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Echocardiographic Assessment of LV Geometric Pattern and Function in Pregnancy-Induced Hypertension

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ABSTRACT

Background and Objectives : Chronic hypertension induces changes in the cardiac structure and function; however, the effects of transient hypertension are unclear. Pregnancy-induced hypertension (PIH) offers a natural and spontaneous model of this condition. **Subjects and Methods :** 20 women, aged 30.3±5.1 years, with pregnancy-induced hypertension, defined as a blood pressure higher than 140/90 mmHg in the third trimester of pregnancy, with no prior history of hypertension, were studied. 20 normal pregnant women (NPW), aged 30.9±3.7 years, were used as the controls. The cardiac chamber dimensions, interventricular septal thickness (IVST), posterior wall thickness (PWT), left ventricular mass index (LVMI), relative wall thickness (RWT), E velocity, A velocity, E/A ratio, isovolumetric relaxation time (IRT), isovolumetric contraction time (ICT), ejection time (ET) and the combined index of myocardial performance (Tei index=IRT+ICT/ET) were calculated by echocardiography. Subjects were considered to have: normal geometry (NG) if both the LVMI and RWT were normal; concentric hypertrophy (CH) if both were elevated; eccentric hypertrophy (EH) if the LVMI was elevated and the RWT normal; and concentric remodeling (CR) if the LVMI was normal and the RWT elevated. **Results :** There were significant differences between the 2 groups in the following parameters; IVST: 10.5±1.3 mm in PIH *vs.* 8.6±1.0 mm in NPW ($p<0.0001$), PWT: 10.0±1.4 mm in PIH *vs.* 8.9±0.9 mm in NPW ($p<0.005$), LVMI: 113.1±20.3 g/m^2 in PIH *vs.* 85.9±14.5 g/m^2 in NPW ($p<0.0001$), RWT: 0.41±0.08 in PIH *vs.* 0.35±0.03 in NPW ($p<0.005$), E/A ratio: 0.95±0.29 in PIH *vs.* 1.56±0.27 in NPW ($p<0.0001$), IRT: 118.8±19.5 msec in PIH *vs.* 83.1±12.4 msec in NPW ($p<0.0001$) and the Tei index: 0.51±0.09 in PIH *vs.* 0.31±0.06 in NPW ($p<0.0001$). The geometric patterns of PIH women were NG in 4 (20%) and abnormal geometry in 16 (80%), of which 10 (50%) had EH. The geometric patterns of NPW were NG in 19 (95%) and abnormal geometry in 1 (5%), which also had EH. **Conclusion :** PIH increases the LVMI due to an increase in the IVST and PWT; the most frequent abnormal geometric pattern was EH. The dimensions of the left ventricle, left atrium and aortic root were unchanged. PIH showed left ventricular dysfunction, mainly diastolic. The IRT and Tei index are the most useful echocardiographic parameters to reveal left ventricular dysfunction in PIH. **(Korean Circulation J 2005;35:718-724)**

KEY WORDS : Geometry ; Tei index ; Hypertension, pregnancy-induced.

Introduction

Left ventricular hypertrophy in systemic hypertension depends on the hemodynamic conditions of the preload, afterload, the left ventricular contractility state, and the severity and duration of the process and also has adverse effects on the rates of cardiovascular events.[1-3] The secondary morphological and functional left ventricular changes induced by chronic hypertension are well known,[4] and patients with chronic hypertension are exposed to long-lasting pressure overload, with enough time to develop changes in the left ventricular structure and function. However, little is known about the changes produced by conditions of acute pressure overload. Pregnancy-induced hypertension (PIH) is a naturally occurring model of acute pressure overload imposed on a

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previously normal heart. Recently, a new easily measurable Doppler index of myocardial performance, which combines the systolic and diastolic time intervals, was introduced. This index has been reported to be simple, reproducible, and independent of the heart rate and blood pressure.[6)7)] In the present study, the left ventricular structure and function in normal pregnant women (NPW) was measured and compared in patients with PIH. The purpose of this study was to assess the impact of a model of transient hypertension, such as PIH, on the systolic and diastolic performance and structure of the left ventricle

