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REVIEW

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Natural Physiological Changes During Pregnancy

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Pregnancy causes physiological changes that support the growing fetus and get the mother ready for labor and delivery. Some of these modifications affect biochemical levels; they are normally stable, while others could imitate symptoms of illness. It is critical to distinguish between pathology associated with disease and typical physiological changes. This review article focuses on the significant changes that occur throughout a typical pregnancy.

INTRODUCTION

The term "pregnancy" describes the process during which an embryo or fetus, or one or more offspring, develops inside a woman's uterus. The expecting mother experiences a range of physical, physiological, and biochemical changes over the course of the pregnancy; some of these changes are transient, others last for a set amount of time even if the pregnancy is terminated, and many others are permanent [1].

The physiological changes that come with pregnancy are a normal reaction to the fetus's development. To support and accommodate the growing fetus, the pregnant woman goes through considerable morphological and physiological changes [2]. Every organ system in the body experiences these changes, which start to occur after conception. Most women who have an uneventful pregnancy find that these alterations disappear after giving birth with few lasting repercussions [1]. Explanations for this include changes in hormones, such as increase in blood volume overall, body weight growth, and the growing size of the baby as the pregnancy advances.

Pregnancy affects respiratory, musculoskeletal, reproductive, endocrine, cardiovascular, neurological, gastrointestinal, and immunological systems in addition to causing changes to the breasts and skin (Table 1). There are variations globally and it is believed that the length of human pregnancies also varies naturally. The whole gestation period is 39-40 weeks and preterm birth is defined as delivery before 37 weeks gestation [3].

RENAL ANATOMICAL CHANGES AND FUNCTIONS

Renal vasodilatation causes a rise in renal plasma flow and glomerular filtration rate (GFR) of 40-65% and 50-85%, respectively, when compared to pre-pregnancy levels. Additionally, the rise in plasma volume results in a reduction in glomerular oncotic pressure, which raises GFR [2]. Although renal plasma flow has significantly

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Abbreviations: GFR, glomerular filtration rate; IVC, inferior vena cava; SVR, systemic vascular resistance; T3, tri-iodothyronine; T4, thyroxine; PTHrP, PTH-related protein; SVR, systemic vascular resistance; PTH, parathyroid hormone.

Keywords: Anatomical Changes, Thyroid hormone changes, Adrenal gland changes, Pituitary gland changes, Cardiac changes, hematological changes, Respiratory changes, Protein metabolism, Glucose metabolism, Lipid metabolism changes

Table. 1 Signs and Symptoms of Pregnancy

Signs and symptoms	Time period
Mild cramping and spotting	week 1 to 4
Missed period	week 4
Fatigue	week 4 or 5
Nausea	week 4 to 6
Tingling or aching breasts	week 4 to 6
Frequent urination	week 4 to 6
Bloating	week 4 to 6
Motion sickness	week 5 to 6
Mood swings	week 6
Temperature changes	week 6
High blood pressure	week 8
Faster heartbeats	week 8 to 10
Extreme fatigue and heart burn	week 9
Breast and nipple changes	week 11
Acne	week 11
Noticeable weight gain	week 11
Pregnancy glow	week 12

increased, glomerular hydrostatic pressure remains constant, delaying the onset of glomerular hypertension. All the renal afferent along with efferent arterioles see a decrease in vascular resistance of glomerular hypertension. Vascular resistance reduces in both the renal afferent and efferent arterioles. The mean values of serum creatinine and urea concentrations decrease to around 44.2 mol/l and 3.2 mmol/l, respectively as the GFR increases.

