

Evaluating functional capacity, and mortality effects in the presence of atrial electromechanical conduction delay in patients with systolic heart failure

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ABSTRACT

Objective: Atrial functions are relatively suppressed in heart failure (HF). We aimed to investigate the associations of intra- and inter-atrial electromechanical conduction delay (EMCD) with functional class and mortality over a 12-month follow-up period.

Methods: The prospective study included 65 patients with systolic HF and 65 healthy subjects with normal sinus rhythm. Left ventricular (LV) systolic functions and left atrial (LA) dimensions and volumes were evaluated by transthoracic echocardiography. Tissue Doppler imaging (TDI) signals at the lateral border of the mitral annulus (lateral PA'), septal mitral annulus (septal PA'), and tricuspid annulus (tricuspid PA') were measured. Intra- and inter-atrial EMCD were calculated.

Results: Mitral inflow velocities were studied using pulsed-wave Doppler after placing the sample volume at the leaflets' tips. The peak early (E wave) and late (A wave) velocities were measured. The septal annular E/E' ratio was relatively higher and lateral, septal, and right ventricular S, E', and A' waves were significantly lower in the HF group than in the control group (12.49±6.03 – 7.16±1.75, p/E/E' <0.0001). Intra-atrial EMCD was detected as 117.5 ms and inter-atrial EMCD as 127.5 ms in patients with prolonged atrial EMCD. A significant increase was found in prolonged intra- and inter-atrial EMCD according to functional capacity increase (p=0.012 and p=0.031, respectively). The incidence of mortality was significantly higher in patients with prolonged atrial EMCD (p=0.025), and 5 patients in the HF group died during the study over the 12-month follow-up period.

Conclusions: In this study, we found a relationship between prolonged atrial conduction time and increased functional class and mortality in patients with systolic HF. (*Anatol J Cardiol* 2016; 16: 579-86)

Keywords: heart failure, atrial electromechanical delay, functional class, mortality

Introduction

The atrium is not only a simple and passive heart space that transports blood to the ventricle but also a dynamic structure. The atria contribute up to 30% of the cardiac output (1); however, atrial function is relatively suppressed in heart failure (HF). Currently, measurement of the left atrial (LA) size is the most commonly used method to estimate the amount of atrial remodeling (2). LA volume and mechanical function have recently been identified as potential indicators of cardiac disease and arrhythmias (3); whereas, for all other aspects, additional parameters are required in the evaluation of atrial remodeling. Intra- and inter-atrial conduction delays and non-homogeneous propagation of

sinus impulses are well-known electrophysiological characteristics of the atria prone to fibrillation (4).

Contrary to LA size, atrial conduction times reflect the amount of both electrical and structural remodeling of the atria.

Among the non-invasive and invasive methods employed for evaluation of inter-atrial conduction, the basic and most frequently used method is evaluation of electrocardiographic P wave duration and morphology (5). The time interval between the electrocardiographic P wave and atrial contraction detected by M-mode or Doppler echocardiography is defined as atrial electromechanical conduction delay (EMCD). Atrial mechanical activity can be detected from different atrial regions by Tissue Doppler imaging (TDI) and echocardiography with high temporal resolution (6).

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Published studies indicate that EMCD, as detected by TDI, increases in different patient groups, such as those with mitral stenosis, paroxysmal atrial fibrillation, and coronary slow-flow pattern (7, 8). There is no clear data about the relationship between the phasic functions of LA and EMCD in systolic HF.

In this study, we aimed to evaluate the relationship of suppressed atrial functions and functional capacity with electrocardiographic findings and echocardiographic parameters of patients with systolic HF. This study also aimed to investigate the correlation between intra- and inter-atrial conduction times measured by TDI. In addition, we investigated morbidity (functional capacity, arrhythmias, cerebrovascular event, or rehospitalization) and mortality in patients with HF.

