



Interatrial conduction time is early marker of disturbed impulse propagation in adults with slightly elevated blood pressure

Kašnjenje električnog impulsa između dve pretkomore je rani marker usporene propagacije impulsa kod odraslih osoba sa blago povišenim krvnim pritiskom

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Abstract

Background/Aim. Interatrial conduction time is early marker of disturbed impulse propagation in adult with elevated blood pressure. The aim of our study was to evaluate significance of noninvasive echocardiographic marker of slow sinus impulse propagation (atrial conduction time) for the identification of persons with slightly elevated blood pressure and hypertension in adults. **Methods.** One hundred and forty nine adults with normal and elevated blood pressure were studied: 46 normotensive adults (group 1), 28 adults with elevated blood pressure and hypertension stage 1 (group 2) and 75 adults with hypertension stage 2 (group 3), based on the Joint National Committee 8 (JNC-8) hypertension guidelines. We studied P wave dispersion, reservoir function of the left atrium (LA), total emptying volume of the LA and total emptying fraction of the LA (LATEF). The atrial conduction time (ACT) was evaluated by the pulsed tissue Doppler, and expressed as interatrial and intraatrial conduction time. **Results.** The LATEF

decreased progressively from the group 3 ($64.8 \pm 4.4\%$) to the group 2 ($59.8 \pm 5.2\%$) and the group 1 ($55.6 \pm 7.3\%$) ($p < 0.001$). The P wave dispersion (55.1 ± 9.8 ms vs. 46.8 ± 3.1 ms vs. 43.1 ± 2.6 ms; $p < 0.01$) and intra ACT were significantly prolonged only in the group 3 compared to the other groups (22.7 ± 11.0 ms vs. 8.4 ± 4.7 ms vs. 5.6 ± 2.4 ms, respectively; $p < 0.001$). Inter ACT significantly increased from the group 1 to the group 2 and the group 3 (15.6 ± 3.9 ms vs. 24.6 ± 5.7 ms vs. 50.4 ± 20 ms, respectively; $p < 0.05$). Using a cut-off level of 19.5 ms, inter ACT could separate adults in the group 2 from the group 1 with a sensitivity of 85%, and specificity of 89% [area under receiver operating characteristic (ROC) curve 0.911]. **Conclusion.** Prolonged ACT estimated with the tissue Doppler may be useful for identification persons with slightly elevated blood pressure, and hypertension stage 1.

Key words: blood pressure; hypertension; echocardiography, doppler; electrocardiography; diagnosis.

Apstrakt

Uvod/Cilj. Kašnjenje električnog impulsa između dve pretkomore je rani marker usporene propagacije impulsa kod odraslih osoba sa povišenim krvnim pritiskom. Cilj ovog rada bio je da se, koristeći novu ehokardiografsku metodu za procenu vremena provođenja impulse kroz pretkomoru, identifikuju odrasle osobe sa blago povišenim krvnim pritiskom i arterijskom hipertenzijom. **Metode.** Ispitivano je 149 odraslih osoba sa normalnim i povišenim krvnim pritiskom: 46 normotenzivnih zdravih osoba (grupa 1), 28 osoba sa blago povišenim krvnim pritiskom i hipertenzijom stepena 1 (grupa 2) i 75 bolesnika sa arterijskom hiperten-

zijom stepena 2 (grupa 3), prema poslednjim *Joint National Committee* (JNC) 8 preporukama. Ispitivana je funkcija rezervoara, prikazana kao totalni volumen pražnjenja leve pretkomore (TEV) i totalna frakcija pražnjenja leve pretkomore (TEF). Vreme provođenja impulsa je izmereno pulsним tkivnim Dopplerom, uključujući vreme provođenja impulsa u levoj pretkomori i vreme provođenja impulse između dve pretkomore. **Rezultati.** Vrednosti TEF su se progresivno smanjivale od grupe 1, preko grupe 2 do grupe 3 ($p < 0,001$). Disperzija P talasa ($55,1 \pm 9,8$ ms vs. $46,8 \pm 3,1$ ms vs. $43,1 \pm 2,6$ ms; $p < 0,01$) i vreme provođenja impulsa unutar leve pretkomore ($22,7 \pm 11,0$ ms vs. $8,4 \pm 4,7$ ms vs. $5,6 \pm 2,4$ ms; $p < 0,001$) su bili značajno

