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THE ANATOMICAL CHANGES DURING PREGNANCY AND COMMON DISORDERS DURING PREGNANCY

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ABSTRACT

The term pregnancy, also known as **Gestation** refers to period of development of an embryo/fetus within the female reproductive system. Development is a continuous process, beginning with the formation of a zygote. During the nine months of pregnancy, the body undergoes various anatomical changes which after the delivery of baby, normalizes within certain period of time. The changes are seen in the pelvis region, protects and supports the pelvic contents, provide s muscle and ligament attachment and facilitates transfer of weight from trunk to legs in standing, and to the ischial tuberosities in sitting. Postural Changes During the pregnancy process, the overall equilibrium of the spine and pelvis alters. These changes persist up to 8 weeks after delivery. The major conditions occurring during pregnancy which leads to harm situation to the mother as well as the baby are Obesity, Anemia, Hypertension and Thyroid Disorder

THE ANATOMICAL CHANGES DURING PREGNANCY AND COMMON DISORDERS DURING PREGNANCY

The term pregnancy, also known as **Gestation** refers to period of development of an embryo/fetus within the female reproductive system. It is the period of the intrauterine life of a fetus, and comprises of developmental changes from conception (fertilization) to birth.

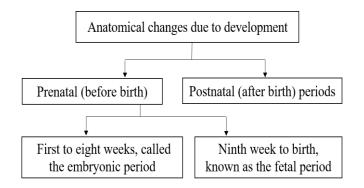


Figure 1.1: The Anatomical changes due to development.

Anatomically, changes due to development can be grouped into: prenatal (before birth) and postnatal (after birth) periods. Changes in these prenatal periods can further be grouped as the first to eight weeks, called the embryonic period and the ninth week to birth, known the fetal period. However, those changes occurring before birth, falls under the changes during gestation.¹

Development is a continuous process, beginning with the formation of a zygote when an **oocyte** (ovum) from a female is fertilized by a sperm (spermatozoon) from a male. Cell division, cell migration, programmed cell death, differentiation, growth, and cell rearrangement transform the fertilized oocyte, into a **zygote** and finally into a multicellular human being.

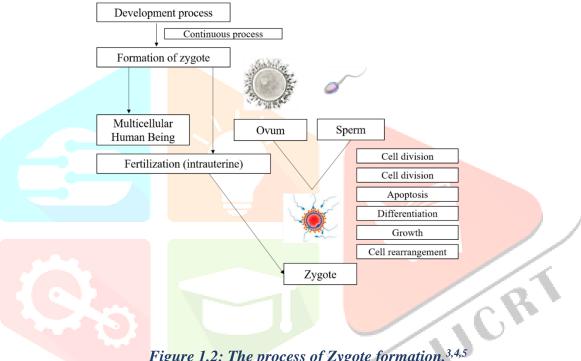


Figure 1.2: The process of Zygote formation.^{3,4,5}

The zygote undergoes multiplication of cells via a process called **cleavage**. Cleavage is the succession of mitotic cell divisions of the zygote to development of early embryonic cells, two cell stage, four cell stage, until the formation of a mass of cells (sixteen to thirty-two cells stage) termed morula. Just as early as embryo enters the uterus, morula stage occurs within 3-4 days after fertilization.¹,

Each of these cells are also known as **blastomeres.** The size of the cleaving zygote remains unchanged since each subsequent cleavage division, the blastomeres become smaller. The blastomeres modifies their shape and tightly align themselves against each other to form a compact ball of cells (morula). This phenomenon of **compaction**, is facilitated by cell surface adhesion glycoproteins.

Blastomere and morula formation

The **blastocele**, develops after the morula enters the uterus, a fluid-filled cavity. This leads to conversion of the morula into a **blastocyst**. The embryonic part of the embryo consists of the centrally located cells, the inner cell mass or embryoblast, while the outer cell mass or the **trophoblast** contributes to the **placenta**. The preimplantation period which is approximately 6 days, of embryonic development is the time between fertilization and the beginning of implantation.²

The embryo makes its way through the fallopian tube towards the uterus, where it implants, and formation of zygote takes place. **Implantation** is the process by which the blastocyst attaches to the endometrium of the mother's uterine tube, the mucous membrane or lining of the uterus, and subsequently embeds in it.⁶

Now, the formation **gastrula starts through the process** called gastrulation. During **gastrulation** a trilaminar embryonic disc or three-layered disc forms. The embryonic period ranges from the third to eighth weeks of development and during this period three germ layers namely, **ectoderm**, **mesoderm**, and **endoderm**; subsequently distinguish into the tissues and organs of the embryo.⁶

The formation of **neural tube** from the neural plate is the stage of **Neurula which occurs** during the third and fourth weeks. This is the stage after gastrula and first appearance of the nervous system. The size of embryos is measured from the vertex of the cranium (crown of head) to the rump (buttocks) on ultrasound and is given as **crown-rump length (CRL**). CRL is used to determine the gestational age of fetus or embryo.

