and give metronidazole (500 mg every eight hours until the woman is fever-free for 48 hours).

A sample partograph for poor progress of labor due to inadequate uterine contractions corrected with oxytocin (Fig. 15.5).

• The woman was admitted in active labor at 10 AM:

— fetal head 5/5 palpable;

— cervix dilated 4 cm;

— two contractions in 10 minutes, each lasting less than 20 seconds.

• At 12 PM:

— fetal head still 5/5 palpable;

— cervix still dilated 4 cm and to the right of the alert line;

— no improvement in contractions.

• At 2 PM:

— poor progress of labor due to inefficient uterine contractions diagnosed;

— augmented labor with oxytocin (15 drops per minute);

— escalated oxytocin until a good pattern of contractions was established.

• At 7 PM:

— fetal head 1/5 palpable;

— cervix dilated 10 cm;

— four contractions in 10 minutes, each lasting 45 seconds.

• Spontaneous vaginal delivery occurred at 8 : 10 PM.

PROLONGED ACTIVE PHASE

When cervical change continues with adequate uterine contractions in the active phase of labor but over a longer time period than anticipated, then a prolonged active phase is the diagnosis. In nulliparous patients, cervical change is < 1.2 cm per hour, whereas in multiparous patients cervical change is occurring at < 1.5 cm per hour. A prolonged active phase may be the result of inadequate uterine contractility, but often both the timing and strength of uterine contractions appear to be normal, and cervix dilation occurs slowly despite oxytocin therapy. The underlying problem may be true CPD or an undiagnosed flexion abnormality. Oxytocin therapy

PARTOGRAPH

Name _____ Gravida _1_ Para _0+0_____

Data of admission _2.5.2000_ Time of admission _10:00 A.M._

Ruptured membranes 13:30 hours

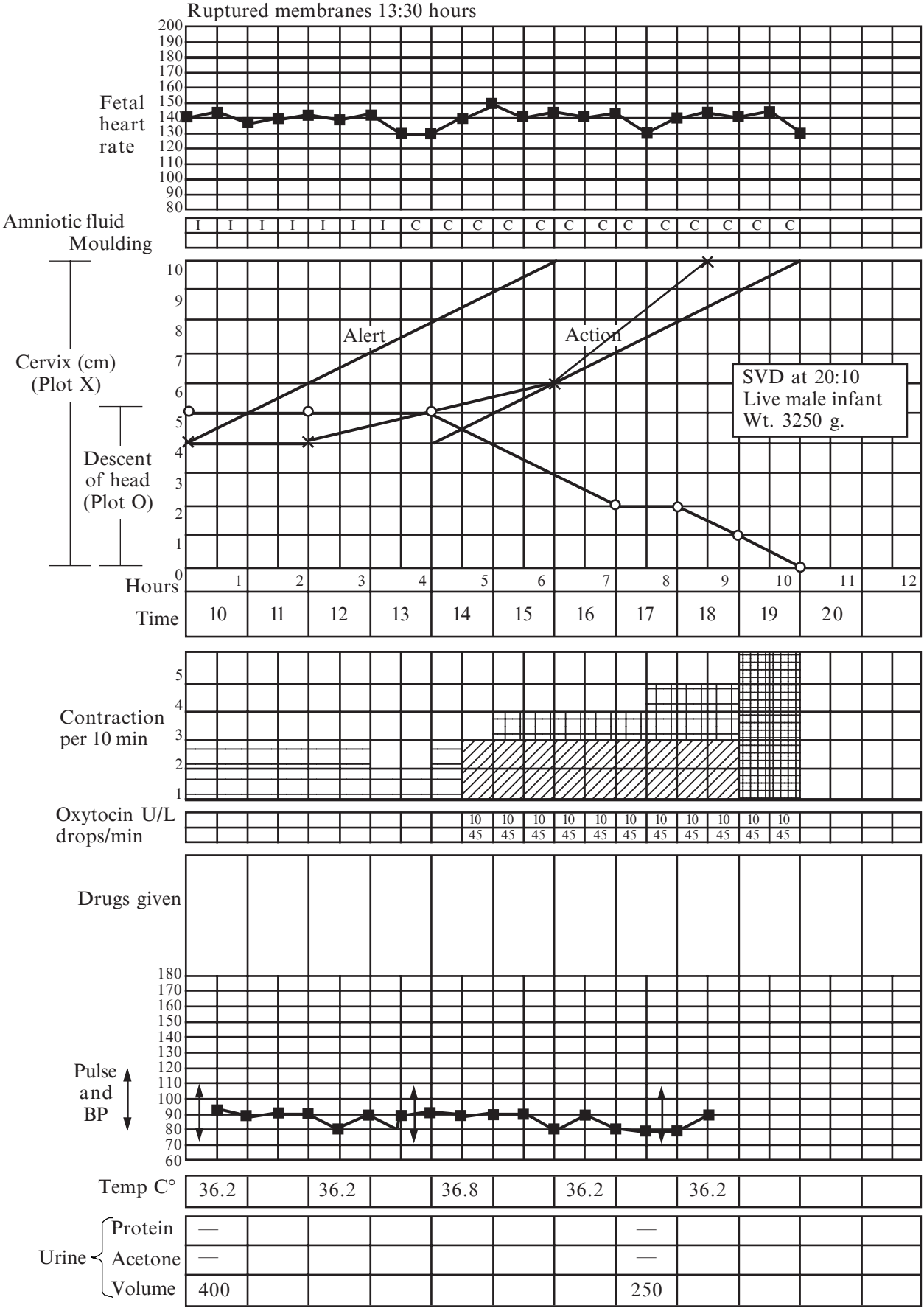


Fig. 15.5

often is not successful in accelerating labor, and an arrest of dilation or descent may be inevitable regardless of the therapies employed. If a protracted active phase leads to an arrest of labor despite oxytocin therapy, cesarean delivery is the best therapeutic course.

Management

• If there are no signs of cephalopelvic disproportion or obstruction and the membranes are intact, rupture the membranes.

• Assess uterine contractions:

— If contractions are inefficient (less than three contractions in 10 minutes, each lasting less than 40 seconds), suspect inadequate uterine activity;

— If contractions are efficient (three or more contractions in 10 minutes, each lasting more than 40 seconds) suspect cephalopelvic disproportion, obstruction, malposition or malpresentation.

• Recommends the use of oxytocin for all protraction and arrest disorders.

OBSTRUCTED LABOR

Obstructed labor is one where in spite of good uterine contractions, the progressive descent of the presenting part is arrested due to mechanical obstruction. This may result either due to factors in the fetus or in the birth canal or both, so that further progress is almost impossible without assistance. In the developing countries, the prevalence is about 1–2% in the referral hospitals.

Etiology

Maternal causes

• Bony obstruction: e.g.

— Contracted pelvis.

— Tumours of pelvic bones.

• Soft tissue obstruction:

— Uterus: impacted subserous pedunculated fibroid, constriction ring opposite the neck of the fetus.

— Cervix: cervical dystocia.

— Vagina: septa, stenosis, tumours.

— Ovaries: Impacted ovarian tumours.

Fetal causes

• Malpresentations and malpositions: e.g.

— Persistent occipito-posterior and deep transverse arrest.

— Persistent mento-posterior and transverse arrest of the face presentation.

— Brow.

— Shoulder.

— Impacted frank breech.

• Large sized fetus (macrosomia).

• Congenital anomalies: e.g.

— Hydrocephalus.

— Fetal ascites.

— Fetal tumours.

• Locked and conjoined twins.

Diagnosis

It is the clinical picture of obstructed labor with impending rupture uterus (excessive uterine contraction and retraction).

Clinical picture: is that of obstructed labor with impending rupture uterus.

History

— Prolonged labor,

— Frequent and strong uterine contractions,

— Rupture membranes.

General examination

It shows signs of maternal distress as:

— Exhaustion,

— High temperature (38°C),

— Rapid pulse,

— Signs of dehydration: dry tongue and cracked lips.

Abdominal examination (Fig. 15.6)

• The uterus:

— is hard and tender;

— frequent strong uterine contractions with no relaxation in between (tetanic contractions);

— the rising retraction ring is seen and felt as an oblique groove across the abdomen.

Pathologic retraction ring (Bandl ring) — when thinning of lower uterine segment is extreme, as in obstructed labor, ring is very prominent [17, 40].

• The fetus:

— fetal parts cannot be felt easily;

— FHS are absent or show fetal distress due to interference with the utero-placental blood flow.

Vaginal examination

— Vulva: is oedematous.

— Vulva: is oedematous.

— Cervix: is fully or partially dilated, oedematous and hanging.

— The membranes: are ruptured.

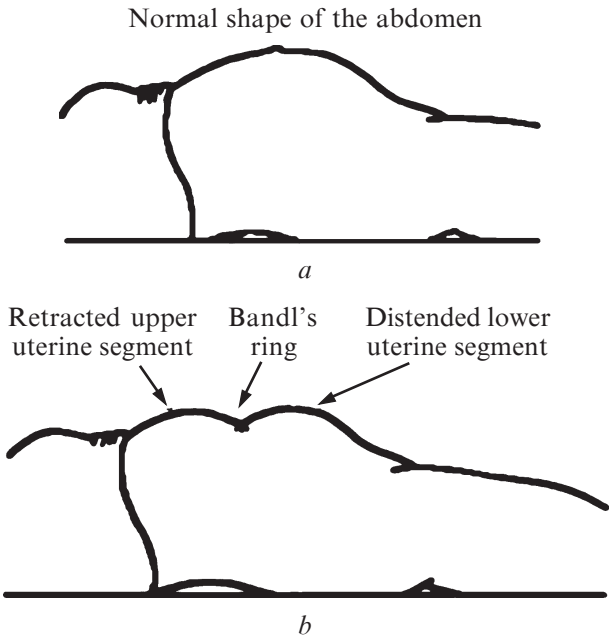


Fig. 15.6

— The presenting part: is high and not engaged or impacted in the pelvis. If it is the head it shows excessive moulding and large caput.

— The cause of obstruction can be detected.

Differential diagnosis

— Constriction ring.

— Full bladder.

— Fundal myoma.

Complications

• Maternal:

— Maternal distress and ketoacidosis.

— Rupture uterus.

— Necrotic vesico-vaginal fistula.

— Infections as chorioamnionitis and puerperal sepsis.

— Postpartum haemorrhage due to injuries or uterine atony.

• Fetal:

— Asphyxia.

— Intracranial haemorrhage from excessive moulding.

— Birth injuries.

— Infections.

Management

— Preventive measures:

Careful observation, proper assessment, early detection and management of the causes of obstruction.

— Curative measures:

Cesarean section is the safest method even if the baby is dead as labor must be immediately terminated and any manipulations may lead to rupture of the uterus.

CONTRACTED PELVIS

Anatomical — It is a pelvis in which one or more of its diameters is reduced below the normal by one or more centimeters.

Obstetric — It is a pelvis in which one or more of its diameters is reduced so that it interferes with the normal mechanism of labor.

Etiology of contracted pelvis

• Common causes of contracted pelvis are:

Nutritional and environmental defects:

— minor variation;

— common major: rachitic and osteomalacic

— rare.

• Disease or injury affecting (Fig. 15.7):

— the bone of the pelvis: fracture, tumors, tubercular arthritis;

— spine: kyphosis, scoliosis, coccygeal deformity;

— lower limbs: poliomyelitis, hip joint disease.

• Developmental defects: Naegele's pelvis, Robert's pelvis.

Classification:

Classified by:

— type of distortion of pelvic architecture,

— degree of contraction.

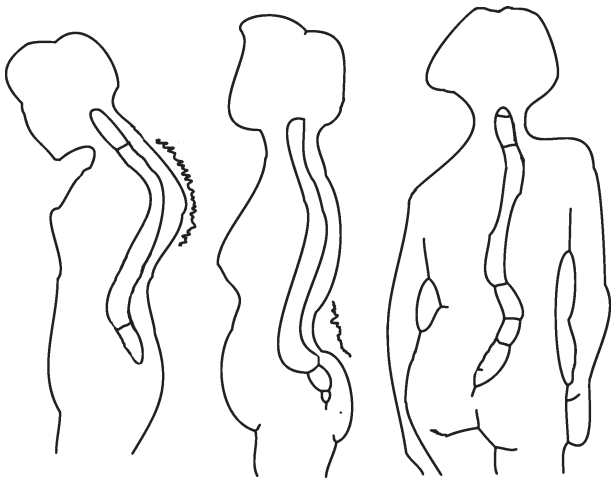


Fig. 15.7. Vertebral disorders

Classification by Pelvic Architecture

1. Pelvis aequabiliter justo minor

• Characterized by general reduction of all diameters; equally shortened usually by 1–2 cm.

• Occurs in short. Also occurs in women with massive skeletal bones and developed muscles, the pelvis has masculine features such as narrow sacrum, narrow pubic outlet (funnel-shaped).

2. Flat Pelvis

• Reduced anteroposterior diameters with normal transverse and oblique diameters.

• Has 2 types of contracture

— Simple flat (or platypelloid) pelvis. Entire sacral platform is dislocated toward the symphysis hence all the anteroposterior diameters of all pelvic planes are reduced.

— Flat rachitic. Anteroposterior diameter of the pelvic inlet only is reduced.

3. Generally Contracted Pelvis:

• All diameters are reduced, but the anteroposterior diameters are shortened greater than the others.

• Usually connected with the rickets of the childhood.

Rare forms of contracted pelvis:

— Otto's pelvis — develop as result of inflammatory process in the hip or knee.

— Beaked (rostrate) pelvis — under development of both sacral wings.

— Spondylolithetic pelvis — formed due to partial dislocation of the last lumbar vertebra in front of the 1st sacral vertebra.

— Osteomalacic pelvis.

— Scoliotic pelvis — only the lumbar region causes deformity of the pelvis. The acetabulum is pushed inwards on the weight bearing side.

Classification by degree of contracture (4 degrees)

— First degree: true conjugate < 11cm but not < 9 cm, spontaneous delivery is possible.

— Second degree: true conjugate = 9–7.5 cm spontaneous delivery possible but complications may arise.

— Third degree: true conjugate 7.5–6 cm spontaneous delivery impossible, use C-section.

— Fourth degree: true conjugate < 6 cm, impossible delivery, only way is C-section; also known as absolutely contracted pelvis.

Diagnosis

1. History:

• Rickets: is expected if there is a history of delayed walking and dentition.

• Trauma or diseases: of the pelvis, spines or lower limbs.

• Infantilism.

• Previous tuberculosis of bones and joints.

• Bad obstetric history: e.g. prolonged labor ended by:

— difficult forceps, cesarean section or stillbirth;

— weight of the baby;

— evidence of maternal injuries such as complete perineal tear, vesico vaginal istula, recto vaginal fistula.

2. General examination:

• Abnormal gait:

— Assess women for stockily built with bull neck.

— Broad shoulder and short thigh.

— Obese and male distribution of hair.

— Stature: women < 150 cm or 5 feet.

3. Abdomen examination:

— Pendulous abdomen in primigravida (Fig. 15.8).

— The fetal head fails to enter a contracted pelvis at the end of pregnancy and floats high above the inlet, failed growth of the uterus deviates upward and anteriorly.

— Non engagement in the last 3–4 weeks in primigravida.

2 shapes of abdomen:

— Acuminate (pointed) abdomen in primigravida with a resilient abdominal wall.

— Pendulous abdomen in multiparous women.

Pendulous abdomen

Acuminate (pointed) abdoimen

Fig. 15.8

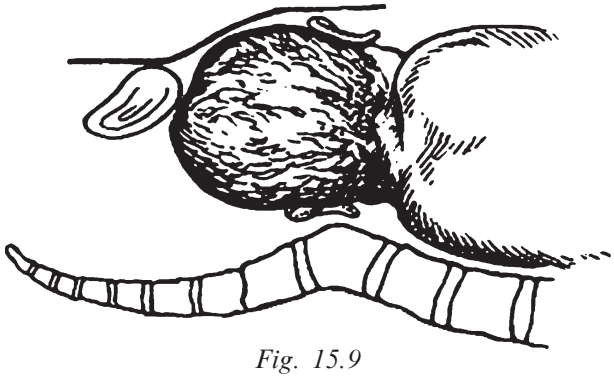


Fig. 15.9

Management

Cesarean section

• Elective cesarean section at term is indicated in:

— major degree of contraction;

— major disproportion;

— absolute contraction.

Patient not fit for trial labor. The operation is done in a planned way any time during the last week of pregnancy.

• Emergency: when trial labor fails.

• Trial labor:

It is the conduction of spontaneous labor in a moderate degree of disproportion, in an institution under supervision with watchful expectancy hoping for a vaginal delivery.

Complications of contracted pelvis

Maternal during pregnancy:

— Incarcerated retroverted gravid uterus.

— Malpresentations.

— Pendulous abdomen.

— Non-engagement.

— Pyelonephritis especially in high assimilation pelvis due to more compression of the ureter.

Maternal during labor:

— Inertia, slow cervical dilatation and prolonged labor.

— Premature rupture of membranes and cord prolapse.

— Obstructed labor and rupture uterus.

— Necrotic genito-urinary fistula.

— Injury to pelvic joints or nerves from difficult forceps delivery.

— Postpartum haemorrhage.

Fetal:

— Intracranial haemorrhage.

— Asphyxia.

— Fracture skull.

— Nerve injuries.

