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ORIGINAL ARTICLE

Left Atrial Strain as a Predictor of Early Left Atrial Dysfunction in Well Controlled Hypertension with Normal Left Atrial Size

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ABSTRACT

Background: Lately, there has been a growing focus on structural and functional changes in the left atrium (LA), which may be early signs of heart damage brought on by hypertension (HTN). Speckle tracking echocardiography can detect a variety of myocardial fiber failure indicators. We aimed to evaluate left atrial function using speckle tracking echocardiography in patients with well-controlled hypertension and normal left atrial size.

Methods: This study included 52 hypertensive patients for early detection of left atrial dysfunction even before left atrial enlargement and compared to 52 normal control subjects. The study population was subjected to complete history taking, full clinical examination, standard 12 leads electrocardiogram and echocardiography. LA stroke volume (LASV) and LA expansion index were calculated. Global peak atrial longitudinal strain, LV systolic function, and LV diastolic function were determined. The early diastolic E′ velocity and late diastolic A′ velocity were estimated by tissue Doppler imaging.

Results: There was a statistically significant difference between the two groups as regard DM and LV diastolic dysfunction; as diabetic patients had lower global PALS and patients with grade II diastolic dysfunction had lower global PALS. A negative correlation between global PALS and age, BMI, SBP, LVMI and LAVI, while there was a statistically significant positive correlation with LA expansion index.

Conclusion: Speckle tracking echocardiography is a helpful new method for identifying early LA malfunction in hypertension before LA enlargement happens.

Keywords: Left Atrial Dysfunction; Hypertension; Left Atrial Strain; Speckle tracking echocardiography.

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INTRODUCTION

Hypertension is the most significant preventable risk factor for cardiovascular disease (CVD) and all-cause mortality worldwide. Due to population aging and increased exposure to lifestyle risk factors like poor diets (i.e., high salt and low potassium intake and lack of physical activity), the prevalence of hypertension is increasing internationally [1]. The mediastinum contains the left atrial (LA), which is positioned behind the right atrium (RA) and leftward. The LA's walls are classified as anterior, septal, left lateral, posterior (infero-posterior), and superior (roof) [2].

There are two primary ways that arterial hypertension can impact the left atrium: hemodynamic and neurohumoral. The development of arterial hypertension first places a greater burden on the left ventricle, which must raise its pressure in order to pump blood into the bloodstream. The pressure in the left atrium may rise as a result of the increased LV filling pressures. Both animal models of

hypertension and individuals with arterial hypertension have been shown to have elevated LA pressures. Furthermore, atrial remodeling and AF are known to be triggered by increased LA pressure and/or atrial stretch [3].

Using speckle tracking echocardiography, we aimed to assess left atrial function in patients with normal left atrial size and well-controlled hypertension

METHODS

This comparative cross-sectional study was carried out at the Cardiology Department, Faculty of Medicine, Zagazig University. 52 age-matched patients and 52 patients, non-hypertensive control volunteers were involved. The study was approved by ethical committee of Faculty of Medicine, Zagazig University (IRB number 9369-23-3-2022(. Informed written consent was obtained from all cases.

Inclusion criteria:

-People with systemic arterial hypertension (defined as a systolic blood pressure of 140 mmHg or a diastolic blood pressure of 90 mmHg) or those with a documented history of hypertension who are taking antihypertensive medication.

-Echocardiographic evidence of normal LA size (defined as LA volume index < 28 ml/m2) was found in all cases.

-Electrocardiograms showed a normal sinus rhythm in every subject.

Exclusion criteria:

-Individuals with a history of cardiomyopathy or valvular heart disease, atrial fibrillation, mitral or aortic valve heart disease, poor imaging quality, or the presence of a significant arrhythmia such as AF, atrial flutter, or SVT are among those with conditions that impact LA size and function, such as documented coronary artery disease.

***Methods*:**

All patients were subjected to a thorough medical history, with special emphasis to risk factors such age, gender, diabetes, high blood pressure, smoking, dyslipidemia, and a positive family history of heart disease, history of drugs, including types, dosages, and lengths of use, history of any cardiac conditions, such as arrhythmias, heart failure, or ischemic heart disorders. Chronic liver disease, bronchopulmonary disorders, or renal issues are examples of extra-cardiac issues. Also, a clinical assessment was done with a focus on the pulse's rate, rhythm, volume, equality on both sides, and unique characteristics. Systolic and diastolic blood pressure were measured with a traditional sphygmomanometer. Subjects were deemed hypertensive if their DBP was greater than 90 mmHg and their SBP was greater than 140 mmHg. A local cardiac examination is necessary to rule out valvular heart disease by looking for murmurs.

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12-Lead surface ECG was done at rest to evaluate the rhythm, ischemia alterations as Q and ST-T waves, abnormalities in the left atrium as p. mitral in left atrial hypertrophy, and changes in the amplitude of the QRS complex as in left ventricular hypertrophy.

***Echocardiography*:**

By using GE Medical Systems' Vivid-S5 or Vivid-9, a typical echocardiographic investigation was conducted utilizing 2D, M-mode, and Doppler techniques along with speckle tracking for LA.

1. Standard assessment of left atrium

The biplane area-length approach is used to determine LA volume from two-chamber and four-chamber apical pictures. Before the mitral valve opens, the maximal LA volume is measured, and after it closes, the minimal LA volume is found. The equipment automatically calculates the length L, measured from the back wall to the line across the mitral valve hinge points (in centimeters), is the input for the LA volume index in milliliters per square meter together with the weight (kg) and height (cm). A1 and A2 are the maximum planimetered LA areas in the corresponding apical 2-chamber (A2C) and apical 4-chamber (A4C) perspectives.

The difference between the maximal and minimal LA volumes is the LA function's total LA stroke volume (LASV). The LA growth index is computed by dividing the total LASV by the minimum LA volume × 100 [4].

2. Speckle tracking echocardiography

Acoustic-tracking software (Echo Pac, GE, USA) was used to process the recordings which allowed for the semi-automated determination of speckle-based strain offline. It is stated that a good ECG is necessary for a good analysis. During breath hold, normal two-dimensional grayscale imaging is used to take apical four-, two-, and three-chamber images. maximizing vision of the LA cavity and avoiding foreshortening of the left atrium while being cautious to expand the LA space in apical perspectives [5].

