P-wave dispersion and its relationship to aortic stiffness in patients with acute myocardial infarction after cardiac rehabilitation

Rezzan Deniz Acar[1], Mustafa Bulut[1], Sunay Ergün[2], Mahmut Yesin[1], Bilal Boztosun[1], Mustafa Akçakoyun[1]

Original Article

Abstract

BACKGROUND: The aim of our study was to investigate the P-wave dispersion from standard electrocardiograms (ECGs) in patients with acute myocardial infarction (AMI) after cardiac rehabilitation (CR) and determine its relation to arterial stiffness.

METHODS: This is a prospective study included 33 patients with AMI and successfully re-vascularized by percutaneous coronary intervention (PCI) underwent CR. Left ventricular ejection fraction (LVEF) was measured by biplane Simpson's method. Left atrium (LA) volume was calculated. The maximum and minimum durations of P-waves (Pmax and Pmin, respectively) were detected, and the difference between Pmax and Pmin was defined as P-wave dispersion (Pd = Pmax–Pmin). Aortic elasticity parameters were measured.

RESULTS: LVEF was better after CR. The systolic and diastolic blood pressures decreased after CR, these differences were statistically significant. With exercise training, LA volume decreased significantly. Pmax and Pd values were significantly shorter after the CR program. The maximum and minimum P-waves and P-wave dispersion after CR were 97 ± 6 ms, 53 ± 5 ms, and 44 ± 5 ms, respectively. Aortic strain and distensibility increased and aortic stiffness index was decreased significantly. Aortic stiffness index was 0.4 ± 0.2 versus 0.3 ± 0.2, P = 0.001. Aortic stiffness and left atrial volume showed a moderate positive correlation with P-wave dispersion (r = 0.52, P = 0.005; r = 0.64, P < 0.001, respectively).

CONCLUSION: This study showed decreased arterial stiffness indexes in AMI patient's participated CR, with a significant relationship between the electromechanical properties of the LA that may raise a question of the preventive effect of CR from atrial fibrillation and stroke in patients with acute myocardial infarction.

Keywords: Cardiac Rehabilitation, P-Wave Dispersion, Aortic Stiffness, Acute Myocardial Infarction

Date of submission: 29 Jan 2013, ***Date of acceptance:*** 27 Apr 2014

Introduction

Increases in the P-wave dispersion from standard electrocardiograms (ECGs) with subsequent development of atrial fibrillation (AF) have been identified in patients with a wide range of cardiovascular disorders.[1] AF is the most common arrhythmia treated in clinical practice and approximately 33% of arrhythmias related hospitalizations are for AF. It is associated with a fivefold increase in the risk of stroke and two-fold increase in the risk of all-cause mortality.[2] The assessment of left atrium (LA) mechanical and electromechanical functions are accepted as risk factors of AF. Among the noninvasive and invasive methods to evaluate the inter-atrial conduction, the basic and the most frequently used one is the electrocardiographic P-wave dispersion.[3]

However, prolonged inter-atrial conduction time (IACT) is associated with the development of atrial fibrillation and abnormal LA function.[4-6] The prolongation of electromechanical delay (EMD) and the inhomogeneous propagation of sinus impulses are well-known electrophysiological characteristics of the atria prone to fibrillation.[7] IACT can be measured by two-dimensional (2D)-Doppler echocardiography, including tissue Doppler imaging. IACT measured by 2D-Doppler echocardiography and its association with indices of

1- Department of Cardiology, Kartal Kosuyolu Education and Research Hospital, Istanbul, Turkey
2- Department of Physical Therapy and Rehabilitation, Kartal Kosuyolu Education and Research Hospital, Istanbul, Turkey
Correspondence to: Rezzan Deniz Acar, Email: denizacar_1999@yahoo.com

LA function has been reported in a few studies in patients with left ventricular (LV) systolic dysfunction.[8-11] Deniz et al. compared the tissue Doppler echocardiography and electrophysiological study in the measurement of atrial conduction times and found a moderate correlation between intra-left atrial conduction time by echocardiography (ILCT-echo) and ILCT by electrophysiology (ILCT-eps), which means that tissue Doppler echocardiography can be used to evaluate atrial conduction time.[12]

In recent times, aortic stiffness was found to influence the diameter of the LA and expose the patient to embolic stroke by increasing their risk of atrial fibrillation (AF). Previous studies revealed an inverse relationship between aortic distensibility and cardiovascular risk factors.[13-15]

Benefits of cardiac rehabilitation (CR) for patients with cardiovascular diseases have been shown by many clinical investigators.[16-18] The effect of CR on total mortality was independent of coronary heart disease diagnosis, type of CR, dose of exercise intervention and length of follow-up.[19] Comprehensive CR program includes not only exercise training, also diet counselling, weight control management, lipid management, smoking cessation, blood pressure monitoring, and psychosocial management that aims to optimize cardiovascular risk reduction.