Subjects and Methods

Study subjects

20 women, aged between 22 and 43 years (mean, 30.3±5.1 years), diagnosed with pregnancy-induced hypertension (PIH) between May 2003 and February 2004 at our institutions, were studied. PIH was diagnosed when the systolic or diastolic pressures were more than 140/90 mm Hg in a resting and sitting position on at least two occasions during the third trimester of pregnancy. None of the women had previous recordings or a history of hypertension and blood pressure (BP) during the first and second trimesters of a normal pregnancy. These measurements were compared to those obtained in 20 aged matched normal pregnant women (NPW) between the ages of 24 and 42 years (mean, 30.9±3.7 years). Diabetes mellitus, essential hypertension and risk factors (family history of ischemic heart disease, dyslipidemia, and smoking) or symptoms of coronary artery disease were excluded. Of the 20 women diagnosed as PIH, 14 were followed up, and underwent echocardiography after delivery for evaluation of the changes.

Echocardiography

All echocardiography examinations were performed on a GE Vivid 7 ultrasound machine (GE Medical System, Horden, Norway), with a 2.5 MHz transducer.

M mode echocardiography

Measurements of the left atrial dimension, left ventricular end diastolic internal dimension (LVIDd), left ventricular end systolic internal dimension (LVIDs), left ventricular end diastolic volume (LVVd), left ventricular end systolic volume (LVVs), interventricular septal thickness (IVSd), left ventricular posterior wall thickness (LVPWTd) and ejection fraction {EF=(LVIDd^3-LVIDs^3/LVIDd^3)} were performed from 2-dimensionally targeted M-mode tracings, according to the recommendations of the American Society of Echocardiography.[8)] The LV mass was calculated using the corrected cube formula of the American Society of Echocardiography[9)] and indexed for body surface area to obtain the LV mass index.

ASE-cube LV mass (g) = 1.04 {(IVSd+LVIDs+LVPWTd)^3-LVIDs^3}
LV mass by corrected ASE method (LVM_{ASE}) = 0.8 (ASE-cube LV mass) +0.6
LV mass index (LVMI) = LVM_{ASE}/BSA (g/m^2)

The relative posterior wall thickness was measured at end diastole as the ratio between the double of the posterior wall thickness to the left ventricular diastolic cavity diameter. The ventricular geometries considered were the four categories of the left ventricular anatomic remodeling, taking into account the relative posterior wall thickness at end diastole and the left ventricular mass index. Patients had a normal left geometry if the relative posterior wall thickness at end diastole and the left ventricular mass index were under the values of those corresponding to normal pregnant women, and had concentric hypertrophy when both were increased. Left ventricular eccentric hypertrophy was identified as an increase in the mass index, with a normal relative posterior wall thickness at end diastole, and concentric remodeling was defined as an increase in the relative posterior wall thickness at end diastole, with a normal left ventricular mass index.

Doppler echocardiography

Conventional Doppler measurements included the peak early and late diastolic mitral flow velocities, the isovolumetric relaxation time and total ejection isovolume index. The flow propagation velocity of the early diastolic flow was calculated using M mode color Doppler.

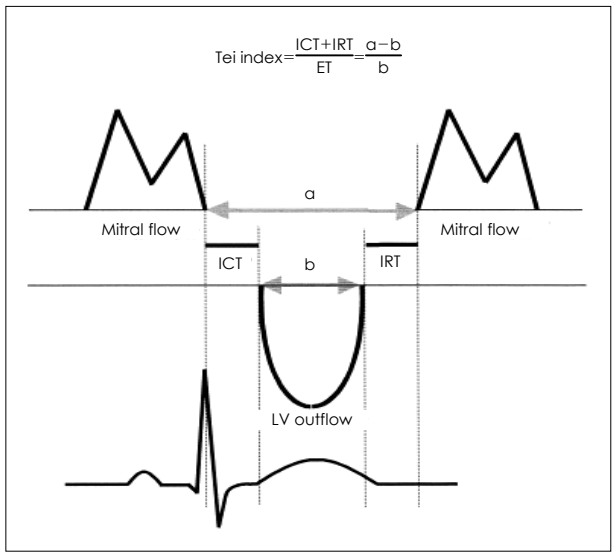

Fig. 1. The interval a (from the cessation to the onset of mitral inflow) is equal to the sum of the isovolumetric contraction time (ICT), ejection time (ET) and IRT. The interval b represents the ET at the left ventricular outflow tract. The Tei index of the myocardial performance was calculated as (a-b)/b. IRT: isovolumetric relaxation time, LV: left ventricular.