Methods

Study population

Between September 2008 and November 2010, the study prospectively enrolled 79 consecutive patients having symptomatic HF with left ventricular ejection fraction (LVEF) measured as <50% with 2D echocardiography. Patients with newly diagnosed systolic HF or those with a known history of systolic HF were included. All patients were in sinus rhythm (SR). However, 14 patients were excluded from the study because of valvular stenosis (n=4), moderate to severe valvular regurgitation (n=6), and detection of atrial fibrillation episodes on 24-h Holter ECG monitoring (n=4). The final study population consisted of 65 patients (44 men and 21 women) with HF who were in normal SR at the time of enrollment. In addition, 65 volunteers (37 men and 28 women) with similar age and sex distributions and normal physical examination findings served as a control group. In the control group, patients over 40 years of age were included only when normal coronary arteries or no critical coronary artery lesions (<40% stenosis) were found on coronary angiography. The study was designed prospectively. Ethical approval was obtained from the Local Ethics Committee, and informed consent was obtained from all subjects. Other exclusion criteria were moderate or severe valvular heart disease, history of asthma or use of bronchodilators, hypo- or hyperthyroidism, hepatic or renal failure (serum creatinine: >2.0 mg/dL), rhythm disturbances including second- or third-degree heart block, sick sinus syndrome, atrial fibrillation, and complete bundle branch block. Detailed family and medical history were collected for all patients those conformed to the inclusion criteria. New York Heart Association (NYHA) functional capacity and medical therapies were recorded. ECGs were also taken. Physical examination, 12-lead ECG, chest X-ray, and laboratory investigations including complete blood count, blood biochemistry, and thyroid function tests were routinely performed for all patients. Twenty-four-hour ambulatory ECG monitoring was performed as an annual routine risk assessment. Transthoracic echocardiography (TTE) was performed for all subjects, and they visited the hospital regularly for monthly check-up after enrollment during the 12-month follow-up period.

Electrocardiography

At the beginning of the study, all subjects underwent standard 12-lead ECG, acquired using Nihon-Kohden Cardiofax Q 3190K (California, USA) electrocardiograph at a paper speed of 25 mm/s and 10 mm/mV. All recordings were performed in the same quiet room through spontaneous breathing with subsequent 20 min of adjustment in the supine position. P wave duration measurements were performed manually by 2 observers using calipers and magnifying lenses to precisely define the ECG deflection, as described in a previous study (9). The beginning of the P wave was defined as the point where the initial deflection of the P wave crossed the isoelectric line, and the end of the P wave was defined as the point where the final deflection of the P wave crossed the isoelectric line. To measure P wave dispersion, 12-lead surface ECGs were obtained for each subject in the supine position at a paper speed of 50 mm/s. The longest atrial conduction time measured on any of the 12 leads was defined as P maximum (Pmax) and the shortest time was defined as P minimum (Pmin). The difference between Pmax and Pmin was calculated and defined as P wave dispersion (PWD=Pmax – Pmin). ECG recordings with a measurable P wave in less than 10 leads were excluded from the analysis.

Echocardiographic examination

All echocardiographic examinations were performed with a Hewlett Packard Sonos 7500 cardiac ultrasound (Philips Medical Systems, Andover, MA, USA) scanner and 2.5–3.5-MHz transducers. All patients were examined by precordial 2-dimensional, Doppler and TDI according to the criteria of the American Society of Echocardiography (10). TTE recordings were performed while the patients were in the left lateral decubitus position. Apical 4-chamber, 2-chamber, and parasternal long-axis images were recorded at expiratory apnea for 3 consecutive cardiac cycles. One-lead ECG was recorded continuously. All echocardiographic assessments were recorded on a VHS video tape; and the records were reanalyzed by 2 physicians who were blind to the study.

Left ventricular (LV) end-systolic and end-diastolic diameters were measured by apical 2- and 4-chamber views. LVEF and left atrial ejection fraction (LAEF) were evaluated by the biplane Simpson's method. All valves were evaluated during echocardiographic examination by continuous wave Doppler color flow and Doppler imaging for insufficiency and stenosis. Pulmonary artery systolic pressure was measured by Bernoulli's equation ($p=4v^2$) from the tricuspid regurgitation jet flow. The estimated pulmonary artery systolic pressure was then calculated by adding 5–20 mm Hg, according to the width of the inferior vena cava, to pulmonary artery systolic pressure (11).

LV diastolic function was evaluated by mitral inflow velocities, namely the E peak, A peak, and E/A ratio, and also by the deceleration time of the E wave and isovolumic relaxation time. In the apical 4-chamber view, the pulsed Doppler sample volume was placed at the level of the right ventricular tricuspid annulus, LV lateral mitral annulus, and septal mitral annulus. At least