The fetal period commences from 57th day (9th week) and ends when the fetus is completely outside the mother. Throughout the fetal period, differentiation and growth of the tissues and organs formed during the embryonic period occur. The anatomical changes are more gross; for example, the **placenta** and the **umbilical cord** evidently marks the link between the developing fetus and the mother.

Structures constituting the **genitalia** that is the phallus, urogenital fold, labioscrotal fold and the perineum are quite recognizable. The fetus accomplishes a CRL of 45-61 mm on average at the tenth week; the face changes into an identifiable human profile with the eyes, ears, nose and mouth in the normal positions. By the 12^{th} week, sex is fully distinguishable. Amplification and growth of formed structure continues, and the fetus is feasible at week thirty on the average, once all the structures of respiration, particularly the alveoli sacs are formed. Increase in weight is the most striking feature during the last two months of gestation.^{6,7}

During the nine months of pregnancy, the body undergoes various anatomical changes which after the delivery of baby, normalizes within certain period of time.

The Pelvis

In females, the pelvis is **wider** and **lower** than that of male counterpart, the changes are seen in the pelvis region which is found between trunk and lower limbs, making it appropriate to accommodate a fetus during both pregnancy and delivery. It protects and supports the pelvic contents, provide s muscle and ligament attachment and facilitates transfer of weight from trunk to legs in standing, and to the ischial tuberosities in sitting.⁸

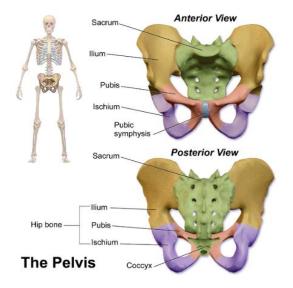


Figure 1.3 The anterior and posterior view of the pelvis.⁸

The cross-sectional anatomy of female pelvis shows five bones:

a. Two hip bones – has 3 parts – ilium, pubis and ischium – these are separated by cartilage at birth and fuse during puberty.

b. Sacrum - located inferiorly to the spinal vertebrae, and posteriorly within the pelvis. It is formed by the fusion of five sacral vertebrae, and transmits the sacral nerve fibers of the cauda equina.

c. Coccyx - The coccyx, commonly referred to as the tailbone, is the smallest of the pelvic bones, and sits inferiorly to the sacrum. It acts as a point of attachment for a few muscles and ligaments.

The **pelvic girdle** is the ring-shaped assortment of these bones at the base of the spine.

The joints are supported by the strongest ligaments in the body which become laxer during pregnancy leading to amplified joint movement and less efficient load allocation over the pelvis.

The pelvic passage at the base of the pelvis is narrower in its slanting span when compared with the pelvic inlet; it comprises of:

- a. The pubic arch,
- **b.** Ischial spines,
- c. Sacro tuberous ligaments, and
- d. Coccyx.⁹

Abdominal corset is four pairs of abdominal muscles combining to form the anterior and lateral abdominal wall.

With the rectus abdominis central, anterior and superficial abdominal oblique, transversus abdominis resides deep to the internal abdominal oblique and external abdominal oblique, external

oblique and transversus abdominis insert into an aponeurosis joining in the midline at the Linea alba. Lumbopelvic cylinder is considered as complete unit of the deep abdominal muscles, together with the pelvic floor muscles, multifidus, and diaphragm which provides support for the abdominal contents and maintains intraabdominal pressure.⁹

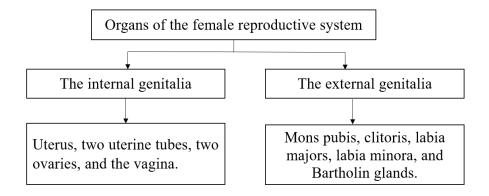


Figure 1.5: The organs of Female reproductive system.