The aim of our study was to investigate P-wave dispersion from standard ECGs and determine its relation to arterial stiffness in patients with acute myocardial infarction (AMI) after CR.

Materials and Methods

Study design

This is a prospective study included 33 patients with AMI and successfully re-vascularized by percutaneous coronary intervention (PCI) underwent CR between October 2012 and April 2013. Each patient had performed intensive out-patient CR program (also known as Phase II CR) for 5 times a week during 6 weeks at the CR center of our education and research hospital. All patients were asymptomatic and had been in a clinically stable condition after discharge period. Lower-risk patients following an acute cardiac event enrolled this study. High risk patients with severe residual angina, severe ischemia, poorly controlled hypertension, hypertensive or any hypotensive systolic blood pressure response to exercise and unstable concomitant medical problems (diabetes prone to hypoglycemia) were excluded from the study. During the training, ECGs were continuously telemonitored. Typical training in CR started with 5 min warm up, followed with 20 min aerobic training and 10-15 min cool down.

This study complied with the Declaration of Helsinki, was approved by the local Ethical Committee and written consent was obtained from each patient before CR.

Electrocardiographic evaluation of atrial conduction

Standard ECG were taken from all patients with sweeping rate of 50 mm/s and amplitude of 1 mV/cm. P-wave durations was measured manually in all simultaneously recorded 12 leads of the surface ECG. The mean P-wave duration for at least three complexes was calculated in each lead. The onset of the P-wave was defined as the point of first visible upward slope from baseline for positive waveforms, and as the point of first downward slope from baseline for negative waveforms. The return to the baseline was considered the end of the P-wave. The Pmax measured in any of the 12 leads of the surface ECG was used as the longest atrial conduction time. The maximum and minimum durations of P-waves (Pmax and Pmin, respectively) were detected, and the difference between Pmax and Pmin was defined as P-wave dispersion (Pd = Pmax–Pmin).

Echocardiography

A Vivid 7 ultrasound system (GE Vingmed Ultrasound, Horten, Norway) was used, and all images and measurements were acquired from the standard views, according to the guidelines of the American Society of Echocardiography. LV end-diastolic volume was measured, and left ventricular ejection fraction (LVEF) was calculated by the Simpson method by apical four-chamber view.

LA maximum anterio-posterior diameter (D1) was measured in the parasternal long-axis views. LA superior-inferior diameter (D2) was measured from the mitral annular plane to the posterior wall of the LA, and medial-lateral diameter (D3) was measured in the apical 4-chamber view (LA volume was calculated with the formula; $D1 \times D2 \times D3 \times 0.523$).

Tissue Doppler echocardiography was performed by transducer frequencies of 3.5-4.0 MHz, adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15-20 cm/s and using the minimal optimal gain. In the apical four-chamber view, the pulsed Doppler sample volume was placed in order at the level of LV lateral mitral annulus, septal mitral annulus, and right ventricular tricuspid annulus. Atrial electromechanical coupling (PA), the time interval from the onset of the P-wave

on the surface ECG to the beginning of the late diastolic wave (Am); was obtained from the lateral mitral annulus (PAlat), septal mitral annulus (PAsep), and tricuspid annulus (PAtricus). The difference between PAlat and PAtricus was defined as the inter-atrial EMD, while the difference between PAsep and PAtricus was defined as the intra-atrial EMD. Every effort was made to align the pulsed wave cursor that the Doppler angle of incidence was as close to 0 as possible to the direction of these walls. All participants in our study also showed no clinical evidence of pulmonary hypertension, and systolic pulmonary artery pressure estimated by Doppler echocardiography was < 35 mmHg.

Systolic and diastolic ascending aortic diameters were measured on M-mode tracings at 3 cm above the aortic valve. An average of three beats was analyzed M-mode traces were recorded at a speed of 50 mm/s and Doppler signals were recorded at a speed of 100 mm/s. Simultaneous electrocardiographic recordings were also taken. Systolic diameter was measured at the maximal anterior motion of the aorta, while diastolic diameter was measured at the peak of the QRS complex on the simultaneous ECG.

