Chapter 5

Non-Dipping Patten of Blood Pressure and Gestational Hypertension

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Abstract

Gestational hypertension (GH) is one of the entities of the hypertensive disorders in pregnancy (HDP), a major cause of maternal, fetal, and neonatal morbidity and mortality. Also, the HDP have been recognized as an important risk factor for cardiovascular diseases. Thus, women who develop GH or preeclampsia (PE) are at increased risk of hypertension, ischemic heart disease and stroke in later life. An ambulatory blood pressure monitoring (ABPM) takes an important role in diagnosing of hypertension in pregnancy. Also, it has been shown that ABPM had higher accuracy in the prediction of GH, premature childbirth and low birth weight, compared with the conventional blood pressure (BP) measurements. In addition, we have found that non-dipping pattern of BP is very highly related with worse pregnancy outcome in a term of preterm delivery and intrauterine growth restriction. Also, it is associated with worse maternal hemodynamics, more impaired systolic function and more pronounced cardiac remodeling compared to women with GH and dipping pattern of BP. This review aimed to explore the (a) current classifications of the HDP; (b) pathogenesis of GH and PE; (c) physiological changes of BP and maternal hemodynamics in pregnancy; and (d) pathophysiological changes of BP and maternal cardiac function, especially in a term on BP pattern.

Keywords: echocardiography, fetal growth restriction, hemodynamics, pregnancy, blood pressure, cardiac function

1. Introduction

The hypertensive disoders in pregnancy (HDP) have a great clinical significance both for mother and for fetus, complicating up to 15% of pregnancies. Approximately 15–33% of the total maternal mortality and quarter of all antenatal admissions during pregnancy is due to HDP [1]. Hypertensive pregnant women are at higher risk of intracranial bleeding, severe organ failure and disseminated intravascular coagulopathy. Hypertension is associated with placental abruption, intrauterine growth restriction (IUGR) and fetal death. Fetal mortality is 4% higher if mother has hypertension during pregnancy, and even 7% more if preeclampsia (PE) develops [2–4]. In addition, preeclampsia is one of the most common causes of preterm delivery and 25% cases of very low birth weight (<1500 g) is due to PE. Also, it has been found that mothers' deaths due to HDP in developing countries, are taking epidemic proportions, and that mortality rates in these countries are 100–200 times higher than in Europe and North America [5].

2. Hypertensive disoders in pregnancy, definition and classification

2.1. Definition of hypertension in pregnancy

The definition of hypertension in pregnancy is based on absolute blood pressure (BP) values according to the JNC 8 classification and is defined as systolic blood pressure (SBP) value ≥140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg [1, 6]. In contrast to the gradation of hypertension of the European Association for Hypertension for a general (non-pregnant) population there are two stages of hypertension in the pregnancy: mild (140–159/90–109 mmHg) and severe (≥160/110 mmHg) hypertension [7, 8].

2.2. Classification of hypertensive disoders in pregnancy

There are several classifications of the hypertensive disoders in pregnancy (HDP) in contemporary literature.

We consider that the classification of the International Society for the Study of Hypertension in Pregnancy (ISSHP) is the most appropriate and least confusing: chronic hypertension, gestational hypertension (GH), preeclampsia (PE)—de novo or superimposed on chronic hypertension and white coat hypertension (WCH) [9].

2.2.1. Chronic hypertension

Chronic hypertension exists before pregnancy or develops before 20 weeks of gestation (GW) and persists 42 days post-partum. It complicates 1–5% of pregnancies and may be associated with proteinuria.

2.2.2. Gestational hypertension

Gestational hypertension (GH) is pregnancy-induced hypertension with occurrence after 20 GW and resolves within 42 days post-partum. This means that after 42 days post-partum, re-assessment to be sure that it is not chronic hypertension, is necessary. It is characterized by poor organ perfusion. It complicates 6–7% of pregnancies. [8].