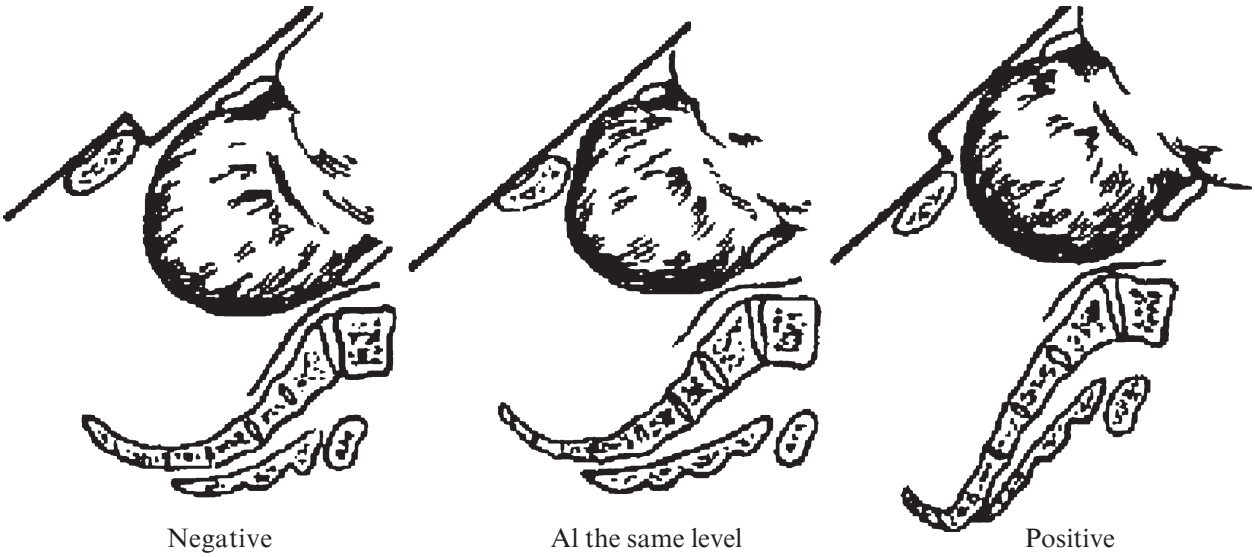
— Intra-amniotic infection.

CEPHALOPELVIC DISPROPORTION (CPD) (Fig. 15.9)

CPD exists if the maternal bony pelvis is not of sufficient size and of appropriate shape to allow the passage of the fetal head.

Clinically or functional contracted pelvis — pelvis with normal dimensions, but vaginally delivery is impossible due to CPD.

CPD is one of the commonest causes of different complications in labor, including prolonged labor, fetal distress, and delayed second stage. CPD is very frequently diagnosed and is a very common indication of cesarean sections, especially when there is failure to progress in labor. But it is very difficult to di-



Negative Al the same level Positive

Fig. 15.10. Vasten's sign

agnose CPD before a woman has started her labor pains since it is very difficult to anticipate how well the fetal head and the maternal pelvis will adjust and mould to each other [23, 26, 50].

Causes:

• Increased Fetal Weight:

• Abnormal Fetal Position:

— Malpresentation.

— Malposition.

• Problems with the Pelvis:

— Small pelvis.

— Abnormal shape of the pelvis due to diseases like rickets, osteomalacia or tuberculosis.

— Abnormal shape due to previous accidents.

— Tumors of the bones.

— Childhood poliomyelitis affecting the shape of the hips.

— Congenital dislocation of the hips.

— Congenital deformity of the sacrum or coccyx.

• Problems with the genital tract:

— Tumors like fibroids obstruct the birth passage.

— Congenital rigidity of the cervix.

— Scarring of the cervix due to previous operations like conisation.

— Congenital vaginal septum.

Diagnosis

Diagnosis of CPD is very difficult. This is because it is difficult to estimate exactly how much the mother's ligaments and joints will 'give' or relax before labor starts.

The fetal head also has a great capacity to mould — the skull bones can overlap to some extent and decrease the diameter of the head. So, a baby who appears to be too big to pass through its mother's birth passage before labor, may do so without much problem when active uterine contractions start.

Degrees of disproportion

— Minor disproportion:

The anterior surface of the head is in line with the posterior surface of the symphysis. During labor the head is engaged due to moulding and vaginal delivery can be achieved.

— Moderate disproportion (1st degree disproportion): The anterior surface of the head is in line with the anterior surface of the symphysis. Vaginal delivery may or may not occur.

— Marked disproportion (2nd degree disproportion): The head overrides the anterior surface of the symphysis. Vaginal delivery cannot occur.

Clinic signs of clinically contracted pelvis:

— Head is arrested in the pelvic inlet (absence of fetal descending in complete cervical dilation and adequate uterine contractions).

— Uterine contractions abnormality.

— Positive Vasten's sign (if disproportion between fetal head and symphysis pubis is prominent — Vasten's sign is positive, if disproportion between fetal head and symphysis pubis is absent — Vasten's sign is negative) (Fig. 15.10).

— Signs of urinary bladder compression.

— Edema of the cervix, and vaginal walls, productions of fistulas.

When the presenting part is firmly wedged into the pelvic inlet but does not advance for a considerable time, portions of the birth canal lying between it and the pelvic wall may be subjected to excessive pressure. As circulation is impaired, the resulting necrosis may become manifest several days after delivery by the appearance of vesicovaginal, vesicocervical, or rectovaginal fistulas.

— Danger of uterine rupture.

When the disproportion between the head and the pelvis is so pronounced that engagement and descent do not occur, the lower uterine segment becomes increasingly stretched, and the danger of its rupture becomes imminent. In such cases, a pathologic contractile ring may develop and can be felt as a transverse or oblique ridge extending across the uterus somewhere between the symphysis and the umbilicus. Whenever this condition is noted, prompt cesarean delivery must be employed to terminate labor and prevent rupture of the uterus.

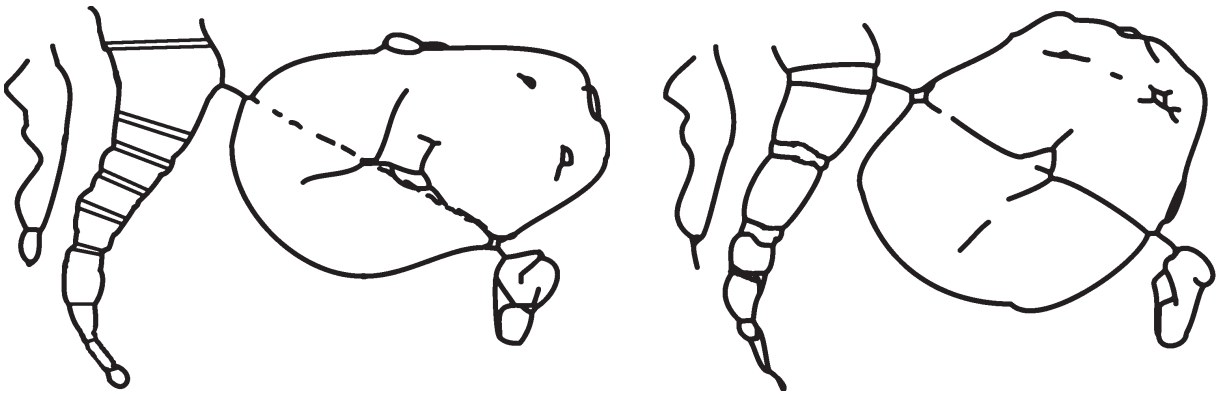


Fig. 15.11

— Pushing occurs if fetal head is situated in the plane of the inlet.

Management in the case of clinically contracted pelvis — only cesarean section.

ASYNCLITISM

In normal pelvis, the fetal head enters with the sagittal suture in the transverse diameter (or occasionally oblique diameter of the brim). If the sagittal suture in between the symphysis pubis and sacral promontory — both parietal bones are felt vaginally at the same level — the head is said to be (synclitic). In such a case the biparietal diameter (9.5 cm) is the diameter of engagement. However, some degree of lateral inclination of the head over the shoulder — (asynclitism) is present normally as the head enters the pelvic inlet (Fig. 15.11).

Asynclitism when the sagittal suture does not lie exactly midway between the symphysis and sacral promontory (Fig. 15.12).

Moderate degrees of asynclitism are the rule in normal labor. Severe asynclitism may lead to cephalopelvic disproportion even with a normal — sized pelvis.

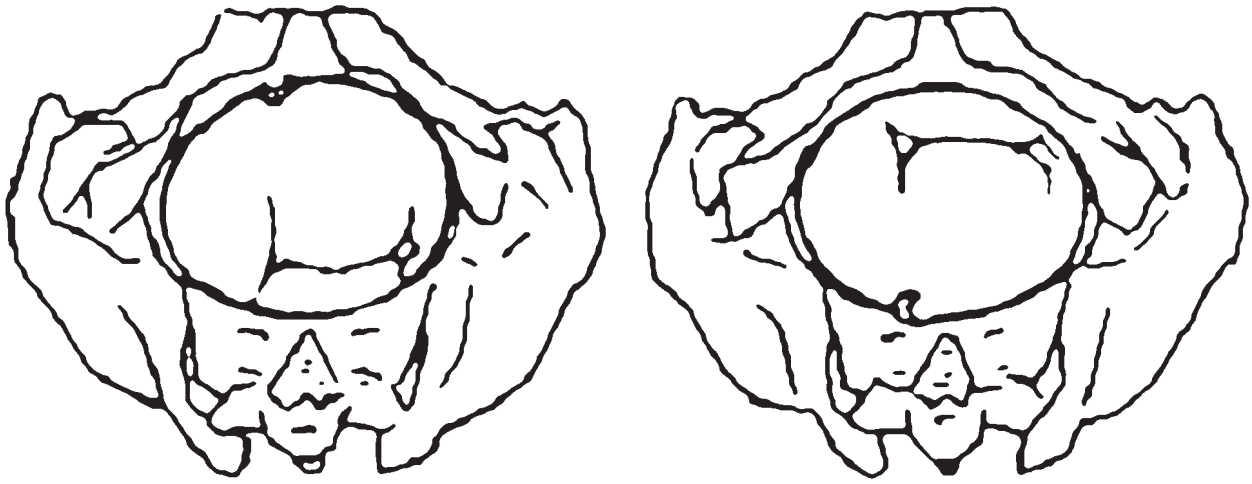
If the sagittal suture lies close to the sacrum and the anterior parietal bone lies over the inlet (Anterior parietal bone presentation) — *anterior asynclitism.*

If the sagittal suture lies close to the symphysis pubis and the posterior parietal bone lies over the inlet (posterior parietal bone presentation) — *posterior asynclitism.*

There is a distinct advantage to having the head engage in asynclitism in certain situations. In a synclitic presentation, the biparietal diameter entering the pelvis measures 9.5 cm; when the parietal bones enter the pelvis in an asynclitic manner, however, the presenting diameter measures 8.75 cm. Therefore, asynclitism permits a larger head to enter the pelvis than would be possible in a synclitic presentation (Fig. 15.13).

LABOR WITH A SCARRED UTERUS

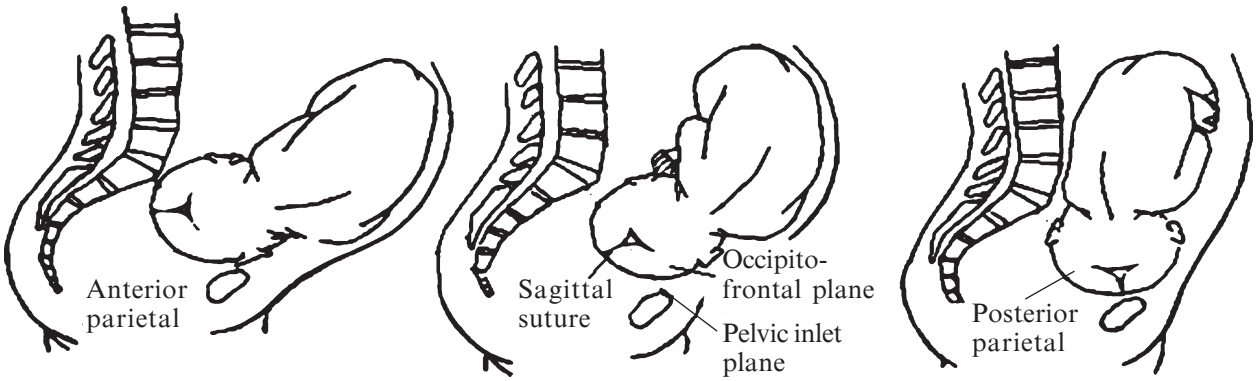
Uterine rupture is an uncommon but potentially catastrophic outcome of pregnancy where the integrity of myometrial wall is breached. Ruptured uterus still remains one of the serious causes of maternal and perinatal mortality and morbidity. Since 1916, the time of Edward Cragin's famous quote, "Once a cesarean, always a cesar-



Anterior asynclitism
Naegele's obliquity

Posterior asynclitism
Litzmann's obliquity

Fig. 15.12



Anterior asynclitism
Naegele's obliquity

Normal synclitism

Posterior asynclitism
Litzmann's obliquity

Fig. 15.13

ean", the medical profession has been concerned about the risks of catastrophic uterine rupture in women with previous cesarean deliveries.

Approximately 50% of all ruptures of classic uterine scars occur before the onset of labor. Studies have shown that more than 60% of cases with low transverse cesarean scars can deliver vaginally. The incidence of uterine rupture is approximately 4–9% of classic scars and 0.7–1.5% of low-transverse scars. Rupture of a classic scar usually is catastrophic, occurring suddenly, totally, and with partial or total extrusion of the fetus into the abdominal cavity. Shock due to internal hemorrhage is a prominent sign. Rupture of the low-transverse scar usually is more subtle and almost always occurs during active labor. The most common presenting sign (present in more than 80% of cases) is a change in the fetal heart rate pattern. A newly recognized finding of variable decelerations or late decelerations should alert the obstetrician. Additional findings that might signal uterine rupture include vaginal bleeding, abdominal pain (especially over the prior incision site), and loss of fetal station. If uterine rupture is suspected, the patient must undergo surgery as soon as possible.

Criteria for vaginal delivery following previous cesarean section:

— Only one previous cesarean section;

— Original indication for cesarean not necessarily recurring in subsequent pregnancies;

— Benign postoperative course;

— Non-complicated current pregnancy (macrosomia, malposition, multiple gestation).

Investigation and assessment:

— To assess the integrity of scar (myometrial thickness > 3.5 mm — normal).

— To assess placental location (absence of sub placental zone — adherent placenta). Doppler may be done for confirmation.

Ultrasonography (USG) plays an important role in the prenatal examination of a patient with previous cesarean section and to diagnose old ruptures. The use of USG to evaluate the scar thickness from previous cesarean helps to forewarn this potential risk. The Montreal study suggested that the combination of single layer closure and full lower uterine segment thickness < 2.3 mm increases the chances of rupture.

Chapter 16
SPECIAL DELIVERY

BREECH PRESENTATION

Breech presentation, which complicates 3–4% of all pregnancies, occurs when the fetal pelvis or lower extremities engage the maternal pelvic inlet. Three types of breech are distinguished, according to fetal attitude. In frank breech, the hips are flexed with extended knees bilaterally. In complete breech, both hips and knees are flexed. In footling breech, 1 (single footling breech) or both (double footling breech) legs are extended below the level of the buttocks.

Fetal position in breech presentation is determined by using the fetal sacrum as the point of reference to the maternal pelvis. This is true for frank, complete, and footling breeches. Eight possible positions are recognized: sacrum anterior (SA), sacrum posterior (SP), left sacrum transverse (LST), right sacrum transverse (RST), left sacrum anterior (LSA), left sacrum posterior (LSP), right sacrum anterior (RSA), and right sacrum posterior (RSP). The station of the breech presenting part is the location of the fetal sacrum with regard to the maternal ischial spines [40, 48, 49].

Before 28 weeks, the fetus is small enough in relation to intrauterine volume to rotate from cephalic to breech presentation and back again with relative ease. As gestational age and fetal weight increase, the relative decrease in intrauterine volume makes such changes more difficult. In most cases, the fetus spontaneously assumes the cephalic presentation to better accommodate the bulkier breech pole in the roomier fundal portion of the uterus.

Causes

Maternal factors:
— Multiparity.
— Contracted pelvis.
— Uterine anomalies.
— Uterine fibroids.

Fetal and Placental factors:
— Prematurity.
— Multiple gestation.
— Poly or Oligohydramnios.
— Fetal abnormality.
— IUGR.
— Placenta Previa.
— Congenital malformations in the fetus.

Diagnosis

— Palpation and Ballottement.

Performance of Leopold's maneuvers and ballottement of the uterus may confirm breech presentation. The softer, more ill-defined breech may be felt in the lower uterine segment above the pelvic inlet. Diagnostic error is common, however, if these maneuvers alone are used to determine presentation.

— Pelvic Examination.

During vaginal examination, the round, firm, smooth head in cephalic presentation can easily be distinguished from the soft, irregular breech presentation if the presenting part is palpable. However, if no presenting part is discernible, further studies are necessary.

— Ultrasound assessment.

Elective cesarean section versus vaginal breech delivery at term

Women should be informed that planned cesarean section carries a reduced perinatal mortality and early neonatal morbidity for babies with a breech presentation at term compared with planned vaginal birth.