Three consecutive cardiac cycles are averaged. To increase the viability of the frame-to-frame tracking method, the frame rate is changed from 60 to 80 frames per second and to combine temporal resolution with adequate spatial clarity. After offline analysis, a point-and-click technique is used to manually trace the LA endocardial surface in both the apical two-chamber and four-chamber views. The algorithm then automatically creates an area of interest (ROI) by tracing the epicardial surface [To trace the ROI in the discontinuity of the LA wall, the orientation of the LA endocardial and epicardial surfaces at the junction with the pulmonary veins and LA appendage is extrapolated. Following human adjustments to the ROI width and shape, the software divided the LA wall into six segments (basal, mid, and apical segments) in the apical 4 and 2 chamber views. Following an automatic scoring system, each segment's tracking quality is classified as either acceptable or unacceptable, with the possibility for further manual modification. The software rejects and removes from the analysis segments when sufficient image quality could not be acquired. Twelve segments in total are then examined in participants with sufficient image quality [7].

R-to-R rating on the ECG was used by software to automatically track the myocardium during the cardiac cycle. For tracking purposes, the region of interest was set to the LA wall thickness that was the smallest. Finally, the software produced a mean curve of all segments and longitudinal strain curves for each segment, which illustrate the pathophysiology of atrial function [8].

LA reservoir strain (LASr), which is determined by subtracting the end-diastole from the beginning of filling, is a measure of the positive atrial strain that occurs during the reservoir phase as the left atrium fills and stretches. It peaks in systole just before the mitral valve opens and at the conclusion of LA filling.

After that, the mitral valve opens, causing passive LA emptying. This lowers atrial strain and causes the strain curve to deflect negatively until it reaches a plateau period, which is comparable to diastasis. This is known as LA conduit strain (LAScd), which is determined by subtracting the onset of atrial contraction from the onset of filling. The strain curve then shows a second negative deflection that corresponds to atrial systole and represents LA contractile strain (LASct), which is determined by dividing end-diastole by the beginning of atrial filling [9]. The average of the corresponding results from both viewpoints was then used to determine the global peak atrial longitudinal strain. The reservoir function, which is most affected by hypertension, is the most dependable indicator of LA function. [10].

3. LV systolic function

It is measured using the biplane method of discs, a modified form of Simpson's rule, in 2D apical 4-chamber and 2-chamber perspectives [4].

4. LV diastolic function

It is computed using the deceleration time, the E/A ratio, and the peak E and A velocities by positioning the sample volume near the mitral valve's septal annulus, Doppler tissue imaging is used to assess the early diastolic E′ and late diastolic A′ velocities and computing the E/E′ ratio Three classifications are assigned to the patients based on LV diastolic dysfunction: poor relaxation (grade 1), pseudo-normal pattern (grade 2), and restrictive pattern (grade 3) [11].

5. LV mass

LV mass (LVM, in grams) is calculated using the Penn formula [12]: LVM = 1.04 ([LVIDD +

PWTD + IVSTD]^3- [LVIDD]^3) -13,6 g. Where, LVIDd is LV end-diastolic internal diameter; PWTd, diastolic posterior wall thickness; and IVSTd, thickness of the diastolic interventricular septum. The LV mass index is then calculated by indexing LVM to body surface area (BSA).

Ethical Consideration:

The Zagazig University Academic and Ethical Committee gave their approval to the study. Written, informed consent was provided by each participant. This study has been conducted in accordance with the Declaration of Helsinki, which is the World Medical Association's Code of Ethics for research involving human subjects.

Statistical analysis:

Microsoft Excel was used to examine the data before it was imported into the Statistical Package for the Social Sciences (SPSS) version 20.0. Quantitative data is represented by mean ± SD, while qualitative data is represented by numbers and percentages. distinctions between Kruskal Wallis and ANOVA for quantitative independent multiples. Quantitative diagnostic tests' sensitivity and specificity that divide instances into two categories: exceptional accuracy (0.90-1(%)), good accuracy (0.80-0.90%), poor accuracy (0.60-0.70(%)), and failed accuracy (0.50-0.60% (%))—are assessed using the receiver operating characteristic (ROC) curve. For significant results, the P value was set at less than 0.05, and for very significant results, it was less than 0.001.

RESULTS

According to various demographic statistics, the two groups in the current study did not differ significantly (P>0.05**).** The two groups' systolic and diastolic blood pressures differed statistically significantly; the HTN group's median SBP and DBP were higher (P<0.001) (Table 1 supplementary).

Regarding LVMI and LV diastolic dysfunction, there was a statistically significant difference between the two groups.; the hypertensive group had a greater mean LVMI (P=0.04). Regarding LV diastolic dysfunction, none of the patients in the normotensive group (0%) showed grade II diastolic dysfunction, but 23.1% of hypertension patients did (P<0.001) (Table 1).

The two groups differed statistically significantly in terms of global PALS, LASV, LA expansion index, and LAVI; the hypertension group had a greater mean of LAVI (P=0.02), while the hypertensive patients had lower means of LASV, Global PALS and the LA growth index (P<0.001). Regarding LASr and LAScd, there was a statistically significant difference between the two groups.; the hypertensive group had higher means for LASr, LAScd, and LASct (P=0.03), P=0.005, and P=0.03), respectively (Table 2).

ACEIs/ARBs were the most commonly used medication, used by 82.7% of patients, followed by CCBs (57.7%) and diuretics (40.4%), while beta-blockers were the least commonly used medication, used by 19.2% of patients (Figure 1).

Patients with diabetes had a statistically significant lower global PALS than patients without diabetes (P=0.04), and patients with grade II diastolic dysfunction had a lower global PALS than patients with grade I diastolic dysfunction and those with normal diastolic function. (P=0.008) (Table 3).

Age (r=-0.467, P<0.001), BMI (r=-0.385, P=0.006), SBP (r=-0.561, P<0.001), LVMI (r=-0.701, P<0.001), and LAVI (r=-0.467, P<0.001) all showed negative correlations with global PALS. Although the LA growth index showed a statistically significant positive connection (r=0.432, P=0.002) (Figure 2).

