Factors affecting left ventricular synchronicity in hypertensive patients: are arterial stiffness and central blood pressures influential?

Hipertansiyonlu hastalarda sol ventrikülün eş zamanlı kasılmasını etkileyen faktörler: Arteriyel katılık ve merkezi kan basıncı etkili mi?

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ABSTRACT

Objectives: Left ventricular (LV) dyssynchrony is a common finding in patients with hypertension and is associated with LV hypertrophy. Arterial stiffness (AS) and central (aortic) blood pressures play a significant role in end-organ damage such as LV hypertrophy caused by hypertension. The objective of this study was to investigate the relationship between AS, central blood pressures (BP) and LV dyssynchrony.

Study design: Thirty-five newly diagnosed hypertensive patients and 40 controls were enrolled in the study. The entire study population underwent a comprehensive echocardiographic study including tissue synchrony imaging. The 12 segmental model was used to measure the time to regional peak systolic tissue velocity (Ts) in the LV and two dyssynchrony indices were computed. Parameters of AS including pulse wave velocity (PWV), augmentation index (AIx@75), and central systolic and diastolic BP were evaluated by applanation tonometry.

Results: The baseline clinical and echocardiographic parameters of both groups were similar except for their BPs. Dyssynchrony indices were prolonged in patients with hypertension as compared to the controls. The standart deviation of Ts of 12 LV segments in patients with hypertension and the controls were 48.7±18.8 *vs.* 25.8±13.1, respectively (p<0.001), and the maximal difference in Ts between any 2 of 12 LV segments was 143.9±52.2 for hypertension patients *vs.* 83.8±39.4 for controls (p<0.001). PWV (11.9±2.5 *vs.* 9.5±1.4, p<0.001), AIx@75 (27.4±8.3 *vs.* 18.3±9, p=0.009), and central systolic (147.6±20.8 *vs.* 105.4±11, p<0.001) and diastolic (99.8±14.4 *vs.* 72.8±9.5, p<0.001) pressures were higher in patients with hypertension than in the controls, respectively. In multivariable analysis, central systolic BP (β=0.496, p=0.03), LV mass index (β=0.232, p=0.027), and body mass index (β=0.308, p=0.002) were found to be independently related to dyssynchrony.

Conclusion: Central systolic BP is an independent predictor of LV dyssynchrony, but AIx@75 did not have an independent effect on LV synchronicity in patients with newly-diagnosed hypertension.

ÖZET

Amaç: Sol ventrikül (SV) eş zamanlı kasılma bozukluğu hipertansiyonlu hastalarda sık rastlanılan bir bulgu olup SV hipertrofisi ile ilişkilidir. Arteriyel katılık (AK) ve merkezi (aortik) kan basıncı, SV hipertrofisi gibi hipertansiyon kaynaklı hedef organ hasarında önemli rol oynar. Bu çalışmada, AK, merkezi sistolik ve diyastolik kan basınçları (KB) ve SV senkronizasyon bozukluğu arasındaki ilişki incelendi.

Çalışma planı: Yeni hipertansiyon tanısı konmuş 35 hasta ve 40 kontrol çalışmaya alındı. Tüm çalışma popülasyonuna 'doku senkronizasyon görüntülemesini' (DSG) de içeren kapsamlı ekokardiyografik inceleme yapıldı. SV'nin pik sistolik doku hız zamanı (Zs) 12 segment modeli ile ölçüldü ve iki eş zamanlı kasılma bozukluğu indeksi hesaplandı. Nabız dalga hızı (NDH) ve artış indeksini (AIx@75) içeren AK parametreleri ile merkezil sistolik ve diyastolik KB aplanasyon tonometresi ile değerlendirildi.