produženi samo u grupi 3. Vreme provođenja impulsa između dve pretkomore značajno se povećavalo od grupe 1, preko grupe 2 do grupe 3 ($15,6 \pm 3,9$ ms vs. $24,6 \pm 5,7$ ms vs. $50,4 \pm 20$ ms; $p < 0,05$ i $p < 0,05$). Vrednost kašnjenja impulsa između dve pretkomore od 19,5 ms može odvojiti odrasle osobe sa povišenim krvnim pritiskom od normotenzivnih zdravih osoba sa osetljivošću od 85%, i specifičnošću od 89% [površina ispod *receiver operating characteristic* (ROC) krive

0,911). **Zaključak.** Produženo vreme provođenja impulsa između dve pretkomore procenjeno pulsним tkivnim Doplerom može biti korisno za identifikovanje osoba sa blago povišenim krvnim pritiskom i arterijskom hipertenzijom prvog stepena.

Ključne reči:
krvni pritisak; hipertenzija; ehokardiografija, doppler; elektrokardiografija; dijagnoza.

Introduction

Elevated blood pressure can easily turn into high blood pressure (hypertension) unless one changes lifestyle habits. Both elevated blood pressure and high blood pressure increase risk of cardiovascular morbidity, mortality and stroke. It is highly prevalent in the general population¹. The prolonged elevation of blood pressure leads to the structural and functional remodeling of the left atrium (LA). LA dilatation and fibrosis lead to impaired impulse conduction and non-homogeneous propagation of impulses, both known as electrophysiological characteristics of atrial fibrillation. Enlargement of the LA and impairment of the LA mechanical function are potential indicators of the presence of cardiac diseases and arrhythmias^{2,3}. Atrial conduction time (ACT) reflect electrical and structural remodeling of the atria. Tissue Doppler imaging, a simple, non-invasive and applicable method, enable the evaluation of impulse propagation through the atria^{4,5}. Sequential analysis of atrial electromechanical coupling by the Tissue-Doppler imaging enables the analysis of atrial electromechanical coupling between the regions. Previous studies have shown that prolongation of interatrial and intra-atrial electromechanical times promotes atrial fibrillation⁶⁻¹⁰. Adults in early hypertensive disease are asymptomatic and it is of importance to establish early diagnosis and to assess the presence of subtle structural changes in their cardiovascular system¹¹.

Our hypothesis was that a noninvasive echocardiographic marker of slow sinus impulse propagation (ACT) may identify adults with early hypertensive disease and hypertensive adults.

Methods

One hundred and forty nine adults with normal and elevated blood pressure were studied: 46 normotensive persons (group 1) [blood pressure (BP) range: systolic blood pressure (SBP) < 120 mmHg, diastolic blood pressure (DBP) < 80 mmHg]; 28 adults with elevated blood pressure (SBP range: 120–129 mmHg, DBP < 80 mmHg) and hypertension stage 1 (SBP 130–139 mmHg, DBP 80–89 mmHg) (group 2), and 75 adults with hypertension stage 2 (group 3) (SBP ≥ 140 mmHg and (or) a DBP ≥ 90 mmHg). Blood pressure classification was based on the Joint National Committee-8 (JNC-8) guidelines, using an average of ≥ 2 readings obtained ≥ 2 occasions (officially based). We examined 243 consecutive participants with elevated blood pressure, and hypertension stage 1 and 2 who were in sinus rhythm referred by echocar-

diography. Patients with hypertension stage 2 were medically treated, i.e. they had pharmacologically regulated hypertension.

Patients with impaired ejection fraction of the left ventricle (LV) less than 50%, valvular heart disease, left or right bundle branch block, electrocardiographic conduction impairment, pericarditis, thyroid dysfunction, anemia, electrolyte disbalance, renal insufficiency, pulmonary disease, poor echocardiographic image as well as patients with documented paroxysms of atrial fibrillation, or those converted to a sinus rhythm pharmacologically or electrically, and who were on antiarrhythmic therapy, were excluded from further analysis. Therefore, the final study population consisted of 149 patients.

Clinical examination included recording of their weight, height, blood pressure and resting heart rate.