The type of incision chosen is extremely important. If the lower uterine segment is well developed, as is usually the case in women at term in labor, a transverse "lower segment" incision is adequate for easy delivery. In premature gestations, in an unlabored uterus, or in many cases of malpresentation, the lower uterine segment may be quite narrow, and a low vertical incision is almost always required for atraumatic delivery.

Unfavorable factors for vaginal breech delivery

— Clinically inadequate pelvis.
— Other contraindications to vaginal birth (e.g. placenta previa).
— Large baby (> 3500g).
— Growth restricted fetus (< 2000g).
— Previous cesarean section.

Spontaneous vaginal delivery

During spontaneous delivery of an infant in the frank breech position, delivery occurs without assistance, and no obstetric maneuvers are applied to the body. The fetus negotiates the maternal pelvis as outlined below, while the operator simply supports the body as it delivers.

Engagement occurs when the bitrochanteric diameter of the fetus has passed the plane of the pelvic inlet. As the fetus descends into the pelvis, the buttocks reach the levator ani muscles of the maternal pelvis. At this point, internal rotation occurs, whereby the anterior hip rotates beneath the pubic symphysis, resulting in a sacrum transverse position. The bitrochanteric diameter of the fetal pelvis is now in an anteroposterior position within the maternal pelvis. The breech then presents at the pelvic outlet and, upon emerging, rotates from sacrum transverse to sacrum anterior. Crowning occurs when the bitrochanteric diameter passes under the pubic symphysis. As this occurs, the shoulders enter the pelvic inlet with the bisacromial diameter in the transverse position. As descent occurs, the bisacromial diameter rotates to an oblique or anteroposterior diameter, until the anterior shoulder rests beneath the pubic symphysis. Delivery of the anterior shoulder occurs as it slips beneath the pubic symphysis. Upward flexion of the body allows for easy delivery of the posterior shoulder over the perineum.

As the shoulders descend, the head engages the pelvic inlet in a transverse or oblique position. Rotation of the head to the occiput anterior position occurs as it enters the midpelvis. The occiput then slips beneath the pubic symphysis, and the remainder of the head is delivered by flexion as the chin, mouth, nose, and forehead slip over the maternal perineum.

As delivery of the breech occurs, increasingly larger diameters (bitrochanteric, bisacromial, biparietal) of the body enter the pelvis, whereas in cephalic presentation, the largest diameter (biparietal diameter) enters the pelvis first. Particularly in preterm labors, the head is considerably larger than the body and provides a better "dilating wedge" as it passes through the cervix and into the pelvis. The smaller bitrochanteric and bisacromial diameters may descend into the pelvis through a partially dilated cervix, but the larger biparietal diameter may be trapped.

Mechanism of labor in breech delivery (Fig. 16.1)

— Right sacrum transverse (RST) at the onset of labor; engagement of the buttocks usually occurs in the oblique or transverse diameter of the pelvic brim.

— Early second stage. The buttocks have reached the pelvic floor, and internal rotation has occurred so that the bitrochanteric diameter lies in the anteroposterior (AP) diameter of the pelvic outlet.

— Late second stage. The anterior buttock appears at the vulva by lateral flexion of the trunk around the pubic symphysis. The shoulders have not yet engaged in the pelvis.

— The buttocks have been delivered, and the shoulders are adjusting to engage in the transverse diameter of the brim. This movement causes external rotation of the delivered buttocks so that the fetal back becomes uppermost.

— The shoulders have reached the pelvic floor and have undergone internal rotation so that the bisacromial diameter lies in the AP diameter of the pelvic outlet. Simultaneously, the buttocks rotate anteriorly through 90 degrees. This is called restitution. The head is engaging in the pelvic brim, and the sagittal suture is lying in the transverse diameter of the brim.

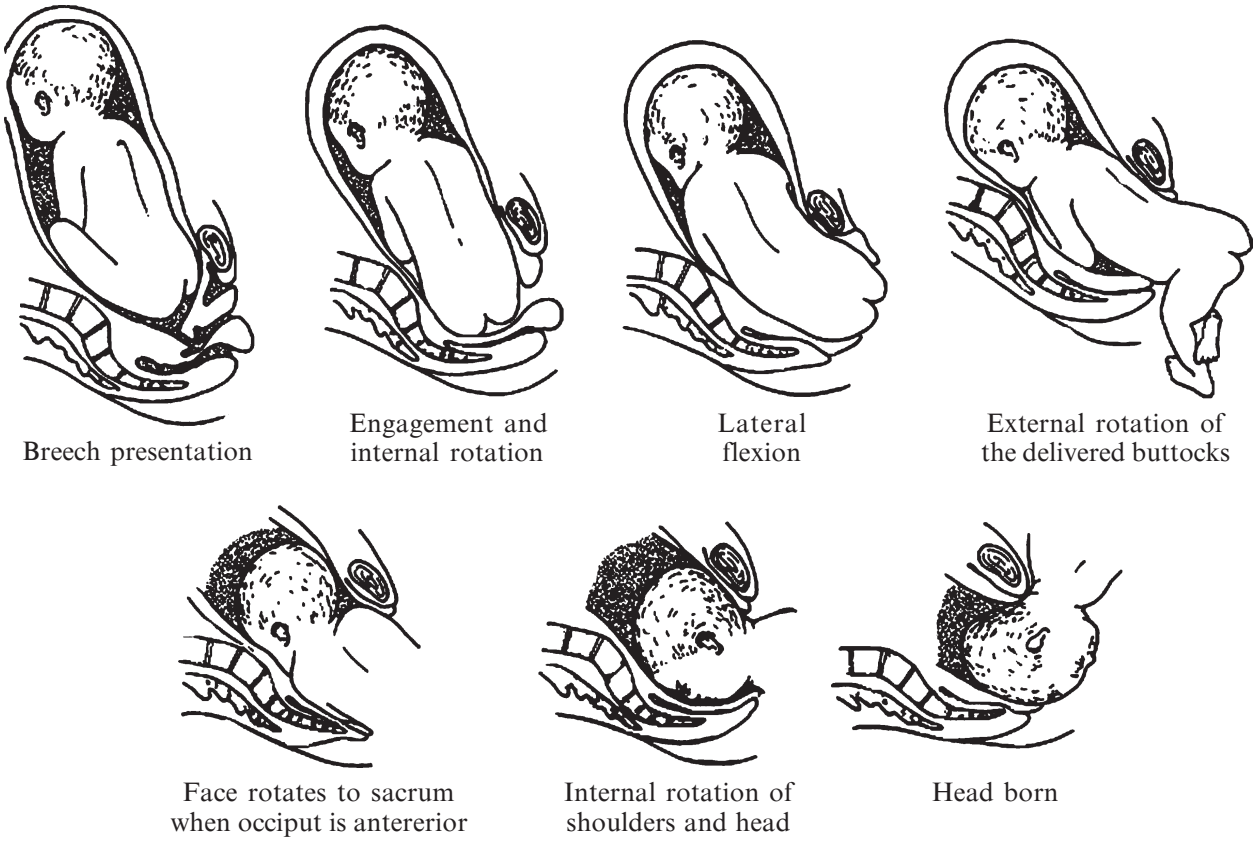


Fig. 16.1

— The anterior shoulder is born from behind the pubic symphysis by lateral flexion of the delivered trunk.

Management of vaginal breech delivery

Spontaneous onset labor increases the chance of successful vaginal delivery. Breech delivery should be conducted by a skilled obstetrician.

First stage.

— The management protocol is similar to that mentioned in normal labor.

— Adequate analgesia is given, epidural is preferred.

— Fetal status and progress of labor are monitored.

— Oxytocin infusion may be used for augmentation of labor.

Second stage.

Principles in conduction:

— Never to rush.

— Never pull from below but push from above.

— Always keep the fetus with the back anteriorly.

Assisted breech delivery

— Dorsal lithotomy position.

— Once the buttocks have entered the vagina and the cervix is fully dilated, tell the woman she can bear down with the contractions.

— The body is allowed to deliver spontaneously up to the level of the umbilicus. The operator then assists in delivery of the legs, shoulders, arms, and head.

— Do not pull the baby while the legs are being delivered.

— Hold the baby by the hips, as shown (Fig. 16.2, *a*).

Do not hold the baby by the flanks or abdomen as this may cause kidney or liver damage.

— The fetal trunk is then wrapped in a towel to support the body. When both scapulae are visible, the body is rotated counterclockwise. The operator locates the right humerus and laterally sweeps the arm across the chest and out the perineum. In a similar fashion, the body is rotated clockwise to deliver the left arm (Fig. 16.2, *b*).

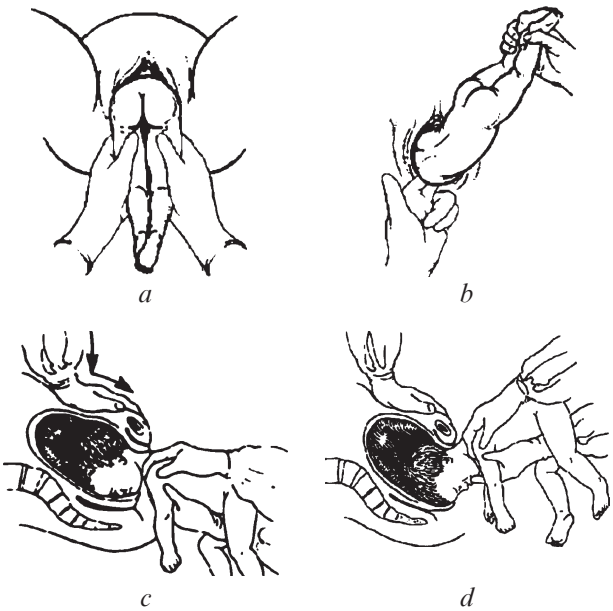


Fig. 16.2. a — hold the baby at the hips; *b* — shoulders engaged, posterior (left) shoulder at lower level in pelvis than anterior shoulder

— The head then spontaneously delivers by gently lifting the body upward and applying fundal pressure to maintain flexion of the fetal head.

— During partial breech extraction, the anterior shoulder may be difficult to deliver if it is impacted behind the pubic symphysis. In this event, the body is gently lifted upward toward the pubic symphysis, and the operator inserts 1 hand along the hollow of the maternal pelvis and identifies the posterior humerus of the fetus. By gentle downward traction on the humerus, the posterior arm can be easily delivered, thus allowing for easier delivery of the anterior shoulder and arm (Fig. 16.2, *c*).

— The operator may elect to manually assist in delivery of the head by performing the Mauriceau-Smellie-Veit maneuver.

In this procedure, the index and middle fingers of 1 of the operator's hands are applied over the maxilla as the body rests on the palm and forearm of the operator. Two fingers of the operator's other hand are applied on either side of the neck with gentle downward traction. At the same time, the body is elevated toward the pubic symphysis, allowing for controlled delivery of the mouth, nose, and brow over the perineum (Fig. 16.2, *d*)

In premature breech presentations, the incompletely dilated cervix may allow delivery to the smaller body, but the relatively larger after coming head may be entrapped. Prompt delivery is mandatory because severe asphyxia leading to death may rapidly ensue.

Complications of Breech Delivery

Maternal Risks

1. Perineal trauma.
2. PPH.

Fetal risks

1. Birth trauma:
— intracranial Hemorrhage;
— spinal cord injuries;
— injury to abdominal organs;
— injuries to limbs;
— brachial plexus injuries;
— others.
2. Birth Asphyxia.
3. Perinatal loss.

Birth Anoxia

Umbilical cord compression and prolapse may be associated with breech delivery, particularly in complete (5%) and footling (15%) presentations. This is due to the inability of the presenting part to fill the maternal pelvis, either because of prematurity or poor application of the presenting part to the cervix so that the umbilical cord is allowed to prolapse below the level of the breech (see below). Frank breech presentation offers a contoured presenting part, which is better accommodated to the maternal pelvis and is usually well applied to the cervix. The incidence of cord prolapse in frank breech is only 0.5% (the same as for cephalic presentations).

Compression of the prolapsed cord may occur during uterine contractions, causing moderate to severe variable decelerations in the fetal heart rate and leading to fetal anoxia or death. Continuous electronic monitoring is mandatory during labor in these cases to detect ominous decelerations. If they occur, immediate cesarean delivery must be performed.

Birth Injury

The incidence of birth trauma during vaginal breech delivery is 6.7%, 13 times that of cephalic presentations (0.51%). Only high forceps and internal version and extraction procedures have higher rates of birth injury than do vaginal breech deliveries. The types of perinatal injuries reported in breech delivery include tears in the tentorium cerebellum, cephalohematomas, disruption of the spinal cord, brachial palsy, fracture of long bones, and rupture of the sternocleidomastoid muscles. Vaginal breech delivery is the main cause of injuries to the fetal adrenal glands, liver, anus, genitalia, spine, hip joint, sciatic nerve, and musculature of the arms, legs, and back.

Factors contributing to difficult vaginal breech delivery include a partially dilated cervix, unilateral or bilateral nuchal arms, and deflexion of the head. The type of procedure used may affect the neonatal outcome.

Partially Dilated Cervix

Delivery of a breech fetus may progress even though the cervix is only partially dilated because the bitrochanteric and bisacromial diameters are smaller than the biparietal diameter. This is true especially in prematurity. The hips and shoulders may negotiate the cervix, but the aftercoming head becomes entrapped, resulting in difficult delivery and birth injury.

Nuchal Arms

During partial breech extraction and more often in total breech extraction, excessive downward traction on the body results in a single or double nuchal arm. This occurs because of the rapid descent of the body, leading to extension of 1 or both arms, which become lodged behind the neck. When delivery of the shoulder is difficult to accomplish, a nuchal arm should be suspected. To dislodge the arm, the operator rotates the body 180 degrees to bring the elbow toward the face. The humerus can then be identified and delivered by gentle downward traction. In cases of double nuchal arm, the fetus is rotated counterclockwise to dislodge and deliver the right arm and rotated clockwise to deliver the left arm. If this action is unsuccessful, the operator must insert a finger into the pelvis, identify the humerus, and possibly extract the arm, resulting in fracture of the humerus or clavicle. Nuchal arms cause a delay in delivery and increase the incidence of birth asphyxia.

Deflexion of the Head

Hyperextension of the head is defined as deflexion or extension of the head posteriorly beyond the longitudinal axis of the fetus (5% of all breech deliveries). Causes of hyperextension include neck cysts, spasm of the neck musculature, and uterine anomalies, but over 75% have no known cause. Although deflexion may be documented by ultrasonographic or x-ray studies weeks before delivery, there is little apparent risk to the fetus until vaginal delivery is attempted. At that time, deflexion causes impaction of the occipital portion of the head behind the pubic symphysis, which may lead to fractures of the cervical vertebrae, lacerations of the spinal cord, epidural and medullary hemorrhages, and perinatal death. If head deflexion is diagnosed prior to delivery, cesarean section should be performed to avert injury. Cesarean section cannot prevent injuries such as minor meningeal hemorrhage or dislocation of the cervical vertebrae, which may develop in utero secondary to longstanding head deflexion.

MULTIPLE PREGNANCY

When more than one fetus simultaneously develops in the uterus then it is called multiple pregnancy. Simultaneous development of two fetuses (twins) is the commonest; although rare, development of three fetuses (triplets), four fetuses (quadruplets), five fetuses (quintuplets or six fetuses (sextuplets)) may also occur.

Varieties:

Monozygotic twins (one-third) results from the fertilization of a single ovum.

Dizygotic twins: is the commonest (two-third) and results from the fertilization of two ova (Fig. 16.3).

Monozygotic multiple gestation

Monozygotic twins, which result from the fertilization of a single ovum by a single sperm, are always of the same sex. However, the twins may develop differently depending on the time of preimplantation division. Normally, monozygotic twins share the same physical characteristics (skin, hair and eye color, body build) and the same genetic features (blood characteristics: ABO, haptoglobin, serum group; histocompatible genes; skin grafting possible), and they are often mirror images of one another (one left-handed, the other right-handed, etc.). However, their fingerprints differ.

The paradox of "identical" twins is that they may be the antithesis of identical. The very earliest splits are sometimes accompanied by a simultaneous chromosomal error, resulting in heterokaryotypic monozygotes, 1 with Down syndrome and the other normal.

Monozygotic triplets result from repeated twinning (also called supertwinning) of a single ovum.

Dizygotic multiple gestation

Dizygotic twins are the product of 2 ova and 2 sperm. The 2 ova are released from separate follicles (or, very rarely, from the same follicle) at approximately the same time. Dizygotic (fraternal) twins may be of the same or different sexes. They bear only the resemblance of brothers or sisters and may or may not have the same blood type. Significant differences usually can be identified over time.