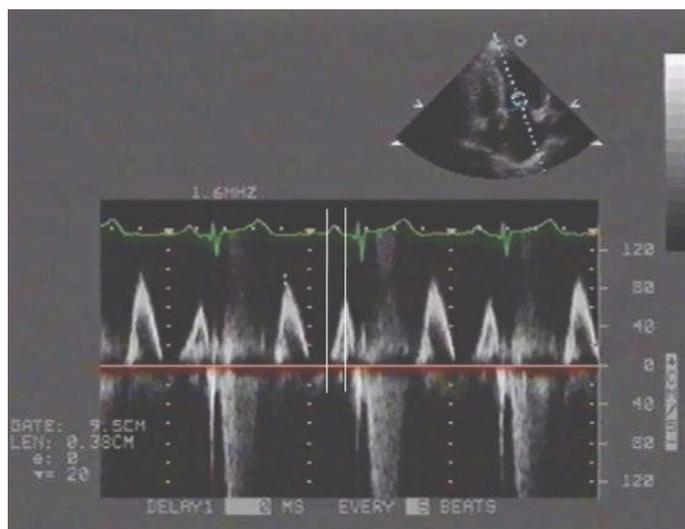


Figure 1. Measurement of inter-atrial conduction time

3 consecutive traces of all annulus were recorded. The tissue Doppler pattern was characterized by a positive (i.e., above the baseline) myocardial systolic wave (S), two negative (i.e., below the baseline) early diastolic waves (E'), and an atrial wave (A'). Every effort was made to align the pulsed-wave cursor so that the Doppler angle of incidence could be as close to zero as possible in the direction of these walls (12).

Inter-atrial conduction time

The time interval from the onset of P wave on the surface ECG to the peak of transmitral A wave (PA), which is defined as the inter-atrial conduction time, was measured in all groups from the apical 4 chambers (Fig. 1) (13).

Atrial EMCD time

The time interval from the onset of P wave on the surface ECG to the beginning of the late diastolic wave (A') was termed as atrial electromechanical coupling (PA'). It was obtained from the right ventricular tricuspid annulus, septal mitral annulus, and lateral mitral annulus and described as tricuspid PA', septal PA', and lateral PA', respectively. The difference between tricuspid PA' and septal PA' was defined as intra-atrial (Δ_{intraA}) EMCD, and the difference between tricuspid PA' and lateral PA' was defined as inter-atrial (Δ_{interA}) EMCD (Fig. 2). All measurements were repeated 4 times, and the average values were obtained for each of the atrial conduction delay times. All measurements were performed by 3 experienced researchers who were unaware about the clinical status of the subjects. If a difference of >5% in any of the variables measured by both the investigators was found, the patient was not included, whereas if the difference was <5%, the measurements were averaged (14, 15).

Twenty-four-hour ambulatory electrocardiographic monitoring

Twenty-four-hour ambulatory electrocardiographic monitoring was performed for all patients with systolic HF over the

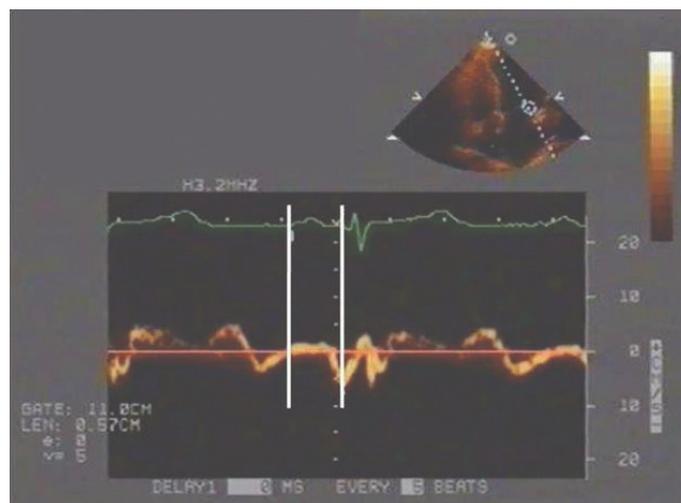


Figure 2. Measurement of intra- and inter-atrial conduction time with the help of electrocardiographic rhythm tracing and pulsed-wave Doppler

12-month study period for annual risk assessment, and presence of arrhythmias was investigated.

Statistical analysis

Statistical analyses were performed using SPSS for Windows, version 18.0 (SPSS, Chicago, IL). All continuous variables were expressed as mean \pm SD, and categorical variables were defined in percentages. Categorical data were compared using the χ^2 test. Continuous variables were compared between the groups using Student's t-test or Mann-Whitney U test depending on whether they distributed normally or not, as tested by the Shapiro-Wilk test. Pearson's correlation analysis was used to estimate the relationship between the test parameters. ROC curve analysis was performed for numerical variables to distinguish the patient and the control groups, and threshold values were also determined. A p value of <0.05 was considered to be statistically significant.