Renal size increases by 1-1.5 cm because of the increased renal blood flow, reaching its maximum size by the middle of pregnancy. The mechanical compressive pressures acting on the ureters cause the renal, pelvic, and calyceal systems to enlarge. These structural alterations are mediated by progesterone, which lowers ureteral tone, peristalsis, and contraction pressure [4]. Renal vasculature, interstitial fluid, and urinary dead space all increase along with an expansion of renal capacity. Additionally, ureter, renal pelvis, including dilatation of calyces is seen in more than 80% of women. Because of the physical circumstances where the right ureter crosses the iliac and ovarian veins at a position before entering the abdominal cavity, before entering the pelvis, there is frequently a right-sided predominance of hydronephrosis. Pregnant women with asymptomatic bacteriuria are more likely to develop pyelonephritis due to urinary stasis in the dilated collecting system [2,3]. The processing of nutrients and wastes in tubular systems has also changed. As in the non-pregnant state, glucose is freely filtered in the glomerulus. Glucose reabsorbing is reduced in the proximal and collecting tubule; efficient and variable excretion occurs during pregnancy. Ninety percent of expectant mothers with normal blood sugar excrete 1-10 g of glucose each day. During healthy pregnancies, the fractional removal of protein may increase to 300 mg/day while the total amount of protein in urine stays within the upper normal range, and increased GFR and/or decreased tubular reabsorption rise above the upper normal range throughout healthy pregnancies. Increased GFR and/or reduced tubular reabsorption both result in an increase in uric acid excretion [4].

CARDIAC CHANGES

Pregnancy causes dramatic changes to the cardiovascular system that start early. By 8 weeks gestation, cardiac output had already increased by 20%. Vasodilatation of the peripheral vessels is likely the main event. Estradiol-upregulated endothelium-dependent factors, such as nitric oxide production and perhaps vasodilatory prostaglandins (PGI2) facilitate this. During pregnancy, peripheral vasodilation causes a 25-30% decrease in systemic vascular resistance, and to make up for this, cardiac output rises by about 40%. This is mostly accomplished by increasing stroke volume, though to a lesser extent, heart rate is also increased. Between 20 and 28 weeks of gestation, the maximum cardiac output is discovered. At term, there is only a slight decline [4].

Pregnancy-related changes in ventricular membrane strength and end-diastolic volume that occur early on but not in end-diastolic pressure raises the possibility of an increase in stroke volume. Myocardial contractility is boosted as the heart physically dilates. Although the stroke volume decreases as the pregnancy progresses to term, the mother's heart rate increases (10-20 bpm), maintaining the higher cardiac output. In the first and second trimesters, blood pressure falls, but in the third trimester, it rises to non-pregnant levels.

Maternal position as the pregnancy nears term has a significant impact on the mother and the fetus's hemodynamic profiles. The inferior vena cava (IVC) is compressed by the gravid uterus while the patient is supine, which lowers the amount of blood returning to the heart and consequently lowers cardiac output and stroke volume [5]. A 25% reduction in cardiac output may occur after shifting from lateral to supine. Therefore, pregnant women should be nursed in the left or right lateral position if possible. To maximize cardiac output and uteroplacental blood flow, the pelvis should be rotated if the mother must be maintained on her back. This will cause the uterus to drop off the IVC and to the side, resulting in decreased cardiac output [6].

Pregnancy causes an increase in blood volume and stroke volume, although it has little effect on pulmonary capillary wedge pressure or central venous pressure. In a

typical pregnancy, pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR) both dramatically decline. Serum colloid osmotic pressure decreases by 10-15%, but pulmonary capillary wedge pressure (PCWP) remains same. Pregnant women are more vulnerable to pulmonary edema due to a 30% reduction in the colloid osmotic pressure/pulmonary capillary wedge pressure gradient. If there is an increase in cardiac pre-load (caused, for example, by fluid infusion) or an increase in pulmonary capillary permeability (caused by pre-eclampsia, for example), or both, pulmonary edema will be precipitated [7]. Further increases in cardiac production are linked to labor (15% to 50% in the second stage, and 25% in the first stage). During uterine contractions, 300-500 ml of blood is automatically returned to circulation, and the sympathetic response to pain and anxiety leads blood pressure and heart rate to rise even higher. Cardiac output rises during and between contractions.

