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3 Physiological Changes During Pregnancy: Main Adaptations, Discomforts, and Implications for Physical Activity and Exercise

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Abstract

Almost the entire female body naturally modifies and changes during pregnancy, and in many cases, these modifications occur normally; however, there may be imbalances that occur that can cause complications or pathologies.

In fact, pregnancy is known as the most changing period in a human life as there is no other time that produces the same quantity and quality of bodily modifications. Due to the varied changes the body experiences, pregnancy and childbirth can determine the future well-being of the mother and her child.

The pregnant body must work for 40 weeks to achieve adequate fetal growth and development and this causes a constant effort to maintain balance in all organs and systems. In summary, due to the many modifications that occur during pregnancy, exercise during pregnancy should be given unique and special considerations when compared to nonpregnant populations. However, from a scientific point of view, none of these modifications contradicts exercise in healthy pregnant women without obstetric contraindications.

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R. Santos-Rocha (ed.), *Exercise and Physical Activity During Pregnancy and Postpartum*, https://doi.org/10.1007/978-3-031-06137-0_3

Keywords

Pregnancy · Physiology · Cardiovascular · Hematologic · Metabolic · Physical activity · Exercise

3.1 Cardiovascular Changes

The human body during pregnancy changes day by day to continuously adapt its functions to ensure the development of an independent individual [1]. It is essential to optimize maternal health during this period.

The fetus is completely dependent on the mother. In a normal pregnancy, the maternal cardiovascular system undergoes essential changes in its structure and function which are necessary to support fetal demands and this leads to a considerable amount of stress on the maternal heart [2]. These changes (see Table 3.1) begin around the fifth week of pregnancy and continue until 1 year after delivery [3, 4].

General hemodynamic changes include an increase in maternal heart rate (30%) [5, 6] and blood volume (40–45%) [7]. Red blood cells also increase during pregnancy but less than blood volume (18–25%) [8], and this produces a decrease in hematocrit known as physiological anemia of pregnancy [5].

Table 3.1 Main cardiovascular changes during pregnancy

			Trimester		
	Change	Magnitude	First T	Second T	Third T
Hemodynamic changes					
Vascular resistance	↓	30%	↓	↓	↑
Blood volume	↑	40–45%	↑	↑	–
Cardiac output	↑	30–50%	↑	↑	NC
Heart rate	↑	15–30%	↑	↑	↑
Stroke volume	↑	20–30%	↑	↑	NC
Systolic blood pressure	–	–	–	–	–
Diastolic blood pressure	↓	10 mmHg	↓	↓/=	↑
Structural changes					
Aorta artery elasticity	↑	30%	↑	–	–
Heart size	↑	30%	↑	↑	↑
Left atrial	↑	16–40%	↑	↑	↑
Left ventricular					
Left ventricular diastolic dimension	↑	20%	↑	–	–
Left ventricular systolic dimension	↑	10%	↑	↑	↑
Left ventricular wall thickness	↑	15–25%	↑	↑	↑
Left ventricular stress	↑	17%	↑	–	↓
Left ventricular mass	↑	50%	↑	↑	↑
Systolic function	NC				
Diastolic function	↓		↑	↑	↓

NC no consensus

Hemodynamic alterations continue to occur throughout pregnancy with different changes taking place in each trimester. Total maternal vascular resistance decreases around 30% up to 28 weeks of pregnancy and then there is a considerable increase until term [6, 9]. This change leads to a reduction in maternal blood pressure (specifically diastolic blood pressure) during the first and second trimesters, and a normal increase during the third trimester [10, 11].

There is a general consensus that maternal cardiac output and stroke volume increase by 30–50% and 20–30%, respectively, in the first and second trimesters. However, previous literature has been inconsistent on what occurs during the third trimester as some authors report a decrease in cardiac output and stroke volume [5, 6], while there is also research supporting an increase [11, 12] or no variation at all [13–15]. Furthermore, it is important to note that most of these hemodynamic changes depend on maternal parity, age, and body mass index (BMI; [16, 17]).