Results

Patient demographics

In this study, 65 patients with systolic HF and 65 healthy subjects as control were compared. There were no statistically significant differences in the etiology of patients with HF [35 patients with ischemic HF (54%) and 30 patients with non-ischemic HF (46%)]. There was no significant difference between both the groups in terms of age, sex, weight, and diastolic blood pressure. Lower functional capacity was found in patients with systolic HF. With respect to the frequency of comorbidities, type 2 diabetes mellitus (DM) and coronary artery disease (CAD) were significantly more common in the HF group, whereas the frequencies of hypertension (HT) and hyperlipidemia (HLP) were similar between the groups. The groups were also compared in terms of medical treatment; it was found that use of statins and angiotensin-converting enzyme inhibitor/angiotensin receptor blocker (ACE-I/ARB) was significantly higher in the HF group. There

Table 1. General characteristics of the systolic HF and control groups

	HF group (n=65)	Control group (n=65)	P
Age, years	55.03±14.3	50.29±12.4	0.12
Sex, male/female	44 / 21	37 / 28	0.087
Body surface area, m ²	1.81±0.16	1.85±0.18	0.06
SBP, mm Hg	115.5±14.8	126.8±16.6	<0.001
DBP, mm Hg	69.5±8.9	71.4±11.7	0.31
NYHA functional class	1.71±0.8	1.01±0.12	<0.001
Disease			
Hypertension	30 (47.6%)	34 (52.3%)	0.59
Hyperlipidemia	26 (41.3%)	22 (33.8%)	0.38
Diabetes mellitus	22 (34.9%)	11 (16.9%)	0.02
Coronary artery disease	34 (53.9%)	5 (7.6%)	<0.001
Drug			
Beta blocker	49 (75.3%)	43 (66.1%)	<0.34
ACE-I/ARB	55 (87.3%)	21 (32.3%)	<0.001
Statin	47 (74.6%)	20 (30.8%)	<0.001
Diuretic	65 (100%)	0 (0%)	
Continuous data are expressed as mean±SD. DBP - diastolic blood pressure; HF - heart failure; NYHA - New York Heart Association; SBP - systolic blood pressure			

was no significant difference between the groups in terms of β -blocker treatment (Table 1). Among the patients, 35 (53%) were taking only loop diuretics, 8 (12%) were taking only aldosterone antagonists, and 22 (33%) were taking both in the HF group. None of the patients were taking antiarrhythmic drugs such as digoxin, propafenone, sotalol, and amiodarone.

The functional class of all patients was evaluated and we found that 35% (23 patients) were in class I, 43% (28 patients) were in class II, and 22% (14 patients) were in class III and class IV. There was no significant difference between the groups in terms of the resting heart rate (74±14.82 vs. 77.69±9.53; $p<0.67$). Five patients in the HF group died during the 12-month follow-up period.

When echocardiography data were compared between the HF and control groups, LVEF was significantly lower in the HF group (33.1%±8.2% vs. 64.2%±5.4%, $p<0.001$). LAEF was also significantly lower in the HF group (39.1%±7% vs. 61%±2%, $p<0.001$). LA volume and maximal diameter were higher in the HF group than in the control group (63.92±26.06 vs. 45.3±1.01, $p<0.0001$ and 4.36±0.65 vs. 3.37±0.35, $p<0.0001$, respectively). Lateral, septal, and right ventricular S, E', and A' waves were significantly lower in the HF group than in the control group. The proportion of E velocity, which was obtained from mitral valve pulsed-wave Doppler flow and E' velocity, obtained from the TDI of septal annular tissue (E/E') as an indicator of LV end-diastolic pressure, was compared and the septal annular E/E' ratio was found to be significantly higher in the HF group than in the control group (12.49±6.03 vs. 7.16±1.75, $p<0.0001$) (Table 2).

Table 2. TTE findings of the systolic HF and control groups

	HF group	Control group	P
LVEF	33.12±7.8	64.27±5.4	0.001
E/E' septal annulus	12.49±6.03	7.16±1.75	0.0001
LA EF	39.19±7.30	61.07±2.39	0.0001
LVDD, cm	5.63±0.98	4.30±0.26	0.0001
LVSD, cm	4.53±1.01	3.28±0.27	0.0001
LA maximal diameter, cm	4.36±0.65	3.37±0.35	0.0001
LA volume, mm ³	63.92±26.06	45.3±1.01	0.0001
RV (S), cm/s	10.10±3.10	13.28±1.14	0.0001
RV (E'), cm/s	8.94±4.47	9.79±2.69	0.0001
RV (A'), cm/s	11.69±4.93	14.57±3.24	0.001
Septal (S), cm/s	5.55±1.89	7.54±1.32	0.0001
Septal (E'), cm/s	5.86±2.63	10.57±3.62	0.0001
Septal (A'), cm/s	6.72±2.85	9.48±1.80	0.0001
Lateral (S), cm/s	5.39±1.87	8.14±2.09	0.0001
Lateral (E'), cm/s	7.11±3.67	12.33±4.30	0.0001
Lateral (A'), cm/s	6.75±3.33	9.45±2.61	0.0001
Continuous data are expressed as mean±SD. HF - heart failure; LA - left atrium; LAEF - left atrium ejection fraction; LVDD - left ventricular diastolic diameter; LVSD - left ventricular systolic diameter, RV - right ventricle			