After delivery, the uterus contracts, releasing blood into the systemic circulation and relieving the IVC obstruction; this causes an immediate increase in cardiac output. Within an hour of delivery, cardiac output rapidly declines to pre-labor levels after increasing by 60 to 80%. Increased venous return and stroke volume result from the transfer of fluid from the extravascular space [8].

The risk of pulmonary edema during the second stage of labor and the first few days after delivery is consequently greatest in women who have cardiovascular impairment. Cardiovascular output has nearly fully returned to pre-pregnancy levels 2 weeks after birth, but some pathological changes (such pre-eclampsia-related hypertension) may take much longer. Those who are not experienced with pregnancy could misunderstand the alterations on a cardiovascular examination caused by the physiological changes as abnormal. The presence of an ejection systolic murmur, which is present in over 90% of pregnant women, and a bounding or collapsing pulse are examples of changes. A third heart sound may occasionally be present, and the first heart sound may be strong and audible throughout the precordium. It is possible to develop peripheral edema and ectopic beats [4]. Normal ECG readings during pregnancy that could partially be related to changes in the heart's location may include:

- Peripheral edema and ectopic beats
- Inverted T wave in lead III and Q wave (small)
- T-wave inversion in the inferior, ST-segment depression, and lateral leads
- Left-axis shift of QRS.

HEMATOLOGICAL CHANGES

As a normal pregnancy progresses, plasma volume gradually rises [9]. The majority of this 50% increase happens between 32 to 34 weeks gestation and is proportionate to the baby's birth weight. Hemoglobin concentration, hematocrit, and red blood cell count decrease because of the expansion in plasma volume being greater than the growth in red blood cell mass. The mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC) are often unaffected by this hemodilution.

During a typical pregnancy, the platelet count tends to gradually decline, albeit it typically stays within normal ranges. The count will rise to 100-150 x 10^9 cells/l by term in a small percentage of women (5-10%), and this happens without any pathogenic mechanism. Therefore, a pregnant woman is not deemed to be thrombocytopenic until her platelet count is below 100 x 109 cells/l [1].

The need for iron increases 2 to 3 times during pregnancy, not only for the fetus, but also for the creation of several enzymes and for the synthesis of hemoglobin. The requirements for folate and vitamin B12 have increased by 10 to 20 times and by 2 times, respectively. Pregnancy-related changes to the coagulation system result in a physiologically hypercoagulable condition [10]. There are increased concentrations of the clotting factors VIII, IX, and X. The production of fibrinogen increases by up to 50%, while fibrinolytic activity decreases. Lower amounts are found in endogenous anticoagulants like protein S and antithrombin. Pregnant and postpartum women are therefore more vulnerable to venous thrombosis because pregnancy shifts the coagulation system's favoring of clotting. Beginning in the first trimester and continuing for at least 12 weeks after delivery, a higher risk exists. Prothrombin time (PT), thrombin time (TT), and activated partial thromboplastin time (APTT) are *in vitro* assays for coagulation that remain normal in the absence of anticoagulants or a coagulopathy [9]. Vasodilation and reduced flow, which are more pronounced on the left, are linked to venous stasis in the lower limbs. This is brought on by the left femoral artery and ovarian artery compressing the left iliac vein. The iliac artery does not cross the vein on the right.

ENDOCRINE CHANGES

Thyroid Hormone Changes

Thyroxine (T4) and tri-iodothyronine (T3) levels rise because of an increase in the liver's production of thyroxine-binding globulin (TBG). Although there is a modest change in serum free T4 and free T3 levels, these changes are typically not clinically significant. However, during the second and third trimesters of pregnancy, there is a little fall in free T3 and T4 levels and a narrowing of the normal ranges [10]. The essential markers of whether a patient is euthyroid are free T3 and free T4, two physiologically significant hormones [11]. In response to the thyrotropic effects of elevated levels of human chorionic

Table 2. Normal Range of Thyroid Stimulating Hormone During Pregnancy

Units	Non-pregnant Adult	First Trimester	Second Trimester	Third Trimester
μIU/mL or	0.34-4.25	0.6-3.4	0.37-3.6	0.38-4.04
mU/L	0.34-4.25	0.6-3.4	0.37-3.6	0.38-4.04

gonadotropin, serum TSH concentrations modestly decline in the first trimester [12].