Organs of the female reproductive system are subdivided into:

a. The internal genitalia consist of the uterus, two uterine tubes, two ovaries, and the vagina.

b. **The external genitalia** mainly consist of the mons pubis, clitoris, labia majors, labia minora, and Bartholin glands.⁸

Reproductive system changes:

The internal genital tract undergoes changes to accommodate the developing fetus. The changes,

a. Uterus

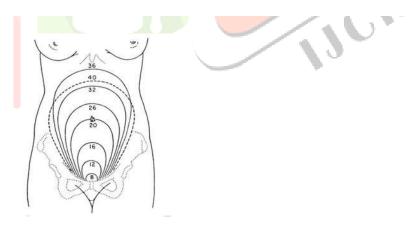


Figure 1.4: The ascending height of Fundus during pregnancy towards abdominal cavity.

As the pregnancy progresses, the uterus leaves the pelvic and ascends to the abdominal cavity.

The abdominal content is displaced in response to the increased size of the uterus which is five times more than normal.

The blood supply to the uterus and uterine muscle activity is increases due to the increased size of uterus.

The size of uterus increases till the 38th week, after that the fundus level starts to descend, preparing for delivery.

The weight of uterus increases from 50mg to 100mg after that it doesn't gets heavier and only stretches to accommodate the fetus and is associated with an increase in the thickness and length of fundus.

b. Cervix

The mucus plug secreted by the mucous glands of the cervix is called as operculum, which acts as a seal for the uterus, protecting it from ascending infection. It also acts as barrier between the vagina and cervix. Later in pregnancy, before delivery the cervix softens in response to the increasing uterine contractions.

c. Vagina

Due to the increased blood supply to the vagina during pregnancy, its color changes from pink to purple, and elasticity increases in second trimester.

Postural Changes

During the pregnancy process, the overall equilibrium of the spine and pelvis alters.¹⁰

There is increase in anteroposterior and medial-lateral sway, the center of gravity no longer falls over the feet¹¹ and women may need to lean backwards to gain equilibrium resulting in disorganization of spinal curves.

The postures include an increase in both lumbar lordosis and thoracic kyphosis or flattening of thoracolumbar spine curve and reduction in lumbar lordosis.

Posterior displacement of the shoulders and thoracic spine, increase anterior pelvic tilting, and increase of the cervical lordosis are due to the compensatory changes to posture in the thoracic and cervical spines, and this combined with the extra weight of the breasts.⁹

These changes persist up to 8 weeks after delivery.

DISEASES/ DISORDERS:

1. OBESITY

Overweight or obesity is defined as abnormal or excess accumulation of fat in adipose tissue in the body. These conditions are caused by a group of genetic, metabolic, behavioral, environmental, cultural, and socioeconomic factors. Overweight and obesity are public health problems that contribute to avoidable deaths each year, and the number of overweight and obese women at reproductive age is increasing in many countries.^{12,13}

Since pregnancy can serve as an aggravating factor for obesity^{14,15}, diagnosing and monitoring the weight status of pregnant women should be a routine prenatal care procedure.^{16,17} A number of factors, such as uterine growth, water retention, formation of fetal tissues and placenta, and increasing amniotic fluid volume, can limit the evaluation of maternal body mass index (BMI) during pregnancy.^{18,19}

Pregnancy weight category	Body Mass Index	Recommended range of total body weight (lb.)	Recommended rates of weight gain in the second and third trimesters(lb) (Mean Range [lg/wk])
Underweight	Less than 18.5	28-40	1 (1-1.3)
Normal Weight	18.5-24.9	25-35	1 (0.8-1)
Overweight	25-29.9	15-25	0.6 (0.5-0.7)
Obese (includes all classes)	30 and greater	11-20	0.5 (0.4-0.6)

Table 1.1: The American College of Obstetrics and Gynecology (ACOG) & Institute of Medicine WeightGain (IOM) recommendations of weight gain during pregnancy.20

Obese pregnant women have higher risks of gestational diabetes mellitus (GDM), hypertension, Cesarean section, shoulder dystocia and early neonatal death²¹. Overweight in pregnancy compromises fetal metabolic programming, increasing the risk of obesity, diabetes and cardiovascular disorders in the offspring.^{22,23}

On the other hand, anemia appears to occur less often in severely obese pregnant women than in normal-weight pregnant women.²⁴