Comparison of inter-atrial conduction time and atrial EMCD

Inter-atrial conduction time and atrial EMCD time were significantly prolonged in the HF group in contrast to the control group. The Pmax ($p<0.001$), Pmin ($p<0.001$), and PWD ($p<0.001$) values were significantly higher in the HF group than in the control group (Table 3). At the end of the study, ROC curve analysis was performed for all EMCD times used in the measurement of intra- and inter-atrial EMCD, and the cut-off value was determined for each of them (Fig. 3, 4). The threshold values of PA, tricuspid PA', septal PA', and lateral PA' were 102.5 ms, 107.5 ms, 127.5 ms, and 137.5 ms, respectively. In a similar manner, the threshold values of Δ intraA and Δ interA was 17.5 ms and 27.5 ms, respectively (Table 4). Ischemic cerebrovascular events were seen in 4 patients (6%) (16). Thirteen patients (20%) were admitted to the hospital again due to HF at the end of the 12-month period. Ten patients (15.9%) reported arrhythmia, including atrial fibrillation (4 patients), ventricular tachycardia (6 patients), and total AV block (1 patient), during the 12-month follow-up period (17). Correlation analysis of PWD with intra- and inter-atrial dyssynchrony and other parameters revealed that PWD was positively correlated with Pmax ($r=0.917$, $p=0.001$), Pmin ($r=0.431$, $p=0.001$), intra-atrial dyssynchrony ($r=0.606$, $p=0.001$), inter-atrial dyssynchrony ($r=0.63$, $p=0.001$), and PA time ($r=0.454$, $p=0.001$), whereas it was negatively correlated with LVEF ($r=-0.277$, $p=0.028$) (Table 5).

Determinants of functional class

The HF group was further divided into 2 groups according to the functional class. Group 1 (51 patients) included func-