Pulsed Doppler measurements were obtained with a transducer in the apical four-chamber view, with the Doppler beam aligned perpendicular to the plane of the mitral annulus. The sample volume was placed between the tips of the mitral leaflets. Five consecutive beats were used to calculate the Doppler variables. Doppler time intervals were measured from the mitral inflow and LV outflow velocity-time intervals, as described by Tei et al.[6] The interval 'a' from the cessation to the onset of mitral inflow was equal to the sum of isovolumetric contraction time (ICT), ejection time and isovolumetric relaxation time (IRT). LV ejection time 'b' was the duration of LV outflow velocity profile. Thus, the sum of ICT and IRT was obtained by subtracting 'b' from 'a' (Fig. 1). The index of the combined LV systolic and diastolic functions (the sum of ICT and IRT divided by ejection time) was calculated as a-b/b. All echocardiograms were performed and analyzed by one observer.

Statistical analysis

All data are expressed as the mean and standard deviation (SD). Data were analyzed using standard statistical software (SPSS package version 11.0), and comparisons of all measurements between NPW and patients with PIH made with paired Student's t- and chi-squared tests. A two-tailed $p<0.05$ was considered significant.

Results

Baseline characteristics

The systolic and diastolic blood pressures, weight, weight gain during pregnancy and body surface area were significantly higher in PIH compared to NPW (all $p<0.01$). There were no significant differences in age, height, hemoglobin level and family history between PIH and NPW (Table 1).

Ventricular geometric patterns

The interventricular septal thickness (10.5±1.3 mm in PIH *vs.* 8.6±1.0 mm in NPW) and posterior wall thickness (10.0±1.4 mm in PIH *vs.* 8.9±0.9 mm in NPW) were significantly higher in patients with PIH (all $p<0.01$), but there were no significant differences in the left atrial dimension, left ventricular dimension, and volume and aortic dimension. The LVMI (113.1±20.3 g/m^2 in PIH *vs.* 85.9±14.5 g/m^2 in NPW) and RWT (0.41±0.08 in PIH *vs.* 0.35±0.03 in NPW) were significantly higher in patients with PIH (all $p<0.01$) (Table 2). The LVMI and RWT of NPW were 85.9±14.5 g/m^2 and 0.39±0.03, respectively, and we considered the mean±2 SD of the upper normal limit of LVMI and RWT of NPW (114.9 g/m^2 and 0.45, respectively). Normal geometric values were obtained in 4 patients with PIH (20%), eccentric hypertrophy in 10 (50%), concentric remodeling in 3 (15%) and concentric hypertrophy in 3 (15%), versus 19 (95%), 1 (5%), 0 (0%) and 0 (0%), respectively, in NPW (Fig. 2).

Table 1. General characteristics of the study patients

Parameter	NPW	PIH	p
Age (years)	30.9±3.7	30.3±5.1	NS
Weight (kg)	65.7±9.6	77.1±11.8	p<0.01
Weight gain (kg)	11.2±4.4	16.3±5.6	p<0.01
Height (cm)	160.7±4.8	161.9±3.9	NS
Body surface area (m^2)	1.7±0.1	1.9±0.2	p<0.01
Weeks of pregnancy	$38^{+2}\pm1^{+2}$	$36^{+1}\pm2^{+1}$	p<0.001
BP systolic (mmHg)	109.0±7.2	164.0±15.4	p<0.001
BP diastolic (mmHg)	69.5±6.1	105.5±10.5	p<0.001
Hemoglobin (g/dL)	10.3±1.4	10.3±1.4	NS
Family history of hypertension	8%	6%	NS