Approximately 75% of dizygotic twins are the same sex. Both twins are males in approximately

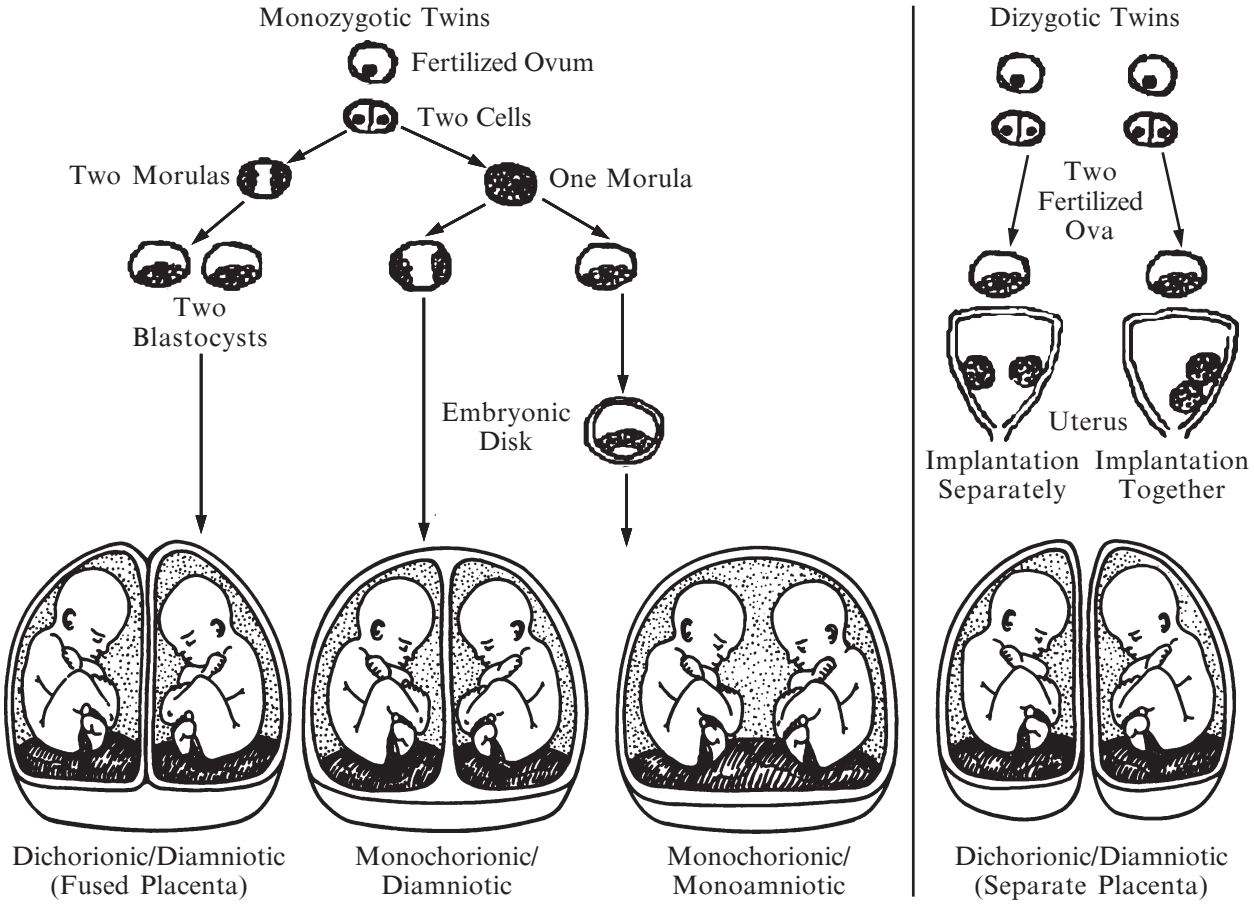


Fig. 16.3. Genesis of Monozygotic and Dizygotic Twins

45% of cases (a lesser preponderance of males in twins than in singletons) and both females in approximately 30%.

Many factors influence dizygotic twinning. Race is a factor, with multiple pregnancy most common in blacks, least common in Asians, and of intermediate occurrence in whites.

Dizygotic multiple pregnancy tends to be recurrent. Women who have borne dizygotic twins have a 10-fold increased chance of subsequent multiple pregnancy. Dizygotic twinning probably is inherited via the female descendants of mothers of twins; the father's genetic contribution plays little or no part. White women who are dizygotic twins or who are siblings of dizygotic twin mothers have a higher twinning rate among their offspring than do women in the general population.

Parity does not influence the incidence of dizygotic twinning but aging does, with the rate of dizygotic twinning peaking between 35 and 40 years of age and then declining sharply. In women who are twins or daughters of twins, the twinning rate peaks at approximately age 35 years, at which time it plateaus until almost age 45 years and then declines.

Placenta and Cord

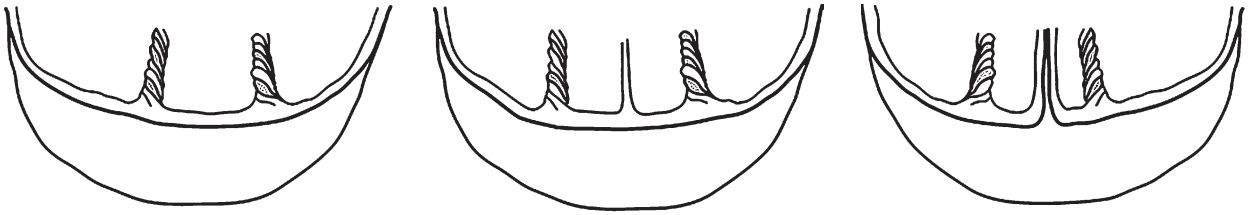
The placenta and membranes of monozygotic twins may vary considerably, depending on the time of initial division of the embryonic disk.

A monochorionic placenta can be identified by stripping away the amnion or amnions to reveal a single chorion over a common placenta. In virtually every case of monochorionic placenta, vascular communications between the 2 parts of the placenta can be identified by careful dissection or injection. In contrast, dichorionic placentas (of dizygotic twinning) only rarely have an anastomosis between the fetal blood vessels.

Placental and membrane examination is a certain indicator of zygosity in twins with monochorionic placentas because these are always monozygotic. Overall, approximately 1% of twins are monoamniotic, and these too are monozygotic. Determination of zygosity is clinically significant in case intertwin organ transplantation is needed later in life, as well as for assessing obstetric risks (Fig. 16.4).

Monochorionic placentation is associated with more disease processes as a result of placental vascular problems. Inequities of the placental circulation in 1 area (marginal insertion, partial infarction, or thinning) may lead to growth discordance between the twins. Because of vascular anastomoses in monochorionic placentation, multifetal reduction can only be performed with dichorionic placentation.

The most serious problem with monochorionic placentas is local shunting of blood — also called ***twin-twin transfusion syndrome*** *(TTTS)*. This occurs because of vascular anastomoses to each twin that are established early in embryonic life. The possible communications are artery to artery, vein to vein, and combinations of these. Artery-to-vein communication is by far the most serious; it is most likely to



Monochorionic, monoamniotic.
No membrane identified.
Shared placenta

Monochorionic, diamniotic.
Thin membrane.
Shared placentation.

Dichorionic, diamniotic.
Thick membrane.
Separate (fused) placentas

Fig. 16.4

cause twin-twin transfusion. In uncompensated cases, the twins, although genetically identical, differ greatly in size and appearance. The recipient twin is plethoric, edematous, and hypertensive. Ascites and kernicterus are likely. The heart, liver, and kidneys are enlarged (glomerulotubular hypertrophy). Hydramnios follows fetal polyuria. Although ruddy and apparently healthy, the recipient twin with hypervolemia may die of heart failure during the first 24 hours after birth. The donor twin is small, pallid, and dehydrated (from growth restriction, malnutrition, and hypovolemia). Oligohydramnios may be present. Severe anaemia, due to chronic blood loss to the other twin, may lead to hydrops and heart failure.

Various modes of treatment have been advocated for twin transfusion syndrome, including amnioreduction, amniotic septostomy, and laser ablation of communicating vessels.

Both twins are threatened by prolapse of the cord. The second twin may be harmed by premature separation of the placenta, hypoxia, constriction ring dystocia, operative manipulation, or prolonged anesthesia.

Fetal

Major malformations are present in approximately 2% of twin infants, compared with 1% of singletons, whereas minor malformations are found in 4% of twins compared with approximately 2.5% in singletons. Monozygotic twins are at higher risk than dizygotic twins.

Conjoined or Siamese twins result from incomplete segmentation of a single fertilized ovum between the 13th and 14th days; if cleavage is further postponed, incomplete twinning (2 heads, 1 body) may occur. Curiously, conjoined twins usually are female.

Each twin and its placenta generally weigh less than the newborn and placenta of a singleton pregnancy after the 30th week, but near term the aggregate weight may approach twice that of a singleton. In general, the larger the number of fetuses, the greater the degree of growth restriction. Normal-weight twins that differ considerably in birthweight commonly have diamniotic-dichorionic placentas. This suggests independent intrauterine growth of co-twins. Low-birth-weight monochorionic twins are the rule rather than the exception. Low birthweight in the various types of multiple pregnancy probably is evidence of growth restriction due to inadequate nutrition [30].

Risk factors of twin pregnancy:

— Increasing maternal age (30–35 years);
— Increasing parity (5 gravida onwards);
— Pituitary gonadotropin;
— Infertility therapy;
— Assisted reproductive technology;
— Genetic, hereditary;
— Nutritional factors;
— BMI > 35;
— Race.

Diagnosis pregnancy

The diagnosis of multiple pregnancy may be suspected on history and clinical examination: a history of infertility treatment or severe hyperemesis in early pregnancy are suggestive. Suspicion may be further raised if the uterus if found to be large for dates. Ultrasound examination in early pregnancy will differentiate these conditions and is the only method of diagnosing multiple pregnancy reliably.

Differential diagnosis

Multiple pregnancy must be distinguished from the following conditions.

— Singleton Pregnancy.
— Polyhydramnios.
— Hydatidiform mole.
— Abdominal Tumors Complicating Pregnancy.
— Fetal macrosomia (late in pregnancy).

Signs

— Uterus larger than expected (> 4 cm) for dates.
— Excessive maternal weight gain that is not explained by edema or obesity.
— Polyhydramnios, manifested by uterine size out of proportion to the calculated duration of gestation, is almost 10 times more common in multiple pregnancy (Fig. 16.5).
— Outline or ballottement of more than 1 fetus.
— Multiplicity of small parts.
— Simultaneous recording of different fetal heart rates, each asynchronous with the mother's pulse and with each other and varying by at least 8 bpm. (The fetal heart rate may be accelerated by pressure or displacement.)

Twins may present in various ways (Fig. 16.6).

Complications

• Maternal complication:
— minor disorders (nausea, vomiting, heart burn);
— anaemia;
— preeclampsia (25%) and eclampsia;

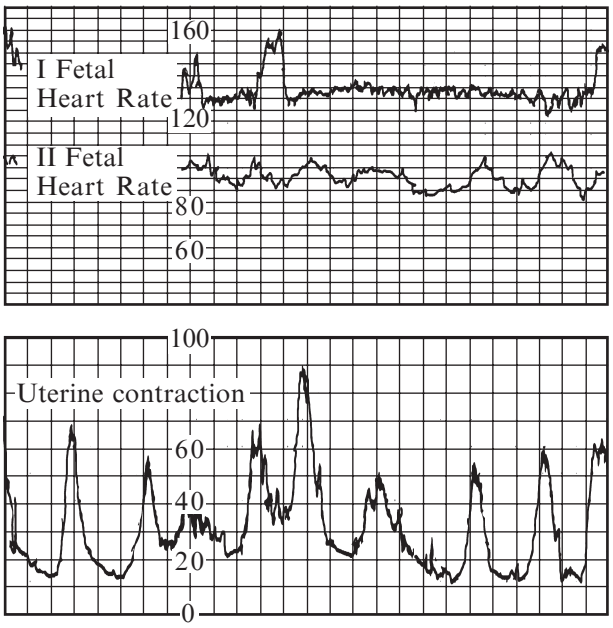


Fig. 16.5

— hydramnios (10%);
— folic acid deficiency;
— malpresentation;
— preterm labor (50%);
— bleeding (intra partum);
— PPH.

• Fetal complication:
— IUGR or intrauterine death;
— fetal anomalies;
— hydrocephalus;
— nicrocephaly;
— down syndrome;
— cardiac anomalies;
— locked twins.

• During puerperium:
— subinvolution;
— infection;
— lactation failure.

The Royal College of Obstetricians and Gynaecologists (RCOG) recommended antenatal care (Table 17).

Timing of delivery

Uncomplicated dichorionic — by 38 weeks. Uncomplicated monochorionic — by 37 weeks.

TTTS — depends on the current situation.

Mode of delivery

• Depends on the presentation of the 1st twin. Both vertex / 1st twin vertex — vaginal delivery.

• Indication for Elective Lower Segment Cesarean Section (LSCS):
— more than 2 fetuses;
— placenta previa;
— Severe preeclampsia;
— 1st twin malpresentation;
— abnormal uterine contractions;
— contracted pelvis;
— scarred uterus;
— monochorionic monoamniotic (MCMA) twin pregnancy;
— conjoint twin;
— IUGR in dichorionic twin;
— both fetuses or even first fetus with non-cephalic presentation;
— twins with complications: IUGR, conjoined twins; monoamniotic twins, monochorionic twins with TTS.

Emergency LSCS:
— fetal distress;
— cord prolapse in 1st baby;

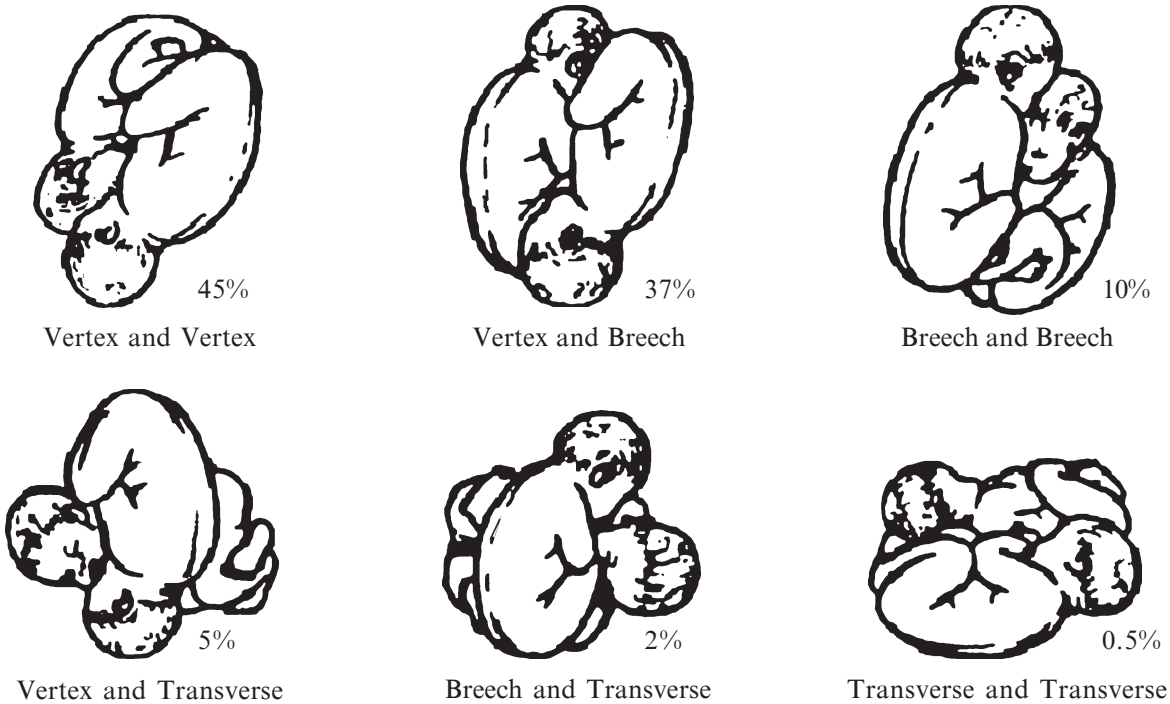


Fig. 16.6

Table 17

Antenatal care by the Royal College of Obstetricians and Gynaecologists (RCOG)

Dichorionic	Monochorionic
Lead clinician with multidisciplinary team	Lead clinician with multidisciplinary team
US at 10–13 weeks: viability, chorionicity, NT: aneuploidy	US at 10–13 weeks: viability,chorionicity, NT: aneuploidy, TTTS
Structural anomaly scan at 20–22 weeks	US surveillance for TTTS and discordant growth at 16weeks and then 2 weekly
Serial fetal growth scan eg:24, 28, 32 then 2–4 weekly	Structural anomaly scan 20–22 weeks.
BP monitoring and urinalysis at 20, 24, 28 and then 2 weekly	Fetal growth scan 2 weekly interval until delivery.
Discussion of mother's/family needs relating to twins	BP monitoring and urineanalysis at 20, 24, 28 then 2 weekly

Table 18

Examination of placenta and membranes

Dizygotic Twin	Monozygotic Twin
Two placenta, either completely separated or more commonly fused at the margin appearing to be one. No anastomosis between the two fetal vessels	Placenta is single Varying degrees of anastomosis between the two fetal vessels
Each fetus is surrounded by the amnion and chorion	Each fetus is surrounded by a separate amniotic sac with the chorionic layer common to both
Intervening membranes consist of 4 layers-amnion, chorion, chorion and amnion	Intervening membrane consists of two layers of amnion only

— non progress of labor;

— the 2nd twin is transverse, version failed after delivery of the 1st twin.

Placental characteristics in twins are presented in Table 18.

Management of difficult cases of twins

Locked twins is a rare, hazardous obstetric complication (Fig. 16.7).