Standard 12-lead electrocardiograms (ECGs) were obtained using a recorder (Cardioexpress SL 12) set at a 25 mm/s paper speed, and 1 mV/cm standardization. We measured P wave duration manually with calipers and a magnifying glass. The mean P wave duration of 3 complexes was calculated in each lead. P maximum and P minimum were measured in 12 leads of the ECG surface. The difference between the P maximum and the P minimum was calculated and defined as P wave dispersion.

All the examinees were screened by an echocardiographer. Transthoracic echocardiographic examination was performed on the Vivid T8 (GEHealthcare) using phased array transducer of 3.5 and 2.5 MHz. During an echocardiographic examination, one ECG lead was continuously recorded.

M mode measurements and Doppler echocardiographic examination were performed according to the criteria of the American Echocardiographic Association^{12,13}. M mode measurements included: the dimensions of the LA, end-systolic and end-diastolic dimension of the LV and dimension of the LA in the parasternal longitudinal section. The LV ejection fraction (LV EF) was determined according to Simpson's rule. By using the pulsed Doppler in the apical four chambered view was measured the mitral flow, early diastolic (E wave) and late diastolic (A wave) inflow, E/A ratio, isovolumetric relaxation time (IVRT) and deceleration time of the mitral E wave (DT).

The volume of the LA (LAV) was measured from the apical four chamber view cross section, by using the biplane area length method¹⁴⁻¹⁶. LAV is indexed in relation to the surface of the body and expressed in mL/m². The LAV maximum (LAV max) was measured at the end of the ventricular systole, at the beginning of the opening of the mitral valve. LAV presystolic (LAV pre A) was measured in the middle

diastole at the beginning of the atrial systole (P ECG wave form) and LAV minimal (LAV min) at the start of closure of the mitral valve.

The parameters of the LA function were calculated from the LAVmax and LAV min. Reservoir function was presented as total emptying volume (TEV) and total emptying fraction (TEF). TEV was calculated as difference between LAVmax and LAVmin. TEF of the LA was calculated as $(LAV_{max} - LAV_{min}) / LAV_{max} \times 100$.

The atrial conduction time was evaluated by the pulsed tissue Doppler. The frequencies of transducers were 3.5 to 4.0 MHz. Adjusting the pulse Doppler signal to a Nyquist limit of 15 to 20 cm/s was done by using the minimum optimal gain. The signal speed of the monitor was set to 50-100 mm/s to optimize the spectral display of myocardial velocity. In the apical four chamber view section of the cursor, pulse tissue surplus was placed on the lateral mitral LV anulus, the septal mitral LV anulus, and the tricuspid anulus of the right ventricle to obtain spectral tissue Doppler image (TDI). Peak early diastolic (Em), and late diastolic (Am) wave were measured out of the surfaces. The same observer performed all echocardiographic measurements.

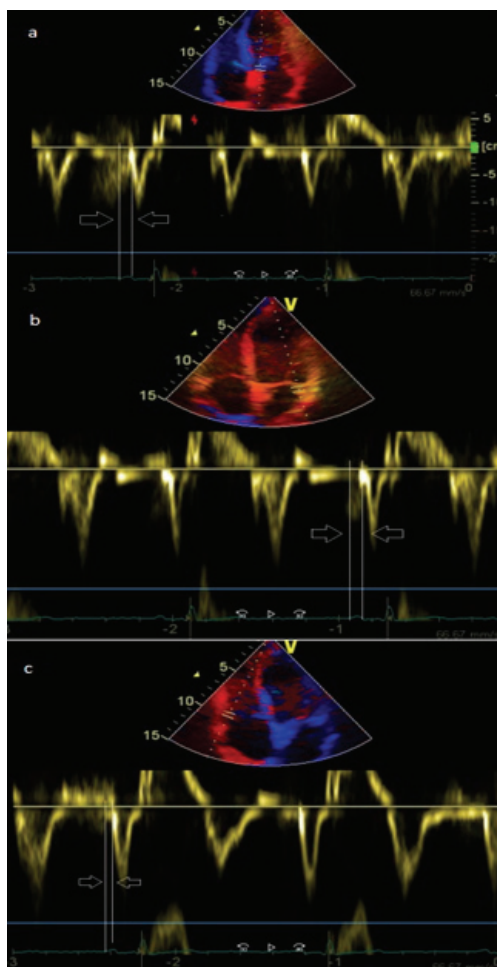


Fig. 1 – Measurement of time interval with tissue Doppler echocardiography, from the onset of P wave on surface of electrocardiogram (ECG) to the beginning of Am wave: a) on mitral septal annulus; b) on mitral lateral annulus; c) on tricuspid annulus.