2.2.3. Preeclampsia: de novo or superimposed on chronic hypertension

If GH is associated with clinically significant proteinuria (≥0.3 g/day in a 24 h urine collection) then it is known as preeclampsia (PE). It is a pregnancy-specific syndrome that occurs after mid-gestation, defined by de novo appearance of hypertension, accompanied by new-onset of significant proteinuria. It is a systemic disorder with both maternal and fetal manifestations. Edema is no longer considered part of the diagnostic criteria, as it occurs in up to 60% of normal pregnancies. Overall, PE complicates 5–7% of pregnancies, but increases to 25% in women with chronic hypertension. It is associated with placental insufficiency, often resulting in IUGR [8, 10]. ISSHP consider that PE is diagnosed when de novo hypertension is accompanied by (a) proteinuria, or (b) evidence of other maternal organ system dysfunction such as impaired GFR, neurological problems, thrombocytopenia, abnormal liver function or (c) fetal growth restriction. Severe PE includes blood pressure ≥ 160 mmHg systolic or ≥110 mmHg diastolic but is not based upon the degree of proteinuria. This is recommended for use in research but in clinical practice all cases of preeclampsia should be considered potentially severe. Early onset preeclampsia is apparent before 34 GW. It is important to note that PE occurs in about 50% of pregnant women in whom GH appeared between 24 and 35 GW [8].

Symptoms and signs of severe preeclampsia include (**Figure 1**): pain in the upper abdomen (due to liver edema/hepatic hemorrhage), headache—visual disturbance (cerebral edema), hyperreflexia—clonus—convulsions (cerebral edema), HELLP syndrome: hemolysis, elevated liver enzymes, low platelet count.

Since proteinuria may appear later, pregnant woman with de novo hypertension accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelet count and abnormal liver enzymes should be treated as PE [9, 11].

The relatively new term is non-proteinuric PE. Recent study highlighted differences between non-proteinuric PE and GH and suggested that the subclassification of "non-proteinuric preeclampsia" should be added to existing classification of HDP. It is worth mentioning that non-proteinuric PE presents significant risk to the mother but less risk to the baby than proteinuric PE [12]. ISSHP recommends a diagnosis of preeclampsia that may not necessarily include proteinuria [9].

2.2.4. White-coat hypertension

White-coat hypertension (WCH) has been recognized in one quarter of patients with elevated office blood pressure (OBP) in the general population [13]. If a diagnosis of WCH is confirmed in the first half of pregnancy, that means normal BP using 24 h ambulatory BP monitoring (ABPM), pregnant women can be managed with regular home blood pressure (HBP)