Hypertensive disorders

Maternal hemodynamic changes in overweight mothers comprise of altered cardiac function, higher arterial blood pressure, and hemoconcentration²⁵. In overweight women, the frequency of hypertension is 2.2–21.4 times higher than in control subjects, and preeclampsia arises 1.22–9.7 times more often.^{24, 26}

Although increased blood pressure in obese women is associated with a decrease in subcutaneous fat of the newborn.²⁷

High Blood Pressure is associated with upper-body rather than with lower-body obesity in nonpregnant subject. Likewise, the regional distribution of fat may modulate the risk of cardiovascular disease in pregnant women. In a study including 22 patients with preeclampsia and 126 control subjects, the ratio of upper-body to lower-body fat was more accurately associated with the development of preeclampsia than was total body fat.²⁸

Infants of overweight mothers

Neonatal parameters Low Apgar scores are slightly more frequent in infants of obese mothers than in infants of normal-weight mothers.^{29,30,31}

Prepregnancy Body Mass Index is a strong predictor of birth weight, and obese mothers deliver large-forgestational-age infants 1.4 to 18 times more frequently than do lean mothers^{32,33}. Neonatal skinfold thickness is also higher in infants born to overweight mothers, signifying that the excess weight in the newborn is due to a higher fat mass³⁴. Macrosomia increases the risk for birth injury, shoulder dystocia, depression of Apgar scores, and perinatal death³⁵. for macrosomic infants, Cesarean deliveries result in fewer birth injuries, but the perinatal death rate remains unaffected.³⁶

High gestational weight gain increases the risk of delivering large-for-gestational-age infants in obese women³⁷. Gestational diabetes also disturbs fetal growth.³⁵

Congenital abnormalities

Maternal obesity is too a risk factor for congenital abnormalities. Data based on 56857 children in an analysis from the National Institute of Neurological and Communicative Disorders and Stroke showed rise in the incidence of major congenital malformations of 35% when mothers were obese and of 37.5% when they were overweight²⁶. This increase was after long found to be accounted for by a higher percentage of neural tube defects; 4 independent studies reported an association between neural tube defects and maternal obesity³⁸⁻³⁹.

Long-term complications

After delivery, overweight mothers are more likely to experience urinary symptoms such as stress incontinence and urgency than normal weight mothers³⁹. Unnecessary weight gain during pregnancy worsens maternal obesity. Weight gain during pregnancy is a strong predictor for sustained weight retention⁴⁰ and weight gains >9 kg is correlated with the amount of weight retained between 2 successive pregnancies.⁴¹

2. ANEMIA

In Obstetrics and Perinatal care, Anemia is a most common problem. If the hemoglobin value is below 10.5 g/ dL, it can be regarded as anemia regardless of gestational age. Reasons for anemia in pregnancy are mainly nutritional deficiencies, parasitic and bacterial diseases and inborn red cell disorders such as thalassemia's. Foremost cause of anemia in obstetrics is iron deficiency, which has a worldwide prevalence between estimated 20-80 %. Phases of iron deficiency are depletion of iron stores, iron scarce erythropoiesis without anemia and iron deficiency anemia, are the most pronounced form of iron deficiency.

Pregnancy anemia can be aggravated by various conditions such as gastro-intestinal bleedings, uterine or placental bleedings, and peripartum blood loss. Beside the overall consequences of anemia, there are specific risks during pregnancy for the mother and the fetus such as prematurity, feto-placental miss ratio, intra uterine growth retardation, and higher risk for peripartum blood transfusion. Along with the importance of prophylaxis of iron deficiency, main therapy options for the treatment of pregnancy anemia are oral iron and intravenous iron preparations.

It is estimated that between 2 and 7% have a value of less than 7 g/dL and 20% of pregnant women have hemoglobin of less than 8 g/dL. The situation is aggravated during the post-partum period because of blood loss during labor and in the puerperium. The American College of Obstetricians and Gynecologists has estimated that 5% of women who give birth lose 1000 mL of blood or more during delivery. The limit value defining puerperal anemia is 1 g/dL lesser, the prevalence of anemia during this period remains comparable with that observed during pregnanc