The time from the beginning of the P wave on the ECG to the beginning of the Am wave of the tissue Doppler signal was accepted as the atrial conduction time (ACT). The difference in ACT of the left mitral lateral annulus and the right ventricle tricuspid annulus was defined as inter-ACT. The difference in ACT of the mitral lateral LV annulus and mitral septal LV annulus was defined as an intra-ACT (Figure 1).

A local Ethics Committee approved the study.

Data analysis

The descriptive statistics, including means and standard deviations of continuous variables, as well as frequencies and percentages of categorical variables were used to characterize the study sample. The differences among groups on continuous and categorical variables were analyzed by the use of one-way analysis of variances and the Pearson χ^2 test, respectively. For atrial conduction time, as the main predictor of interest for the detection of adults with elevated blood pressure vs. normotension, we calculated measures of diagnostic accuracy, including sensitivity, specificity and area under curve (AUC). The R environment for statistical computing (R Core Team, 2016) and IBM SPSS software (version 22) were used to conduct statistical analyses. Significance level (alpha level) was set at 0.05.

Interobserver variability was analyzed using the interclass coefficient (Table 1).

Table 1

Intraobserver variability	
Variable	ICC
LAVImax	0.998
LAVImin	0.997
LAVIpreA	0.999
ACT septal (ms)	1.000
ACT lateral (ms)	0.899
ACT tricuspid (ms)	0.877

ICC – interclass coefficient; ACT – atrial conduction time; LAVImax – left atrial volume index maximum; LAVImin – left atrial volume index minimum; LAVIpreA – left atrial volume index presystolic.

Results

There was no difference in terms of age, sex, resting heart rate, LV EF, BMI among all three groups (all $p > 0.05$). Through the study design, systolic blood pressure was significantly higher in the group 2 (all $p < 0.001$). Diastolic blood pressure was higher in the group 3 and the group 2 ($p < 0.001$) compared to that in the group 1 (Table 2).

Patients in the group 3 had higher LA diameter, LAVI max, LAVI pre A, LA TEV and LVS Em than those of the groups 2 and 1 (all $p < 0.001$). E/A ratio and LVS Em in the group 3 were significantly lower than those of the groups 1 and 2. E/Em velocity and LAVI min increased progressively from the group 1 to the groups 2 and 3 (all $p < 0.001$). LA TEF decreased progressively from the group 1 to the groups 2 and 3 (all $p < 0.01$). Peak mitral E wave was significantly higher in the group 1 compared to the groups 2 and 3 ($p < 0.001$) (Table 3).

Table 2

Baseline characteristics of the study population

Characteristics	Group 1	Group 2	Group 3	p^a	p^b	p^c
Age (years), mean \pm SD	51.0 \pm 9.3	50.6 \pm 10.5	53.5 \pm 9.8	0.986	0.401	0.422
Gender, n (%)				0.106	0.047	1.00
male	24 (54)	20 (77)	50 (75)			
female	20 (46)	6 (23)	17 (25)			
BMI (kg/m ²), mean \pm SD	26.8 \pm 2.1	25.6 \pm 1.4	26.2 \pm 2.8	0.100	0.239	0.671
SBP (mmHg), mean \pm SD	106.8 \pm 3.3	135.5 \pm 2.1	123.2 \pm 5.8	< 0.001	< 0.001	< 0.001
DBP (mmHg), mean \pm SD	72.2 \pm 2.6	84.3 \pm 2.2	81.8 \pm 6.5	< 0.001	< 0.001	0.067
Heart rate (beat/min), mean \pm SD	74.4 \pm 3.5	75.3 \pm 3.9	74.1 \pm 7.9	0.839	0.965	0.689
LVEF (%), mean \pm SD	66.4 \pm 3.8	64.4 \pm 9.3	64.1 \pm 8.1	0.523	0.237	0.978

Group 1 – normotensive adults; Group 2 – adults with elevated blood pressure and hypertension stage 1;

Group 3 – adults with hypertension stage 2.