Iodine insufficiency is correlated with pregnancy. Iodine active transfer from the mother to the fetoplacental unit and increased iodine excretion in the urine are the causes of this. The World Health Organization advises increasing the daily recommended intake of iodine for pregnant women from 100 to 150-200 mg [10]. The size of the thyroid gland does not alter during pregnancy if iodine intake is maintained, therefore goiter should always be investigated. Patients with iodine deficiency have thyroid glands that are 25% larger than normal (Table 2).

Adrenal Gland Changes

Mineralocorticoids, glucocorticoids, and sex steroids are the three types of steroids that are produced by the adrenal glands. The renin-angiotensin-aldosterone system (RAAS) is triggered by lower blood pressure and vascular resistance, which increases aldosterone levels by three times in the first trimester and ten times in the third [13]. Deoxycorticosterone, corticosteroid-binding globulin (CBG), adrenocorticotropic hormone (ACTH), cortisol, and free cortisol all rise during pregnancy. A physiological hypercortisolism syndrome is the result of these modifications, and its clinical symptoms include striae, facial plethora, elevated blood pressure, and impaired glucose tolerance [14]. At the end of the first trimester, total cortisol levels have tripled compared to pre-pregnancy levels. The placenta's release of corticotropin-releasing hormone, which is one of the labor-inducing factors and contributes to hypercortisolism in late pregnancy. The levels of ACTH and cortisol exhibit daily fluctuations. During pregnancy, the response to exogenous glucocorticoids in the hypothalamic-pituitary axis is diminished [2].

Pituitary Gland Changes

Pregnancy causes the pituitary gland to grow, primarily because of the anterior lobe's proliferating prolactin-producing cells. At term, serum prolactin levels are 10 times higher than during the first trimester. Most likely, rising serum estradiol concentrations during pregnancy are to blame for the rise in prolactin. Because of the negative feedback from the increased amounts of estrogen, progesterone, and inhibin during pregnancy, levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are undetectable [15]. Due to growth hormone production from the placenta, pituitary growth hormone output declines while serum levels of growth hormone rise. Oxytocin and arginine vasopressin (AVP) are produced by the posterior pituitary. Pregnancy raises oxytocin levels, which peak at term. Antidiuretic hormone (ADH) levels do not vary, but osmolality does because pregnancy causes a drop in salt content. Osmoreceptors for ADH release and thirst are therefore reset [16].

BODY WATER METABOLISM CHANGES

Pregnancy-related arterial under-filling stimulates arterial baroreceptors, which in turn activates the sympathetic and RAA excretion rises because of changes in GFR and glomerular capillary permeability to albumin. The nervous systems. As a result, the hypothalamus releases AVP non-osmotically. These modifications cause the kidneys to retain water and sodium, which results in the hypervolemic, hyperosmolar state that is typical of pregnancy [17]. Both plasma volume and extracellular volume rise by 30-50%. Maternal blood volume gets rises to 1200-1600 ml, or 45% more than non-pregnant levels. By the late third trimester, plasma volume rises by more than 50%-60% but red blood cell mass increases only slightly. As a result, plasma osmolality decreases by 10mol/kg. To maintain blood pressure, circulating blood volume, and uteroplacental perfusion during pregnancy, an increase in plasma volume is essential [18].