Hemodynamic modifications on the maternal heart persist for a longer time than other hemodynamic and functional changes that occur during pregnancy. The increase in stroke volume is attributed to an increase in blood volume and preload, as demonstrated by the increase in left atrial and left ventricular end-diastolic dimensions and consequently the size of the heart gradually increases by approximately 30% [5, 18]. A slight increase in the left ventricular systolic dimension is also observed in a normal pregnancy [19].

To minimize stress on the heart, the walls of the heart increase in thickness beginning in the first weeks of gestation [2]. This response leads to a change in geometric left ventricular patterning usually toward eccentric hypertrophy and this is typically associated with an exercise stimulus and pregnancy. Abnormal structural adaptations may also lead to concentric remodeling or concentric hypertrophy and this may cause health complications during and after pregnancy [20].

Additional structural modifications include the aorta artery which increases in its flexibility by approximately 30% [21], in response to the normal reduction in total maternal vascular resistance [2].

In regards to maternal heart function, literature has been inconsistent on what potential changes occur with some research suggesting an improvement in systolic function [19] However, there are also reports of impaired systolic function [22] or no change taking place [6, 9]. As pregnancy progresses, the heart's capacity to relax increases which implies an enhancement in diastolic function up to the beginning of the third trimester. Following this, a normal decline in diastolic function until labor has been observed [11, 15].

3.1.1 Implications of Exercise During Pregnancy on the Maternal Heart

The changes that occur in the maternal heart during pregnancy are reversible in healthy women, but it is estimated that approximately 1% of pregnancies are complicated by heart disease in Europe [23]. There are some risk factors associated with an unhealthy lifestyle during pregnancy that may compromise maternal and fetal health.

A growing body of evidence supports that adopting or continuing a sedentary lifestyle during pregnancy increases the risk for developing gestational hypertension or diabetes and gaining excessive weight, and these are considered the risk factors for heart failure and cardiovascular dysfunction [24]. During labor, physically inactive women show limitations in the intensity and duration of pushes which lead to greater stress on the maternal heart and this may increase the risk of developing cardiovascular disease later in life [25].

Maintaining or starting to exercise during pregnancy may have great physiological benefits for the mother and the newborn. However, it is important to keep in mind some exercise considerations to accommodate for the naturally occurring maternal cardiovascular response to pregnancy and in order to avoid any additional stress on the maternal heart.

3.1.1.1 Maternal Cardiovascular Response to Exercise

During aerobic exercise, vascular resistance is reduced and this produces an increase in cardiac output, stroke volume, and heart rate [26]. This hemodynamic response depends on the gestational age, intensity, modality, and fitness level [27].

Importantly, maximal maternal heart rate is lower in pregnant women compared to nonpregnant women during submaximal exercise [28]. The physiological increase in maternal heart rate at rest and the maximal heart rate reduction produce a decrease in maternal heart rate reserve [29]. Exercise programs for pregnant women should control the intensity of the activity not only by a heart rate monitor but also by using the Borg Scale of Perceived Exertion [30] to ensure the safety of the woman.

Maternal position during exercise also plays an essential role in the cardiovascular response. Aortocaval compression occurs when the gravid uterus compresses the maternal abdominal aorta and inferior vena cava. This impedes venous return which decreases cardiac output and stroke volume and may reduce uteroplacental perfusion resulting in fetal acidosis [31]. This phenomenon occurs in 90% of pregnant women when they adopt a supine position or are lying on their right side [31].

3.1.1.2 Maternal Cardiovascular Adaptations to Exercise

There is limited evidence on the cardiovascular adaptations that occur in response to exercise during pregnancy [32, 33]. Findings from both studies were consistent, especially in the third trimester where results showed that the effects of exercise are hidden by the physiological pregnancy adaptations. No significant improvement in hemodynamic, functional, or structural results was observed; however, authors clarify that moderate regular exercise from late first trimester to term does not produce an additional change or adverse impact on the maternal heart [32, 33].