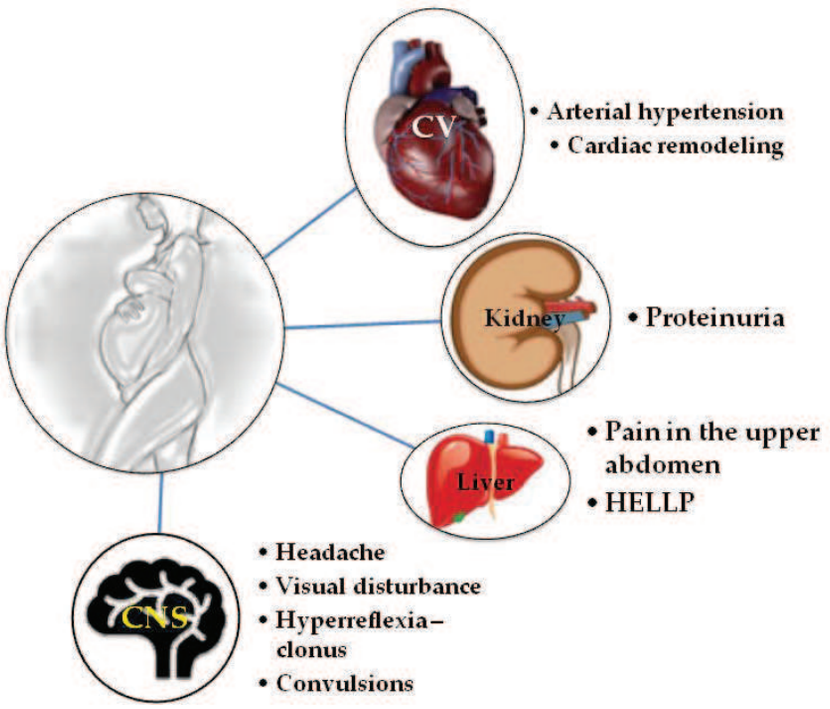

Figure 1. Signs and symptoms of severe preeclampsia (HELLP: hemolysis, elevated liver enzymes, low platelet count).

assessments. Antihypertensives can be avoided, at least up to BP levels of 160–170/110 mmHg. It is considered that near half women with WCH will develop true GH or PE [14].

ISSHP recommends that the criterion for defining hypertension in pregnancy depends on the method of measuring BP. If OBP measurement is ≥140/90 mmHg before 20 GW, it is necessary to preform ABPM. If values are:

- awake BP ≥ 130 /80 mmHg
- sleep BP ≥ 115/70 mmHg,

it is a diagnose of chronic hypertension and risk of PE is 25%. There is a need to monitor with HBP measurement if a white-coat effect is apparent on ABPM.

If values are:

- awake BP ≤ 130 /80 mmHg
- sleep BP ≤ 115/70 mmHg,

it is a diagnose of WCH, risk of GH is 50% and risk of PE is 8%.

For HBP measurement ≥135 /85 mmHg after 20 GW, hypertension is diagnosed [15].

3. Pathogenesis in gestational hypertension and preeclampsia

It is still common to consider GH and PE as "diseases of many theories" [16].

The most consistent findings indicate that an inadequate function of trophoblast, plays an important role in their origin. Actually, in normotensive pregnancies trophoblasts invade the wall of the spiral arteries and this process takes place in two phases. The first one is during the first trimester when there is a significant transformation of the decidual parts of the spiral arteries. There is a degeneration of an inner, elastic layer, and consequently the destruction of a middle, muscular, and external layer. Destroyed structures of the arterial wall are replaced by hyaline and fibrin.

The second phase coincides with the second trimester. At that time, the endovascular invasion of trophoblast involves the segment of the arcuate arteries, belonging to the myometrium. Unlike the first phase, the invasion takes place only to the muscular layer. Process is the most intense between 16 and 20 GW, and at the same time there is the largest drop in resistance of uteroplacental circulation. These morphological changes allow maximum blood flow with the least resistance through dilated blood vessels to the fetus. On the other hand, morphologically altered blood vessels become relatively insensitive to vasoconstrictor substances because they have very few smooth muscles.

The lack of endothelin 1 (ET1) in trophoblast cells during the first trimester causes inadequate proliferation and invasion of trophoblast cells, causing the absence of physiological changes in the spiral arteries of the uterus, the musculoelastic layer of spiral arteries remains unchanged, and the arteries remain narrowed throughout the pregnancy and sensitive to vasoconstrictor substances. As a consequence, there is a reduced blood supply of the placenta and hypoxia of the placenta and the fetus. This causes increased secretion of ET1 with an increase in its concentration in the bloodstream and consequent vasoconstriction. The pathophysiological mechanism itself further leads to so-called "vicious cycle" (**Figure 2**) because vasoconstriction provokes insufficiency of the placenta. This is an oxidative stress that causes an endothelial dysfunction, leads to reduced secretion of vasodilatory substances (nitric oxide—NO, prostacyclin, thromboxane A2), with simultaneous increased secretion of vasoconstrictor substances (ET1, serotonin, neuropeptide Y). Another, not less important reason for provocation and

