



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

María Perales, Taniya Singh Nagpal, and Ruben Barakat

Contents

3.1	Introduction.....	46
3.2	Cardiovascular Changes.....	46
3.2.1	Implications of Exercise During Pregnancy on the Maternal Heart.....	48
3.2.2	Maternal Cardiovascular Response to Exercise.....	48
3.2.3	Maternal Cardiovascular Adaptations to Exercise.....	49
3.3	Hematological Changes.....	49
3.4	Respiratory Changes.....	50
3.5	Metabolic Changes.....	51
3.6	Locomotor Changes.....	53
	References.....	54

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 modi-

M. Perales (✉)

University Camilo José Cela, Madrid, Spain

Research Institute Hospital 12 de Octubre ('i+12'), Madrid, Spain

T. S. Nagpal

Faculty of Health Science, University of Western Ontario, London, ON, Canada

e-mail: tnagpal@uwo.ca

R. Barakat

Faculty of Sciences for Physical Activity and Sport, Technical University of Madrid, Madrid, Spain

fications. 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.

Keywords

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

3.1 Introduction

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.

3.2 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].

Table 3.1 Main cardiovascular changes during pregnancy

	Change	Magnitude	Trimester		
			First T	Second T	Third T
<i>Hemodynamic changes</i>					
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	↓	↓/=	↑
<i>Structural changes</i>					
Aorta artery elasticity	↑	30%	↑	–	–
Heart size	↑	30%	↑	↑	↑
Left atrial	↑	16–40%	↑	↑	↑
<i>Left ventricular</i>					
Left ventricular diastolic dimension	↑	20%	↑	–	–
Left ventricular systolic dimension	↑	10%	↑	↑	↑
Left ventricular wall thickness	↑	15–25%	↑	↑	↑
Left ventricular stress	↑	17%	↑	–	↓
Left ventricular mass	↑	50%	↑	↑	↑
<i>Systolic function</i>	NC				
<i>Diastolic function</i>	↓		↑	↑	↓

NC no consensus

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].

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 trimester and a normal increase during the third trimester [10, 11].

There is general consensus that maternal cardiac output and stroke volume increase by 30–50% and 20–30%, respectively, in the first and second trimester. 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 regard 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].

3.2.1 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 risk factors for heart failure and cardiovascular dysfunction [24]. During labor, physically inactive women show limitations in the intensity and duration of pushes which leads to greater stress on the maternal heart, and this may increase the risk for 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.

3.2.2 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 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, produces 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 on 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].

3.2.3 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 changes or adverse impact on the maternal heart [32, 33].

A nonsignificant trend was found in regard 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.

3.3 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 of blood volume or plasma (around 1500 ml) and polycythemia (around 350 ml). This “hemodilution” will maintain adequate uteroplacental flow [35].

An additional gram of iron (daily) is required during pregnancy due to the increase in red blood cells (polycythemia) 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. 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].

3.4 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 structure. 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 pregestation values. As there is 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 PCO_2 . 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 PCO_2 and thus generate a mild maternal alkalosis that ensures placental gas exchange and prevents fetal acidosis [36, 40].

3.5 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. Usually total maternal weight gain of 10–13 kg was considered as adequate (with many individual variations), although 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, and 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

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

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 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 likelihood for 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].

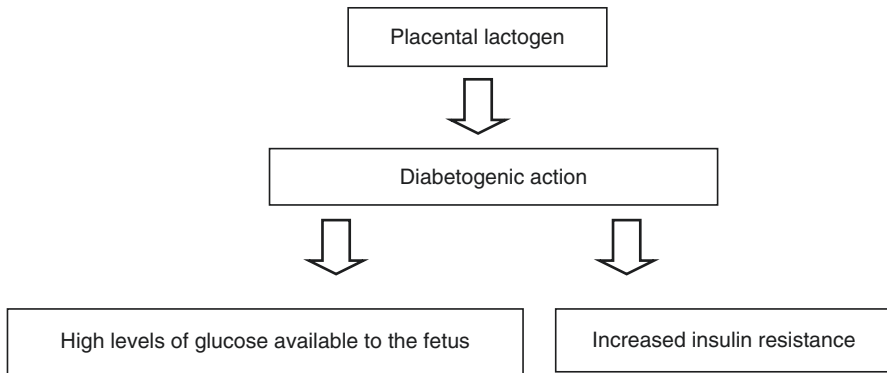


Fig. 3.1 Metabolism of carbohydrates in the second half of pregnancy [40]

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].