A nonsignificant trend was found in regards to maternal heart structure. Sedentary women tend to increase the proportion of abnormal left ventricular patterning, particularly concentric remodeling, which was not observed in active women [33]. This result is potentially relevant because cardiac remodeling during pregnancy is associated with a higher risk for maternal and fetal complications such as preeclampsia, hypertension, and preterm delivery [20].

Regular moderate exercise has also shown to be effective for the prevention of important cardiovascular risks during pregnancy such as antenatal depression and excessive gestational weight gain [33].

Further studies related to cardiovascular adaptations during pregnancy in response to exercise are necessary to clarify the most effective dose of exercise required based on maternal characteristics for the most physiological benefit overall.

3.2 Hematological Changes

Hematological modifications occur in response to pregnancy to meet the bodily requirements for both the mother and growing fetus such as nutrient transport to maintain fetal well-being [34].

Blood volume increases by 45% (1800 mL), and this includes an increase in blood volume or plasma (around 1500 mL) and cythemia (around 350 mL). This "hemodilution" will maintain adequate uteroplacental flow [35].

An additional gr of iron (daily) is required during pregnancy due to the increase in red blood cells (citemia) and fetal–placental iron requirements. This need is more essential from the second half of gestation (extra caution is recommended when engaging in strenuous physical activity) [36].

During pregnancy, hemoglobin concentration below 11 g/100 mL is considered anemia. Normal pregnancy in a woman with a deficit of iron reserves can lead to iron deficiency anemia [36].

During pregnancy, there is a decrease in folates, which are essential for fetal development. It is advisable for a woman who is trying to conceive to take a certain amount of folic acid a few months before pregnancy [37].

There is also a state of hypercoagulability (increased fibrinogen and other factors of coagulation). These changes are required for coagulation at the time of delivery. An increase of plasma fibrinogen justifies the elevation for the rate of globular sedimentation. A mild leukocytosis is established in 20% of pregnant women. Lymphocytes decrease in number and in absolute percentage, especially at the beginning of pregnancy, and continue throughout [36].

3.3 Respiratory Changes

The physiological requirements and normal modifications that occur during pregnancy include a relevant interaction between cardiovascular and respiratory functions. During exercise, this interaction also exists [34].

Oxygen and carbon dioxide are transported in both directions between the atmosphere and maternal and fetal cells. This is a complex process with challenges and anatomical and physiological implications that occur due to pregnancy [35].

The changes in the respiratory system cause alterations in anatomical and functional structures. These changes occur in early pregnancy due to hormonal influence and small volumetric alterations. These modifications include variations in lung dimension and capacity and respiratory mechanisms [35, 38].

The upper respiratory tract is (in some cases) affected by changes in the mucosa of the nasopharynx, for example, hyperemia, edema, and excessive secretion. This generates obstructive symptoms for normal breathing functions [36].

Furthermore, as a result of the expansion of the uterus, the average diaphragmatic position when the pregnant woman is standing is elevated by 4 cm [39].

As pregnancy progresses, the growing uterus significantly increases intra-abdominal pressure and this causes the ribs to become more horizontal. However, the ribs compensate for this by increasing the anteroposterior and transverse diameters of the rib cage by approximately 2 cm. The substernal angle is also increased by about 70° in the first trimester and 105° in the final stage of gestation and the circumference of the thoracic cage increases by about 5–7 cm [39].

At the beginning of pregnancy, the woman breathes more deeply but not more frequently, primarily due to an increase in progesterone. The consequence of this is that alveolar ventilation increases above pregestational values. As there is a higher tidal volume, the volume of expiratory reserve decreases, but vital capacity is maintained by a slight increase in inspiratory capacity [36].

Another characteristic of pregnancy at the respiratory level is the increase of oxygen consumption by 10–20% and a reduction in PCO2. This increases the tidal volume while decreasing the residual volume and functional residual capacity. The combination of reduced residual functional capacity and increased oxygen consumption results in reduced oxygen reserves. There is also an increase in oxygen uptake when breathing due to an increase in diaphragmatic work [36].