To find the best cutoff value for detecting left atrial impairment, ROC analysis was performed. The results indicated that global PALS had the maximum sensitivity (98%) and specificity (86%) at < 37, with an area under the curve of 0.929 (Figure 3).

According to univariate analysis, global PALS had a negative correlation with LA impairment, whereas age, DM, SBP, LVMI, LAVI, LASV, and the LA expansion index had a positive correlation. Global PALS had a negative correlation with LA impairment, but SBP had a positive correlation, according to multivariate

analysis. Therefore, Global PALS and SBP can be utilized separately to indicate LA impairment (Table 4).

Table 1: General echocardiographic data among studied groups

Variables		HTN group (n=52)	No-HTN group (n=52)	P Value
EF *(%)*	*Mean ± SD*	64.6 ± 6.52	63.04 ± 6.71	
	Range	(57 – 70)	(55 – 70)	0.23
LVMI *(gm/m^2)*	*Mean ± SD*	82.6± 20.9	75.21± 14.2	
	Range	(56 – 137)	(57 – 95)	0.04
E *(m/s)*	*Mean ± SD*	0.81 ± 0.16	0.85 ± 0.17	
	Range	(0.6 – 0.9)	(0.6 – 0.9)	0.23
A *(m/s)*	*Mean ± SD*	0.8 ± 0.18	0.7 ± 0.26	
	Range	(0.2 – 1.35)	(0.2 – 1.35)	0.06
E/A ratio	*Mean ± SD*	0.99 ± 0.44	1.11 ± 0.36	
	Range	(1.2 – 2.2)	(0.9 – 1.8)	0.07
DT *(ms)*	*Mean ± SD*	205 ± 47.6	189 ± 36.9	
	Range	(85 – 240)	(73 –210)	0.06
LV diastolic dysfunction *(n. %)*	None	11 (21.2%)	34 (65.4%)	
	Grade I	29 (55.8%)	18 (34.6%)	
	Grade II	12 (23.1%)	0 (0%)	<0.001
LVEDD *(cm)*	*Mean ± SD*	5.26 ± 0.45	5.11 ± 0.32	
	Range	(4.25 – 6.22)	(3.9 – 6)	0.06
LVESD *(cm)*	*Mean ± SD*	4.1 ± 0.5	3.9± 0.99	
	Range	(2.7 – 4.9)	(2.1 – 4.7)	0.21
LVEDV *(mL)*	*Mean ± SD*	134 ± 17.3	129 ± 14.4	
	Range	(104 – 166)	(102 – 165)	0.11
LVESV *(mL)*	*Mean ± SD*	40.01 ± 1.26	39.92 ± 1.44	
	Range	(24 – 64)	(21 – 60)	0.74
LA *(cm)*	*Mean ± SD*	3.76 ± 0.47	3.74 ± 0.39	
	Range	(2.8 – 4.1)	(2.8 – 4.1)	0.82

Table 2: Left atrium parameters and Left atrium strain among studied groups

LAVI (mL/m^2)	Mean ± SD	20.41 ± 4.55	18.78 ± 1.98	
	Range	(13 – 27)	(13 – 23)	0.02
LASV(mL)	Mean ± SD	26.14 ± 4.88	29.42 ± 3.47	
	Range	(17 – 35)	(22 – 36)	<0.001
LA expansion index(%)	Mean ± SD	231.4 ± 38.9	255.7 ± 18.1	
	Range	(143 – 287)	(190 – 287)	<0.001
Global PALS (%)	Mean ± SD	26.11 ± 8.43	39.44 ± 3.46	
	Range	(8 – 36)	(35 – 46)	<0.001
LASr(%))	Mean ± SD	33.1 ± 5.92	35.7 ± 6.8	
	Range	(18.4 – 44.8)	(18.8 – 44.8)	0.03
LAScd(%)	Mean ± SD	13.5 ± 5.93	17.3 ± 7.36	
	Range	(6.8 – 32)	(6.8 – 32.1)	0.005
LASct(%)	Mean ± SD	13.9 ± 5.61	16.1 ± 4.33	
	Range	(4.3 – 22.2)	(4.3 – 22.2)	0.03

Table 3: Relation of global PALS with different risk factors among studied patients

Variables		**Global PALS (%) Mean ± SD**	**P Value**
Sex	**Male**	**22.1 ± 5.68**	**0.57[1]**
	Female	**22.3 ± 7.73**	
Smoking status	**Non-smokers**	**21.47 ± 4.49**	**0.22[1]**
	Smokers	**20.37 ± 3.96**	
DM	**No**	**29.2 ± 5.52**	**0.04[1]**
	Yes	**25.8 ± 7.58**	
Family history	**No**	**28.9 ± 5.15**	**0.18[1]**
	Yes	**26 ± 7.86**	
LV diastolic dysfunction	**None**	**28.9 ± 5.81**	**0.008[2]**
	Grade I	**26.2 ± 6.47**	
	Grade II	**18.6 ± 11.33**	

Table 4: Logistic regression analysis for predictors of LA impairment

Variables	Univariate analysis		Multivariate analysis	
	P value	Odds (CI 95%)	P value	Odds (CI 95%)
Age (years)	0.008	0.78 (0.54 – 0.99)	0.74	0.78 (0.18 – 3.33)
Sex	0.12	3.57 (0.73 – 17.47)	-	-
Smoking	0.12	3.57 (0.73 – 17.47)	-	-
DM	<0.001	0.34 (0.18 – 0.95)	0.18	0.78 (0.54 – 1.13)
BMI(kg/m^2)	0.02	0.98 (0.97 – 1.19)	0.21	0.74 (0.81 – 1.62)
SBP(mmhg)	<0.001	0.97 (0.96 – 0.99)	0.01	1.21 (1.04 – 1.4)
EF (%)	0.39	1.03 (0.96 – 1.099)	-	-
LVMI (gm/m^2)	<0.001	0.02 (0.002 – 0.14)	0.12	1.09 (0.98 – 1.23)
LAVI (mL/m^2)	0.006	0.55 (0.36 – 0.84)	0.22	1.56 (0.77 – 3.79)
LASV(mL)	<0.001	0.97 (0.96 – 0.99)	0.52	1.002 (0.99 – 1.007)
LA expansion index(%)	<0.001	9.55 (3.2 – 28.5)	0.54	1.006 (0.99 – 1.027)
Global PALS(%)	<0.001	0.16 (0.06 – 0.47)	<0.001	0.98 (0.96 – 0.99)