3.6 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].

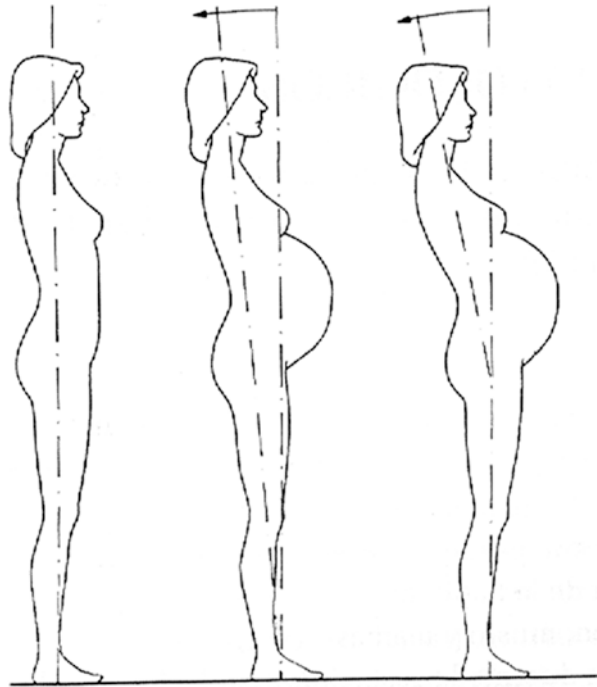
Currently, it is considered that this hyperlordosis is only apparent because the pregnant woman compensates for the deviation of her center of gravity, not by means of hyperlordosis but by displacing the entire craniocaudal axis backward (Fig. 3.2). This new position can cause low back pain, especially 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 [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

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



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]. Further development on this topic is in Chap. 5.

References

1. Ezcurdia GM. Ejercicio físico y deportes durante el embarazo [Spanish]. En: Grupo de trabajo sobre asistencia al embarazo normal. Sección de Medicina Perinatal. Cap. 11. Sociedad Española de Ginecología y Obstetricia. Manual de asistencia al embarazo normal. Ed. E. Fabre Gonzalez; 2001.
2. Melchiorre K, Sharma R, Thilaganathan B. Cardiac structure and function in normal pregnancy. *Curr Opin Obstet Gynecol.* 2012;24(6):413–21.
3. Duvekot JJ, Cheriex EC, Pieters FA, Menheere PP, Peeters LH. Early pregnancy changes in hemodynamics and volume homeostasis are consecutive adjustments triggered by a primary fall in systemic vascular tone. *Am J Obstet Gynecol.* 1993;169(6):1382–92.
4. Clapp JF, 3rd, Capeless E. (1997). Cardiovascular function before, during, and after the first and subsequent pregnancies. *Am J Cardiol* 80(11):1469-1473.
5. Kametas NA, McAuliffe F, Cook B, Nicolaides KH, Chambers J. Maternal left ventricular transverse and long-axis systolic function during pregnancy. *Ultrasound Obstet Gynecol.* 2001;18(5):467–74.
6. Geva T, Mauer MB, Striker L, Kirshon B, Pivarnik JM. Effects of physiologic load of pregnancy on left ventricular contractility and remodeling. *Am Heart J.* 1997;133(1):53–9.