The ventilation/minute also increases, resulting in respiratory alkalosis, again in this case due to progesterone and additionally by the increase in estrogen. Despite this respiratory alkalosis, the acid–base state is maintained by compensatory metabolic acidosis. The arterial pH remains at 7.44 as a result of primary respiratory alkalosis and compensatory acidosis. The main purpose of these maternal respiratory mechanisms is to reduce arterial PCO2 and thus generate a mild maternal alkalosis that ensures placental gas exchange and prevents fetal acidosis [36, 40].

3.4 Metabolic Changes

The body's goal during pregnancy is to ensure fetal growth and development and this generates continuous adjustments in the maternal metabolic system throughout the 40 weeks of gestation. From a general point of view, normal metabolic processes are altered during pregnancy to adapt to the exact needs of the developing fetus [34, 35, 41].

During pregnancy, the protein content in body tissue is increased. Carbohydrates accumulate in the liver, muscles, and placenta. Under the skin, fat deposits increase, especially in the chest and buttocks area. The concentration of both types of cholesterol and blood fat is also increased. The pregnant body accumulates salts of various minerals essential for the normal development of the fetus, including calcium, phosphorus, potassium, and iron. In addition, hormonal changes favor the retention of water in tissues [35].

Weight gain is the most obvious change during pregnancy. Traditionally, total maternal weight gain of 10–13 kg was considered adequate (with many individual variations). However, currently the recommendations are based on maternal prepregnancy BMI [34, 42] (Table 3.2).

Many parameters influence maternal weight gain during pregnancy (Table 3.3), including the interstitial fluid and the increase of fatty tissue (deposit).

Maternal weight gain determined by the increase in fat reserves has significant variability. When the total weight gain is 11 kg, the average fat deposit at the end of pregnancy is 1800 g, but it can be much higher (3–4 kg or even more), it can be null, or even negative; this means that the pregnant woman's body consumes the reserve fat that she had previously accumulated [40, 43].

In healthy pregnant women during the first half of gestation (anabolic phase), the weight gain depends mostly on the accumulation of fatty deposits and normal changes in the maternal body in response to pregnancy. In this phase, the contribution that fetal weight has on total maternal weight gain is not significant. As pregnancy progresses, weight gain is primarily due to fetal growth and less due to maternal bodily changes [40].

Fat reserves accumulated in the first half of gestation are used during the third trimester, mainly in the last 4 weeks when the fetus has a rapid increase in body fat percentage. From the biological point of view, maternal weight gain during pregnancy should be lower when prepregnancy BMI is higher; that is, when a woman's

Table 3.2 Institute of medicine weight gain recommendations for pregnancy

Prepregnancy body mass index category	Recommended intervals of total weight gain (kg)
<18.5	12.5–18
18.5–24.9	11.5–16
25–29.9	7–11.5
≥30	5–9

Table 3.3 Analysis of maternal weight gain during pregnancy [40]

	Weight gain in grams			
	Week 10	Week 20	Week 30	Week 40
Fetus	5	300	1500	3400
Placenta	20	170	430	650
Amniotic fluid	30	350	750	800
Uterus	140	320	600	970
Mamas	45	180	360	405
Blood	100	600	1300	1250
Interstitial liquid	0	30	80	1680
Fat deposits	310	2050	3480	3345
Total weight gain	650	4000	8500	12,500