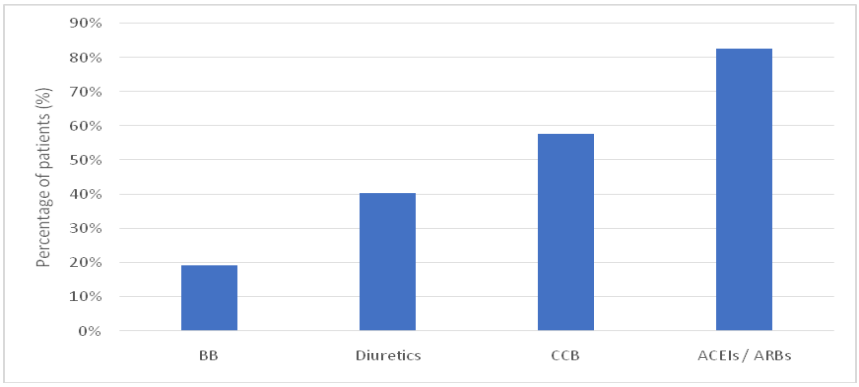
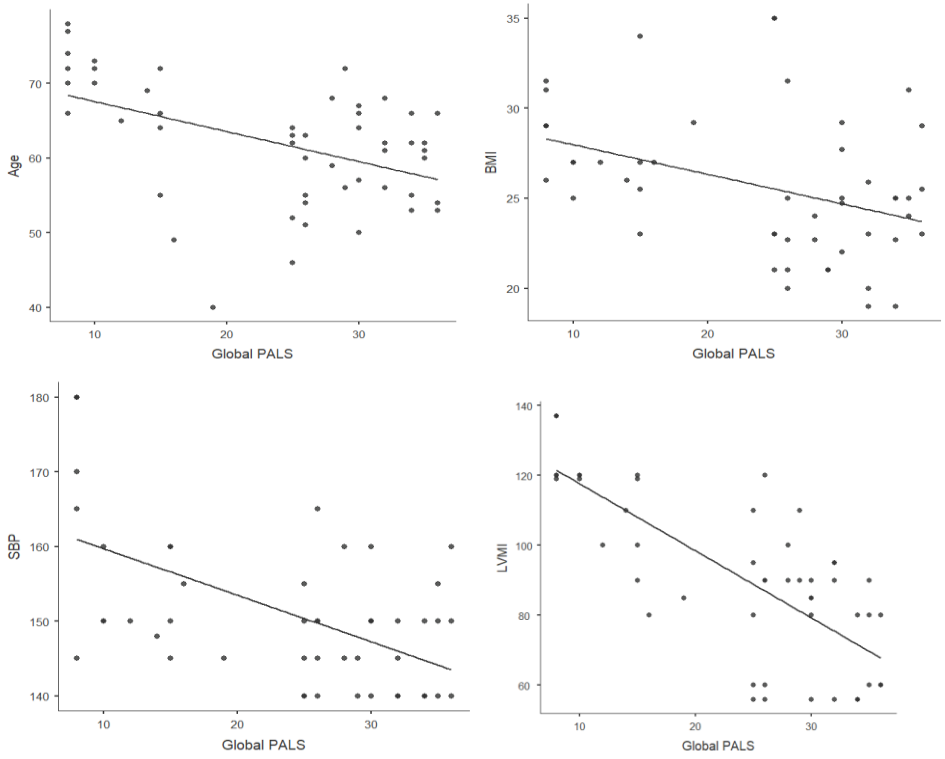


Figure (1): Drug history among hypertensive patients



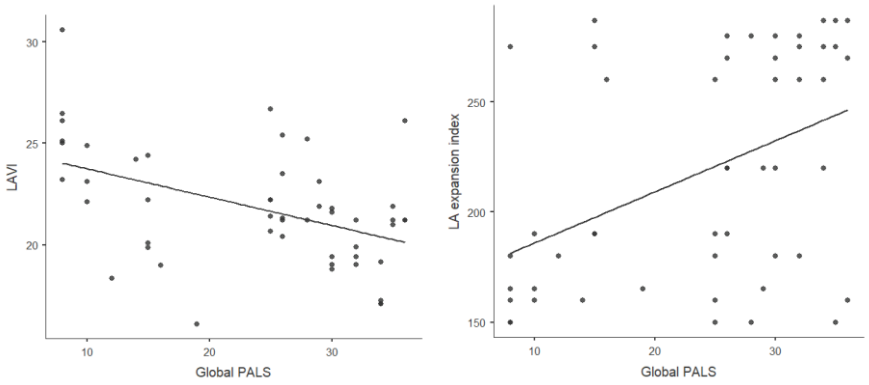


Figure (2): Scatter plots showing correlation between global PALS and different parameters among hypertensive patients