7. Thornburg KL, Jacobson SL, Giraud GD, Morton MJ. Hemodynamic changes in pregnancy. *Semin Perinatol.* 2000;24(1):11–4.
8. Hytten F. Blood volume changes in normal pregnancy. *Clin Haematol.* 1985;14(3):601–12.
9. Gilson GJ, Samaan S, Crawford MH, Qualls CR, Curet LB. Changes in hemodynamics, ventricular remodeling, and ventricular contractility during normal pregnancy: a longitudinal study. *Obstet Gynecol.* 1997;89(6):957–62.
10. Atkins AF, Watt JM, Milan P, Davies P, Crawford JS. A longitudinal study of cardiovascular dynamic changes throughout pregnancy. *Eur J Obstet Gynecol Reprod Biol.* 1981;12(4):215–24.
11. Mesa A, Jessurun C, Hernandez A, et al. Left ventricular diastolic function in normal human pregnancy. *Circulation.* 1999;99(4):511–7.
12. Mabie WC, DiSessa TG, Crocker LG, Sibai BM, Arheart KL. A longitudinal study of cardiac output in normal human pregnancy. *Am J Obstet Gynecol.* 1994;170(3):849–56.
13. Mashini IS, Albazzaz SJ, Fadel HE, et al. Serial noninvasive evaluation of cardiovascular hemodynamics during pregnancy. *Am J Obstet Gynecol.* 1987;156(5):1208–13.
14. Robson SC, Hunter S, Boys RJ, Dunlop W. Serial study of factors influencing changes in cardiac output during human pregnancy. *Am J Physiol.* 1989;256(4 Pt 2):H1060–5.
15. Valensise H, Novelli GP, Vasapollo B, et al. Maternal cardiac systolic and diastolic function: relationship with uteroplacental resistances. A Doppler and echocardiographic longitudinal study. *Ultrasound Obstet Gynecol.* 2000;15(6):487–97.
16. Turan OM, De Paco C, Kametas N, Khaw A, Nicolaidis KH. Effect of parity on maternal cardiac function during the first trimester of pregnancy. *Ultrasound Obstet Gynecol.* 2008;32(7):849–54.
17. Dagher FJ, Lyons JH, Finlayson DC, Shamsai J, Moore FD. Blood volume measurement: a critical study prediction of normal values: controlled measurement of sequential changes: choice of a bedside method. *Adv Surg.* 1965;1:69–109.
18. Sociedad Europea de cardiología (ESC). Guía de práctica clínica de la ESC para el tratamiento de las enfermedades cardiovasculares durante el embarazo [Spanish]. *Rev Esp Cardiol.* 2012;65(2):171. e1–e44.
19. Savu O, Jurcut R, Giusca S, et al. Morphological and functional adaptation of the maternal heart during pregnancy. *Circ Cardiovasc Imaging.* 2012;5(3):289–97.
20. Novelli GP, Valensise H, Vasapollo B, et al. Left ventricular concentric geometry as a risk factor in gestational hypertension. *Hypertension.* 2003;41(3):469–75.
21. Hall ME, George EM, Granger JP. The heart during pregnancy. *Rev Esp Cardiol.* 2011;64(11):1045–50.
22. Schannwell CM, Zimmermann T, Schneppenheim M, Plehn G, Marx R, Strauer BE. Left ventricular hypertrophy and diastolic dysfunction in healthy pregnant women. *Cardiology.* 2002;97(2):73–8.
23. Roos-Hesselink JW, Duvekot JJ, Thorne SA. Pregnancy in high risk cardiac conditions. *Heart.* 2009;95(8):680–6.
24. Wolfe LA, Weissgerber TL. Clinical physiology of exercise in pregnancy: a literature review. *J Obstet Gynaecol Can.* 2003;25(6):473–83.
25. Sohnchen N, Melzer K, Tejada BM, et al. Maternal heart rate changes during labour. *Eur J Obstet Gynecol Reprod Biol.* 2011;158(2):173–8.
26. Artal R, Platt LD, Sperling M, Kammula RK, Jilek J, Nakamura R. I. Maternal cardiovascular and metabolic responses in normal pregnancy. *Am J Obstet Gynecol.* 1981;140(2):123–7.
27. Pivarnik JM. Cardiovascular responses to aerobic exercise during pregnancy and postpartum. *Semin Perinatol.* 1996;20(4):242–9.
28. Soultanakis HN, Artal R, Wiswell RA. Prolonged exercise in pregnancy: glucose homeostasis, ventilatory and cardiovascular responses. *Semin Perinatol.* 1996;20(4):315–27.
29. Wolfe LA, Mottola MF. Aerobic exercise in pregnancy: an update. *Can J Appl Physiol.* 1993;18(2):119–47.
30. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc.* 1982;14(5):377–81.
31. Lee SW, Khaw KS, Ngan Kee WD, Leung TY, Critchley LA. Haemodynamic effects from aortocaval compression at different angles of lateral tilt in non-labouring term pregnant women. *Br J Anaesth.* 2012;109(6):950–6.