Locked twins usually occur when the after-coming head of the first breech fetus is locked with the head of the second cephalic fetus. Of the different etiological factors, the most important are the age and parity of the mother and the size of the twins. A large pelvis with relatively small infants and decreased liquor volume following rupture of membranes are thought to be factors favoring interlocking. This complication of twin delivery occurs rarely. Cesarean with abdominal delivery of both fetuses may be the safest route.

Management Interlocking Commonest: after coming head of first baby getting locked with forecoming head of the second baby. Vaginal manipulation to separate chins of the fetuses Decapitation of first baby (dead), pushing up decapitated head, followed by delivery of second baby and lastly, delivery of decapitated head. Occasionally, two heads of both vertices get locked at the pelvic brim preventing engagement of either of the heads. Disengagement of the higher head: under general anesthesia, if fails, cesarean section is the alternative.

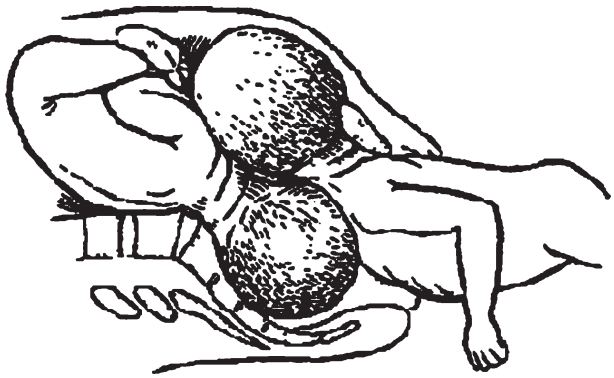


Fig. 16.7. Locked twins

Conjoined twins. It takes place extremely rare. Often diagnosed during pregnancy. Antenatal diagnosis is important. Benefits are: reduces maternal trauma and morbidity, improves fetal survival, helps to plan method of delivery, allows time to organize pediatric surgical team.

Fetus papyraceous. Sometimes a twin does not develop but becomes amorphous or shrivelled and flattened. This is called fetus papyraceous or compressus. It may be readily apparent or may be found wrapped in the membranes of the placenta.

Chapter 17
OBSTETRIC EMERGENCIES

AMNIOTIC FLUID EMBOLISM

This rare complication occurs when amniotic fluid suddenly enters the maternal circulation during labor or delivery. It carries a high maternal mortality (up to 80%) and is associated with multiparity, precipitate labor, uterine stimulation and cesarean section. Clinically there is sudden dyspnoea, fetal distress and hypotension, followed within minutes by cardiorespiratory arrest with or without seizures. It is often followed by hemorrhage from disseminated intravascular coagulation (DIC) and uterine atony, and may lead to acute renal failure (ARF) and adult respiratory distress syndrome (ARDS). It is often diagnosed by exclusion, but is ideally identified by the presence of fetal squamous cells on a blood film from a central line [5, 40, 48–50].

The unpredictable and catastrophic consequences are following: acute cardiopulmonary embarrassment, coagulation failure.

Symptoms:
— sudden onset of severe chest discomfort;
— difficult breathing;
— pallor;
— cyanosis;
— cardiovascular collapse.

Signs:
— venous congestion with raised JVP;
— output failure with tachycardia, hypotension, and peripheral vasoconstriction;
— coagulation failure, petechial skin;
— bleeding at puncture site, vaginal bleeding;
— coagulopathic signs may be the presenting features with out other symptoms.

Investigations:
— no time for investigations;
— 30 % will die in the first hour;
— suspicion when cardiorespiratory collapse during labor or soon after delivery;
— diagnosis only confirmed at postmortem , by finding pulmonary vasculature packed with amniotic debris and trophoblast or aspirating blood from the pulmonary artery and examine for trophoblastic tissue;
— coagulation profile requested.

Differential diagnosis: Thromboembolism.
Management
— Artificial ventilation.
— Cardiopulmonary resuscitation.
— Circulatory support.
— Dopamine, steroids may be useful.
— Correct acidosis.
— Treating coagulopathy.
— If the patient survive taken to the intensive care, anticoagulant, antifibrinolytics.
— Fetus is unlikely to survive.
— After stabilizing the maternal condition vaginal delivery is preferable.

Prognosis: Maternal mortality 90%.
Prevention is by avoiding excessive uterine contraction with oxytocin.

CORD PROLAPSE

Cord prolapse is defined as the descent of the umbilical cord through the cervix alongside or past the presenting part in the presence of ruptured membranes.
Cord presentation is the presence of the umbilical cord between the fetal presenting part and the cervix, with or without intact membranes (Fig. 17.1).

The incidence of cord prolapse is about 0.2–0.5% of all births.
Etiology
Anything which interferes with perfect adaptation of the presenting part to the lower uterine segment, disturbing the ball valve action may favor cord prolapse. Too often, more than one factor operates. The following are the associated factors:
— Malpresentations — the commonest being transverse (5–10%) and breech (3%) especially with flexed legs or footling and compound (10%) presentation.
— Placental factor — minor degree placenta previa with marginal insertion of the cord or long cord,
— Contracted pelvis.
— Prematurity.
— Twins.
— Hydramnios.
— Iatrogenic — low rupture of the membranes, manual rotation of the head.

Specific management (cord prolapse):

• Baby living or dead:

— Abnormal fetal heart tones (variable decels with cord prolapse)

— If the cord is pulsating, the fetus is alive (Fig. 17.2)

— If the cord is not pulsating, the fetus is dead. Deliver in the manner that is safest for the woman.

• Diagnose stage of labor by an immediate vaginal examination:

If the woman is in the first stage of labor, in all cases:

— insert a hand into the vagina and push the presenting part up to decrease pressure on the cord and dislodge the presenting part from the pelvis;

— place the other hand on the abdomen in the suprapubic region to keep the presenting part out of the pelvis;

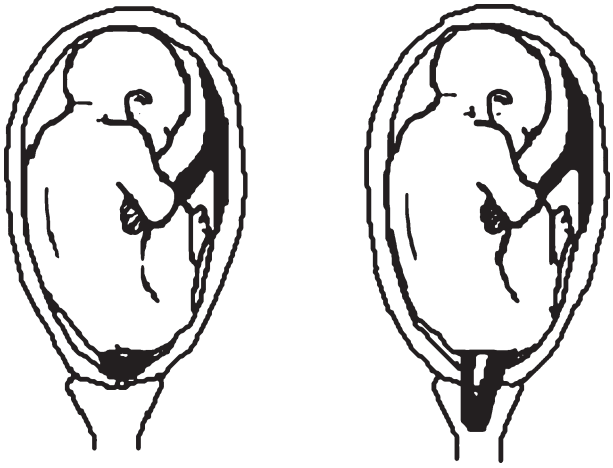
— once the presenting part is firmly held above the pelvic brim, remove the other hand from the vagina. Keep the hand on the abdomen until cesarean section;

— perform immediate cesarean section.

If the woman is in the second stage of labor:

— Expedite delivery with episiotomy and vacuum extraction or forceps.

|The best way to prevent is by not rupturing membranes before the presenting part engaged.



Cord presentation Cord prolapse

Fig. 17.1

SHOULDER DYSTOCIA

The term shoulder dystocia is defined to describe a wide range of difficulties encountered in the delivery of the shoulders. Shoulder dystocia occurs when either the anterior or the posterior fetal shoulder impacts on the maternal symphysis or on the sacral promontory. Shoulder dystocia cannot be predicted. Previous shoulder dystocia, prolonged first or second stage of labor are the important ones. Maneuvers to prevent shoulder dystocia may be used prophylactically in cases where it is anticipated.

Diagnosis

— The fetal head is delivered but remains tightly applied to the vulva.

— The chin retracts and depresses the perineum.

— Traction on the head fails to deliver the shoulder, which is caught behind the symphysis pubis.

Management

— Ask for help. Urgently mobilize all available personnel.

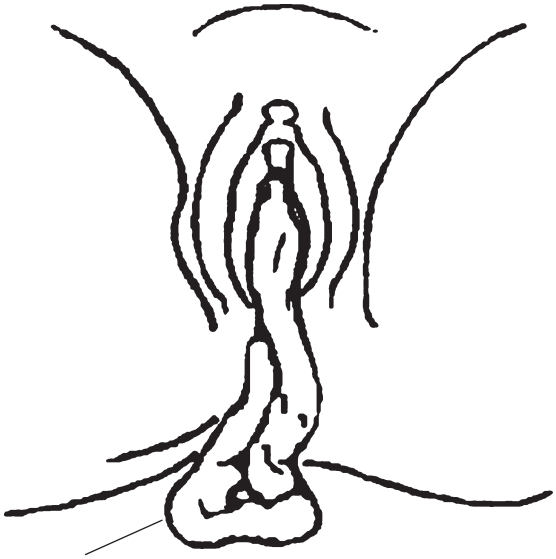
— Make an adequate episiotomy to reduce soft tissue obstruction and to allow space for manipulation (Fig. 17.3).

— With the woman on her back, ask her to flex both thighs, bringing her knees as far up as possible towards her chest (McRoberts position).

— Wearing high-level disinfected or sterile gloves:

— Apply firm, continuous traction downwards on the fetal head to move the shoulder that is anterior under the symphysis pubis.

Note: Avoid excessive traction on the fetal head as this may result in brachial plexus injury.



Umbilical cord

Fig. 17.2

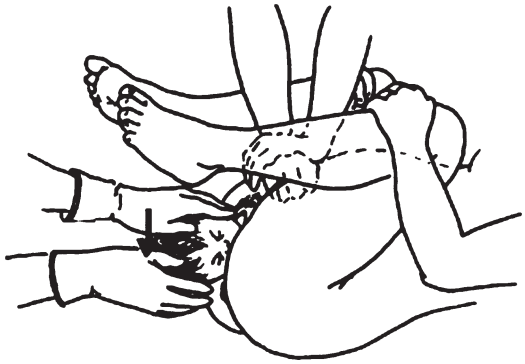


Fig. 17.3

— Have an assistant simultaneously apply suprapubic pressure downwards to assist delivery of the shoulder.

Note: Do not apply fundal pressure. This will further impact the shoulder and can result in uterine rupture.

Fig. 17.4

• If the shoulder still is not delivered:

— Insert a hand into the vagina along the baby's back.

— Apply pressure to the shoulder that is anterior in the direction of the baby's sternum to rotate the shoulder and decrease the diameter of the shoulders.

— If needed, apply pressure to the shoulder that is posterior in the direction of the sternum.

• If the shoulder still is not delivered despite the above measures:

— insert a hand into the vagina;

— grasp the humerus of the arm that is posterior and, keeping the arm flexed at the elbow, sweep the arm across the chest. This will provide room for the shoulder that is anterior to move under the symphysis pubis (Fig. 17.4);

• If all of the above measures fail to deliver the shoulder, other options include:

— fracture the clavicle to decrease the width of the shoulders and free the shoulder that is anterior;

— apply traction with a hook in the axilla to extract the arm that is posterior.

Chapter 18
OPERATIVE DELIVERY

An operative delivery refers to an obstetric procedure in which active measures are taken to accomplish delivery. Operative delivery can be divided into operative vaginal delivery and cesarean delivery. The last several years have seen a trend toward a decrease in the operative vaginal delivery rate but a climb in the cesarean section rate. In addition, the use of a vacuum during delivery has become more common than forceps. A vacuum generally requires less anesthesia and pain-relieving medications than forceps. Use of a vacuum is associated with less risk for a cesarean delivery compared to forceps. It's also associated with less risk of maternal death and maternal trauma [40, 48–50].

The success and safety of these procedures are based upon operator skill, proper timing, and ensuring that proper indications are met while contraindications are avoided. This chapter explains how each procedure is performed, the indications and contraindications to the procedure, the potential complications, and how to minimize complications.

Forceps are placed around the baby's head. A vacuum extractor uses suction to adhere to the baby's head. With either device, the baby is gently pulled as the woman pushes (Fig. 18.1).

The ability to perform an operative vaginal delivery with forceps or vacuum remains a vital skill for family physicians who provide maternity care. The World Health Organization considers operative vaginal delivery to be a critical part of basic emergency obstetric care.

Although rates of operative vaginal delivery are dropping, vacuum has emerged as the most popular delivery instrument.

VACUUM EXTRACTION (VENTOUSE)

The vacuum extractor works by allowing the external traction forces applied to the fetal scalp to be transmitted to the fetal head. The traction on the vacuum apparatus increases the forces of delivery and facilitates passage of the fetus through the pelvis. In order for delivery to be accomplished, both traction on the fetal scalp and compression of the fetal head occur.

TYPES OF VACUUM DEVICES

Originally, vacuum devices had a rigid metal cup with a separate suction catheter attached laterally and connected to a foot-operated pedal. Today's vacuum cups can be soft or rigid and can be different shapes and sizes (Fig. 18.2).

The flatter cup allows for better placement at the flexing position on the fetal head, which is usually much further back in the sacral hollow during occipitoposterior presentation. These handheld devices are intended for single use and are disposable.

Flexible vacuum delivery system is given at the Fig. 18.3.

Indications for vacuum delivery:

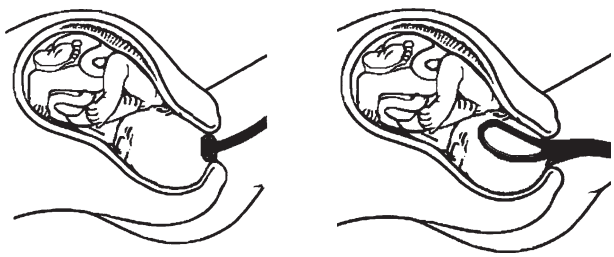
— maternal distress in 2nd stage of labor;

— fetal distress in 2nd stage of labor;

— prolonged 2nd stage of labor;

— for prophylactic use in mothers with cardiovascular, respiratory, cerebrovascular disorders.

Contraindications to use of vacuum for operative vaginal delivery:



Vacuum extractor Forcepts

Fig. 18.1

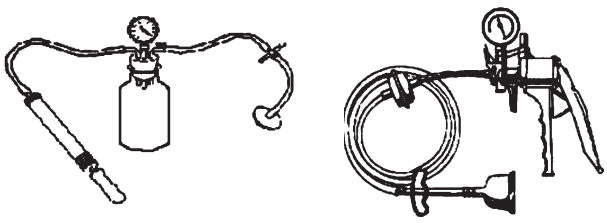


Fig. 18.2. Examples of different vacuum devices

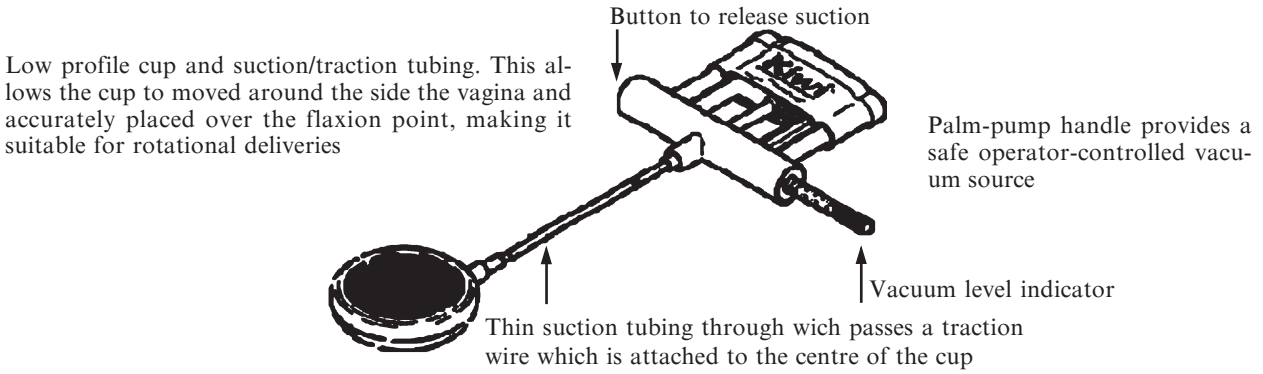


Fig. 18.3. Flexible vacuum delivery system

— Cephalopelvic disproportion;
— Gestational age less than 34 weeks;
— Known fetal conditions that affect bone mineralization or bleeding disorder;
— Noncephalic or facial presentation.

Review for conditions:
— Vertex presentation;
— Cervix fully dilated;
— The amniotic membranes ruptured;
— Fetal head at least at 0 station or no more than 2/5 palpable above symphysis pubis;
— Adequate anesthesia.
— The patient must be placed in the dorsal lithotomy position. The legs should be comfortably placed in stirrups with the hips flexed and abducted.
— Check all connections and test the vacuum on a gloved hand.
— Provide emotional support and encouragement.
— Assess the position of the fetal head by feeling the sagittal suture line and the fontanelles.

Landmarks of the fetal skull:
— The center of the cup should be on the flexion point of the head, which is a point located on the sagittal suture, 3 cm in front of the posterior fontanelle (Fig. 18.4).
— Traction at this point results in maximum flexion (flexing median application) (Fig. 18.5).