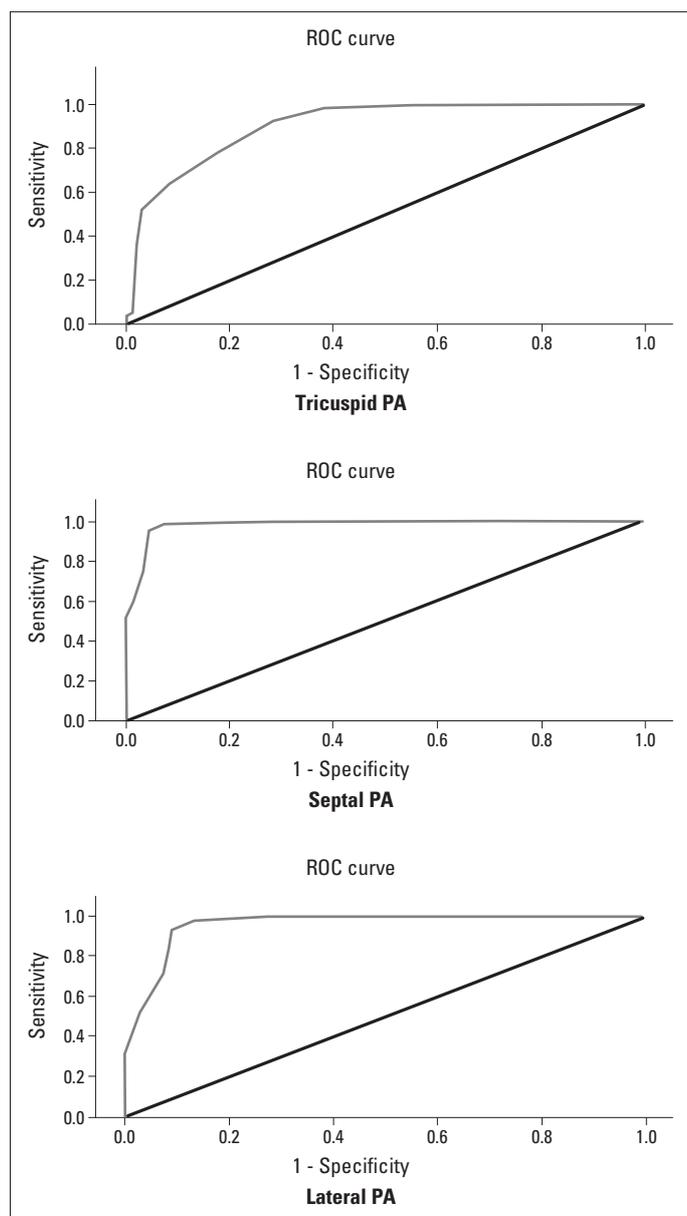


Figure 3. ROC curve analysis of tricuspid PA', septal PA', and lateral PA' tional classes I and II. Group 2 (12 patients) included functional classes III and IV. Group 1 contained a significantly lower number of patients who had prolonged intra- and inter-atrial EMCD ($p=0.004$ and $p=0.012$, respectively). The functional class of patients with prolonged intra- and inter-atrial EMCD was increased during the 12-month follow-up period ($p=0.012$ and $p=0.031$, respectively).

Relationship between inter-atrial EMCD and morbidity

Patients with atrial EMCD had a high morbidity rate during the 12-month follow-up period, but it was not statistically significant ($p_{\Delta\text{intraA}}=0.211$, $p_{\Delta\text{interA}}=0.280$).

Relationship between inter-atrial EMCD and mortality

Patients with atrial EMCD had a high mortality rate during

Table 3. Comparison of inter-atrial conduction time and atrial EMCD

Time, msn	HF group	Control group	P
PA time	125.56±16.2	117.63±20.5	0.017
Lateral PA'	148.81±9.2	120.62±12.1	<0.001
Septal PA'	138.73±8.9	108.85±11.68	<0.001
Tricuspid PA'	117.14±7.7	98.54±12.4	<0.001
ΔintraA	21.51±3.8	10.46±6.23	<0.001
ΔinterA	31.67±4.3	22.08±9.3	<0.001
Heart rate	77.69±9.53	75.34±14.82	0.67
Pmax, msn	109.20±5.84	95.94±4.77	<0.001
Pmin, msn	64.78±2.11	62.38±1.43	<0.001
PWD, msn	43.79±4.94	33.5±3.02	<0.001
PR, msn	164.89±26.71	161.88±19.84	0.42

Continuous data are expressed as mean±SD.
EMCD - electromechanical conduction delay; HF - heart failure; PWD - P wave dispersion; ΔinterA - interatrial electro-mechanical delay; ΔintraA - intraatrial electro mechanical delay

Table 4. Threshold value of inter-atrial conduction time and atrial EMCD

Time, msn	Threshold value
PA time	102.5
Lateral PA'	107.5
Septal PA'	127.5
Tricuspid PA'	137.5
ΔintraA	17.5
ΔinterA	27.5

ΔinterA - interatrial electromechanical delay; ΔintraA - intraatrial electromechanical delay; EMCD - electromechanical conduction delay

the 12-month follow-up period, and it was statistically significant ($p_{\Delta\text{intraA}}=0.025$, $p_{\Delta\text{interA}}=0.017$).

Discussion

In the present study, we demonstrated that TDI echocardiography, intra- and inter-atrial EMCD are significantly related in the presence or absence of systolic HF. Furthermore, LA mechanical functions were impaired in patients with HF. TDI echocardiography was used as a non-invasive technique to evaluate atrial conduction times because of its high temporal and spatial resolution (18). Using this method, we observed a significant increase in LA, intra-atrial and inter-atrial dyssynchrony in patients with systolic HF compared with patients without HF. Dyssynchrony was related to LAEF and LVEF. We found that ΔintraA and ΔinterA detected by tissue Doppler echocardiography were higher in patients with systolic HF than in healthy control subjects. In addition, lateral PA', mitral septal PA', and tricuspid PA' were higher in the HF group. The inter-atrial conduction time was also higher in the HF group, but there was no statistically significant difference between the 2 groups. Sanders et al. (19) found a prolonged total