32. Wolfe LA, Preston RJ, Burggraf GW, McGrath MJ. Effects of pregnancy and chronic exercise on maternal cardiac structure and function. *Can J Physiol Pharmacol.* 1999;77(11):909–17.
33. Perales M, Santos-Lozano A, Sanchis-Gomar F, Luaces M, Pareja-Galeano H, Garatachea N, Barakat R, Lucia A. Maternal cardiac adaptation to a physical exercise program during pregnancy. *Med Sci Sports Exerc.* 2016;48(5):896–906.
34. Barakat R. El ejercicio físico durante el embarazo [Spanish]. Madrid: Ed. Pearson Alhambra; 2006.
35. Artal R, Wiswell R, Drinkwater B. Exercise in pregnancy. 2nd ed. Williams and Wilkins: Baltimore; 1991.
36. Villaverde Fernandez S, Rodriguez Melcon A, Villaverde Baron S. Modificaciones de la sangre en el embarazo. Cambios circulatorios y respiratorios. Alteraciones de los sistemas digestivos y urinarios. Sistema óseo y dientes. Cambios en la piel. Otras modificaciones. En: Tratado de Ginecología, Obstetricia y Medicina de la Reproducción [Spanish]. Tomo 1. Ed. Panamericana. Sociedad Española de Ginecología y Obstetricia; Madrid; 2003.
37. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY. Hematological changes. In: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY, editors. *Williams obstetrics.* 23rd ed. New York: McGraw-Hill; 2010. p. 114.
38. Alaily AB, Carrol KB. Pulmonary ventilation in pregnancy. *Br J Obstet Gynecol.* 1978;85:518–24.
39. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY. Respiratory tract. In: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY, editors. *Williams obstetrics.* 23rd ed. New York: McGraw-Hill; 2010. p. 121.
40. De Migue LJ, Sánchez M. Cambios fisiológicos y adaptación materna durante el embarazo [Spanish]. En : Grupo de trabajo sobre asistencia al embarazo normal. Sección de Medicina Perinatal. Cap. 4. Sociedad Española de Ginecología y Obstetricia. Manual de asistencia al embarazo normal, 2ª edición. Ed. E. Fabre Gonzalez; 2001.
41. Barakat R, Perales M, Garatachea N, Ruiz JR, Lucia A. Exercise during pregnancy. A narrative review asking: what do we know? *Br J Sports Med.* 2015;49(21):1377–81.
42. Rasmussen KM, Yaktine AL (editors). Institute of Medicine (committee to reexamine IOM pregnancy weight guidelines, Food and Nutrition Board and Board on Children, Youth, and Families) weight gain during pregnancy: reexamining the guidelines. Provides new guidelines for weight gain during pregnancy that are based on minimizing the risks of inadequate or excessive gains to mothers as well as their infants. Washington, DC: National Academy Press; 2009.
43. Cerqueira M. Metabolismo en el embarazo. Modificaciones endocrinas. Modificaciones psíquicas. En: Tratado de Ginecología, Obstetricia y Medicina de la Reproducción [Spanish]. Tomo 1. Sociedad Española de Ginecología y Obstetricia. Ed. Panamericana; 2003.
44. Barakat R, Perales M. Resistance exercise in pregnancy and outcome. *Clin Obstet Gynecol.* 2016;59(3):591–9.
45. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY. Other system. Musculoskeletal system. In: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY, editors. *Williams obstetrics.* 23rd ed. New York: McGraw-Hill; 2010. p. 129.