Bulgular: Kan basınçları dışında her iki grubun temel klinik ve ekokardiyografik parametreleri benzerdi. Eş zamanlı kasılma bozukluğu indeksleri hipertansiyonlu hastalarda kontrol grubuna göre uzamıştı: 12 segmentin Zs'nın standart sapması (48.7±18.8 ve 25.8±13.1, p<0.001); herhangi iki segmentin maksimum Zs farkı (143.9±52.2 ve 83.8±39.4, p<0.001), NDH (11.9±2.5 ve 9.5±1.4, p<0.001), AIx@75 (27.4±8.3 ve 18.3±9, p=0.009), merkezi sistolik (147.6±20.8 ve 105.4±11, p<0.001) ve diyastolik (99.8±14.4 ve 72.8±9.5, p<0.001) basınçlar hipertansiyonlu hastalarda kontrol grubundan daha yüksekti. Çok değişkenli analizde, merkezi sistolik KB (β=0.496, p=0.03), SV kitle indeksi (β=0.232, p=0.027) ve beden kütle indeksi (β=0.308, p=0.002) eş zamanlı kasılma bozukluğu ile bağımsız ilişkili bulundu.

Sonuç: Merkezi sistolik KB, yeni tanı konmuş hipertansiyonlu hastalarda SV'nin eş zamanlı kasılma bozukluğunun bağımsız öngördürücüsüdür. Ancak AIx@75, SV eş zamanlı kasılması üzerinde doğrudan etkiye sahip değildir.

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Hypertension is a major health problem that can lead to cerebrovascular events, coronary artery disease, and heart failure. Heart failure caused by hypertension is generally associated with diastolic dysfunction and preserved ejection fraction.[1] Left ventricular (LV) systolic dyssynchrony suggests the loss of the simultaneous peak contraction of corresponding cardiac segments. In hypertensive patients, the presence of systolic and diastolic LV dyssynchrony and their adverse effects on cardiac performance have been shown.[2-5]

Arterial stiffness (AS) represents the decreased elasticity of the aorta and its branches, and it plays an important role in target organ damage. AS leads to an increased central (aortic) systolic blood pressure (SBP) and decreased diastolic blood pressure (DBP), which result in a broader pulse pressure.[6] Because of increased afterload and decreased coronary perfusion, the effects of AS on the heart are left ventricular hypertrophy (LVH) and myocardial ischemia.[6] LVH and myocardial ischemia are also important etiologic factors for dyssynchrony.[2,5,7] Hypertension is one of the most common causes of AS. Therefore, AS may play a role in the development of LV dyssynchrony in patients with hypertension.

In this study, the relationship between LV dyssynchrony, AS, and central aortic pressures was assessed in this population.

PATIENTS AND METHODS

Patients

Thirty five newly diagnosed hypertensive patients who had narrow QRS complexes on electrocardiography (ECG) and normal left ventricular ejection fractions (LVEF) on echocardiography were enrolled in the study between October 2009 and December 2011. Patients having two separate office blood pressure measurements >140/90 mmHg were diagnosed with hypertension (stage 1: 140-159/90-99 mmHg and stage 2: ≥160/100 mmHg).[8] Forty healthy subjects were in the control group. Full demographic data, biochemical blood tests, and ECG were obtained from the entire study population. The clinical examination included height and body weight measurements. Body mass index (BMI) was calculated as weight (kilograms) divided by the square of height (meters squared). Patients having a BMI ≥30 kg/m^2 were defined as obese. SBP and DBP were measured three times in the sitting position after 15 minutes of rest, and the mean was taken for all cases. Participants were advised to avoid cigarette smoking, alcohol, caffeinated beverages, and exercise for at least 30 minutes before their blood pressure measurements. Diabetes mellitus, atrial flutter or fibrillation, cardiac pacing, prolonged QRS duration (≥120 msn), reduced LVEF (<%50), significant valvular heart disease, hypertrophic cardiomyopathy, chronic renal disease, heart failure, coronary artery disease including previous myocardial infarction, angina pectoris, percutaneous coronary intervention, and poor echocardiographic imaging were all exclusion criteria. In addition, patients using any anti-hypertensive drug that may affect the AS measurement were excluded from the study. The study protocol was approved by the local Ethics Committee and the informed consent was obtained from the entire study population.