Values are the mean±SD. NPW: normal pregnant women, PIH: pregnancy induced hypertension, BP: blood pressure, NS: no significance

Ventricular functional changes

There were no significant differences in the ejection

Table 2. M-mode assessment of the left ventricular geometry in PIH and NPW

Parameter	NPW	PIH	p
LAD (mm)	34.8±4.4	37.1±3.9	NS
LVDd (mm)	47.6±4.9	49.6±6.1	NS
LVDs (mm)	30.9±3.6	33.1±6.5	NS
LVVd (cm^3)	102.2±12.7	98.9±11.4	NS
LVVs (cm^3)	38.1±10.5	35.8±9.8	NS
IVSTd (mm)	8.6±0.9	10.5±1.3	p<0.001
PWTd (mm)	8.9±0.9	10.1±1.3	p<0.01
LVMI (g/m^2)	85.9±14.5	113.1±20.3	p<0.001
RWT	0.39±0.03	0.41±0.08	p<0.01
ARD (mm)	28.2±2.1	30.6±3.7	NS

Values are the mean±SD. NPW: normal pregnant women, PIH: pregnancy-induced hypertension, LAD: left atrial dimension, LVDd: left ventricular diastolic dimension, LVDs: left ventricular systolic dimension, LVVd: left ventricular end diastolic volume, LVVs: left ventricular end systolic volume, IVSTd: interventricular septal thickness, PWTd: left posterior wall thickness, LVMI: left ventricular mass index, RWT: relative wall thickness, ARD: aortic root dimension, NS: no significance

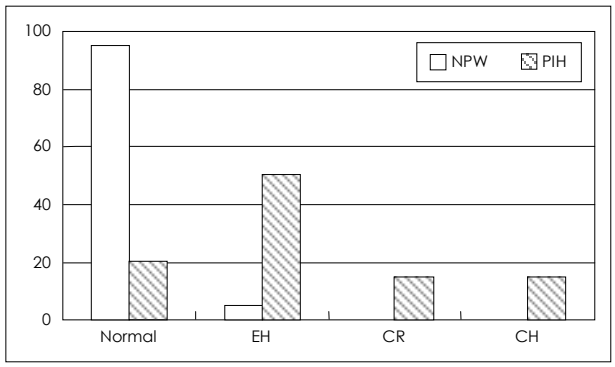

Fig. 2. Distribution of hypertensive pregnant women according to their geometric patterns of ventricular hypertrophy, considering the mean±SD. NPW: normal pregnant women, PIH: pregnancy-induced hypertension, EH: eccentric hypertrophy, CR: concentric remodeling, CH: concentric hypertrophy.

fraction, shortening fraction and peak E velocity between NPW and PIH. The peak A velocity was significantly higher in patients with PIH (83.3±23.0 msec) than NPW (58.2±10.2 msec) ($p<0.01$), and the E/A ratio was significantly lower in patients with PIH (0.95±0.29 in PIH *vs.* 1.56±0.27 in NPW, $p<0.01$). The IRT (118.8±19.5 msec in PIH *vs.* 83.1±12.4 msec in NPW) and Tei index (0.51±0.09 in PIH *vs.* 0.31±0.06 in NPW) were significantly higher in patients with PIH (0.001) (Table 3). The variables that more clearly separated NPW from patients with PIH were the IRT and Tei index. The IRT reached maximal significance, and there was minimal overlapping between groups. The Tei index was borderline (0.43) in only 3 NPW, and normal in the rest. Conversely, this was found to be abnormal in all of the PIH patients (Fig. 2).

Clinical course and follow-up echocardiography

Of the 20 PIH women, 14 were able to undergo follow-up echocardiography after 6 month for evaluation of the geometric and functional changes of LV. 2 women progressed to pre-eclampsia during 34th and 35th gestational weeks, respectively, and showed reduced systolic function, but recovered normal systolic function within 3 months of delivery, with normal follow-up echocardiograms. 10 showed transient hypertension, but recovered normal blood pressure within 3 months of delivery, and the follow-up echocardiograms showed a normal LV geometry and Tei index. 2 progressed to chronic hypertension and had persistent high blood pressure after 6 months of delivery, and follow-up echocardiography showed an abnormal geometry and high Tei index (0.53 and 0.52, respectively). However, the PIH women who recovered normal blood pressure after delivery, showed normal, but still higher RWT (0.40±0.03 in PIH *vs.* 0.39±0.06 in NPW, p=NS) and Tei index (0.36±0.10 in PIH *vs.* 0.34±0.06 in NPW, p=NS) compared to the NPW after delivery on follow-up echocardiography (Table 4).