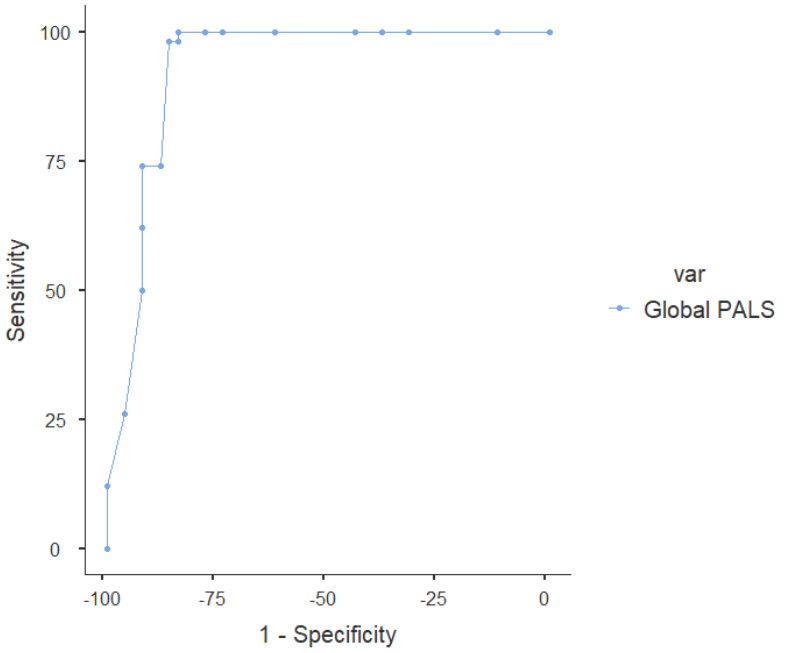


Figure (3): ROC curve analysis of global PALS in detecting left atrium impairment among hypertensive patients

DISCUSSION

Worldwide, hypertension is a serious health issue that is becoming more common. Regretfully, its mechanism and clinical trajectory remain unclear [13]. Williams et al. [14] shown that hypertension has a detrimental effect on target organ damage, it explains the poor prognosis and elevated incidence of cardiovascular morbidity and death in people with hypertension, including myocardial infarction, stroke, atrial fibrillation, and sudden cardiac death.

In individuals with hypertension, a thorough evaluation of left atrial phasic function may be clinically significant and beneficial for risk stratification. Indeed, based on LA enlargement and LA functional abnormalities, these patients may be at risk for atrial fibrillation and cerebrovascular strokes [15].

Because they were not thoroughly examined, relatively little is known about how hypertension affects left ventricular functions and its prognostic effects, despite the fact that morphological and functional alterations in the left ventricle during hypertension are well documented [8].

Even in patients with well-controlled hypertension, structural alterations of the LA and LV are preceded by both diastolic and atrial dysfunction in the left ventricle. Several standard methods, including computed tomography, echocardiogram, magnetic resonance imaging, and, can be used to assess atrial structural and functional alterations brought on by hypertension. The most widely used noninvasive imaging methods for assessing LA size and function are two-dimensional and three-dimensional echocardiography [13].

One imaging method that can be used to analyze left atrial function is speckle tracking echocardiography (STE). Since direct and angle-independent analysis of myocardial deformation allows for sensitive and repeatable indices of myocardial fiber dysfunction that circumvent the majority of the limitations of Doppler-derived strain measures, a more accurate assessment of the myocardium in patients with hypertension is essential for the successful diagnosis and treatment of cardiac dysfunctions in patients with hypertension [13].

To better understand and manage hypertensive patients, clinicians may find it useful to use bidimensional (2D) strain to assess LA function in patients who do not exhibit LA enlargement. This is because it might offer additional details for the extremely early identification of anomalies in the LA, which could aid in the prediction of cardiac events and atrial fibrillation (AF). The predictive effect of LA deformation in hypertension patients using STE has not been well studied [16].

We used speckle tracking echocardiography in 52 patients with well-controlled hypertension and normal left atrial size to assess left atrial function even before left atrial hypertrophy. This study, in my opinion, is the first to address this topic at Zagazig University Hospitals. All of the patients were selected based on their systemic arterial hypertension and echocardiograms showing normal sinus rhythm and LA size. Furthermore, 52 healthy people had no prior history of cardiovascular disease or hypertension.

The HTN group's average age was 61.7 ± 8.16. According to our findings, there was no discernible difference between the two groups' demographics (sex and age). In agreement, research conducted by Mondillo et al. [8], Kokubu et al. [17], Sahebjam et al. [18], and Miyoshi et al. [19], both groups were age matched.

Kamal et al. [20] Speckle tracking echocardiography was used to In hypertensive patients with normal left atrial size, detect left atrial dysfunction early. The average age of the HTN group was 56.04 ± 10.72. In both groups, age and gender were comparable (non-significant difference).

Also, Hennawy et al. [21] Speckle tracking echocardiography was used to identify left atrial dysfunction early in hypertensive patients with normal left atrial size. Age and gender were similar in both groups (non-significant difference).

Also, Taamallah et al. [13] identified early alterations in left atrial longitudinal strain using speckle tracking echocardiography in patients with hypertension. Fifty hypertension patients and fifty-two age-matched normotensive controls participated in the study. The HTN group's average age was 58.86 ± 11.61. However, Sahebjam et al. [18] did not match their candidates regarding gender.

Regarding diabetes mellitus, which is more prevalent in the hypertension group, and smoking, our study found no discernible differences between the two groups. Furthermore, Kamal et al. [20] and Hennawy et al. [21] discovered that whilst smoking was more prevalent in the control group, diabetes mellitus was more prevalent in the hypertension group. Miljkovic et al. [22] LAS's sensitivity and specificity were evaluated in order to ascertain cut-off values and their correlation with DD and elevated LAP in individuals with well-treated arterial hypertension. The incidence of smoking history was substantially greater in the DD group with elevated LAP ($p < 0.0001$).

In terms of body surface area and body mass index, our analysis revealed no discernible differences between the two groups. Furthermore, Kamal et al. [20] and Hennawy et al. [21] discovered that there was no significant difference between the two groups' BMI and BSA. However, in research conducted by Mondillo et al. [8] and Miyoshi et al.

[19], both groups were not matching in BMI. Sahebjam et al. [18] Their candidates were matched in terms of BSA but not BMI. It was not possible to examine BMI as a negative influence on LA function in these trials since it was not matched between the two groups.

Systolic and diastolic blood pressure in our study differed statistically significantly between the two groups; the HTN group had higher medians for both SBP and DBP. Furthermore, Taamallah et al. [13] discovered that the hypertension group's systolic and diastolic blood pressures were noticeably higher than those of the control group.

Both groups in our study had HRs that were comparable (non-significant difference). Furthermore, Sahebjam et al. [18], Kamal et al. [20] and Hennawy et al. [21] found that the HR of the two groups did not differ significantly.