Abbreviations:

2D	*2-dimensional*
AIx	*Augmentation index*
AS	*Arterial stiffness*
BMI	*Body mass index*
DBP	*Diastolic blood pressure*
LV	*Left ventricular*
LVEF	*Left ventricular ejection fraction*
LVH	*Left ventricular hypertrophy*
LVM	*Left ventricular mass*
LVMI	*Left ventricular mass index*
PWV	*Carotid-femoral pulse wave velocity*
Ts	*Time to peak myocardial velocity*

Echocardiography

Transthoracic 2-dimensional (2D), conventional Doppler, and tissue Doppler echocardiography were performed according to the recommendations of the American Society of Echocardiography[9] using a commercially available system (Vivid 7, GE Vingmed Ultrasound AS, Horten, Norway). Subjects were examined in the left lateral recumbent position using standard parasternal (short-and long-axis) and apical views (two chamber, four chamber, and long axis). LV dimensions were measured by 2D guided M mode echocardiography. LV function was assessed by LVEF using the modified biplane Simpson's rule. Left ventricular mass (LVM) was calculated using the Devereux formula.[10] LVH was evaluated by sex-specific left ventricular mass index (LVMI) criteria.[11] To obtain LV diastolic filling patterns, the Doppler sample volume was put in the middle of the LV inflow tract 1 cm below the plane of mitral annulus in the apical four chamber view. Transmitral filling velocities including peak early (E) and late (A) diastole were obtained.

The assessment of LV dyssynchrony

LV systolic dyssynchrony was assessed via the TSI method as previously defined by Yu et al.[12] TSI shows regional dyssynchrony on 2D echocardiography and allows for the immediate visual assessment of regional delay in systole. In addition, quantitative measurement of regional delay was derived from 2D-TDI images in which the time to peak myocardial velocity (Ts) was automatically color-coded from green to red with reference to the QRS signal.[12,13] Ts suggests the time to reach regional peak systolic tissue velocity. A quantitative measurement device allows for the calculation of the median Ts within a 6-mm sample volume manually positioned within the 2D TSI image for 12 LV segments. Initially, to prevent the TSI system from measuring peak systolic velocities outside the ejection phase, the event-timing tool was used to manually adjust the start and end times of the aortic valve ejection. Later, at least three consecutive beats on TSI were stored and the images were analyzed offline with a customized software package (EchoPac for PC, GE Vingmed Ultrasound). The six-basal-six-midsegmental model was used.[12,13] Two indices of systolic dyssynchrony were computed with the software by two independent cardiologists who were blinded to the patient's data. These indices were the standard deviation of Ts of the 12 LV segments (Ts-SD-12) and the maximal difference in Ts between any 2 of the 12 LV segments (Ts-12). Ts-SD-12 is the most widely used parameter to assess left ventricular dyssynchrony.[12,13]

The measurements of arterial stiffness and central pressures

Arterial stiffness parameters and central pressures were noninvasively measured by the SphygmoCor system (AtCor Medical, Sydney, Australia) and applanation tonometry after an overnight fast and 24 hours after refraining from any antihypertensive medications. Subjects were asked to omit caffeinated beverages, smoking, and alcohol for at least 12 hours before the assessment. All measurements were performed in a quiet, temperature controlled room. Carotid-femoral pulse wave velocity (PWV) and augmentation index (AIx) are the main indices used to assess AS. Carotid-femoral PWV is a non-invasive measurement of the distensibility of the aorta. The carotid-femoral PWV was measured by sequential recordings of the arterial pressure waveform at the carotid and femoral arteries with a hand-held micromanometer-tipped probe on the skin at the site of maximal arterial pulsation after 10-15 minutes of the participant resting in the supine position. Gating of the recordings at these two sites to the ECG allowed the PWV to be measured. The distances from the carotid sampling site to the suprasternal notch and from the suprasternal notch to the femoral artery were measured.[14] The carotid-femoral PWV (in meters per second) was calculated automatically as the distance between the carotid and the femoral sampling site divided by the time interval between the systolic R-wave and the femoral systolic upstroke minus the time interval between the systolic R-wave and the carotid systolic upstroke. The carotid-femoral PWV was determined as the mean of at least three consecutive beats recorded during 10 seconds of data acquisition. AIx is another AS parameter that is defined by the difference between early and late pressure peaks divided by central pulse pressure. To measure the AIx, an ascending aortic pressure waveform was derived from the radial artery waveforms recorded at the wrist using applanation tonometry with a high-fidelity micromanometer. The aortic augmentation pressure was measured as the difference between the first and second systolic peaks of the ascending aortic waveform, and AIx was calculated. Then, AIx was normalized for a heart rate of 75 beats/min (AIx@75) since it is influenced by heart rate.[15] All measurements were performed by the same investigator without knowledge of the clinical and echocardiographic data. For reliable results, only high-quality recordings were included in the analysis. High-quality recordings were defined as having the presence of acceptable waveform curves on visual inspection and an in-device quality index of 80% or more.