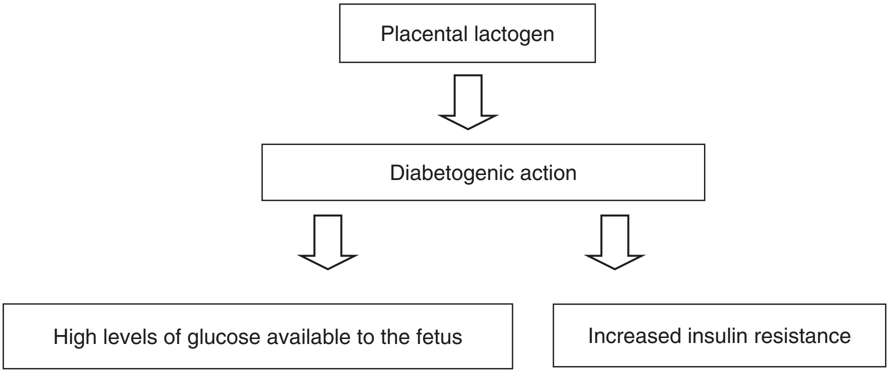

Fig. 3.1 Metabolism of carbohydrates in the second half of pregnancy [40]

fat deposit is higher before pregnancy, her BMI will be greater, and therefore less weight gain is recommended during pregnancy [34].

Metabolism of carbohydrates: In nondiabetic women, pregnancy is associated with profound metabolic changes, which can be studied in both fasting and postprandial situations (after the first intake) [40].

After an overnight fast, glucose levels are lower in pregnant women than nonpregnant women, especially in the second and third trimesters. The decrease in glucose decreases the level of insulin and this exaggerates ketosis by starvation. Therefore, after fasting during pregnancy, the levels of beta-hydroxybutyric acid and acetoacetic acid are higher than in nonpregnant women [40]

When pregnant women are fasting, there is an increase in the likelihood of hypoglycemia (very important), hypoinsulinemia, and hyperketonemia. This is due to facilitated diffusion of glucose from the mother to the fetus as a basic mechanism, and the existence of a greater volume of maternal distribution for glucose.

Thus, in the postprandial state, the maternal response is characterized by hyperinsulinemia, hyperglycemia, hypertriglyceridemia, and decreased sensitivity to insulin (insulin resistance) [40]. Pregnancy is, therefore, a diabetogenic experience. The factors responsible for this diabetogenic effect are placental hormones (placental lactogen), especially in the second half of pregnancy (Fig. 3.1) [34, 35, 40].

Metabolism of fats: Plasma lipids increase in the second half of pregnancy. This increase affects total lipids, cholesterol, phospholipids, and free fatty acids [40].

3.5 Locomotor Changes

Changes in the locomotor system are responsible for many common symptoms during pregnancy. Paresthesia and pain in the upper extremities occur as a consequence of an accentuated cervical lordosis and collapse of the scapular belt. These complications occur more frequently in the third trimester [34, 35, 44].

Traditionally, hyperlordosis has been considered a complication or even a pathology that occurs during pregnancy [34].

Currently, it is considered that this hyperlordosis is only apparent because the pregnant woman compensates for the deviation of her center of gravity, not by means of hyperlordosis but by displacing the entire craniocaudal axis backward. This new position can cause low back pain, especially if there is poor postural hygiene. Occasionally, a lumbosciatica originates due to compression of the sciatic nerve, and this may cause an increase in pain and functional disability (Fig. 3.2) [40].

Carpal tunnel syndrome is caused by the compression of the median nerve as it passes through the carpal tunnel on the anterior side of the wrist. It is characterized by pain and paresthesia usually experienced at night in the territory that is innervated by the median nerve and after delivery this reverts back to its normal state [40].

The rectus abdominis muscles are occasionally separated from the midline, creating a diastasis of the recti of variable extension. Sometimes, the uterus is only covered by a thin layer of peritoneum, fascia, and skin [34].

The mobility of the sacroiliac joints increases during pregnancy due to hormonal action, especially due to an increase in relaxin. The increase in joint relaxation can potentially diffuse pain. At the end of pregnancy, paresthesia may occur in the lower extremities (thigh and back of the leg), as a consequence of compressive changes (edema of the sheaths, pressure of the fetal head). In fact, this may contribute to the amount and quality of physical activity that a pregnant woman can perform in the final stage of pregnancy [45].