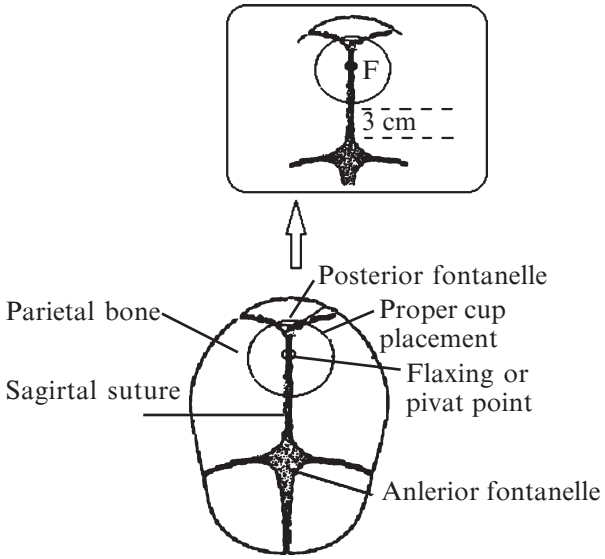


Fig. 18.4

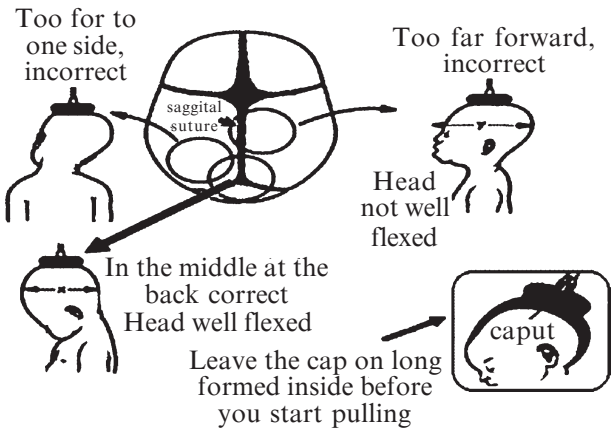


Fig. 18.5.

VACUUM APPLICATION

— An episiotomy may be needed for proper placement at this time. If an episiotomy is not necessary for placement, delay the episiotomy until the head stretches the perineum or the perineum interferes with the axis of traction. This will avoid unnecessary blood loss (Fig. 18.6)
— Check the application. Ensure there is no maternal soft tissue (cervix or vagina) within the rim.
— With the pump, create a vacuum of 0.2 kg/cm^2 negative pressure and check the application.
— Increase the vacuum to 0.8 kg/cm^2 and check the application.
— After maximum negative pressure, start traction in the line of the pelvic axis and perpendicular to the cup. If the fetal head is tilted to one side or not flexed well, traction should be directed in a line that will try to correct the tilt or deflexion of the head (i.e. to one side or the other, not necessarily in the midline) (Fig. 18.7).
— With each contraction, apply traction in a line perpendicular to the plane of the cup rim. Place

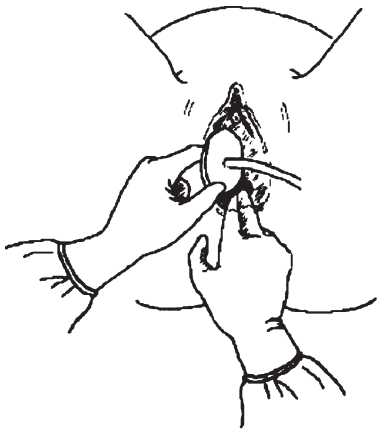


Fig. 18.6

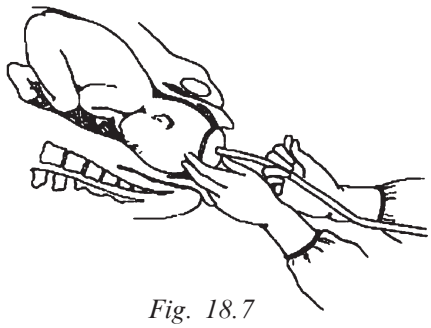


Fig. 18.7

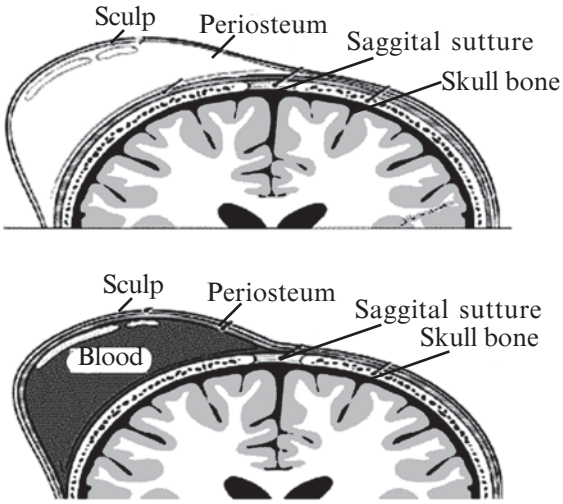


Fig. 18.8

a finger on the scalp next to the cup during traction to assess potential slippage and descent of the vertex.

— Between contractions check:

— fetal heart rate;

— application of the cup.

Note:

— Never use the cup to actively rotate the baby's head. Rotation of the baby's head will occur with traction.

— The first pulls help to find the proper direction for pulling.

— Do not continue to pull between contractions and expulsive efforts.

— With progress, and in the absence of fetal distress, continue the "guiding" pulls for a maximum of 30 minutes.

• Vacuum extraction failed if the:

— fetal head does not advance with each pull;

— fetus is undelivered after three pulls with no descent, or after 30 minutes;

— cup slips off the head twice at the proper direction of pull with a maximum negative pressure;

— every application should be considered a trial of vacuum extraction. Do not persist if there is no descent with every pull.

Complications

Complications usually result from not observing the conditions of application or from continuing efforts beyond the time limits stated above.

Fetal complications

• A localized scalp edema (caput succedaneum or chignon) under the vacuum cup is harmless and disappears in a few hours (Fig. 18.8).

• Cephalohematoma is a collection of blood between the skull bones and tissue of a baby's head. These complications are associated with trauma during birth.

Cephalohematoma requires observation and usually will clear in three to four weeks.

• Scalp abrasions (common and harmless) and lacerations may occur.

• Intracranial bleeding is extremely rare and requires immediate intensive neonatal care.

Maternal complications

• Tears of the genital tract may occur.

FORCEPS DELIVERY

The obstetric forceps is an instrument designed to assist with delivery of the baby's head. It is used either to expedite delivery or to assist with certain abnormalities in the cephalopelvic relationship that interfere with advancement of the head during labor. The primary functions of the forceps are to assist with traction of the fetal head and/or to assist with rotation of the fetal head to a more desirable position.

In fact, many investigators are concerned that the use of forceps is becoming a lost art. The reasons often cited as contributing to the decline in the use of forceps are:

— medicolegal implications and fear of litigation;

— reliance on cesarean section as a remedy for abnormal labor and suspected fetal jeopardy;

— perception that the vacuum is easier to use and less risky to the fetus and mother;

— decreased number of residency programs that actively train residents in the use of forceps.

THE OBSTETRIC FORCEPS

The obstetric forceps consist of 2 matched parts that articulate or "lock". Each part is composed of a blade, shank, lock, and handle. Each blade is designed so that it possesses 2 curves: the cephalic curve, which permits the instrument to be applied accurately to the sides of the baby's head, and the pel-

vic curve, which conforms to the curved axis of the maternal pelvis. The tip of each blade is called the toe. The front of the forceps is the concave side of the pelvic curve. The blades are referred to the left and right according to the side of the mother's pelvis on which they lie after application. During application, the handle of the left blade is held in the left hand, and the blade is applied to the left side of the mother's pelvis. Conversely, the handle of the right blade is held in the right hand and inserted so as to lie on the right side of the mother's pelvis. When the blades are inserted in this order, the right shank comes to lie atop the left so that the forceps articulate, or lock, as the handles are closed (Fig. 18.9).

Physicians have been modifying 1 or more of the 4 basic parts since forceps were first invented. Although more than 600 kinds of forceps have been described, only a few are currently in use. Although it is beyond the scope of this chapter to discuss all the different varieties of forceps and their indications, a brief comment on the more common types of forceps is appropriate. Simpson or Elliot forceps are most often used for outlet vaginal deliveries, whereas Kielland or Tucker-McLane forceps are used for rotational deliveries. Piper forceps are used in the United States for delivery of the after coming head. The pelvic and cephalic curve, shank, blade, lock, and handle are different for each type of forceps. These features determine the type of forceps that is best suited for the appropriate indication. For example, Piper forceps, which are specifically designed for breech deliveries, have a reverse pelvic curve compared to other forceps. Simpson forceps are suited for application to the molded fetal head, whereas Tucker-McLane forceps or Kielland forceps are more appropriate for application to the fetal head with little or no molding (Fig. 18.10).

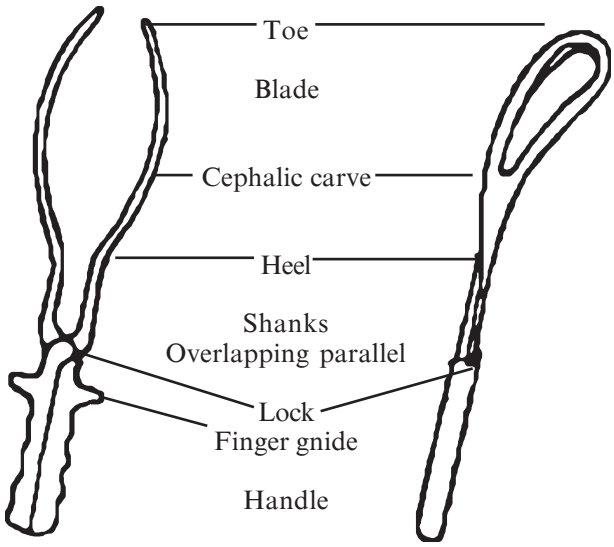
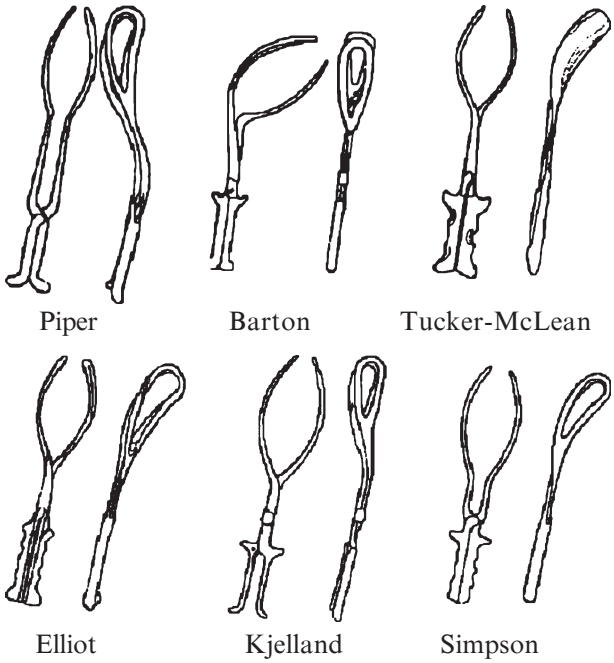


Fig. 18.9. De Lee moditication of Simpson forceps



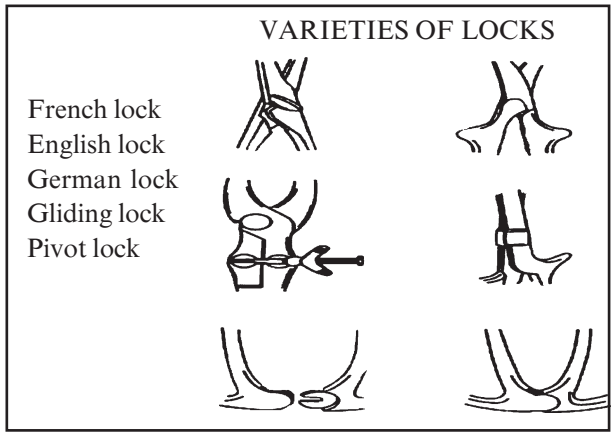


Fig. 18.10

CLASSIFICATION OF FORCEPS DELIVERIES

In 1988 the American College of Obstetricians and Gynecologists redefined the classification of forceps. This classification uses the leading bony point of the fetal skull and its relationship to the maternal ischial spines in centimeters as the point of reference. Each station of the fetal head refers to the relationship of the leading bony part of the fetal skull with respect to the ischial spines. The fetal head is said to be at 0 station when the head is at the level of the spines. When the head is above this level, the station is described as –1 through –5, corresponding to the number of centimeters above the level of the ischial spines. When the head is below this level, the station is described as +1 through +5, corresponding to the number of centimeters below the level of the ischial spines.

THE CLASSIFICATION OF FORCEPS

Outlet forceps is the application of forceps when (a) the fetal scalp is visible at the introitus without separating the labia, (b) the fetal skull has reached the pelvic floor, (c) the sagittal suture is in the anteroposterior diameter or in the right or left occiput anterior or posterior position, and (d) the fetal head

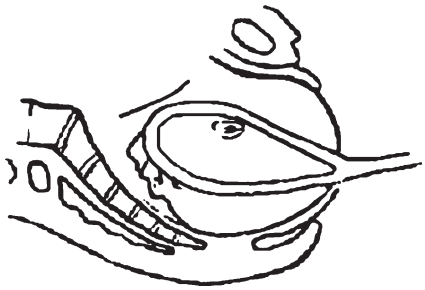


Fig. 18.11. Low forcepts

Fig. 18.12. Mid forcepts

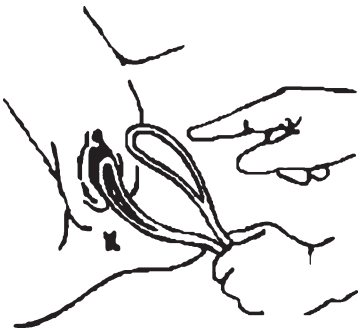


Fig. 18.13

is at or on the perineum. According to this definition, rotation of the fetal head must be equal to or less than 45 degrees (Fig. 18.11).

Low forceps are the application of forceps when the leading point of the fetal skull is at station +2 or greater and not on the pelvic floor. Low forceps have two subdivisions: (a) rotation less than or equal to 45 degrees and (b) rotation greater than 45 degrees (Fig. 18.11).

Mid forceps are the application of forceps when the head is engaged but the leading point of the fetal skull is above station +2.

Only rarely should an attempt be made at forceps delivery above station +2. Under unusual circumstances, such as sudden onset of severe fetal or maternal compromise or transverse arrest, application of forceps above station +2 can be attempted while simultaneously initiating preparation for a cesarean delivery in case the forceps maneuver is unsuccessful (Fig. 18.12).

Under no circumstances should forceps be applied to an unengaged head.

Indications for forceps delivery:

— nonreassuring fetal heart rate pattern;
— shortening of the second stage of labor for fetal or maternal reasons;
— prolonged 2nd stage of labor;
— maternal exhaustion.

Contraindications for forceps:

— absence of full dilatation of cervix;
— cephalopelvic disproportion;
— hydrocephalic infant;
— gestational age less than 34 weeks;
— known fetal conditions that affect bone mineralization or bleeding disorder;
— malpresentation (noncephalic or facial presentation);
— preeclampsia.

Identification of blade of forceps

• Take the blade of forceps.

• Place it in front of the maternal pelvis, tip of the forceps directed towards the maternal head, concavity of the pelvic curve directed toward the midline of the pelvis.

• The blade which corresponds to the left side of the mother is the left blade and to the right side — the right blade respectively.

Review for conditions:

— vertex presentation;
— cervix fully dilated;
— the amniotic membranes ruptured;
— fetal head at +2 or +3 station or 0/5 palpable above the symphysis pubis. At a minimum, the sagittal suture should be in the midline and straight, guaranteeing an occiput anterior or occiput posterior position. Fetal head at least at 0 station or no more than 2/5 palpable above symphysis pubis (Fig. 18.13);
— adequate anesthesia.

• The patient must be placed in the dorsal lithotomy position. The legs should be comfortably placed in stirrups with the hips flexed and abducted.

• Assemble the forceps before application. Ensure that the parts fit together and lock well.

• Provide emotional support and encouragement.

• Assemble the forceps before application. Ensure that the parts fit together and lock well.