There were no appreciable variations in EF between the two groups, according to our examination of the echocardiogram data. According to Kamal et al. [20], the LVEF of every patient under study was normal.

In terms of LVMI and LV diastolic dysfunction, our investigation revealed a statistically significant difference between the two groups; the hypertensive group had a greater mean LVMI. Regarding LV diastolic dysfunction, none of the patients in the normotensive group (0%) showed grade II diastolic dysfunction, but 23.1% of patients in the hypertension group did. This was consistent with a previous study conducted by Hassanin in Egypt at Cairo University [23]. This was also consistent with the study conducted by Miyoshi et al. [19], They discovered that the hypertension group had a bigger LV mass and a higher grade of LV diastolic dysfunction.

Also, Kamal et al. [20] discovered that the hypertensive group had a significantly higher LV mass index, which led to a significantly higher prevalence of LV diastolic dysfunction. Hennawy et al. [21] discovered that while all of the subjects under study had normal LVEF, the hypertensive group had a significantly higher LV mass index, which in turn led to worse LV diastolic dysfunction. However, Another study discovered that while there was no discernible change in LVEF between the two groups, the hypertension group had greater LV mass and lower diastolic performance. [24].

Taamallah et al. [13] discovered that the ascending aortic diameter and the LVEF, LVED, and the two groups' LVES diameters were identical. Furthermore, hypertension individuals exhibited greater LV mass and thicknesses of the septal and posterior walls; 69.7% (n=76) of patients had normal LV geometry. The two groups differed significantly in all diastolic function measures. The hypertensive group had a markedly elevated systolic pulmonary artery pressure (SPAP).

Global PALS (peak atrial longitudinal strain), LASV (the LA's contractile function), LA expansion index (the LA's reservoir function), and LAVI were among the left atrium parameters that showed a statistically significant difference between the two groups. The group with hypertension had a higher mean LAVI. whereas the mean of LASV, LA expansion index, and global PALS was lower in the hypertensive patients. Making sure that every person in our study had a normal LA size was crucial, and the LA volume index is the best way to describe this. Prior to LA expansion, this was crucial for the early identification of LA malfunction. Similar to a study by Eshoo et al., this crucial aspect was also apparent in a study by Mondillo et al. [8], in which every individual had a normal LA size. According to Mondillo et al. [25], the hypertensive group's LA volume index was higher than that of the control group.

The hypertension group in our current study demonstrated impaired global PALS compared to the control group. In patients with arterial hypertension, the reservoir function is mainly affected early on is represented by global PALS. The findings of Mondillo et al. [8], who discovered that hypertension patients showed early LA strain anomalies in comparison to normal subjects, were consistent with this.

Additionally, a study by Cameli [26] showed that preclinical LA dysfunction was indicated by the affection of LA deformation parameters expressed in longitudinal strain. was one of the early impacts of hypertension. Additionally, a different study by Hassanin (2015) found that the hypertension group had a significantly significant P-value (i.e., < 0.001) lower LA longitudinal strain, which represents its reservoir function.

Kokubu et al. [17] tested the impact of RAS inhibition on LA function and found that RAS inhibitors might maintain or enhance LA reservoir function (as measured by LA speckle tracking) and that hypertension patients had lower LA deformation indices. According to Sahebjam et al. [18], hypertension had a significant impact on LA reservoir function as determined by LA speckle

tracking, and this effect was unaffected by heart rate, age, sex, LV mass index, or LVEF.

Also, Kamal et al. [20] and Hennawy et al. [21] claimed that even though the LA volume index was within the normal range for all of the subjects under study, the hypertensive group's total LA stroke volume was significantly lower, indicating that hypertensive patients' contractile function is impacted even before their LA size enlarges. Additionally, the hypertensive group's LA expansion index was significantly lower, indicating that their reservoir function is impacted before their LA enlargement occurs. Global PALS, which characterizes the reservoir function of LA and is a measure of LA global dysfunction, was significantly lower in the hypertension group. This indicates that alterations in global PALS and other markers of LA dysfunction occurred prior to anomalies in LA volume. The hypertension group had significantly reduced LA size (LA volume index) and the several indices of LA dysfunction (Total LA stroke volume, LA expansion index, and global PALS), even though all research participants had acceptable LV volume indices.

But, this was different than a study by Cameli [26] However, there was no appreciable variation between the two groups' LA volume indices. Despite having a larger LA volume, the hypertensive group in a study by Tsai et al. [27] did not significantly vary from the other group in terms of the associated parameters of the LA function (such as the LA expansion index). This discrepancy could be explained by the fact that there were twice as many hypertension patients than there were in the control group.

Taamallah et al. [13] Although all participants in the two groups had normal LA size and volume, the hypertensive group had significantly greater LA area, LAV max, LAV min, and LAVp than the control group. The LA anteroposterior diameters of the two groups were similar. Hypertensive patients had lower levels of LASV, LAEF, LAEI, LApEF, and LASi than controls. There was no difference between the two groups in terms of LAAEF.

The hypertensive group had significantly reduced peak strain (PS) measured in apical 4c and 2c views during reservoir phase (PS-S) and conduit phase (PS-E), with values of 31.23±9.93 against 46.43±11.06 and 14.26±2.91% compared 21.41±2.8%, respectively. Peak strain, as determined by apical four-chamber and two-chamber views during the contractile phase (PS-A), was greater in individuals with hypertension but did not differ significantly between the two groups [13]. The values were 16.73 ± 3.84% and 15.29 ± 2.75%, respectively.

Regarding DM and LV diastolic dysfunction, our study found a statistically significant difference between the two groups; patients with diabetes had lower global PALS, and patients with grade II diastolic dysfunction had lower global PALS. Additionally, there was a statistically significant positive link with the LA expansion index and a statistically significant negative correlation with age, BMI, SBP, LVMI, and LAVI. According to Mondillo et al. [8], DM had a negative impact on LA strain. These alterations were not influenced by LA dilatation and were more noticeable in patients who both had hypertension and DM. Using speckle tracking echocardiography, Tadic and Hassanin [28] reported the impact of type II DM on LA remodeling as indicated in LA strain. On the other hand, a study by Sahebjam et al. [18] discovered that in hypertensive patients, DM had no effect on LA strain. The fact that around 50% of the hypertension patients in this study were using RAAS inhibitors may help to explain why their LA strain values were comparatively greater.