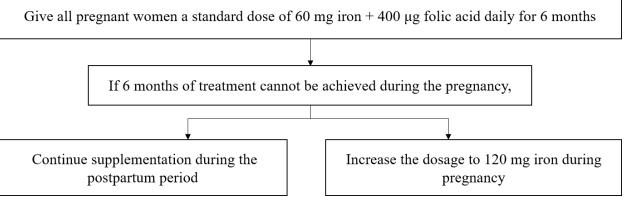


Figure 1.6: WHO recommendation of Iron intake during pregnancy ⁴²

Iron needs in pregnancy

The average total iron requirement, during pregnancy has been estimated to be approximately 1200 mg for an average weight of 55 kg in a pregnant woman. The iron is utilized mainly for the increase in maternal erythrocyte mass (450 mg), placenta (90 -100 mg), fetus (250-300 mg) general losses (200-250 mg) and a blood loss at delivery corresponding to 150 mg iron (300-500 mL blood loss). Iron absorption necessities in the first trimester are around 0.8 mg/ day, increasing to 7.5 mg/ day at third trimester

Any disorder that leads to anemia signifies an increased risk of an abnormal course of pregnancy and higher maternal and infant morbidity and mortality. According to WHO data, anemia is linked with 40% of maternal deaths worldwide.⁴³

According to the CHERG report on iron deficiency, anemia is linked to maternal mortality, i.e., the risk of maternal mortality decreases significantly for every hemoglobin increase of 1 g/dL.⁴⁴

Hemoglobin levels of more than 11.0 g/dL and less than 9.0 g/dL are related with a 2-3 times greater risk of a light-for-dates neonate. Hemoglobin levels of more than 12.0 g/dL at the end of the second trimester are associated with an increased risk of pre-eclampsia and intrauterine growth retardation, probably due to a lack of plasma volume expansion. Maternal hemoglobin levels below 9.0 g/dL increase the risk of premature births (PMB), intrauterine growth retardation (IUGR) and intrauterine fetal death (IUFD).

Where the prevalence of anemia in pregnancy is over 40%, advise the woman to continue the prophylaxis for three months in the postpartum period.

Give iron supplementation even if folic acid is not available.

Examine or screen all women for anaemia during antenatal and postpartum visits.

Treat anemia with doses of 120 mg iron daily for three months.

Follow up in two weeks to check clinical progress, test results and compliance and again four weeks later all women with severe anemia that have been treated with iron and folate.

Refer women with severe anemia to a higher level of care if they are in the last month of pregnancy, have signs of respiratory distress or cardiac abnormalities such as oedema, or when the condition do not improve or worsen after one week of iron/ folate therapy.

Provide advice on consumption of iron rich foods and vitamin C.

Figure 1.7: WHO, prophylaxis for treating Anemia 45

Basic diagnostic tests Hemoglobin and erythrocyte indices.

The first test for the investigation of anemia comprises the hematological profile with the following "classic" parameters:

- Hemoglobin concentration
- Hematocrit (not needed if Hb-levels are properly tested)
- MCV (if below 70 fl and ferritin is normal, indicator for thalassemia or hemoglobinopathy [HbC, HbE])
- MCH (no additional information, if MCV is tested)
- Erythrocyte count
- Occasionally: reticulocyte count

Prevention of iron deficiency

Majority guidelines recommend an increase in iron consumption by about 15 mg/day (to about 30 mg/day), an amount readily met by most prenatal vitamin formulations. This is adequate supplementation for non-anemic and non-iron deficient women. In a 2012 systematic review, daily iron supplementation reduced the risk of maternal anemia at term by 70 percent and iron deficiency at term by 57 percent.

According to WHO guidelines, daily oral iron and folic acid supplementation is recommended as part of the antenatal care to reduce the risk of low birth weight, maternal anemia and iron deficiency⁴⁵. Intermittent iron supplementation (one to three times per week) appears to be as effective as daily supplementation for preventing anemia at term and is better tolerated. Women with iron deficiency anemia (first or third trimester hemoglobin [Hb]

3. HYPERTENSION

In pregnant women, chronic hypertension is defined as raised blood pressure that is present and recognized before pregnancy. In women whose prepregnancy blood pressure is unidentified, the diagnosis is founded on the presence of sustained hypertension before 20 weeks of gestation, defined as either systolic blood pressure (SBP) of at least 140 mm Hg or diastolic blood pressure (DBP) of at least 90 mm Hg on at least two occasions measured at least 4 hours apart.