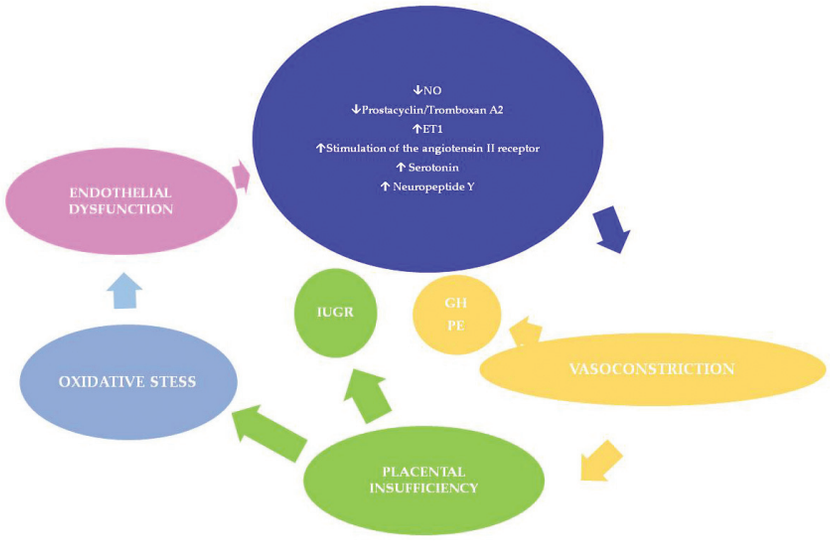

Figure 2. Pathophysiology of gestational hypertension and preeclampsia—"vicious cycle".

maintenance GH, is an increased reactivity of blood vessels to angiotensin II in women with this type of hypertension (in normotensive pregnant women, reactivity to this most powerful vasoconstrictor is physiologically reduced) [16–20].

4. Physiology changes in arterial blood pressure in pregnancy

There are numerous changes in the body of the pregnant woman as a result on an adaptation to the newborn condition. In the first trimester of pregnancy, due to a development of a new vascular network, relaxation of the blood vessels and increased influence of mediators such as NO, prostacyclin, thromboxane A2, peripheral vascular resistance decreases, causing a systemic vasodilatation which results in a physiological fall in arterial BP in that period. Systolic BP drops during the first two trimesters, with an increase in the third. Due to declining tonus of blood vessels, the decrease of diastolic BP is more prominent than systolic.

This may mask the chronic hypertension and, when hypertension is recorded later in pregnancy, it may be interpreted as gestational.

Although there is an increase in plasma renin activity during pregnancy, blood vessels of pregnant women are refractory to the vasoconstrictor effect of angiotensin II. In the further course of gravidity, there is an increase of BP, but always in the reference values [21–25]. Mean arterial pressure (MAP), as well as peripheral vascular resistance, are also decreased during the first two trimesters, and elevated in the third trimester [26].

5. Changes of blood pressure in gestational hypertension

As it has been already mentioned, in contrast to normotensive pregnancy characterized by systemic vasodilatation, there is systemic vasoconstriction that caused the increase in the total vascular resistance (TVR) in GH [27, 28]. More frequent absence of dipping profile of BP in women who develop hypertension in pregnancy was registered by performing ABPM [29–31].

5.1. 24-h arterial blood pressure pattern

5.1.1. Classification

There is a predictable pattern of BP in healthy individuals—BP is normally lower during the night-time and higher during the daytime. A dipping pattern represents a drop of nocturnal BP for >10%, of the daytime BP—their ratio is between 0.8 and 0.9 [32]. Absence of the night-time BP drop (<10% of the daytime BP, i.e., their ratio is between 0.9 and 1—non-dipping pattern) is a crucial risk factor for the cardiac and cerebrovascular events, also for the remodeling of the left ventricle (LV) in general population [33]. An increase in the prevalence of dipping profile by 10% reduces cardiovascular morbidity for 25% [34].