BMI – body mass index; SBP – systolic blood pressure; DBP – diastolic blood pressure; LVEF – left ventricle ejection fraction; SD – standard deviation; p^a – significance of the difference between groups 1 and 2; p^b – significance of the difference between groups 2 and 3; p^c – significance of the difference between groups 1 and 3.

Table 3

Left ventricle and left atrial structural and functional parameters in the study population

Variables	Group 1 (mean \pm SD)	Group 2 (mean \pm SD)	Group 3 (mean \pm SD)	p^a	p^b	p^c
P-WD (msec)	43.1 \pm 2.6	46.8 \pm 3.1	55.1 \pm 9.8	0.096	< 0.001	< 0.001
E/A ratio	1.4 \pm 0.03	1.3 \pm 0.14	0.9 \pm 0.28	0.109	< 0.001	< 0.001
LA diameter (mm)	3.2 \pm 0.39	3.4 \pm 0.27	3.8 \pm 0.40	0.110	< 0.001	< 0.001
LAVI max. (mL/m ²)	24.7 \pm 6.9	28 \pm 7.5	40 \pm 6.8	0.092	< 0.001	< 0.001
LAVI min. (mL/m ²)	8.7 \pm 2.2	11.3 \pm 2.8	17.8 \pm 5.2	0.030	< 0.001	< 0.001
LAVIpreA (mL/m ²)	11.9 \pm 2.6	14.7 \pm 3.4	29.4 \pm 7.3	0.110	< 0.001	< 0.001
LATEV (mL/m ²)	16.0 \pm 2.5	16.7 \pm 2.5	22.1 \pm 2.5	0.702	< 0.001	< 0.001
LATEF (%)	64.8 \pm 4.4	59.8 \pm 5.2	55.6 \pm 7.3	0.004	< 0.001	0.011
LV DT (msec)	142.5 \pm 8.1	145.1 \pm 12.3	194.6 \pm 29.1	0.882	< 0.001	< 0.001
IVRT (msec)	101.9 \pm 18.2	110.1 \pm 14.3	128.8 \pm 15.8	0.105	< 0.001	< 0.001
LVSEm (m/s)	10.5 \pm 1.8	9.8 \pm 1.3	6.7 \pm 1.7	0.267	< 0.001	< 0.001
LVSE/Em	7.2 \pm 1.4	9.5 \pm 2.5	12.5 \pm 2.1	< 0.001	< 0.001	< 0.001
Peak mitral Ewave (m/s)	8.3 \pm 0.5	7.5 \pm 0.9	7.1 \pm 0.9	0.001	< 0.001	0.153
Peak mitral A wave (m/s)	8.6 \pm 1.7	9.0 \pm 1.6	9.2 \pm 1.4	0.577	0.107	0.799

Group 1 – normotensive adults; Group 2 – adults with elevated blood pressure and hypertension stage;

Group 3 – adults with hypertension stage 2.

PWD – P wave dispersion; LA – left atrium; LV – left ventricle; LAVI – left atrial volume index; IVRT – isovolumetric relaxation time; DT – deceleration time; LATEV – left atrium total emptying volume; LATEF – left atrium total emptying fraction; LVSEm – septal tissue Doppler early diastolic wave; E – early diastolic wave; A – late diastolic wave; SD – standard deviation; min. – minimum; max. – maximum; p^a – significance of the difference between groups 1 and 2; p^b – significance of the difference between groups 2 and 3; p^c – significance of the difference between groups 1 and 3.

Table 4

Atrial conduction time assessed by the tissue Doppler imaging in the study population

Variables	Group 1 (mean \pm SD)	Group 2 (mean \pm SD)	Group 3 (mean \pm SD)	p^a	p^b	p^c
ACT septal (ms)	20.9 \pm 5.0	32.6 \pm 6.5	58.3 \pm 16.7	0.001	< 0.001	< 0.001
ACT lateral (ms)	26.6 \pm 5.6	41.2 \pm 6.9	81.1 \pm 28.3	0.006	< 0.001	< 0.001
ACT tricuspid (ms)	10.9 \pm 3.1	16.8 \pm 4.9	29.8 \pm 8.6	0.001	< 0.001	< 0.001
ACT lateral-ACTseptal (ms)*	5.6 \pm 2.4	8.4 \pm 4.7	22.7 \pm 11.0	0.345	< 0.001	< 0.001
ACT lateral-ACTtricuspid (ms)†	15.6 \pm 3.9	24.6 \pm 5.7	50.4 \pm 20.0	0.034	< 0.001	< 0.001

Group 1 – normotensive adults; Group 2 – adults with elevated blood pressure and hypertension stage;

Group 3 – adults with hypertension stage 2.