The judgement may be difficult in women with previously undiagnosed chronic hypertension who begin prenatal care after 16 weeks' gestation because a physiologic decrease in blood pressure usually begins at that time. This decrease may result in normal blood pressure findings at that time, which will eventually increase again during the third trimester. These women are more likely to be inaccurately diagnosed as having gestational hypertension. ⁴⁶

Hypertension in pregnancy is associated with increased risk of placental abruption, intracerebral hemorrhage, intrauterine growth retardation, intrauterine death and prematurity.

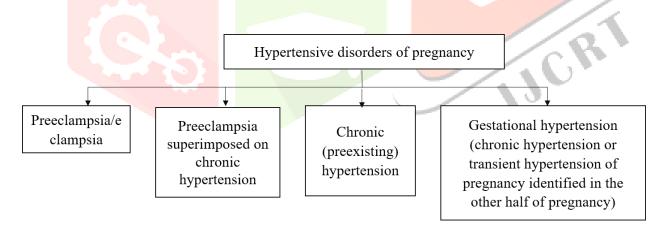


Figure 1.8 Hypertensive disorders during pregnancy

There are four groupings of hypertensive disorders of pregnancy:

- 1. Preeclampsia/eclampsia
- 2. Chronic (preexisting) hypertension
- 3. Preeclampsia superimposed on chronic hypertension

4. Gestational hypertension (chronic hypertension or transient hypertension of pregnancy identified in the other half of pregnancy)

Diagnosis Hypertensive disorders of pregnancy are diagnosed by systolic blood pressure (BP) of 140 mmHg or greater and/or diastolic BP of 90 mmHg or greater on at least two occasions more than 4 h apart while resting. Systolic BP of 160 mmHg or greater and/or diastolic BP of 110 mmHg or greater measured on two separate occasions are generally agreed to represent severe hypertension.⁴⁸ However, the degree of hypertension is not associated with the risk of devastating eclamptic outcomes. Preexisting hypertension may not be evident in the first and second trimesters owing to the physiologic reduction in BP, thus causing confusion with gestational hypertension.

The etiology and severity of chronic hypertension are significant considerations in the management of pregnancy. Chronic hypertension is further divided into primary (essential) and secondary. Primary hypertension is by far the most common cause of chronic hypertension seen more underlying disorders such as collagen vascular disease (lupus, scleroderma), renal disease glomerulonephritis, interstitial nephritis, polycystic kidneys, renal artery stenosis), endocrine disorders (diabetes mellitus with vascular involvement, pheochromocytoma, thyrotoxicosis, Cushing disease, hyperaldosteronism), or coarctation of the aorta.^{49,50}

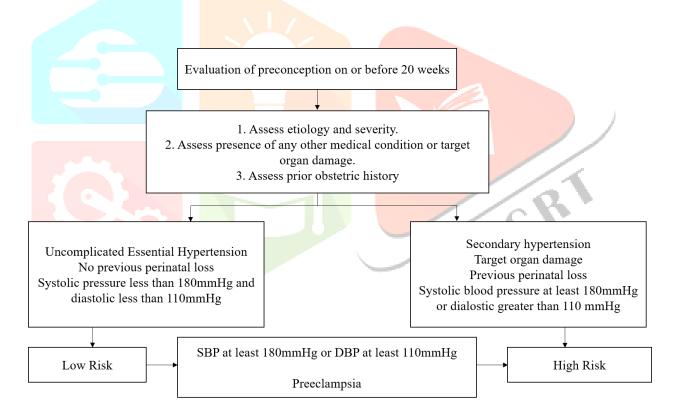
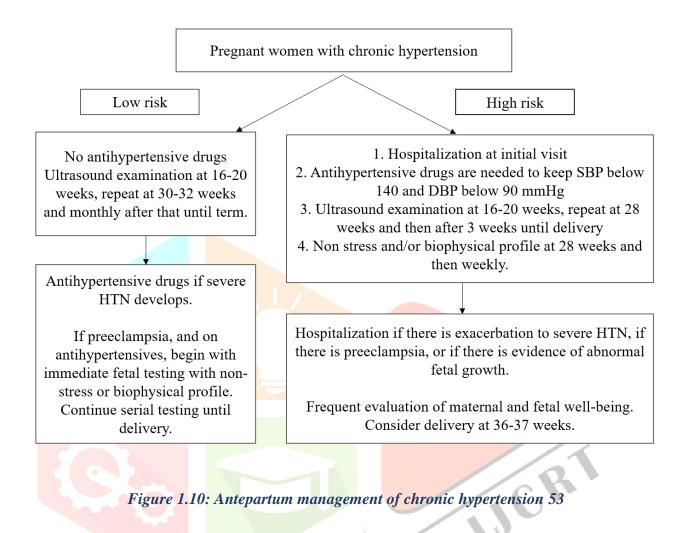


Figure 1.9: Initial evaluation of women with chronic hypertension

Women with high-risk chronic hypertension are at risk for postpartum complications such as hypertensive encephalopathy, pulmonary edema, and renal failure.^{51,52}

These risks are particularly increased in women with a target organ involvement, superimposed preeclampsia, or abruptio placentae.⁵² In these patients, blood pressure must be thoroughly controlled for at least 48 hours after delivery.