Increased plasma levels of aldosterone because of RAA system activation cause salt and water retention in the distal tubule as well as collecting duct. Along with the kidneys producing more renin, early pregnancy causes the ovaries and uteroplacental unit to create renin's inactive precursor protein [19]. The liver's production of angiotensinogen is stimulated by the estrogens the placenta produces, leading to proportionally higher amounts of aldosterone than renin. Aldosterone levels in plasma rise gradually throughout pregnancy and have a good correlation with estrogen levels. The rise in plasma volume during pregnancy is caused by an increase in aldosterone [18]. Despite aldosterone's ability to retain sodium, progesterone, a powerful aldosterone antagonist, permits natriuresis. The increase in GFR also facilitates the excretion of extra sodium by increasing distal sodium transport. Due to changes in tubular reabsorption caused by progesterone's ant-kaliuretic actions, potassium excretion is maintained constant throughout pregnancy while total body potassium increases [1].

Early in pregnancy, increased relaxin levels lead to

an increase in the release of hypothalamic AVP. The collecting duct's aquaporin 2 channels allow AVP to mediate an increase in water absorption. When the thresholds for hypothalamic AVP secretion and thirst are adjusted to lower plasma osmolality levels, the hypo-osmolar state that is characteristic of pregnancy results. These modifications are mediated by relaxin and human chorionic gonadotropin (HCG) [20].

GLUCOSE METABOLISM CHANGES

Changes in the mother's nutritional metabolism start to show in the early weeks following conception and continue all the way through the pregnancy. A lot of these modifications are done to make sure the fetus has access to nutrients during times when its needs are higher. The genetically defined sequences of fetal tissue growth and development correspond to different nutritional needs [21]. The kinds and quantities of nutrients necessary for the development of embryonic structures as well as the operation of metabolic pathways determine the kinds and quantities of essential nutrients. When the genes that regulate fetal growth and development express themselves, nutrients need to be accessible [22]. Increased insulin resistance, moderate hypoglycemia during fasting, and extended hyperglycemia following meals are characteristics of a normal pregnancy [21].

Maternal insulin resistance begins to develop in the second trimester and peaks in the third. This is brought on by an increase in the production of the hormones that induce diabetes, such as human placental lactogen, growth hormone, progesterone, cortisol, and prolactin. These hormones lessen the sensitivity of peripheral tissues, such as adipocytes and skeletal muscle, to insulin by interfering with insulin receptor signaling [23]. Postpartum, when there is a sharp decline in insulin resistance, the impact of the placental hormones on insulin sensitivity is clearly visible [24]. In pregnancy, insulin levels rise both postprandial and when fasting. However, fasting glucose levels are reduced because of [25]:

- Increased peripheral glucose use
- Increased storage of tissue glycogen
- Decrease in glucose production by the liver
- Uptake of glucose by the fetus.

Lipolysis is caused by insulin resistance and relative hypoglycemia, which enables the pregnant mother to preferentially utilize fat for energy while conserving the fetus's access to glucose and amino acids and reducing protein catabolism. Large lipids cannot pass through the placenta, however glucose, amino acids, and ketones can all be transferred to the fetus. Gestational diabetes develops when a woman's endocrine pancreas function is compromised and she is unable to overcome the insulin resistance brought on by pregnancy [26].

The placenta produces the aminopeptidase vasopressinase, which is four times more prevalent in middle and late pregnancy. These adjustments control the amounts of active AVP and improve the metabolic clearance of vasopressin [2]. A temporary form of diabetes insipidus may appear in situations where the placenta produces more vasopressinase, such as pre-eclampsia or twin pregnancies [20]. As a result of this volume growth, atrial natriuretic peptide secretion rises by 40% in the third trimester and continues to climb throughout the first week following delivery. Pregnant women with pre-eclampsia and persistent hypertension had greater amounts of natriuretic peptides [27].

LIPID METABOLISM CHANGES

Total serum cholesterol and triglyceride levels rise throughout pregnancy. Triglyceride levels rise mostly because of the liver's increased production and decreased lipoprotein lipase activity, which prevents adipose tissue from being broken down. At term, levels of low-density lipoprotein (LDL) cholesterol reach 50%. Concentrations of high-density lipoprotein rise in the first half of the pregnancy and decline in the third, but they are still 15% higher than those in non-pregnant women [12].