A study conducted by Okamatsu et al. [29] determined that LA size and function (as measured by LA strain) are negatively impacted by aging. However, in hypertensive individuals, Sahebjam et al. [18] could not find any association between age and LA strain. This might be because the study's mean age of 54 was significantly lower than that of the elderly age category, which includes people over 65.

Tadic and Cuspidi [30] claimed that compared to hypertension patients with a normal BMI, those who were overweight or obese had inferior LA function as determined by 2D strain. Miyoshi et al. [19] discovered that BMI had an independent effect on LA strain using 2D speckle tracking echocardiography.

In a study by Miyoshi et al. [19], hypertensive individuals' systolic blood pressure had a negative impact on their LA strain. Additionally, Tadic and Cuspidi [30] discovered a correlation between decreased LA strain and blood pressure fluctuation, or inadequate blood pressure management, in hypertension.

A research by Miyoshi et al. [19] discovered a strong link between LA systolic strain and LV

diastolic dysfunction (E/e'). According to a research by Tsai et al. [27], LV diastolic dysfunction has a detrimental effect on LA conduit function.

According to a study by Xu et al. [31], there was a substantial correlation between the LV mass index and all of the LA deformation indices; that is, a greater LV mass index corresponds to a lower LA function as measured by speckle tracking. Additionally, LV mass index had a negative impact on LA strain in the study conducted by Miyoshi et al. [19]. In contrast, Kokubu et al. [17] investigated two hypertension groups with and without a high LV mass index and observed no relationship between the two variables and LA systolic strain.

According to a study by Cameli, a higher LA volume index was an independent predictor of a lower LA systolic strain [26]. By using 2D speckle tracking, Miyoshi et al. [19] discovered a comparable relationship between LA size and LA function as expressed in LA strain. Since systemic hypertension impacts LA hemodynamics through two arms—changes in LA size and function—this correlation makes some sense.

In a study by Saraiva et al. [32], they discovered a strong correlation between the global LA strain, which represents the LA conduit function, and the LA expansion index. This may be explained by the fact that the global LA strain and the LA expansion index both reflect the function of the LA conduit, which is impacted early in hypertension individuals.

LVEF and LA strain did not correlate, according to Kokubu et al. [17]. Furthermore, a research by Mondillo et al. found that LVEF had no discernible effect on LA deformation indices via speckle tracking. [8]. On the other hand, Dogan et al. [33] discovered that LVEF significantly affected LA strain.

According to Kamal et al. [20], patients with LV diastolic dysfunction and diabetics both showed decreased global PALS. Global PALS was lower in older participants, those with greater systolic blood pressure, and those with higher body mass index (all of which are risk factors for LA dysfunction). Global PALS was decreased in subjects with increased LV mass index, a frequent consequence of hypertension. Despite the fact that all LA volume index values fell within the normal range, a larger LA volume index was linked to a lower global PALS. While total LA stroke volume had no discernible effect on global strain, participants with lower LA expansion index had lower global PALS according to conventional measures of LA function (since global PALS primarily represents the reservoir function of LA more than its contractile function).

Hennawy et al. [21] Patients with diabetes and those with higher grades of LV diastolic dysfunction were shown to have lower global PALS. Older participants, those with greater systolic blood pressure, those with higher body mass index (BMI), and those with higher LV mass index had lower global PALS. (a common consequence of hypertension), and, lastly, those with a larger LA volume index "even though all values of LA volume index were within the normal range." While total LA stroke volume had no discernible effect on global strain, participants with lower LA expansion index had lower global PALS according to conventional measures of LA function (since global PALS primarily represents the reservoir function of LA more than its contractile function).

Taamallah et al. [13] discovered no relationship between LA diameter and the contractile, conduit, and the peak strain and strain rate during reservoir stages. During the contractile stage, the only variables that showed a correlation with LA area were pic strain and strain rate. With the exception of LASV and LAAEF, all of the volumetric study's echocardiographic measures showed a strong correlation with PS-S. PS-E was found to be significantly correlated with IVS, Em/Am, E/Ea, LAV max, LAV min, LApV, LASV, LAEI, LAEF, LApEF, and SPAP. While there was no statistically significant link between PS-A and Em/Am, Em/Ea, LApV, LASV, LAEI, or LApEF, there was a significant correlation between PS-A and LAVmin, LAV max, LAEF, and LAEI.

Significant correlations were found between PSR-S and LAVmax, LASV, LAEF, LApEF, Em/Am, Em/Ea, and SPAP. There was a correlation between PSR-E and LAVp, LApEF, Em/Am, Em/Ea, and SPAP. There was no significant link between PSR-A and Em/Am, Em/Ea, LAV max, LApV, stroke volume, LAEI, LAEF, and LApEF, with the exception of a correlation between PSR-A and LAAEF and LA minimum volume [13].

We considered determining a global PALS cutoff value below which, even in hypertension individuals with normal LA size, we could identify impairment of LA function as measured by global PALS. In order to identify the ideal cutoff value for detecting left atrial impairment, ROC analysis (Receiver Operation Curve) was performed. The results indicated that global PALS had the highest

sensitivity (98%) and specificity (86%) at < 37, with an area under the curve of 0.929. Additionally, the receiver operating characteristic (ROC) curve was used by Kamal et al. [20] and Hennawy et al. [21] to show that the cutoff value for global PALS was less than 35% with a sensitivity of 98% and a specificity of 98%. With a 98% sensitivity and a 98% specificity below this number, this suggests that the participant of the study had LA dysfunction before LA enlargement.

Miljkovic et al. [22] evaluated using ROC analysis the sensitivity and specificity of various echocardiographic markers in DD diagnosis with high LAP. LAS, E/e', LVGS, and DTE were statistically significant predictors of DD with increased LAP based on the area under the curve.

SBP and Global PALS can be used as independent factors for predicting left atrial impairment (LAI) after logistic regression analysis for predictors of LAI has been used. According to Miljkovic et al. [22], DTE ($p = 0.021$) and LAS ($p < 0.0001$) were statistically significant predictors of DD with increased LAP.