It is necessary to know that there are so-called extreme dippers—when a nocturnal drop of BP is >20%, (average nightly and average daily BP ratio is less than 0.8) and inverse dippers—there is no drop in BP during the night, on the contrary, there is an increase over the daily BP values (the ratio of average nightly and average daily BP is greater than 1) [32, 35].

5.1.2. The causes of the non-dipping pattern of blood pressure

There are several causes of the non-dipping pattern of BP: endocrinological disorders, renal dysfunction, disorder of the autonomic nervous system, salt-sensitivity hypertension, preeclampsia, malignant hypertension, heart transplantation, menopause, ethnicity, sex, metabolic syndrome, obesity, age, and smoking.

Some of the listed reasons can be of importance for developing GH.

5.1.2.1. Disorder of the autonomic nervous system

It is known that excessive sympathetic activity or decreased parasympathetic activity has an inadequate drop in BP during the night [36]. There is the greatest sympathetic activity overnight in inverse dippers [37], i.e., there is a significant negative correlation between sympathetic activity and a fall of BP during the night. Non-dippers have a lower drop in catecholamine levels in the urine overnight compared with dippers, and a higher activity of an α1-adrenergic receptors. [38].

5.1.2.2. Sensitivity to NaCl

Hypertensive patients, sensitive to NaCl intake do not have an adequate fall in night-time BP, while they are eating a food rich in salt. If they reduce the NaCl intake, they become dippers. The opposite, people who are resistant to NaCl intake, has no significant change in BP overnight regardless of salt intake [39, 40].

5.1.2.3. Obesity

The body mass index is inversely proportional to the drop in the night-time BP, and the prevalence of the non-dipping pattern is greater among obese people [41]. The possible cause is an increase in the concentration of catecholamines in the blood of obese people [42].

5.1.2.4. Gender

Hypertensive women with non-dipping profile have a significantly higher risk of cardiovascular events in the future than women with dipping pattern. There is no such difference in men [43].

5.1.2.5. Preeclampsia

It has been shown that there is a connection between non-dipping profile in the first trimester of pregnancy in normotensive pregnant women with a subsequent onset of hypertension and PE, but also with IUGR [44, 45]. Eight of hypertensive pregnant women whose pregnancy were complicated with PE, had an increased activity of the sympathetic autonomic nervous system [46].

5.2. Role of 24-h ambulatory blood pressure monitoring

ABPM provides the most accurate and reliable determination of the BP pattern. The results obtained by ABPM significantly more correlated with target organ damage, as well as with the prognosis of cardiovascular events, than the results obtained during an OBP measurements [33, 47–50]. In addition, ABPM is also recommended for the detection of the WCH [13].

It has been shown that ABPM is superior to OBP in the prognosis of premature termination of pregnancy, low birth weight and onset of proteinuria later in pregnancy [51–53]. A prospective double-blind study, revealed that differences in the daily-night BP pattern in hypertensive pregnant women can be helpful in determining the severity of PE and that the increase in night-time BP predominantly occurs in PE [54].

It is well known that nocturnal hypertension is associated with an exacerbation of endothelial damage in PE [55]. On the other hand, recent study has shown that the non-dipping pattern of BP in GH is associated with IUGR, preterm delivery and with the deterioration of maternal hemodynamics [56].

6. Physiology changes in cardiac function and geometry in pregnancy

Due to so-called systemic vasodilatation, characteristic of the first and the second trimester of pregnancy and decreased resistance of peripheral arteries, activation of the compensatory homeostatic mechanisms of blood flow—sympathetic nervous system, renin-angiotensin-aldosterone system, and non-osmotic secretion of vasopressin occurs. It leads to retention of sodium and water, and consequently to a purposefully increase of intravascular fluid to provide sufficient uteroplacental circulation in order to assure development and growth of the fetus [57]. This expansion of the intravascular volume leads to an increase in stroke volume (SV), which reaches the highest values between 30 and 36 GW. Due to this increase, but also because of the rise in heart rate, the cardiac output also (CO) increases. Compared with the period before pregnancy, the heart rate is 16–35% higher during pregnancy [58–61], as a compensatory mechanism due to vasorelaxation, to provide an adequate CO [23].