Statistical analysis

Continuous variables were described as mean ± SD and analyzed with an unpaired-t test and Mann-Whitney U-test when appropriate. A chi-squared test was used for categorical variables. Pearson and Spearman correlation coefficients were used to assess the relationship between dyssynchrony and other parameters including clinical, echocardiographic, and AS. Multiple linear regressions were used to detect the factors that were independently related to LV dyssynchrony. A p value <0.05 was considered statistically significant. An SPSS software program (SPSS, 13.0, Inc, Chicago, Illinois) was used for statistical analysis.

RESULTS

Patient characteristics

The study consisted of 35 patients with hypertension and 40 control subjects. The hypertension and control groups had similar demographic and biochemical characteristics such as age, gender, smoking, and fasting blood glucose (Table 1). Nine (25.7%) patients had stage 1 hypertension and 26 (74.3%) patients had stage 2. In addition, BMI was mildly higher in the hypertension group than in the control group, but the prevalence of obesity was similar in both groups (51.4% *vs.* 30%, p=0.097).

Echocardiographic parameters

The echocardiographic parameters of both groups are given in Table 2. There were no differences in baseline M-mod or 2D echocardiographic parameters such as LV diameters and LVEF. LVMI and LVH prevalence (42.9% *vs.* 7.5%, p<0.001) were higher in the hypertensive group than in the controls. In conventional Doppler echocardiography, E velocity was similar in both groups, but A velocity was higher in the hypertensive group than in the controls.

LV dyssynchrony

TS-SD-12 was used to assess the presence of dys-

Table 1. Baseline demographic characteristics of the study population

	Hypertension (n=35)	Control (n=40)	*p*
Age (years)	51.9±8.3	49.3±6.7	0.13
Gender (Male/Female)	23 / 12	31 / 9	0.30
Smoking [n (%)]	9 (25.7)	11 (27.5)	1.0
Fasting glucose (mg/dl)	96.1±13	95.5±13.6	0.83
Creatinin (mg/dl)	0.9±0.2	1.1±1.4	0.28
Heart rate (beat/sec)	73.9±11	69.5±9.1	0.06
QRS duration (msn)	96.4±7.1	94.8±7.9	0.36
Systolic BP (mmHg)	159.4±20.3	111.9±19.7	**<0.001**
Diastolic BP (mmHg)	98.3±14	71.4±9.4	**<0.001**
Body mass index	29.9±4	28.1±3.6	0.04

BP: Blood pressure.

Table 2. Conventional echocardiography and asynchrony parameters of the two groups

	Hypertension (n=35) Mean±SD	Control (n=40) Mean±SD	*p*
LVESD (mm)	29.1±4.5	30.6±3.4	0.12
LVEDD (mm)	47.1±4.0	48.2±3.6	0.20
Ejection fraction (%)	67.7±6.7	66.2±5.5	0.26
IVS (mm)	12±1.9	10.1±1.2	**<0.001**
PW (mm)	10.7±1.2	9.4±1.2	**<0.001**
LV mass index (gr/m^2)	103.1±20.9	85.6±13.5	**<0.001**
E (cm/s)	74.5±14.9	75.2±18	0.86
A (cm/s)	76.1±13.6	60±15.1	**<0.001**
Ts-SD-12	48.7±18.8	25.8±13.1	**<0.001**
Ts-12	143.9±52.2	83.8±39.4	**<0.001**

A: Late diastolic mitral inflow velocity; E: Early diastolic mitral inflow velocity; IVS: Interventricular septum; LV: Left ventricle; LVEDD: Left ventricular end-diastolic diameter; LVESD: Left ventricular end-systolic diameter; PW: Posterior wall; Ts:, Time to peak tissue velocity; Ts-SD-12: Standard deviation of Ts of the 12 LV segments; Ts-12: Maximal difference in Ts between any 2 of the 12 LV segments.