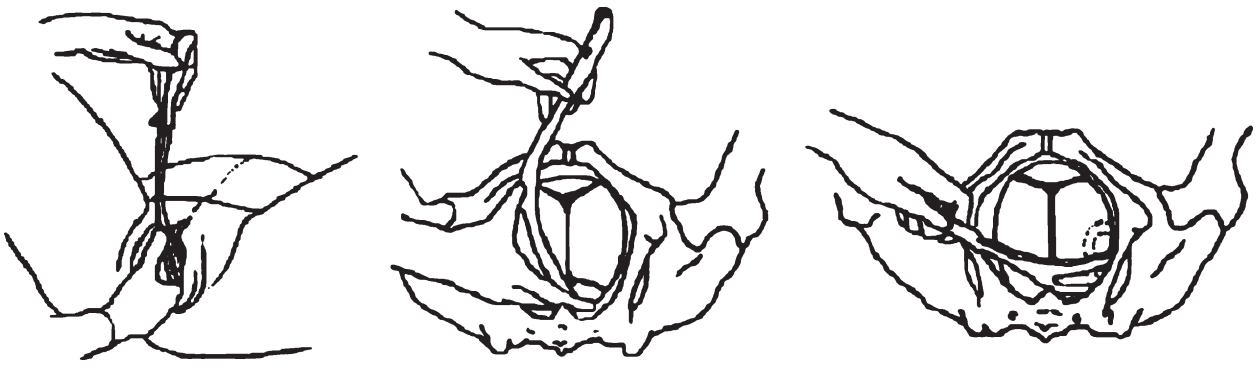


Fig. 18.14. Applying the left blade of the forceps

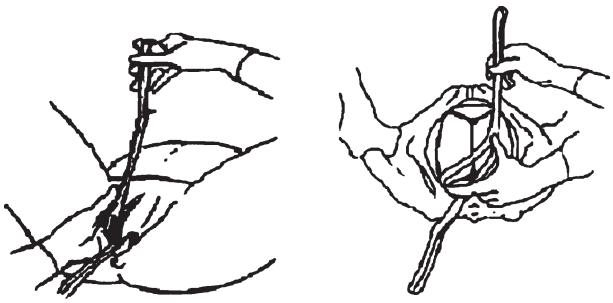


Fig. 18.15. Applying the right blade of the forceps

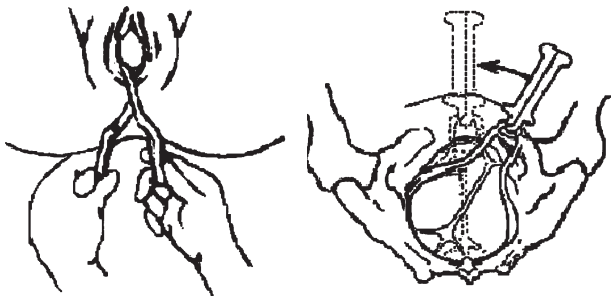


Fig. 18.16

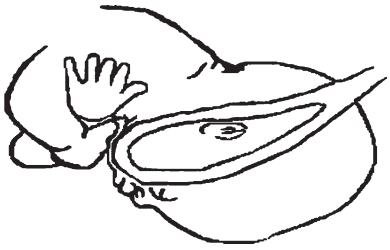


Fig. 18.17. Correct cephalic application

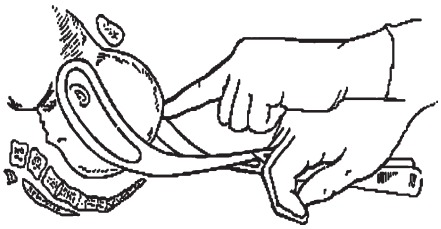


Fig. 18.18

Fig. 18.19

• Insert two fingers of the right hand into the vagina on the side of the fetal head. Slide the left blade gently between the head and fingers to rest on the side of the head (Fig. 18.14).

• The right blade is held by the right hand and is applied between the left hand that protects the vagina and the head by negotiating the cephalic and pelvic curve (Fig. 18.15).

• Forceps correctly applied along the occipitomental diameter of the head (Fig. 18.16).

• Depress the handles and lock the forceps.

Clinical checks for correct forceps application:

• The sagittal suture lies in the midline of the shanks (Fig. 18.17).

• The operator cannot place more than a fingertip between the fenestration of the blade and the fetal head.

The posterior fontanelle is not more than one finger-breadth above the plane of the shanks.

Difficulty in locking usually indicates that the application is incorrect. In this case, remove the blades and recheck the position of the head. Reapply only if rotation is confirmed.

— After locking, apply steady traction with each contraction.

Traction:

— Steady and intermittent traction to be applied during contraction, first downwards (horizontal), backwards, forwards and lastly upwards (Fig. 18.18).

— In outlet forceps — only two fingers are to be introduced. Traction is applied straight horizontal, upward and then forwards (Fig. 18.19).

• Between contractions check:

— fetal heart rate;

— application of forceps.

• Removal of blades — right blade should be removed first.

Failure

• Forceps failed if:

— fetal head does not advance with each pull;

— fetus is undelivered after three pulls with no descent or after 30 minutes.

• Every application should be considered a trial of forceps. Do not persist if the head does not descend with every pull.

Complications of obstetric forceps delivery

Fetal complications

• Injury to facial nerves requires observation.

• Lacerations of the face and scalp may occur.

• Fractures of the face and skull.

Maternal complications

• Tears of the genital tract may occur.

Examine the woman carefully and repair any tears to the cervix or vagina or repair episiotomy.

CESAREAN SECTION

A cesarean section refers to the delivery of a fetus, placenta, and membranes through an abdominal and uterine incision. The first documented cesarean section on a living person was performed in 1610. The patient died 25 days later. Since that time, numerous advances have made cesarean section a safe procedure. In the past 35 years, the rate of cesarean section has steadily increased from 5% to approximately 25%. Over this time, the maternal mortality ratio (maternal deaths per 100,000 births) has decreased from almost 300 to less than 10 [11, 32, 47, 50].

The following factors are often cited as contributing to the increasing cesarean section rate:

— lower vaginal delivery rates;

— lower rates of vaginal birth after cesarean section;

— fewer vaginal breech deliveries.

In order for the practitioner to perform this common operation safely, he or she must be aware of the indications, risks, operative technique, and potential complications of the procedure.

INDICATIONS

Cesarean section is used in cases where vaginal delivery either is not feasible or would impose undue risks to the mother or baby. Some of the indications for cesarean section are clear and straightforward, whereas others are relative. In some cases, fine judgement is necessary to determine whether cesarean section or vaginal delivery would be better. It is not practical to list all possible indications; however, hardly any obstetric complication has not been dealt with by cesarean section. The following indications are currently the most common (Fig. 18.20).

The absolute indications for cesarean section:
— fetal distress;
— cord prolapse;
— fetal malposition and malpresentation;
— failed induction of labor;
— failure to progress of labor;
— labor dystocia;
— cephalopelvic distortion;
— previous uterine surgery (myomectomy);
— prior uterine rupture;
— outlet obstruction (fibroids);
— placenta previa, large placental abruption.

The relative indications for cesarean section:
— fetal anomaly, hydrocephalus;
— elective repeat cesarean section;
— maternal disease;
— severe preeclampsia;
— cardiovascular, cerebrovascular disorders.

TYPES OF CESAREAN SECTION INCISION

• Joel Cohen (Fig. 18.21)
Advantages:
— Shorten surgical time,
— Minimization of tissue damage,
— Less blood loss.
Disadvantage:
— Limited ability to extend laterally to enlarge the incision.

• Pfannenstiel
Advantages:
— Reduced risk disruption (and thereby reduced risk of wound infection)
— Reduced risk of incisional hernia
— Improved cosmetic result
— Reduced risk of hypertrophic scars
— Reduced postoperative pain
Disadvantage:
— Less visualization of the uterus

Vertical
Advantages:
— Quicker to perform
— Better visualization on the uterus
— Can quickly extend upward for greater visualization if needed
Disadvantage:
— Early visible when healed
— Greater chance of dehiscence and hernia formation

Low-Transverse Cesarean Section (Fig. 18.22)
Because the low-transverse uterine incision is associated with less blood loss and the risk of subsequent uterine rupture is less than with a classic cesarean section, this type of cesarean delivery is performed more frequently.

— After the peritoneal cavity is opened and the uterus identified, the bladder fold of the peritoneum is picked up with tissue forceps and incised transversely. The bladder is bluntly separated from the anterior aspect of the uterus inferiorly for a distance of 3–4 cm. The bladder is held away from this area by a specially designed bladder retractor.

— A transverse incision is made through the anterior uterine wall with the scalpel. Using either bandage scissors or fingers, the transverse incision is extended in a semilunar fashion and extended superiorly at the lateral edges in order to avoid the uterine vessels.

— The loose vesicouterine serosa is grasped with the forceps. The loose serosa above the upper margin of the bladder is elevated and incised laterally.

— The myometrium is incised carefully to avoid cutting the fetal head.

— After entering the uterine cavity, the incision is extended laterally with bandage scissors or with fingers.

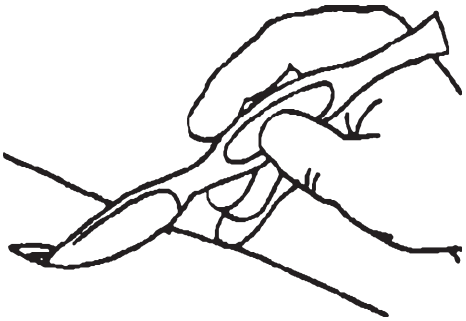


Fig. 18.20

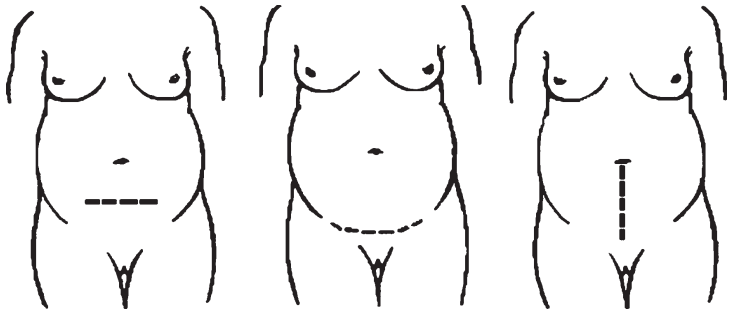


Fig. 18.21. Types of C-cection incision

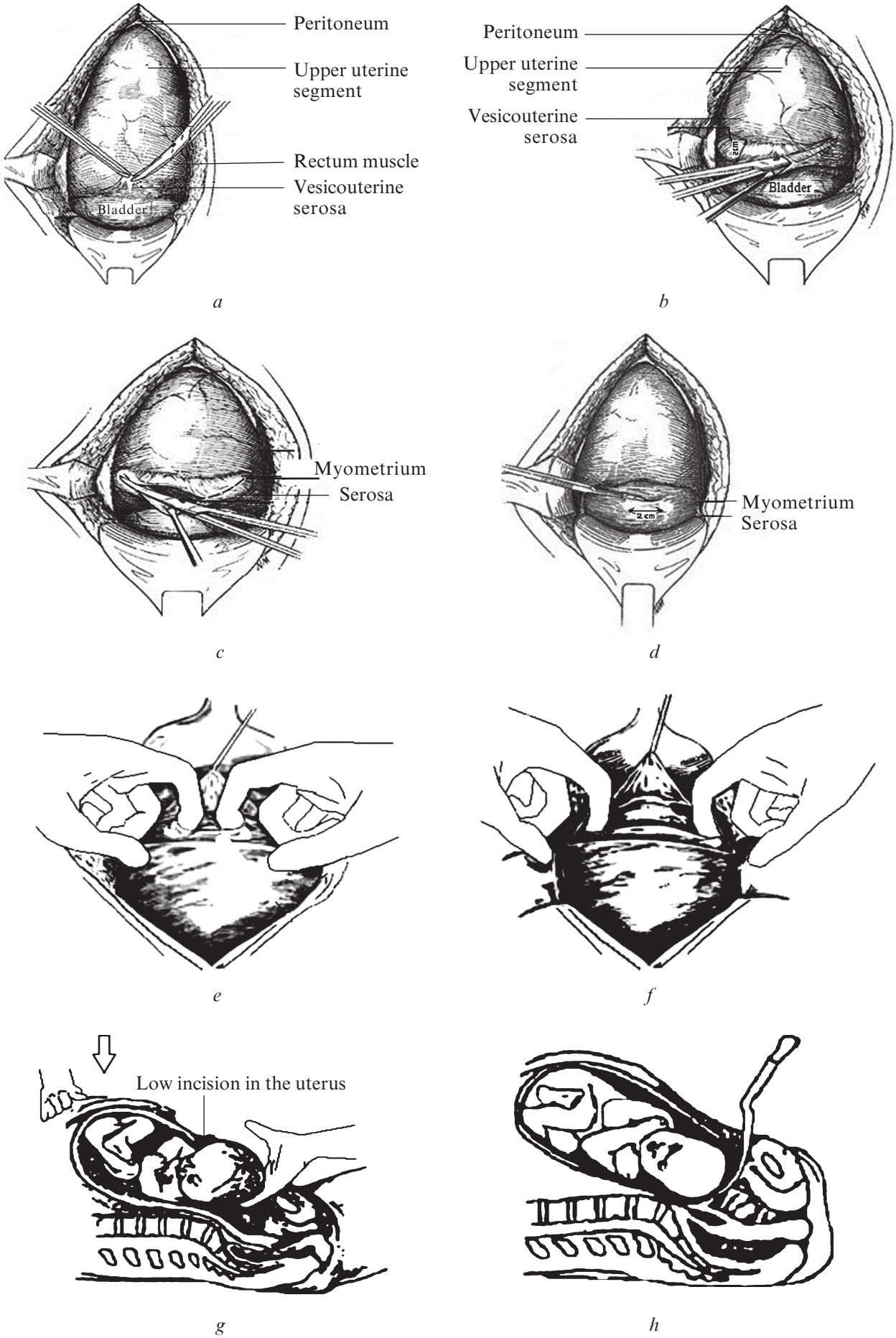

Peritoneum
Upper uterine segment
Rectum muscle
Vesicouterine serosa
Bladder
a
Peritoneum
Upper uterine segment
Vesicouterine serosa
2 cm
Bladder
b
Myometrium
Serosa
c
2 cm
Myometrium
Serosa
d
e
f
Low incision in the uterus
g
h

Fig. 18.22, a–h

— Immediately after incising the uterus and rupturing the fetal membranes, the fingers are insinuated between the symphysis pubis and the fetal head until the posterior surface is reached.

• The head is lifted carefully anteriorly and, as necessary, superiorly to bring it from beneath the symphysis forward through the uterine and abdominal incisions. As the fetal head is lifted through the incision, pressure usually is applied to the uterine fundus through the abdominal wall to help expel the fetus. The shoulders are delivered, and the oxytocin infusion is begun (Fig. 18.23).

• If the maneuver can be easily done, the fetal presenting part is elevated with the hand, making sure not to flex the wrist, thereby increasing the possibility of extension of the incision inferiorly towards the cervix. If the head is located deep in the pelvis, the head can safely be pushed up by an assistant inserting a hand into the vagina to elevate the fetal head for ease of delivery.

• Placenta bulging through the uterine incision as the uterus contracts.

• After the baby and placenta are delivered, the uterus is exteriorized and clamps are placed on the cut edge of the uterus in areas of significant bleeding from the uterine sinuses. The uterine incision is generally closed (1 layer).

• After adequate hemostasis has been achieved, the bladder peritoneum either is reapproximated with suture or left in place. Before the uterus is returned to the peritoneal cavity, the adnexa should be inspected for the presence of any pathology, such as ovarian cysts.

Complications:

— postpartum hemorrhage;

— endometritis, and wound infection.

Administering prophylactic antibiotics and ensuring hemostasis prior to closure of the abdomen have helped decrease the incidence of these complications. The major factors affecting healing of the uterine incision are hemostasis, accuracy of apposition, quality and amount of suture material, and avoidance of infection and tissue strangulation. It can generally be stated that the longer the operative procedure, the greater the likelihood of postoperative complications. Disasters following cesarean section are rare. Some clearly are not preventable. Others are the direct result of faulty surgical technique, especially lack of attention to hemostasis, inept or ill-chosen anesthesia, inadequate blood product replacement or