All mentioned leads to changes in cardiac morphology and systolic and diastolic function during pregnancy. Myocardial contractility increases, resulting in a shortening of the pre-ejection time with the prolongation of the left ventricular (LV) ejection time (ET), which is consequence of an increased SV. Most studies have shown that the parameters of systolic function, such as an ejection fraction (EF), end-diastolic volume of the LV (LVEDV), SV, ET, the systolic velocity of the mitral-septal and lateral anulus (s'), progressively increase during pregnancy, with a slightly lower value in the third trimester [62–64]. There is an increase in the volume of the left and right atrium, the left and right chambers, and the thickening of the walls of the LV, which with an increased preload in the first half of the pregnancy and an increased afterload in the last trimester of pregnancy, leads to physiological cardiac hypertrophy and to increase of myocardial mass. The LV hypertrophy becomes visible in the second trimester, while maximum values are reached toward the end of the pregnancy [23, 25, 61, 62, 64]. Myocardial mass is 12–30% higher than before pregnancy [23, 25, 61]. Due to increased preload, and therefore increased LVEDV, according to Frank Starling's law, there is an increase

in the strength of muscle contraction during the systole. Also, due to the increase in LVEDV and end-diastolic left ventricular diameter, there is an increase of the pressure on the walls of the LV, that leads to increased CO and oxygen demands. According to Laplace's law, wall stress is directly proportional to the pressure on the wall of the LV and the radius of the chamber, and inversely proportional to the thickness of the walls. In order to reduce wall stress, the walls of the left ventricle become thicker [65–67].

An increased preload in the first and the second trimester of pregnancy also affects changes in diastolic function, and there is an increase of the velocity of an early filling of the LV (E), but also an increase of the velocity of a late filling of the LV (A). Thus, during this period, the ratio E/A remains unchanged. In the last trimester of pregnancy, when there is an increase of MAP and peripheral vascular resistance, and consequently increase of afterload, the early stage of diastolic filling slows down. It is reflected in the reduction of E wave velocity and the deceleration time of E wave (DTE). As a consequence, there is a greater retention of blood in the left atrium (LA) at the end of the diastole, and consequently increase of LA work that leads to an increase of A wave velocity. The increase of A wave is also affected by an increase of heart rate. During this period there is a decrease of E/A ratio [63–69]. While some authors suggested that there is prolongation of the isovolumetric relaxation time of the LV (IVRT), others did not show significant changes in it during pregnancy [70].

7. Changes in cardiac function in gestational hypertension/preeclampsia

There are two hemodynamic disorders, characteristic for GH and PE, both the consequences of the endothelial dysfunction: reduction of CO and increasing of TVR. The first one is a result of the reduction of the total plasma volume [71–73]. The second one occurs because of vasoconstriction, increased sensitivity of blood vessels to angiotensin II and increased peripheral vascular resistance. It is interesting to note that the transition from a hypervolume state with increased CO and decreased TVR into a condition characterized by low CO and high TVR coincides with the clinical manifestation of symptoms and signs in women whose pregnancies are complicated by hypertension and preeclampsia [26, 28, 74, 75].

7.1. Systolic function in gestational hypertension/preeclampsia

Mentioned hemodynamic changes affect the function and morphology of the LV. According to the literature data, which are unfortunately, due to the specificity of the problem, still scarce and done in a small number of cases, there is mainly a change of the diastolic function of LV in GH, while the data on the change of the systolic function are fewer and more controversial [76–79].