The lipid metabolism is altered to meet the needs of the growing fetus. Increasing triglyceride levels meet the mother's energy requirements while saving glucose for the fetus. The process of placental steroidogenesis depends on a rise in LDL cholesterol [28].

PROTEIN METABOLISM CHANGES

Increased protein consumption is necessary for pregnant women. To meet the requirements of the growing fetus, amino acids are actively transferred through the placenta. As fat reserves are required to fuel energy metabolism during pregnancy, protein catabolism is reduced [25].

CALCIUM METABOLISM CHANGES

The transportation of calcium ions from the mother to the fetus against a concentration gradient is the placenta main function in fetal calcium metabolism. The fetal skeleton grows more easily when there is a comparatively high ionic calcium level in the fetus because it stimulates calcitonin (CT) and suppresses parathyroid hormone (PTH). About 30g of calcium are needed on average by the fetus to maintain its physiological functions. Most of this calcium comes from the mother's enhanced food intake and is passed to the fetus throughout the third trimester [21]. During pregnancy, the total serum calcium concentration decreases. This is mostly caused

by hemodilution, which lowers serum albumin levels and lowers the albumin-bound portion of calcium. The physiologically significant portion, serum ionized calcium, is, nevertheless, unaffected [30]. Thus, during pregnancy, mother serum calcium levels are kept constant, and increased intestine absorption—which doubles after 12 weeks of gestation—meets the needs of the developing fetus. But only during the third trimester does calcium consumption reach its peak. The maternal skeleton may be able to store calcium in advance thanks to this early increase in calcium absorption [20].

25-hydroxyvitamin D is metabolized more into 1.25-dihydroxyvitamin D when serum levels rise. The rise in intestinal calcium absorption can be directly attributed to the increase in 1.25-dihydroxyvitamin D [29]. For 12 weeks, there has been an increase in calcium excretion in the urine along with increased calcium absorption. Urinary calcium levels are low or normal during fasting times, demonstrating that hypercalciuria results from increased absorption [30]. Consequently, kidney stones are at increased risk during pregnancy.

RESPIRATORY CHANGES

The need for oxygen rises noticeably throughout a typical pregnancy. This is brought on by a 15% rise in metabolic rate and a 20% rise in oxygen demand. The minute ventilation has increased by 40-50%, primarily because of an increase in tidal volume rather than respiratory rate. As a result of the maternal hyperventilation, the arterial pO2 and arterial pCO2 rise and the serum bicarbonate decreases to 18-22 mmol/l. Therefore, a modest totally compensated pulmonary alkalosis (arterial pH 7.44) is typical during pregnancy [31].

In late pregnancy, diaphragmatic elevation causes a reduction in functional residual capacity, although diaphragmatic excursion and hence vital capacity are unaffected. Early in pregnancy, increasing tidal volume causes a reduction in the amount of inspiratory reserve; however, in the third trimester, increased functional residual capacity causes an increase. Pregnancy has no impact on forced expiratory volume in one second (FEV1) or peak expiratory flow rate [32]. In addition to a subjective experience of being out of breath without hypoxia, pregnancy may also cause being shortness of breath. Although it usually begins in the third trimester, this physiological condition can occur at any point throughout the course of pregnancy. Typically, the shortness of breath occurs at rest or when speaking, and contrary to expectation, may get better during light activity.

SKELETAL AND BONE DENSITY CHANGES

The fetal need for calcium, which is only partially reliant on calcitriol, is supplied by an increase in maternal intestine absorption. PTH and PTH-related protein (PTHrP) are primarily responsible for the control of minerals. The synthesis of PTH and PTHrP is controlled by the calcium-sensing receptor will cause the mother's bones to resorb if her calcium intake is inadequate. Following delivery, fetal to neonatal homeostasis is transitioned from through an increase in PTH and calcitriol, as well as a developmental adaptation of the kidneys and intestines, with bone turnover adding more minerals to the bloodstream. The absorption of calcium becomes increasingly active and reliant on calcitriol. Osteosis can appear briefly in the postnatal skeleton, although full repair is typically possible with a sufficient mineral diet [13,25].