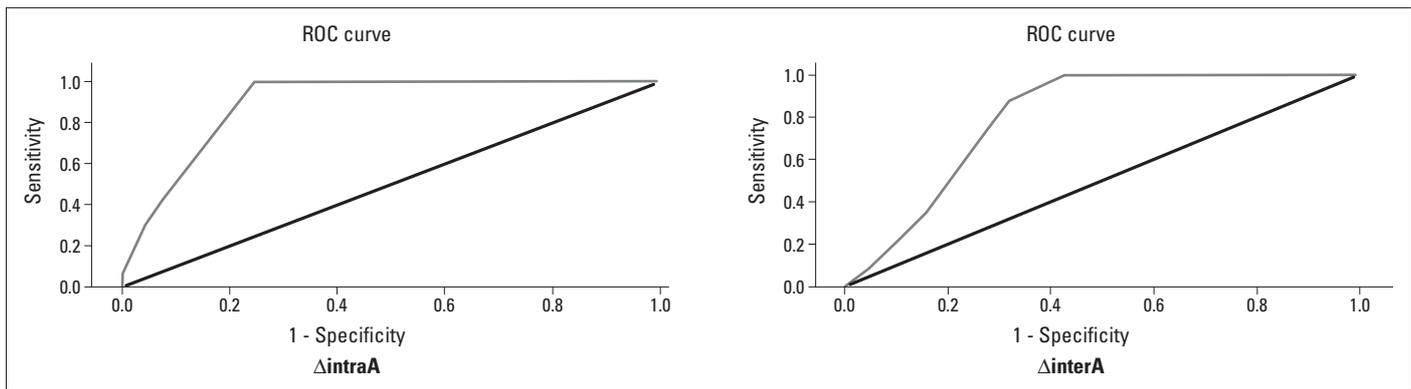


Figure 4. ROC curve analysis of intra- and inter-atrial EMCD time

right atrium (RA) activation time in patients with HF compared with the control group. Furthermore, Van Beeumen et al. (8) have shown slowing of conduction in patients with HF independent of the atrial tissue mass or surface area. These observations appear to be related to the structural remodeling of the atria, characterized by the occurrence of fragmented atrial activity, regions of low voltage, atrial conduction slowing, and interstitial atrial fibrosis. Matrix metalloproteinase and angiotensin II levels appear to be key factors in atrial remodeling and systolic HF. Moreover, changes in atrial histopathology appear to be dependent on the severity of hemodynamic overload of the atria (20, 21).

Atrial EMCD has been evaluated with TDI by measuring the temporal relation between atrial myocardial regional motions and electrocardiographic P waves in some cardiac disorders such as paroxysmal atrial fibrillation (PAF) and congestive HF. Omi et al. (22) showed that patients with PAF had longer tricuspid lateral annular atrial EMCD and bigger right atrial dimensions than the control group. However, Pala et al. (23) found that while mitral septal annular EMCD and mitral lateral annular EMCD were significantly prolonged in the non-ischemic dilated cardiomyopathy group rather than in the control group, there was no significant prolongation of tricuspid lateral annular EMCD (24). Furthermore, Özer et al. (7) found that atrial EMCD in patients with mitral stenosis also revealed a significant positive correlation between prolonged mitral lateral annular EMCD and an increase in the LA size. Similarly, Çağlar et al. (25) observed a positive correlation between the right atrial area and tricuspid lateral annular EMCD and LA EMCD in patients with chronic obstructive pulmonary disease. This indicates atrial size as an important factor for determining atrial EMCD. In a recently published study, De Vos et al. (14) followed 249 people with SR for nearly 2 years; 15 of them (6%) developed AF and showed prolonged atrial EMCD as compared with those who did not develop AF.

In purpose of these findings, prolonged atrial EMCD by TDI is considered as a predictor for the development of AF. Öztürk et al. (26) found prolonged intra- and inter-atrial EMCD in patients with overt hypothyroidism in their study. As a consequence, prolonged intra- and inter-atrial EMCD may be associated with an increased risk of arrhythmias in patients with overt hypothyroidism. However, in our study, we excluded all patients with hyper- or hypothyroidism.

In this study, we found that patients with systolic HF had a higher heart rate, NYHA class, coronary artery disease, and history of using β -blockers, ACE-I/ARB, diuretics, and statins. LAEF was also significantly lower in the patient group. LA volume and LV diastolic and systolic diameters were greater in the HF group than in the control group. In addition, lateral, septal, and tricuspid S, E', and A' waves were significantly lower, whereas the septal annular E/E' ratio was significantly higher in the HF group. This indicates that there was a correlation between the enlargement of the left atrium or left ventricle and intra- and inter-atrial EMCD. Moreover, we found that P wave duration and dispersions were significantly higher in the HF group than in the control group. In contrast, systolic blood pressure was lower in the systolic HF group despite high endothelin and angiotensin II levels in patients with HF.