The systolic function is determined by the ability of the heart muscle to make contraction and to pump the blood (stroke volume) into the arterial system. One of the reasons for an inconsistent data about systolic function of the LV in GH is that in most studies the systolic function was evaluated using standard parameters such as EF and SV, which are dependent, besides the contractility of the heart, on volume and heart rate. Besides, the heart loses its classical ellipsoid shape during the pregnancy [23, 80]. In order to avoid the influence of geometric remodeling, but also the influence of preload, the longitudinal systolic function of the LV has

to be evaluated (**Figure 3**). Myofibrils of the LV are arranged mainly longitudinally and oblique in the subendocardial and subepicardial layers, and circumferently in the middle layers. LV subendocardial fibers are more susceptible than the circumferential fibers to the effects of ischemia or pressure-load. First, there is a contraction of the longitudinal and the oblique myofibrils at the onset of the systole, causing a spherical LV shape, and then contraction of the circumferential myofibrils, which are responsible for the ejection [81].

The longitudinal systolic velocity—s′ and end-systolic elasticity of the left ventricle (Ees), parameters independent of the volume, are more precise measure of contractility of the heart than EF and SV [64, 82–86]. More recently, the relationship between effective arterial elasticity (Ea), which is the measure of afterload, and the elasticity of the LV at the end of the systole—Ea/Ees, has been used. The elasticity of the LV at the end of the systole shows how much the end-systolic volume of the LV increases, and the SV decreases in response to an increase of end-systolic pressure [86]. The velocity of the contraction of circumferential myofibrils (Vcf) is also a parameter that indicates the condition of the left ventricular systolic function. Similarly as the longitudinal systolic function, Vcf also decreases towards the end of the pregnancy, but never below the reference values. On the other hand, the pressure-load parameter—end-systolic wall stress (ESS) increases, especially in the third trimester, so the ratio between Vcf and ESS decreases [64, 87].

More precise data on the myocardial contractility are obtained using these parameters in patients who have a preserved EF (i.e., a preserved pump function of the heart), as the subendocardial layers of the LV are more sensitive to ischemia.

Most of the authors consider that there is depression of systolic function either as decrease of EF, SV and CO, either as decrease of longitudinal systolic velocity s′ [69, 78, 85].

It has been shown that regional longitudinal systolic function is markedly reduced in pre-eclamptic women, without regional systolic abnormalities in GH (had not observed an impact of the non-dipping pattern) [88]. Similarly, we have revealed that longitudinal systolic function is significantly reduced in women with GH and non-dipping pattern of BP, compared with both, normotensive pregnant women and those who developed GH with dipping pattern of

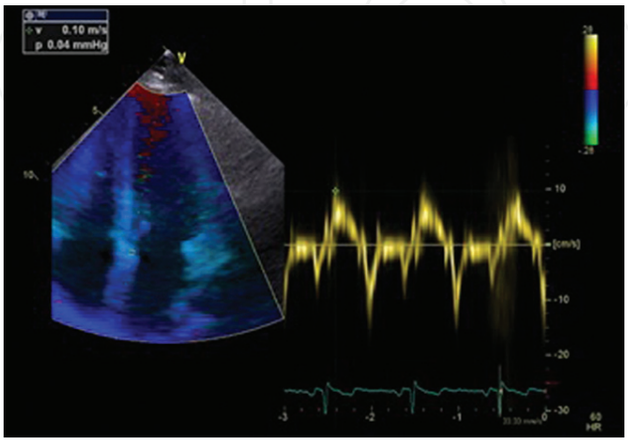

Figure 3. The longitudinal systolic function of the LV evaluated by tissue Doppler measurement—s′—longitudinal systolic velocity at mitral valve annulus.

BP, without the difference between normotensive and dippers in GH, as well as Vcf. Also, CO index was the most reduced, while Ees was the most increased in non-dippers [56].

7.2. Diastolic function in gestational hypertension/preeclampsia

The diastolic function is the ability of the LV to fill up to the normal end-diastolic volume, both at rest and in effort, with the mean pressure in LA ≤ 12 mmHg. The optimal function of the left ventricle depends on two cycles: its ability to relax and its compliance. Ability of the LV to relax, allows filling of the LV chamber from the LA in diastole. An increase in the chamber's compliance due to a sudden increase in pressure in the LV, enables the ejection of the SV into the arterial system in the systole. Since the LV relaxation process is more dependent on energy than the contraction of the heart muscle, it is logical that abnormalities of the diastolic function occur before systolic dysfunction in all situations in which myocardial circulation is compromised (ischemia, increased myocardial mass, hypertrophy) [89, 90].