ACT – atrial conduction time, interval from the onset of P wave on the surface of electrocardiogram to the beginning of the late diastolic wave (Am wave) assessed by the tissue Doppler imaging; p^a – significance of the difference between groups 1 and 2; p^b – significance of the difference between groups 2 and 3; p^c – significance of the difference between groups 1 and 3; *intraatrial conduction time; †interatrial conduction time.

P wave dispersion in the group 3 was significantly higher than that in the groups 1 and 2. Atrial conduction time findings measured by TDI are shown in Table 4.

Atrial conduction time on septal, lateral and tricuspid annulus were significantly increasing from the group 1 to the groups 2 and 3. There was no difference in inter ACT between the group 1 and the group 2. Patients in the group 3 had significantly prolonged intra ACT than those in the groups 1 and 2. Related to the group 1, inter ACT was prolonged in the group 2 ($p = 0.034$) and further prolonged in the group 3 ($p < 0.001$).

Using a cut off level of 19.5 ms, interatrial ACT could separate adults with slightly elevated blood pressure and hypertension stage 1 from normotensive persons with a sensitivity of 85%, and specificity of 89% [area under receiver operating characteristic (ROC) curve (AUC) 0.911] (Figure 2).

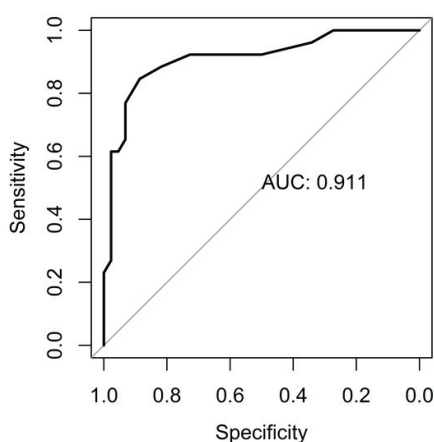


Fig. 2 – The area under receiver operating characteristic (ROC) curve (AUC) of the interatrial conduction time for predicting presence of early hypertensive disease vs. normotension was 0.911 (sensitivity 85%, specificity 89%). Optimum cut-off point was 19.5 ms.

Discussion

The principal finding of this study is that interatrial conduction time is a predictor of the presence of early hypertensive disease in adults. A cut off 19.5 ms for inter ACT had a sensitivity 85% and specificity 89% in identifying early hypertensive disease in adults. According to available sources, this is a new finding, which has not been previously reported.

In our study, we demonstrated that impairment in propagation of sinus impulses is present in hypertension. However, adults with slightly elevated blood pressure had a slowing of sinus impulse propagation. It was demonstrated that ACT on lateral, septal mitral, and tricuspid annulus, inter and intra ACT were prolonged in hypertension compared to normotension¹⁷. Atrial electromechanical delay and P wave dispersion are longer in hypertensions with elevated pulse pressure and without significant structural remodeling¹⁸. Inter and intra ACTs are prolonged in hypertension¹⁹, and in early stages of hypertension without significant prolongation of maximum P wave duration on 12 lead ECG. This is an explanation for presence of earlier

period of atrial electrical impairment, before the appearance of P wave prolongation on 12 lead ECG⁶. Our study shows that P wave dispersion was similar between normotensive persons and those with slightly elevated blood pressure, while it was significantly prolonged in patients with hypertension stage 2. Slowing atrial conduction velocity and atrial conduction delay can occur due to atrial fibrosis, remodeling and can be a trigger for atrial fibrillation. Atrial conduction delay can be easily discovered with tissue Doppler, and may be an indicator for the presence of arrhythmogenic substrate¹⁶. In our study we demonstrated significantly prolonged intra ACT, inter ACT and P wave dispersion in patients with hypertension, even when it is medically regulated. We found prolonged intra ACT in patients with early hypertension disease, without increase in P wave dispersion on 12 channel ECG, which is known as electrophysiological marker of inhomogenous propagation of sinus impulses. Our findings suggest that inter ACT conduction time measured by tissue Doppler is the first sign of impaired conduction of sinus impulses in the early stage of hypertension, even in persons with slightly elevated blood pressure.