Table 3. Arterial stiffness analysis in the study groups

	Hypertension (n=35) Mean±SD	Control (n=40) Mean±SD	*p*
AIx@HR75 (%)	27.4±8.3	18.3±9	0.009
PWV (m/s)	11.9±2.5	9.5±1.4	**<0.001**
Central aortic pressures			
Systolic (mmHg)	147.6±20.8	105.4±11	**<0.001**
Diastolic (mmHg)	99.8±14.4	72.8±9.5	**<0.001**

AIx@75: Heart rate-corrected augmentation index; CF-PWV: Carotid-femoral pulse wave velocity.

synchrony. A cutoff value (>52 ms) derived from the control group using the formula 'mean+2SD' was compatible with previous studies.[5,13,16] For Ts-12-SD, intra- and inter- observer variability were assessed by the method described by Bland-Altman[17,18] and were found to be 4.8% and 5.6%, respectively. Both LV systolic dyssynchrony indices were significantly prolonged in patients with hypertension as compared with controls (Table 2). The prevalence of LV dyssynchrony was higher in the hypertension group than in the control group (40% *vs.* 5%, p<0.001). The hypertensive patients with LVH had higher LV dyssynchrony than those without LVH (66.7% *vs.* 20%, p=0.005). But, the patients with stage 1 and 2 had similar prevalence (44.4% *vs.* 38.5%, p=1.0).

LV dyssynchrony was also higher in subjects that were obese than in those that were not obese (33% *vs.* 13%, respectively, p=0.038). In addition, the prevalence of LVH and LV dyssynchrony was higher in obese hypertensive patients than in obese controls (44.4% *vs.* 8.3%, p=0.049 and 50% *vs.* 8.3%, p=0.24, respectively).

Arterial stiffness and central aortic pressures

The central systolic and diastolic pressures were higher in the hypertensive group than in controls. AS indices including PWV and AIx@75 (%) were also higher in the hypertensive group than in controls (Table 3).

Relationship between dyssynchrony and other parameters

Correlation analysis revealed that Ts-SD-12 was related to LVMI (r=0.338, p=0.003), BMI (r=0.34, p=0.003), AIx@75 (r=0.372, p=0.001), and central systolic (r=0.543, p<0.001) and diastolic pressure (r=0.452, p<0.001). However, in multivariable analysis, LVMI (beta=0.232, p=0.02), BMI (beta=0.308, p=0.002) and central SBP (beta=0.496, p=0.03) were independently related to Ts-SD-12 (Table 4). For this model, the statistical power of study was computed to be 92%.

DISCUSSION

In this study, the effects of AS and central pressures on left ventricular synchronicity were investigated in

Table 4. The results of multiple linear regression analyses showing independent predictors of Ts-SD-12

	Beta	95% CI		*p*
		Lower	Upper	
Body mass index	0.308	0.576	2.560	0.002
LVMI	0.232	0.028	0.445	0.027
AIx@HR75	0.087	-0.295	0.647	0.459
Aortic SBP	0.496	0.036	0.694	0.030
Aortic DBP	-0.175	-0.620	0.241	0.383

CI: Confidence interval; AIx@75: Heart rate-corrected augmentation index; DBP: Diastolic blood pressure; LVMI: Left ventricular mass index; SBP: Systolic blood pressure.

patients with newly-diagnosed hypertension. As expected, AS parameters and central blood pressures were significantly higher in the hypertensive group than in the control group. LV dyssynchrony indices were also significantly prolonged in patients with hypertension as compared to controls. A significant correlation was found between dyssynchrony and AIx, central SBP and DBP, and BMI and LVMI. The central SBP, LVMI, and BMI were found to be independent predictors of dyssynchrony.

The presence of LV dyssynchrony has been reported in different hypertensive populations.[2-5] For example, Tan et al.[2] reported impaired ventricular synchronicity in hypertensive patients with ventricular hypertrophy, while Yang et al.[5] reported LV dyssynchrony in hypertensive patients without congestive symptoms. In these studies, LVH was considered to be the factor causing dyssynchrony. However, in a recent publication, Kwon et al.[19] reported the presence of systolic dyssynchrony in never-treated hypertensive patients without LVH. Therefore, we considered that factors other than LVH might have an effect on synchronicity.