If there is an increased need, for example during physical effort, pregnancy, the SV is increased without a significant increase in pressure in the LA [91]. This optimal situation is possible due to the cyclic interaction of myofilaments and the competence of the mitral and aortic valve [92]. Increased afterload will lead to decreased relaxation, especially if there is an increased preload, and this will contribute to increase of the LV filling pressure. This increase in pressure is the main consequence of the diastolic dysfunction [92, 93].

As it is mentioned, during pregnancy there is an increase in preload and myocardial mass. In hypertensive pregnancies, due to increased after-load and peripheral vascular resistance, hemodynamics is further complicated. There is a more pronounced decrease in the E/A ratio, prolongation of IVRT and DTE, changes of volume and dimensions of the LA [77–81]. In normotensive pregnant women, increased preload and decreased afterload lead to improved discharge of the LV during systole and reduction of end-systolic pressure. This results in a decrease of the pressure gradient between the LA and the LV, that reduces the required time for the drop of the pressure in the LV below the values of the pressure in the LA. As a result, filling of the LV in the diastole is done under the best conditions [25, 94]. In GH, increased afterload and TVR are followed by reduction in the LV discharge, leading to increased end-systolic volume and then to increased end-systolic pressure. This explains the prolonged IVRT because it takes longer time for the drop of the LV pressure below the LA pressure values. The delayed opening of the mitral valve and reduced LV compliance lead to reduced filling of the LV in the diastole.

It was revealed that diastolic function is more impaired in non-dippers with GH, compared to dippers, as well as global cardiac function and cardiac remodeling [56].

8. Conclusions

Recent studies have shown that the determination of the non-dipping pattern of BP, and therefore the role of ABPM, is of a great importance in women with GH.

Being an important risk factor for the remodeling of the LV in general population, the non-dipping profile of BP is also associated with a deterioration of maternal hemodynamics in GH. It is revealed that a depression of systolic function, an impaired diastolic function and remodeling of the LV are more pronounced in non-proteinuric women with non-dipping pattern of BP then in women with GH and dipping profile of BP. Besides, the non-dipping pattern was related with IUGR and preterm delivery.

According to the fact that, until nowadays, there are no data about the reversibility of these changes after delivery in the term on BP pattern, further research is needed to reveal that.138728

Conflicts of interest

None declared.

Abbreviations

A	peak velocity of the A wave
ABPM	ambulatory blood pressure monitoring
BP	blood pressure
DBP	diastolic blood pressure
DTE	deceleration time of the E wave
E	peak velocity of the E wave
Ea	effective arterial elastance
E/e′	index of the left ventricular filling pressure
Ees	left ventricular end-systolic elastance
EF	ejection fraction of the left ventricle
ESS	end-systolic wall stress
ET	ejection time of the left ventricle
ET1	endothelin 1
GH	gestational hypertension
HDP	hypertensive disoders in pregnancy
ISSHP	International Society for the Study of Hypertension in Pregnancy
IVRT	isovolumetric relaxation time of the LV

CO	cardiac output
GW	gestational week
HBP	home blood pressure
HR	heart rate
IUGR	intrauterine growth restriction
LA	left atrium
LV	left ventricle
LVEDV	left ventricle end-diastolic volume
MAP	mean blood pressure
mass	left ventricle myocardial mass
NO	nitric oxide
OBP	office blood pressure
PE	preeclampsia
SBP	systolic blood pressure
s′	longitudinal systolic velocity at mitral valve annulus
SV	stroke volume
TVR	total vascular resistance
Vcf	circumferential systolic velocity
WCH	white coat hypertension

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