In addition to morphologic remodeling known to be present in hypertensive patients, even slightly elevated blood pressure also cause electrical remodeling of atria which are responsible for slowing conduction of sinus impulses. LA enlargement causes slowing of sinus impulse conduction because of structural and electrophysiological changes in the atrial myocardium.

Our study also showed impairment of the mechanical function of LA in hypertension but in adults with slightly elevated blood pressure as well. Impairment of the LA mechanical function and LA enlargement were common findings in hypertension^{18, 19}. Decreased mechanical function of LA has been observed in recently diagnosed hypertensive patients and it was related to an increased LAVI min²⁰. The LA volume and active systolic function assessed by real-time three-dimensional echocardiography (RT3DE) were significantly increased in prehypertension¹⁶. Some sources²¹ suggested that the occurrence of paroxysmal atrial fibrillation in hypertensive patients is associated with enhanced LA reservoir and conduit function and worsening booster pump function. In our study population dimensions of the LA were normal. Using pulsed wave Doppler methods [DT, isovolumic relaxation time (IVRT), LV E/A, mitral A wave], we did not find complete impairment of diastolic function of the LA in the group 2, but we found it in the group 3. All the examinees including those with hypertension had normal LA dimensions. By analyzing the LA volume in various phases of the heart cycle, we showed that there was a deterioration of the mechanical function of the LA in the group 2, and significant impairment in the group 3, even though they were pharmacologically regulated. There was continuous trend in the progression of reduction in the atrial reservoir function from slightly elevated blood pressure to hypertension.

Our study demonstrated the presence of disturbance of atrial conduction in adults with slightly elevated blood pressure and significant impairment of interatrial and intraatrial sinus impulse conduction in medically controlled hypertensive patients.

We found that inter ACT measured by tissue Doppler may be useful echocardiographic marker for the identification of adults with slightly elevated blood pressure.

The limitation of this study is that atrial conduction time was not investigated by invasive electrophysiological techniques and compared to echocardiographic examination.

Another limitation was a single operator acquired echocardiographic and electrocardiographic measurements, that unabled us to compare interobserver variability.

Conclusion

Mechanical and electrical function of the LA is impaired in patients with early hypertensive disease and significantly impaired in hypertension. Prolonged atrial conduction time estimated with a tissue Doppler may be useful for distinguishing normotensive persons from those with slightly elevated blood pressure and those with hypertension stage 1.