According to Zhao et al. [34], aberrant LA deformation occurs before LVH and LA hypertrophy. Peak strain and strain rate were shown to be considerably lower during reservoir and conduit phases in hypertensive patients with normal LA size.

Numerous studies have demonstrated that atrial function is impaired during the contraction phase in addition to the decreased strain during reservoir and conduit.

It was shown that the LA contractile function was impaired even prior to the development of LA enlargement [35]. A possible noninvasive measure of left ventricular end-diastolic compliance was proposed: LA function during atrial contraction. Compared to those with normal LV geometry, patients with concentric LVH showed a marked increase in LA contraction function [36].

PS-S and LV diastolic function are highly correlated, and elevated LV filling pressures are also a good indicator of LV diastolic dysfunction, as is the decreased LA strain. Diastolic dysfunction can be accurately diagnosed and categorized with the use of LA strain measures [13].

In patients with white coat hypertension, a disruption in LA function was also seen. Tadic et al. [37] showed that whereas reservoir function was normal, patients with white coat hypertension had an enhanced pomp function and a decreased conduit function. When they compared the LA function of patients with white coat hypertension, chronic hypertension, and controls, the alterations in LA function were more noticeable in the former group. From controls to persistent hypertensives, LA and aortic stiffness progressively rise.

In situations of pregnant hypertension, a reduction in LA strain was also seen. Global left atrial peak strain has been shown to have an additive predictive value in women with new-onset pregnancy hypertension, according to Sonaglioni et al. [16], who also documented reduced LA function in these patients.

It is commonly recognized that hypertension is a risk factor for the development of AF from a prognosis perspective. Apart from LA enlargement, atrial fibrillation may also be predicted by LA functional abnormalities. In hypertensive patients, LA strain metrics may be helpful indicators of the likelihood of AF. Therefore, greater monitoring is required to identify arrhythmias in patients with compromised atrial function.

Hennawy et al. [21] determined that even before LA enlargement happens, speckle tracking echocardiography is a valuable new method for identifying LA malfunction in hypertension. Ikejder et al. [38] Two-dimensional volumetric metrics were used to assess LA size and phasic function in individuals with critical hypertension. Nine percent of patients had dilated LA in univariate analysis. In 82% and 85% of patients, respectively, the pump and LA reservoir functions were improved. Eighty percent of patients had poor LA conduit function. Diabetes, obesity, and hypertension medications were the most potent predictors for this effect in bivariate analysis; individuals receiving calcium antagonist treatment had markedly reduced LA pump function. They showed that AHT caused dilatation of the LA, improved function of the pump and reservoir, and decreased function of the conduit; these effects seem to be associated with LVH and the extent of LV diastolic dysfunction. Obesity, diabetes, and hypertension medications are the main causes of this effect.

Taamallah et al. [13] demonstrated that left atrial longitudinal strain during the reservoir and conduit periods is impaired in patients with hypertension despite normal cavity size and before the detection of other echocardiographic changes. Speckle-tracking echocardiography may be considered a promising tool for the early detection of LA strain abnormalities in these patients.

Limitations:

-Duration of HTN was difficult to be assessed accurately in the study population.

-A relatively small study population number was included and a further study with large number is warranted

-There was no way for detecting level of patient adherence to treatment strategy including medications and life style influence.

Recommendations:

a) It would be interesting to conduct further studies with larger populations and follow-up data to precise the discriminatory role of LA 2D strain in the AF and heart failure with preserved ejection fraction risk stratification. Indeed, identifying hypertensive patients at risk for developing these complications by early diagnosis of LA dysfunction, and prompt institution of effective treatment should be the goal when considering this patient population.

b) The clinical usefulness of LA function by STE in these patients merits further investigations to better precision of the role of the LA study in the prediction of atrial fibrillation, and the risk of heart failure with preserved ejection fraction.

c) Future studies dealing with a large number of patients are needed to verify the above results and to throw more light into this important issue.

Conclusions:

Even before LA enlargement happens, speckle tracking echocardiography is a helpful new method for identifying early LA dysfunction in hypertension. It may be utilized to identify mild impairment of LA function in hypertensive patients. Impaired reservoir, conduit, and contractile LA function are linked to hypertension.

Future research with a large number of patients is required to confirm the aforementioned findings and provide more light on this crucial matter.

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Conflict of interest:

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Table 1 Supplementary: Baseline data among studied groups

Variables		HTN group (n=52)	No-HTN group (n=52)	P Value
Age (years)	Mean ± SD	61.7 ± 8.16	60.1 ± 9.49	0.36[1]
	Range	(40 – 78)	(38 – 85)	
Sex (n. %)	Male	30 (57.7%)	33 (63.5%)	0.54[2]
	Female	22 (42.3%)	19 (36.5%)	
Smoking status (n. %)	Non-smokers	22 (42.3%)	25 (48.1%)	0.42[2]
	Smokers	30 (57.7%)	27 (51.9%)	
DM (n. %)	No	20 (38.5%)	21 (40.4%)	0.84[2]
	Yes	32 (61.5%)	31 (59.6%)	
Family history (n. %)	No	28 (53.8%)	34 (65.4%)	0.22[1]
	Yes	24 (46.2%)	18 (34.6%)	
BMI(kg/m^2)	Mean ± SD	25.7 ± 3.98	25.9 ± 4.01	0.87[1]
	Range	(19 – 35)	(19 – 35)	
BSA (m^2)	Mean ± SD	1.87 ± 0.02	1.96 ± 0.01	0.23[1]
	Range	(1.26 – 2.01)	(1.26 – 2.01)	
SBP (mmhg)	Median (IQR)	150 (10)	120 (17.5)	<0.001
	Range	(140 – 180)	(100 – 135)	
DBP (mmhg)	Median (IQR)	90 (10)	80 (10.8)	<0.001
	Range	(90 – 120)	(60 – 90)	
HR (beat/m)	Median (IQR)	75 (16.8)	72.5 (13.8)	0.71
	Range	(58 – 99)	(60 – 93)	

Citation

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