Table 3. Left ventricular diastolic variables in PIH and NPW

Parameter	NPW	PIH	p
EF (%)	65.8±7.5	64.7±6.9	NS
FS (%)	35.8±2	34.9±4.2	NS
ET (ms)	288.9±28.9	303.7±30.7	NS
E (msec)	85.5±23.6	77.1±19.9	NS
A (msec)	58.2±10.2	83.3±23.0	p<0.01
E/A	1.6±0.3	0.9±0.3	p<0.01
IRT (ms)	83.1±12.4	118.8±19.5	p<0.001
Tei index	0.3±0.06	0.5±0.09	p<0.001

Values are mean±SD. NPW: normal pregnant women, PIH: pregnancy-induced hypertension, ET: ejection time, E: peak E velocity, A: peak A velocity, IVRT: isovolumetric relaxation time, NS: no significance

Discussion

When the heart faces a hemodynamic burden, it can act in the following way to compensate: 1) use the Frank-Starling mechanism to increase crossbridge formation; 2) augment muscle mass to bear the extra load; and 3) recruit neurohormonal mechanisms to increase contractility. The type of overload to which the heart is subjected determines the pattern of hypertrophic growth. Volume overload generally produces an increased ventricular cavitary volume, and pressure overload increases the left ventricular mass out of proportion to the volume.[10] Hypertrophy, secondary to pressure overload, has a great impact on the cardiovascular system, and may develop with different patterns, generically called ventricular geometries.[11-13] The heart must adapt its wall

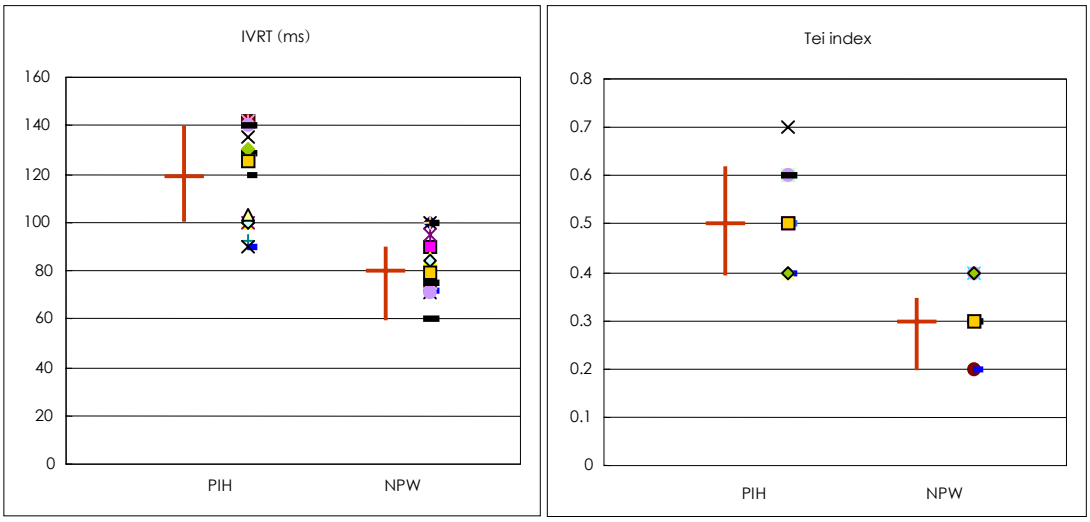

Fig. 3. Comparison of the isovolumetric relaxation time (IRT) and the Tei index in pregnancy-induced hypertension (PIH) and normal pregnant women (NPW). The IRT reached maximal significance, and there was minimal overlapping between groups. The Tei index was borderline in three of NPW subjects, but normal in rest. Conversely, this was found to be normal in only one of the PIH patient, borderline in three, and abnormal in the rest. IVRT: isovolumetric relaxation time.