R E F E R E N C E S

1. *Liszka HA, Mainous AG 3rd, King DE, Everett CJ, Egan BM.* Prehypertension and cardiovascular morbidity. *Ann Fam Med* 2005; 3(4):294–9.
2. *Hof I, Chilukuri K, Arbab-Zadeh A, Scherr D, Dalal D, Nazarian S,* et al. Does Left Atrial Volume and Pulmonary Venous Anatomy Predict the Outcome of Catheter Ablation of Atrial Fibrillation? *J Cardiovasc Electrophysiol* 2009; 20(9): 1005–10.
3. *Abecasis J, Dourado R, Ferreira A, Saraiva C, Cavaco D, Santos KR,* et al. Left atrial volume calculated by multi-detector computed tomography may predict successful pulmonary vein isolation in catheter ablation of atrial fibrillation. *Europace* 2009; 11(10): 1289–94.
4. *Todaro MC, Choudhuri I, Belohlavek M, Jahangir A, Carerj S, Oreto L,* et al. New echocardiographic techniques for evaluation of left atrial mechanics. *Eur Heart J Cardiovasc Imaging* 2012; 13(12): 973–84.
5. *Yavuz B, Deniz A, Ertugrul DT, Devci OS, Yalcin AA, Ata N,* et al. A novel echocardiographic marker in hypertensive patients: is diastolic dysfunction associated with atrial electromechanical abnormalities in hypertension? *J Clin Hypertens (Greenwich)* 2010; 12(9): 687–92.
6. *Avci BK, Gulmez O, Donmez G, Pehlivanoglu S.* Early Changes in Atrial Electromechanical Coupling in Patients with Hypertension: Assessment by Tissue Doppler Imaging. *Chin Med J (Engl)* 2016; 129(11): 1311–5.
7. *Omi W, Nagai H, Takamura M, Okura S, Okajima M, Furusbo H,* et al. Doppler tissue analysis of atrial electromechanical coupling in paroxysmal atrial fibrillation. *J Am Soc Echocardiogr* 2005; 18(1): 39–44.
8. *De Vos CB, Weijts B, Crijs HJ, Cheriex EC, Palmans A, Habets J,* et al. Atrial tissue Doppler imaging for prediction of new-onset atrial fibrillation. *Heart*. 2009; 95(10): 835–40.
9. *Cui QQ, Zhang W, Wang H, Sun X, Wang R, Yang HY,* et al. Assessment of atrial electromechanical coupling and influential factors in nonrheumatic paroxysmal atrial fibrillation. *Clin Cardiol* 2008; 31(2): 74–8.
10. *Kinay O, Nazli C, Ergene O, Dogan A, Gedikli O, Hoscan Y,* et al. Time interval from the initiation of the electrocardiographic P wave to the start of left atrial appendage ejection flow: A novel method for predicting atrial fibrillation recurrence. *J Am Soc Echocardiogr* 2002; 15(12): 1479–84.
11. *Bajpai JK, A P S, A K A, A K D, Garg B, Goel A.* Impact of prehypertension on left ventricular structure, function and geometry. *J Clin Diagn Res* 2014; 8(4): BC07–10.
12. *Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA,* et al. Recommendations for Chamber Quantification: A Report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, Developed in Conjunction with the European Association of Echocardiography. *J Am Soc Echocardiogr* 2005; 18(12): 1440–63.
13. *Haffajee JA, Lee Y, Alsheikh-Ali AA, Kuvin JT, Pandian NG, Patel AR.* Pre-operative left atrial mechanical function predicts risk of atrial fibrillation following cardiac surgery. *JACC Cardiovasc Imaging* 2011; 4(8): 833–40.
14. *Gulmez O, Parildar H, Cigerli O, Demirağ N.* Assessment of left atrial function in patients with type 2 diabetes mellitus with a disease duration of six months. *Cardiovasc J Afr* 2018; 29(2): 82–7.
15. *Aktiürk E, Ermis N, Yağmur J, Aciogöz N, Kurtoglu E, Cansel M,* et al. Early left atrial mechanics and volume abnormalities in subjects with prehypertension: a real time three-dimensional echocardiography study. *Echocardiography* 2012; 29(10): 1211–7.
16. *Cimen T, Sunman H, Efe TH, Akyel A, Yayla K, Şaban HF,* et al. Early changes in atrial conduction times in hypertensive patients with elevated pulse pressure. *Rev Port Cardiol* 2017; 36(6): 453–9. (English, Portuguese)
17. *Kokubu N, Yuda S, Tsuchihashi K, Hashimoto A, Nakata T, Miura T,* et al. Noninvasive assessment of left atrial function by strain rate imaging in patients with hypertension: a possible beneficial effect of renin-angiotensin system inhibition on left atrial function. *Hypertens Res* 2007; 30(1): 13–21.
18. *Dernellis JM, Vjysoulis GP, Zacharoulis AA, Toutouzas PK.* Effects of antihypertensive therapy on left atrial function. *J Hum Hypertens* 1996; 10(12): 789–94.
19. *Aljizeeri A, Gin K, Barnes ME, Lee PK, Nair P, Jue J,* et al. Atrial remodeling in newly diagnosed drug-naive hypertensive subjects. *Echocardiography* 2013; 30(6): 627–33.
20. *Cui Q, Wang H, Zhang W, Wang H, Sun X, Zhang Y,* et al. Enhanced Left Atrial Reservoir, Increased Conduit, and Weakened Booster Pump Function in Hypertensive Patients with Paroxysmal Atrial Fibrillation. *Hypertens Res* 2008; 31(3): 395–400.

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