High Blood Pressure during pregnancy complicates 5-10 % of pregnancies and is the common cause of maternal death. The pathogenesis of hypertension comprises of inadequate cytotrophoblastic invasion of the myometrium, which results in placental hypoperfusion and diffusion of maternal endothelial dysfunction.



Hypertensive disorders of pregnancy are the second commonest cause of direct maternal death in the developed world. High Blood Pressure is also the commonest medical complication encountered during pregnancy, and complicates about 5-10 % of pregnancies.⁵¹

4. THYROID

TSH (thyrotropin)-release from the anterior pituitary rapidly decreases in the first trimester as a result of the increasing human chorionic gonadotrophin (HCG)⁵⁴. HCG is structurally alike TSH, and has thyroid-stimulating properties⁵⁵. TSH falls in the first trimester, returning gradually to normal by term. Hyperemesis gravidarum in the first trimester may be associated with a biochemical hyperthyroidism with suppressed TSH and high levels of free thyroxine, because of HCG's thyrotropic activity.

Since estrogen causes a two-fold upsurge in the synthesis of thyroxine-binding globulin from the liver, circulating levels of total thyroxine (T4) and triiodothyronine (T3) increases. Levels of free-T4 and free-T3 though remain unaffected during pregnancy⁵⁴. Pregnancy is associated with a state of iodine-deficiency because of renal excretion and increased active transport across the feto-placental unit.

During normal pregnancy, thyroid gland endures physiological changes. Hyperthyroidism occurs in about 2 of every 1000 pregnancies and if untreated can lead to neonatal mortality and low birth weight infants in 6% of cases 111). Antithyroid drugs and p-blockers can be used. Radioactive iodine is unconditionally contraindicated during pregnancy. Intolerable side effects of medical therapy are the only suggestion for surgery during pregnancy TSH levels are normal or suppressed early and rise to normal levels during third trimester ⁵⁶. Hypothyroidism during pregnancy is also important to be documented because of its association with congenital malformations and perinatal mortality affecting both mental and somatic development of the fetus.⁵⁷

Despite the rise in production of thyroid hormones, the size of the thyroid gland remains normal, and the presence of any goiter should always be inspected⁵⁸. The increase in the production of thyroxine-binding globulin (TBG) by the liver is noted, resulting in increased levels of tri-iodothyronine (T3) and thyroxine (T4). Serum free T4 (fT4) and T3 (fT3) levels are to some extent altered but have no clinical significance. Levels of free T3 and T4 decreases slightly in the second and third trimesters of pregnancy and the normal ranges are reduced. Free T3 and T4 are the physiologically significant hormones and are the chief determinants of whether a patient is euthyroid.

Serum concentrations of TSH are reduced slightly in the first trimester in response to the thyrotropic effects of augmented levels of human chorionic gonadotropin. Levels of TSH rises again at the end of the first trimester, and the upper limit in pregnancy is increased to $5.5 \mu mol/l$ compared with the level of $4.0 \mu mol/l$ in the non-pregnant state. Pregnancy is related with a relative iodine deficiency. The reason behind this is active transport of iodine from the mother to the foeto-placental unit and raised iodine excretion in the urine.

The World Health Organization recommends an increase in iodine consumption in pregnancy from 100 to 150–200 mg/day.⁵⁸ If iodine intake is continued in pregnancy, the size of the thyroid gland remains unaffected and therefore the presence of goiter should always be investigated. The thyroid gland is 25% grander in patients who are iodine scarce.⁵⁹

The most recent guidelines issued by the American Thyroid Association⁶⁰ suggested that all pregnant women should ingest ~250 μ g iodine daily, and women who are planning pregnancy or are currently pregnant, should add to their diet a daily oral supplement that contains 150 μ g of iodine.

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