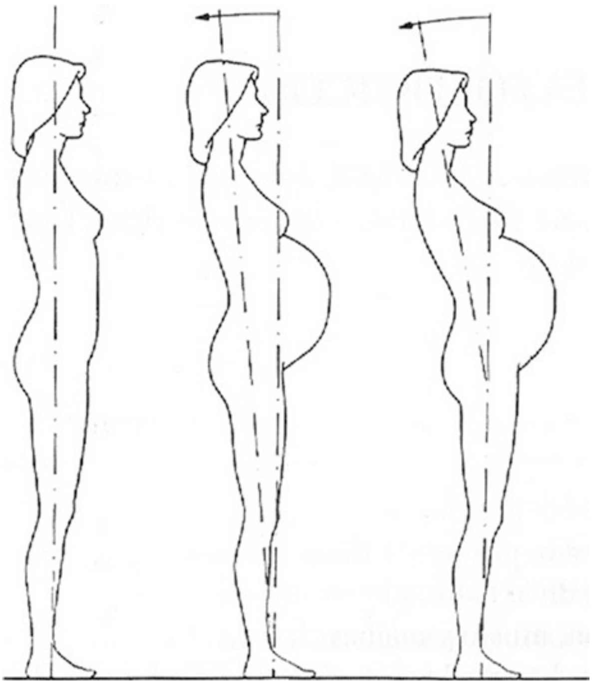

Fig. 3.2 Displacement of the center of gravity [40]

3.6 Microbiome Changes During Pregnancy

The human body has particular microbial population, including bacteria, archaea, fungi, and viruses. It is an important health factor due to mothers share their microbes and microbiome metabolites with the fetus in utero, during delivery and during lactation. This factor is also essential because it that is influenced and influences in maternal physiological changes during gestation with a high impact in maternal and fetus health from the beginning of life by altering pregnancy outcomes [46].

The womb is not sterile and microbial exposure may begin in utero. The placenta and amniotic fluid have been reported to serve as potential sources of intrauterine microbial transmission from the mother to the developing fetus. In healthy pregnant women, microbiota change dramatically, not only in bacterial load but also in its composition, from the first to the third trimester. Notably, this change is a key to promote a healthy pregnancy and normal fetal development [46]. Microbiota changes during pregnancy are characterized by increased abundance of members of the Actinobacteria and Proteobacteria phyla, as well as a reduction in individual richness [47]. These changes include placenta, gut, vaginal, and oral microbiota and are affected by gestational age.

Interestingly, microbiota during pregnancy is affected by such important factors as diet and physical exercise, not only during pregnancy but also prior it. Factor such as women body mass index prior to pregnancy correlated to neonatal gut microbiota composition in vaginally delivered, but not C-section [48]. The maternal gut microbiota differs between women with normal weight and obesity particularly in the latter half of pregnancy, with overweight women demonstrating increases in the Firmicutes phylum (Staphylococcus) as well as increases in some Proteobacteria (Escherichia coli) [49].

However these are not the unique factors, maternal weight gain during pregnancy showed a significant alteration in gut microbiota [46], as well as maternal health status and metabolism, mode of delivery, genetics, and use of antibiotics [50].

Exercise as a modify factor is recommended for healthy pregnancy with a great impact on intestinal microbiota. The scope of exercise biology extends well beyond energy expenditure and has emerged as an effective tool in the prevention and treatment of common diseases. Most studies to understand the impact of exercise on microbiota were performed by animal model. However, the anti-inflammatory effect of regular exercise, particular a combination of aerobic and resistance exercise, has been reported by much high-quality studies [51]. Exercise prescription is increasingly recognized as a safe and effective treatment for many diseases by adding further support for the potential influence of exercise on the brain-gut-microbiome axis [51].

Keeping in mind that microbiota can be "trained" even during pregnancy with a great effect in the present and future health generation [51], pregnancy, and also the postnatal period, are critical windows of opportunity for the prevention of metabolic diseases in the next generation.

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