Table 4. Follow-up echocardiographic findings and clinical features in hypertensive pregnant women after delivery

Case	Preeclampsia before delivery	BP (mm/Hg)	LV geometric pattern (LVMI/RWT)	IVRT (ms)	Tei index
1	Yes	120/70	Normal (90/0.40)	90	0.37
2	No	130/80	Normal (100/0.41)	88	0.35
3	No	110/70	Normal (95/0.35)	98	0.32
4	No	100/70	Normal (90/0.38)	95	0.28
5	No	120/80	Normal (98/0.40)	87	0.39
6	No	130/85	Normal (102/0.42)	102	0.40
7	No	140/100	EH (125/0.42)	155	0.53
8	Yes	120/80	Normal (90/0.38)	98	0.36
9	No	120/75	Normal (102/0.40)	100	0.42
10	No	110/75	Normal (95/0.39)	85	0.38
11	No	150/100	CH (120/0.48)	135	0.52
12	No	100/70	Normal (100/0.39)	98	0.34
13	No	130/80	Normal (90/0.40)	76	0.28
14	No	120/70	Normal (100/0.40)	88	0.13
Mean values			LVMI; 99.8±10.7 RWT; 0.40±0.03	99.6±20.8	0.36±0.10

BP: blood pressure, LVMI: left ventricular mass index, RWT: relative wall thickness IVRT: isovolumetric relaxation time, EH: eccentric hypertrophy, CH: concentric hypertrophy, LV: left ventricular

thickness to this increase in pressure load, to decrease the parietal stress, despite the short lasting overload.

During normal pregnancy a number of hemodynamic changes take place, such as increases in the blood and stroke volumes together with the heart rate, and decreases in the peripheral resistance and mean. As a consequence of these changes, the heart of a woman is structurally altered (remodeled) during pregnancy. These modifications include increases in the end-diastolic volume and functional changes in the left ventricle after the first half of the pregnancy.[14)15)] The hemodynamic pattern is quite different in PIH. The peripheral resistance increases, whereas the plasmatic and cardiac volumes may remain normal or even decrease.[16)] These changes are transient, produce an acute pressure overload on the left ventricle, and normalize rapidly after delivery. Therefore, PIH may be an adequate model for evaluating the consequences of a transient pressure overload on the myocardium during a short period of time.

In relation to the left ventricular structure and function in PIH, published studies are not only scarce, but controversial on several topics. Sanchez et al[17)] and Thompson et al[18)] found no significant changes in the left ventricular mass in these patients, whereas Vazquez Blanco et al[19)] found that in PIH, the left ventricular mass and RWT were increased and the geometric pattern of the left ventricle modified. In the present study, patients with PIH had a significant increase in the left ventricular mass, a situation that especially reflects an increase in the septal and posterior wall thicknesses, without changes in the left ventricular diastolic diameter. The left ventricular mass was not known before the pregnancy; however, it can be assumed it was previously normal, as the patients had no history of hypertension or any other condition that could cause an increase. The mean±2 SD of the LVMI and RWT of NPW were considered as the upper normal limits of the LVMI and RWT of NPW (114.9 g/m^2 and 0.45, respectively), which differ from those reported by Ganau et al,[11)] who consider 106 g/m^2 and 0.44, respectively, as the normal upper limit for the left ventricular mass, whereas these were found by So et al[20)] and Park[21)] to be 100.5 and 103.6 g/m^2, respectively, in Koreans. These different results may have been due to differences in the population, because the pregnant patients in this study had other hemodynamic modifications that could have produced different changes in the structure and function of the left ventricle.