AS plays an important role in the development of end-organ damage caused by hypertension such as heart failure and cerebrovascular disease.[6] The important adverse effects of AS on the heart include myocardial damage, LV hypertrophy, impaired coronary flow reserve, and myocardial ischemia.[20-22] Gedikli et al.[20] reported a relationship between AS and myocardial damage in patients with newly diagnosed essential hypertension. Saito et al.[22] suggest that AS is linked to a reduction of coronary flow reserve. Increased pulse pressure and afterload due to increased central SBP and decreased central DBP are the basic mechanisms in target organ damage caused by AS. In this study, we found a moderate relationship between dyssynchrony and SBP. In multivariable analysis, only central aortic SBP was found to be an independent predictor for dyssynchrony. The central aortic SBP represents the systolic load on the heart (afterload), which is a main determinant of left ventricular structure and function. In the long-term, the increase in afterload may lead to the enhancement of myocardial wall thickness and oxygen demand followed by LVH. There is an accumulation of collagen and myocardial fibrosis as well as myocyte hypertrophy[23] when LVH is caused by overload. Myocardial fibrosis[24,25] may cause dyssynchrony because it may impair the myocardial electrical coupling[26] and lead to asynchronous contractiles. In addition, Zang et al.[27] reported that cardiac structure was significantly associated with central BP components. Wang et al.[28] also suggested that aortic SBP was more valuable than other BP variables in the prediction of cardiovascular mortality. There is a weak relationship between dyssynchrony, AIx@75, and aortic DBP in this study, and no independent relationship was found between these parameters. This situation may partially be explained by the stage of disease. We only included newly diagnosed (early stage) hypertensive patients in the study, and the adverse effects of hypertension on the arterial tree may not fully occur in this stage of disease. According to our findings, it may be proposed that central SBP is an important indicator of cardiac involvement in the early stages of hypertension.

BMI was another independently associated parameter of LV dyssynchrony in this study. The prevalence of LV dyssyncrony was higher in obese subjects than those that were not obese. The relationship between obesity and LV dyssynchrony was investigated in previous studies. Marfella et al.[29] reported that impaired LV synchronization was caused by inappropriate cytokine release in premenopausal obese women. Purushottam et al.[30] recently reported ventricular dyssynchrony in obese patients who have narrow QRS duration but do not have cardiac disease. Obesity has many adverse cardiovascular effects such as increased total blood volume, cardiac output, cardiac workload, and filling pressure and volume that lead to ventricular hypertrophy.[31] Therefore, obesity may be considered to be a contributing factor for LV dyssynchrony caused by hypertension. In addition, obese hypertensive patients had higher LVH and LV dyssynchrony than the obese controls in our study. Thus, it may be concluded that LVH as well as inappropriate cytokine release may play a role in the development of LV dyssynchrony associated with obesity.

Lastly, LV dyssynchony has been proposed as an early finding of cardiac involvement in disorders such as diabetes mellitus, hypothyroidism, hyperthyroidism, and hyperparathyroidism.[32-35] Our study consisted of hypertensive patients who were newly diagnosed and never treated. Also, the LVMI of our patients was lower than in other studies that report impaired LV synchronicity in hypertensive patients.[2,3,5]

Therefore, it may be proposed that LV dyssynchrony may indicate early myocardial involvement in hypertension.

Limitations

The most important limitation of our study is the relatively small sample population. However, the statistical power of this study was calculated to be 92%. In addition, we did not evaluate strain or strain rate parameters for LV dyssynchrony. However, analysis of strain and strain rate is not applicable for routine clinical use and the interpretation of strain images is rather difficult.[12] The TSI method, which was used in this study, is reliable, practical and less time-consuming. Lastly, the presence of ischemia was not assessed by a quantitative method such as coronary angiography or myocardial scintigraphy in this study. However, patients with any suspicion of ischemia were excluded from the study to reduce the impact of this limitation.

Conclusion

The central SBP is an independent predictor of LV dyssynchrony, but AIx does not have a direct effect on LV synchronicity in patients with newly-diagnosed hypertension. LV mass index and BMI are other important factors that affect the synchronicity in these patients. In addition, central SBP may be a more important indicator of cardiac involvement than AS in the early stages of hypertension.

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Key words: Blood flow velocity; blood pressure; body mass index; echocardiography, Doppler/methods; heart failure/epidemiology; hypertension; hypertrophy, left ventricular; pulse wave velocity; vascular resistance; ventricular dysfunction, left/etiology.

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