Pregnant women maintain elevated levels of certain minerals, specifically calcium and phosphorus, in their blood to enable the growing skeleton to absorb sufficient mineral content. It is for this reason that the placenta actively transfers minerals. The impact of pregnancy on maternal bone loss is a topic of debate [13]. When the fetus calcium requirements rise in the third trimester, bone turnover rises from a low level in the first trimester. Third-trimester calcium comes from previously stored skeletal calcium [25].

According to a previous study, pregnancy alters the micro-architectural pattern of bone, but not the total amount of bone mass [25]. To support the growing fetus, the maternal skeleton must be resistant to bending forces as well as the metabolic stressors. Other musculoskeletal changes seen in pregnancy include:

- Excessive shoulder flexion in the downward direction, lordosis of the lower spine, and neck flexion. Joint laxity in the anterior and longitudinal ligaments of the lumbar spine.
- The pubic symphysis and sacroiliac joints are widened and more mobile.

ADAPTIVE CHANGES IN RENAL VASCULATURE

A significant decrease in systemic vascular resistance (SVR), which occurs by week 6 of pregnancy, serves as the main adaptation mechanism. The renal vasculature is also impacted by the 40% drop in SVR [33]. Because 85% of the volume is in the venous circulation, there is an arterial under-filling condition even if there is a significant rise in plasma volume during pregnancy [34]. Pregnancy alone causes this under-filling of the vascular system. This contrasts with other conditions of arterial under-filling, such as cirrhosis, sepsis, or arterio-venous fistulas, where the decrease in SVR is accompanied by an increase in renal blood flow [11]. Relaxin, a peptide hormone created by the placenta, decidua, and corpus

luteum, plays a critical role in regulating the metabolism of water and hemodynamic throughout pregnancy. Serum levels of relaxin, which are already high during the luteal phase of the menstrual cycle, increase after conception, reaching a peak at the end of the first trimester, before decreasing to an intermediate level throughout the second and third trimesters. When relaxin stimulates the formation of endothelin, which in turn causes the renal arteries to dilate, nitric oxide (NO) is created. Although the renin-angiotensin-aldosterone (RAA) system is activated in the early stages of pregnancy, concurrently growing relative angiotensin II resistance balances the vasoconstrictive impact and allows for considerable vasodilatation. The pregnancy-related alterations in the angiotensin I receptors, the effects of progesterone and vascular endothelial growth factor-mediated prostacyclin production, and insensitivity to angiotensin II may all be to blame. AVP and adrenergic agonists may both share the vascular refractoriness to angiotensin II in their effects as vasoconstrictors [35]. The second half of pregnancy's vasodilatory state is likely to be maintained in part by placental vasodilators [36].

HEADACHES

Common headaches are typically brought on by hormonal and postural changes. Other potential reasons of headaches during pregnancy include fatigue, stress, and lack of sleep. Pre-eclampsia may occasionally be indicated by a headache during pregnancy. It can be controlled by consuming lots of fluids, getting enough sleep, and if required, seeking medical attention [37,38].

CONCLUSION

Pregnancy-related physiologic alterations are the outcome of hormonal and metabolic adjustments required to maintain the growing fetus. Additionally, they result in a range of cutaneous abnormalities that are readily noticeable to the patient and the treating physician. Obstetricians and dermatologists can provide appropriate comfort and avoid needless concern and intervention by recognizing the natural cutaneous changes. Most of these benign alterations are treated cosmetically, however many of these treatments have not been tested in women who are pregnant or nursing. Moreover, by the postpartum period, these abnormalities are usually resolved. Therefore, waiting until after birth for non-urgent and cosmetic procedures may be reasonable. Often, patients' discomfort can be reduced by teaching them about the physiological basis of these alterations in the skin and associated structures.

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