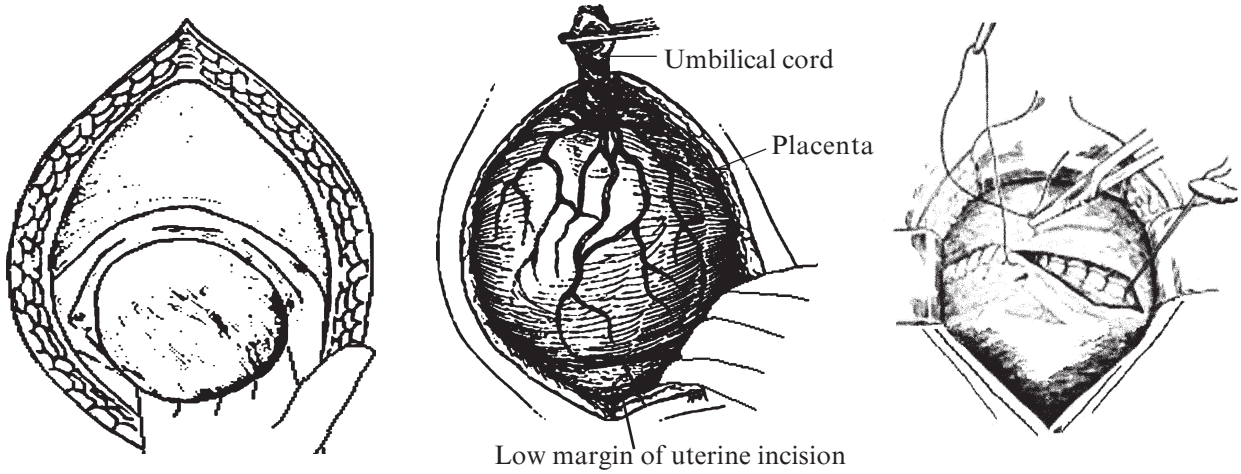


Fig. 18.23

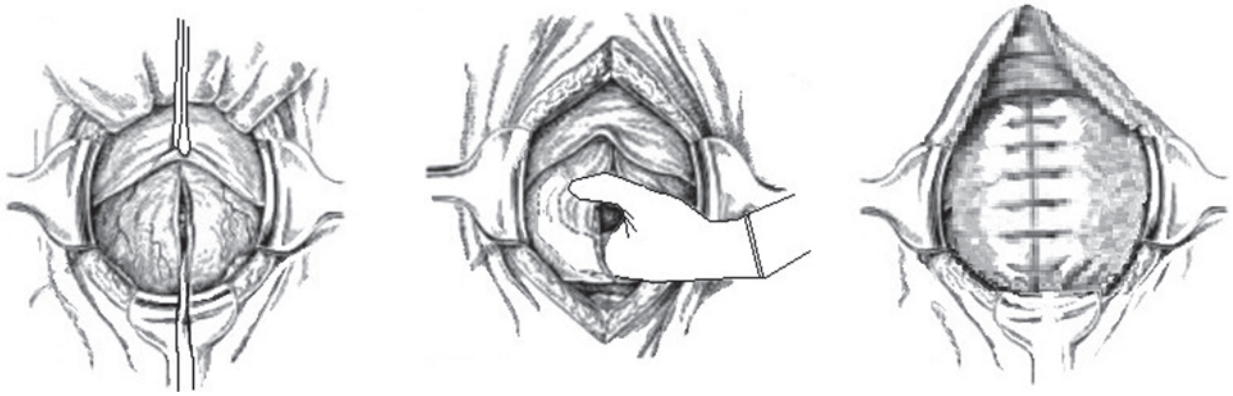


Fig. 18.24

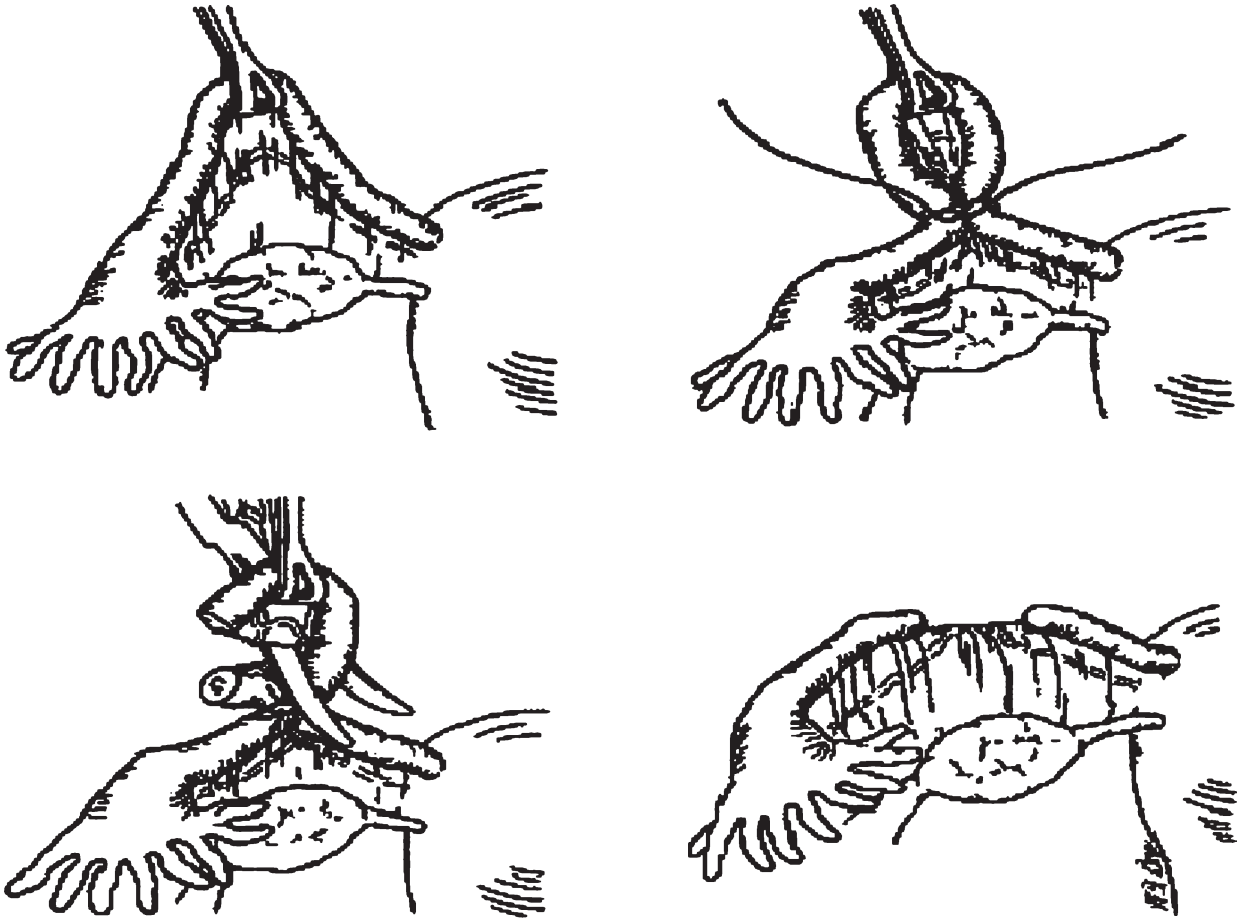


Fig. 18.25

transfusion of mismatched blood, and delayed diagnosis or mismanagement of infection.

Unfortunately, little information about the integrity of a particular scar in a subsequent pregnancy is gained by inquiry into the presence or absence of postoperative infection and location of the incision. In a later pregnancy, pain in the area of the scar may suggest dehiscence.

High vertical ("classical") incision (Fig. 18.24)

• Open the abdomen through a midline incision skirting the umbilicus. Approximately one-third of the incision should be above the umbilicus and two-thirds below.

• Make the uterine incision in the midline over the fundus of the uterus. The incision should be approximately 12–15 cm in length and the lower limit should not extend to the utero-vesical fold of the peritoneum.

• Cut down to the level of the membranes and then extend the incision using scissors. After rupturing the membranes, grasp the baby's foot and deliver the baby.

• Deliver the placenta and membranes.

• Close the incision using at least three layers of suture:

— close the first layer closest to the cavity, but avoiding the decidua, with a continuous 0 chromic catgut (or polyglycolic) suture;

— close the second layer of uterine muscle using interrupted 1 chromic catgut (or polyglycolic) sutures;

— close the superficial fibres and the serosa using a continuous 0 chromic catgut (or polyglycolic) suture with an atraumatic needle.

— close the abdomen as for the lower segment cesarean section.

Note:

The woman should not labor with future pregnancies.

TUBAL LIGATION AT CESAREAN SECTION

Tubal ligation can be done immediately following cesarean section if the woman requested the procedure before labor began (during prenatal visits).

Adequate counselling and informed decision-making and consent must precede voluntary sterilization procedures; this is often not possible during labor and delivery (Fig. 18.25) [49].

• Grasp the least vascular, middle portion of the fallopian tube with forceps.

• Hold up a loop of tube 2.5 cm in length.

• Crush the base of the loop with artery forceps and ligate it with 0 plain catgut suture.

• Excise the loop (a segment 1 cm in length) through the crushed area.

• Repeat the procedure on the other side.

REFERENCES

1. Abuhamad A. Ultrasound in obstetrics and gynecology: a practical approach. 2014. 322 p.

2. American Institute of Ultrasound in Medicine. AIUM practice guideline for the performance of ob-stetric ultrasound examinations. Revised 2017. http://www.aium.org/resources/guidelines/reproductivemed.pdf.

3. Coutin Anne-Sophie. Essential obstetric and newborn care. Practical guide for midwives, doctors with obstetrics training and health care personnel who deal with obstetric emergency. 2015 edition. 271 p. Available from : http://refbooks.msf.org/msf_docs/en/obstetrics/obstetrics_en.pdf

4. Clement Philip B., Young Robert H. Atlas of Gynecologic Surgical Pathology. 3rd edition. 2014. 584 p. Available from https://www.rcog.org.uk/globalassets/documents/guidelines/gtg_31.pdf

5. Rymer J., Bottomley C. 100 cases in Obstetrics and Gynaecology. Great Britain, 2008. 265 p.

6. Chesley's Hypertensive Disorders in Pregnancy / Jan Pincombe, Carol Thorogood, Sally K. Tracy, Sally Pairman. Midwifery. E-Book : Preparation for Practice. Elsevier Australia. 4th edition. 2014. 484 p.

7. Chestnut's Obstetric Anesthesia: Principles and Practice / David H. Chestnut, Cynthia A. Wong, Lawrence C. Tsen [et al.]. 5th edition. 2014. 1328 p.

8. Cruickshank M., Shetty A. Obstetrics and Gynaecology. Clinical cases uncovered. UK, 2009. 216 p.

9. Current Diagnocsis & Treatment: Obstetrics & Gynecology / Alan H. Decherney, Lauren Nathan, T. Murphy Goodwin [et al.]. 11th Edition. The United States of America, 2013. 1024 p.

10. Danforth's Obstetrics and Gynecology / Ronald S. Gibbs, Beth Y. Karlan, Haney A. F., Nygaard Ingrid E. 10th edition. 2008. 375 p.

11. Hamilton-Fairley D. Obstetrics and Gynaecology. Lecture notes. 3rd edition. 2009. 366 p.

12. Kurjak A., Cherve-nak F. A.. Donald School Textbook of Ultrasound in Obstetrics and Gynaecology. Jaypee Brothers Medical Pub. 4th edition. 2017. 1097 p.

13. Dutta D. C. Textbook of Obstetrics: textbook. 7th edition. India, 2011. 688 p.

14. Food & Medication Interactions / Z. M. Pronsky, M. S. Fada, J. P. Crowe [et al.]. L. A., FMI, 2012. 410 p.

15. Ganong F. W. Review of Medical Physiology. 22nd edition. 2005. 912 p.

16. Ganong's review of medical physiology / Kim E. Barrett, Heddwen L. Brooks, Scott Boitano, Susan M. Barman. 23rd edition. New York: McGraw-Hill Medical, 2010. 727 p.

17. Gomella T. L., Eyal Fabien G., Zenk Karin E. Neonatology: Management, Procedures, On-Call Problems, Diseases, and Drugs. 7th edition. The United States of America, 2013. 1114 p.

18. Neville Hacker F., Joseph Gambone C., Calvin Hobel J. Hacker & Moore's essentials of obstetrics & gynecology. 6th edition. 2016. 474 p.

19. Hypertensive Disorders in Pregnancy (HDP). Guideline Summary. New York State Department of Health. 2013. 56 p. Available from : https://www.health.ny.gov/professionals/protocols_and_guidelines/hypertensive_disorders/2013_hdp_executive_summary.pdf

20. Iron deficiency anaemia: assessment, prevention and control. Available from : http://www.who.int/nutrition/publications/micronutrients/anaemia_iron_deficiency/WHO_NHD_01.3/en/#

21. Jeffery S. Procedures in Obstetrics and Gynaecology. Creative Commons Attributive Licence, 2010. 268 p.

22. Edmonds D. K. Dewhurst's textbook of obstetrics and gynaecology. 7th edition. 2007. 717 p.

23. Kevin P. Hanretty. Obstetrics Illustrated. 7th edition. China, 2009. 451 p.

24. Modified from American Congress of Obstetricians and Gynecologists. Chronic hypertension in Pregnancy. Practice Bulletin number 125. Obstetrics and Gynecology 2012. N 119 (2 Part 1). P. 396–407.

25. Troiano Nan H., High Risk and Critical Care Obstetrics. Harvey Carol J., Flood Bonnie Chez. Published with the Association of Women's Health, Obstetrics & Neonatal Nurses (AWHONN), Philadelphia. 3d edition. 2012. 448 p.

26. National Institute for Health and Care Excellence (NICE). NICE Guidelines CG 62. Antenatal Care: Routine Care for the Healthy Pregnant Woman. London : NICE, 2010a. Available from : https://www.nice.org.uk/guidance/cg107/resources/guidance-antenatal-care-pdf; Accessed 11 September 2014.

27. National Institute for Health and Care Excellence (NICE). NICE Guidelines CG 107. Hypertension in Pregnancy: The Management of Hypertensive Disorders During Pregnancy. London : NICE; 2010b, Last updated : January 2017. Available from : https://www.nice.org.uk/guidance/cg107/resources/guidance-hypertension-inpregnancy-pdf; Accessed 11 September 2014.

28. National Institute for Health and Clinical Excellence (NICE). NICE Guidelines CG 70. Induction of labor. London: RCOG Press, 2008. Available from : http://www.nice.org.uk/nicemedia/live/12012/41255/41255.pdf

29. National Institute for Health and Clinical Excellence (NICE). NICE Guidelines. Performing induction : NICE, 2013. Available from : http://pathways.nice.org.uk/pathways/inductionof-labor 5

30. Obstetrics and gynecology / Charles R. B. Beckmann, Frank W. Ling, Barbara M. Barzansky [et al.]. American College of Obstetrics and Gynecology (ACOG). 6th edition, 2010. 497 p.

31. Obstetrics: Normal and Problem Pregnancies / Mark Landon, Steven Gabbe, Jennifer Niebyl [et al.]. 7th edition. 2007. 1320 p.

32. Oxford Handbook of Obstetrics and Gynaecology / Sally Collins, Sabaratnam Arulkumaran, Kevin Hayes [et al.]. 3rd edition. 2013. 824 p.

33. Pansky B. Review of medical embryology. Available from : https://discovery.lifemapsc.com/library/review-of-medical-embryology

34. Pitkin J., Peattie A. B., Magowan B. A. Obstetrics and Gynaecology. An illustrated colour text. Elsevier Science Limited, 2003. 172 p.

35. Prevalence of anaemia among pregnant women. Index Mundi. Available from : http://www.indexmundi.com/facts/indicators/SH.PRG.ANEM

36. Narendra Malhotra N., Shah P. K., Divakar Hema. Principles and practice of Obstetrics and Gynecology for postgraduates. 4th edition. 2014. 874 p.

37. Risk calculator for fetal growth restriction. Fetal Medicine Foundation. Available from : https://courses.fetalmedicine.com/calculator/sgafilocale=en

38. Royal College of Obstetricians and Gynaecologists (RCOG). The Investigation and Management of the Small for Gestational Age Fetus. Royal College of Obstetricians and Gynaecologists Green Top Guide-line № 31. Revised 2014. London : RCOG, 2013.

39. Royal College of Obstetricians and Gynaecologists. Pre-eclampsia — study group consensus statement. Date of publication — September 2011. Available from : file:///C:/Users/Acer/Desktop/Diagnosis-and-Management-of-Pre-eclampsia-and-Eclampsia%-202011.pdf

40. The ALARM Course Manual is published by the society of obstetricians and gynaecologists of Can-ada. 22nd edition ALARM Course Manual. 2015–2016. 636 p. Available from : https://alarm.cfpc.ca/wp-content/uploads/2015/05/22nd-Ed-Manual-Printable-Version.pdf

41. The American College of Obstetricians and Gynecologists (ACOG). Management of preterm labor. ACOG practice bulletin. № 127. Washington, DC : ACOG, 2012. https://www.acog.org/About-ACOG/ACOG-Departments/Deliveries-Before-39-Weeks/ACOG-Clinical-Guidelines

42. The American College of Obstetricians and Gynecologists (ACOG). Medically indicated late-preterm and early-term deliveries. Committee Opinion, 2013. № 560. Available from : https://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Obstetric-Practice/Medically-Indicated-Late-Preterm-and-Early-Term-Deliveries

43. The American College of Obstetricians and Gynecologists (ACOG). ACOG Practice bulletin № 145: antepartum fetal surveillance, 2014. Available from : http://journals.lww.com/greenjournal/Citation/2014/0-7000/Practice_Bulletin_No__145___Antepartum-_Fetal.35.aspx

44. The American College of Obstetricians and Gynecologists (ACOG). Ultrasound in pregnancy. Practice Bulletin № 175, 2016. Available from : http://journals.lww.com/greenjournal/Fulltext/2016/12000/-Practice_Bulletin_No__175___Ultrasound_in.53.aspx

45. The Clinical Protocol approved by the Ministry of Health of Ukraine obstetrics and gynecology. Available from : www.moz.gov.ua

46. WHO recommendations for augmentation of labor. 2015. Available from : http://apps.who.int./iris/bitstream/10665/174001/1/WHO_RHR_15.05_eng.pdf

47. WHO recommendations on antenatal care for a positive pregnancy experience. 2016. http://apps.who.int./iris/handle/10665/250796

48. WHO recommendations on interventions to improve preterm birth outcomes. 2015. Available from : http://www.who.int/reproductivehealth/publications/maternal_perinatal_health/preterm-birth-guideline/en/

49. Williams Obstetrics / F. G. Cunningham, K. J. Leveno, S. L. Bloom [et al.]. 24th edition. 2014. 1358 p.

50. Zaporozhan V. M., Mishchenko V. P. Obstetrics and Gynaecology (in 2 books). Odessa : Odessa state medical university, 2007. 374 p.

CONTENTS

Навчальне видання

ГЛАДЧУК Ігор Зіновійович,
АНЧЕВА Ірина Анатоліївна

АКУШЕРСТВО

Навчальний посібник
Англійською мовою

Провідний редактор ***О. В. Бровкін***
Редактор ***Р. В. Мерешко***
Художній редактор ***А. В. Попов***
Художник ***І. К. Каневський***
Технічний редактор ***А. А. Гречанова***
Коректор ***О. В. Титова***

Електронне видання на диску. Формат 60x84/8.
Ум.-друк. арк. 19,87. Тираж 1. Зам. 2136.

Видавець і виготовлювач Одеський національний медичний університет.
65082, Одеса, Валіховський пров., 2.
Свідоцтво суб'єкта видавничої справи ДК № 668 від 13.11.2001.