Pregnancy-induced hypertension appears within a short time, is short lasting, and affects a healthy cardiovascular system free of previous pathological influences, so the organism does not have enough time to adapt itself to this pressure overload. Pregnancy imposes a volume overload on the circulatory system, represents a physiological explanation for this trend and for the greatest incidence of eccentric hypertrophy, as observed in our series. This pattern of eccentric hypertrophy (cavity dilatation with a decrease in ratio of wall thickness/chamber dimension) is initially compensatory, such that the heart can meet the demand to sustain a high stroke volume, and also a form of left ventricular failure due to pressure overload.[22)] In the present study, no patient had overt cardiac failure, although the possibility that this type of geometric remodeling may hide a latent form of contractile failure can not be ruled out. Our study showed 2 out of 3 women with PIH to have a geometric pattern of concentric remodeling progressed to preeclampsia, which is consistent with the mechanism sug-

gested by Ganau et al,[11] who proposed that the increase in the peripheral resistance with a decrease in preload is caused by a contraction of the intravascular volume. Concentric Hypertrophy is the type of geometric pattern expected in patients with an increased afterload.[23] However, it was less frequently observed in our series, which may be explained by pregnancy-induced hypertension being a short lasting phenomenon.

The majority of chronic hypertensive patients, even those with a relatively prolonged form of the disease, and patients with PIH, show a normal geometric pattern. It may be possible that other issues, such as the contractile state of the myocardium, and even genetic causes can explain the lack of structural changes seen in patients with essential hypertension, as well as in patients with PIH.

Studies related to the myocardial function in PIH[24] are scant, and in some cases controversial. Discrepancies in the findings related to the diastolic function are evident, even in normal pregnancy,[25] as some investigators highlight significant changes in the diastolic function; whereas, others fail to find any,[26] but there is agreement in the literature pertaining to alteration in the diastolic function in PIH. Vazquez Blanco et al[27] concluded that the E/A ratio tended to be lower in PIH; the E velocity did not change, but the A velocity was higher and the IRT markedly increased in this group of patients; these findings were consistent with our results.

A new Doppler index was recently introduced by Tei,[6][7] which included the duration of the systolic and diastolic time intervals to globally evaluate myocardial performance.

The systolic time intervals have been found to correlate with other systolic function parameters, such as the stroke volume, cardiac output, ejection fraction and positive dP/dt of the left ventricle, with the ICT more reliably reflecting the myocardial contractility.[28] In the evaluation of the diastolic function, the IRT correlates with a negative dP/dt and with the tau constant of relaxation. The Tei index consists of the sum of the IRT and ICT, divided by the ejection time. The IRT and ICT are prolonged, whereas the ejection time tends to be shorter in cardiac diseases.[29] Thus, the index has the potential to unmask lesser degrees of left ventricular myocardial dysfunction. The index correlates closely with both positive and negative dP/dt, the ejection fraction, tau constant, functional class and the prognosis.[30] Its usefulness, added to the fact it can be obtained in a simple and reproducible manner, means it can easily be added to clinical practice.

Only one recent study[7] has been reported concerning hypertension using the Tei index in a chronic hypertensive population. These investigators found a close relationship between the index and the RWT, in that the Tei index was found to be increased significantly compared to normal in patients with concentric remodeling or concentric hypertrophy, irrespective of the systolic function. We also found that in PIH patients the RWT was increased, and one of the more frequent left geometric patterns is eccentric hypertrophy; therefore, an alteration to the index is expected in this group of patients. The present study showed that the BP of PIH patients decreased after delivery, as is usual, although the RWT and Tei index recovered to normal values, but remained high compared to the NPW at the time of the follow-up echocardiography. However, our results suggest that the structural and functional changes produced by this transient pressure overload persist, even after the BP normalizes. To our knowledge, the present investigation is the first to evaluate the ventricular geometry and Tei index in PIH after delivery. Although a normal pattern is common in the ventricular geometry is found in patients with PIH, this short lasting pressure overload is capable of inducing changes in the structure of the left ventricle. However, the cavitary dimensions of the left ventricle, atrium and aortic root remain unchanged, indicating PIH does not have enough time to produce changes in the left atrium and the aortic root. It is surprising that only one patient in the PIH group had a normal left ventricular function. Thus, this study confirms that the IRT (which reflects diastolic relaxation) and the Tei index of global myocardial performance are altered in PIH, and the most powerful parameters for unmasking functional ventricular impairment in PIH patients.

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