In our study, patients with systolic HF had prolonged inter-atrial conduction time (PA) measurements compared with patients in the control group. Lateral PA', septal PA', tricuspid PA', Δ interA, and Δ intraA EMCD was significantly higher in the HF group. In addition, according to our study, intra- and inter-atrial electromechanical conduction times were prolonged in the HF group compared with the control group. Patients with prolonged atrial conduction time in the HF group had worse functional class. Functional class increased during the 12-month follow-up period. In addition, patients with prolonged atrial conduction time in the HF group had higher morbidity during the 12-month follow-up period. However, neither an increase in morbidity nor an increase in the functional class was statistically significant. In this study, mortality was found to be higher in the systolic HF group with prolonged atrial conduction time during the 12-month follow-up period.

Study limitations

The most important limitation of our study was the small number of patients and a short follow-up period. A computer-assisted accounting system was not used in the calculation of the atrial conduction time and the observer variability was not evaluated. Using diuretics may affect LA volume and atrial function.

Table 5. Correlation of PWD and intra- and inter-atrial dyssynchrony with other parameters

Variables	Pwd		Intra-atrial dyssynchrony		Inter-atrial dyssynchrony	
	<i>R</i>	<i>P</i>	<i>R</i>	<i>P</i>	<i>R</i>	<i>P</i>
HR	0.109	0.395	0.007	0.959	0.017	0.892
P max	0.917	0.001	0.571	0.001	0.604	0.001
P min	0.431	0.001	0.186	0.144	0.243	0.055
PR interval	0.050	0.699	-0.070	0.585	0.022	0.867
Pwd	-	-	0.606	0.001	0.631	0.001
LVEF	-0.277	0.028	-0.284	0.024	-0.251	0.047
LAEF	-0.100	0.435	0.032	0.806	-0.058	0.651
Intra-atrial dyssynchrony	0.606	0.001	-	-	0.835	0.001
Inter-atrial dyssynchrony	0.631	0.001	0.835	0.001	-	-
LA max	0.142	0.268	0.187	0.143	0.152	0.235
LA min	-0.017	0.894	0.065	0.612	0.064	0.620
LA diastolic volume	0.313	0.013	0.484	0.001	0.544	0.001
LA systolic volume	0.332	0.008	0.440	0.001	0.547	0.001
QRS	0.079	0.536	0.161	0.208	0.098	0.445
QTc	-0.002	0.985	0.124	0.335	0.192	0.133
E/E' septal annulus	0.066	0.609	0.056	0.665	0.046	0.723
Tricuspid PA	0.016	0.931	0.072	0.557	0.095	0.460
Septal PA	0.294	0.019	0.527	0.001	0.469	0.001
Lateral PA	0.330	0.008	0.480	0.001	0.583	0.001
PA time	0.454	0.001	0.589	0.001	0.462	0.001
LVDD	0.061	0.637	0.226	0.075	0.281	0.026
LVSD	0.041	0.747	0.254	0.045	0.253	0.045
RVS	0.059	0.647	0.012	0.924	0.004	0.976
RVE	-0.064	0.617	-0.081	0.526	-0.136	0.288
RVA	-0.036	0.778	0.032	0.805	-0.092	0.473
Septal S	-0.060	0.639	0.105	0.414	0.023	0.856
Septal E	-0.100	0.435	0.055	0.671	-0.148	0.249
Septal A	-0.039	0.759	0.092	0.474	-0.039	0.761
Lateral S	-0.043	0.734	-0.053	0.679	-0.045	0.727
Lateral E	0.080	0.531	0.270	0.033	0.199	0.118
Lateral A	-0.011	0.932	-0.010	0.938	-0.074	0.563
E	0.057	0.655	0.089	0.487	-0.051	0.692
A	0.069	0.591	0.045	0.726	-0.046	0.720

Continuous data are expressed as mean±SD.

HR - heart rate; LAEF - left atrial ejection fraction; LVDD - left ventricular diastolic diameter; LVSD - left ventricular systolic diameter; LVEF - left ventricular ejection fraction; PA time-onset of P-wave on surface ECG to the peak of trans mitral A-wave (PA - interatrial conduction time); Pmin - minimum P wave duration; Pmax - maximum wave duration; Pwd - P wave dispersion; QTc - corrected QT

Conclusions

The atrial conduction time delay was seen more frequently in patients with systolic HF than in healthy subjects. This indicates an increased atrial arrhythmia risk in patients with systolic HF. In this study, we found a relationship between prolonged atrial conduc-

tion time and increased functional class, morbidity, and mortality in systolic HF patients. As a result, we believe that intra- and inter-atrial conduction time can be used in the follow-up of patients with systolic HF and determination of the functional class and prognosis. However, our findings need to be supported by other studies including a large number of patients and a long-term follow-up period.

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