Aortic elasticity parameters were calculated using the following formulas:

Aortic strain (%) = (aortic systolic–diastolic diameter) × 100/aortic diastolic diameter

Aortic stiffness index = (systolic/diastolic blood pressure)/aortic strain

Aortic distensibility ($cm^2/dyne/10^6$) = 2 × aortic strain/ (systolic–diastolic blood pressure).

Statistical analyses

All values were expressed as a mean ± SD. Data were analyzed using the SPSS for Windows (version 15.0, SPSS Inc., Chicago, IL, USA) and considered as significant if $P < 0.05$. Statistical analysis was performed using Student's t-test. Linear regression and Pearson correlation analysis were used for correlation of variables of interest. P-value < 0.05 was considered to indicate statistical significance.

Results

Thirty-three participants in sinus rhythm after AMI and successfully revascularization by PCI were recruited into the study. The mean age of the patients was 57 years. Infarct related artery was left anterior descending in 13 patients, circumflex coronary artery in 6 patients, and right coronary artery in 14 patients. No changes were done in medical therapy of the patients during the follow-up, and there were no complications or arrhythmia in subjects during the study period. Patient demographics and clinical characteristics are presented in table 1.

Table 1. Patient demographics and clinical characteristics (n = 33)

Patients	Value
Age (year)	57 ± 7 (mean ± SD)
Gender	
Male, n (%)	27 (81)
Female, n (%)	6 (19)
Diabetes, n (%)	13 (39)
Hypertension, n (%)	19 (57)
Hyperlipidemia, n (%)	14 (42)
Smoking, n (%)	15 (45)
IRA	
LAD	13 (40)
CX	6 (18)
RCA	14 (42)

SD: Standard deviation; IRA: Infarct related artery; LAD: Left anterior descending; CX: Circumferential coronary artery; RCA: Right coronary artery

LVEF was improved with CR ($P < 0.001$). In comparison with the baseline, Pmax, and Pd values were significantly shorter after the CR program ($P = 0.001$ and $P = 0.019$, respectively). Furthermore, mitral lateral EMD (PAlat), septum EMD (PAsep), and tricuspid EMD (PAtricus) were decreased with the CR. Calculated inter-atrial and intra-atrial EMD were significantly lower after the CR compared to the baseline (21 ± 5 vs. 18 ± 4 ms , $P < 0.001$; 6 ± 2 vs. 4 ± 2 ms, $P < 0.001$). LA volume was decreased with exercise based CR ($P < 0.001$). IACT and P-wave dispersion showed a moderate positive correlation with left atrial volume ($r = 0.591$; $P < 0.001$, $r = 0.615$; $P < 0.001$, respectively). P-wave measurements and atrial EMD parameters at baseline and after the CR are set out in table 2.

The systolic and diastolic blood pressures decreased after CR, these differences were statistically significant. Aortic strain and distensibility increased and aortic stiffness index was decreased significantly after CR ($P = 0.001$). Aortic stiffness showed a moderate positive correlation with P-wave dispersion ($r = 0.52$; $P = 0.005$). Aortic elasticity parameters and blood pressure measurements of patients before and after CR are represented in table 3.

Table 2. Electromechanical delay and P-wave dispersion before and after cardiac rehabilitation

Echocardiographic and ECG parameters	Before CR (mean ± SD)	After CR (mean ± SD)	P
LVEF (ejection fraction)	51.4 ± 9.9	54.6 ± 9.3	< 0.001
LA volume (ml)	34.0 ± 9.7	33.0 ± 8.5	< 0.001
Mitral lateral (PAlat) EMD (ms)	62.0 ± 7.7	57.0 ± 7.8	0.001
Septum (PAsep) EMD (ms)	47.0 ± 10.0	43.0 ± 8.0	0.049
Tricuspid (PAtricus) EMD (ms)	40.0 ± 9.0	39.0 ± 7.0	0.418
Intra-atrial EMD (ms)	6.0 ± 2.0	4.0 ± 2.0	< 0.001
Inter-atrial EMD (ms)	21.0 ± 5.0	18.0 ± 4.0	< 0.001
Pmax (ms)	102.0 ± 8.0	97.0 ± 6.0	0.001
Pmin (ms)	55.0 ± 5.0	53.0 ± 5.0	0.044
Pd (ms)	46.0 ± 5.0	44.0 ± 5.0	0.019

ECG: Electrocardiograms; CR: Cardiac rehabilitation; SD: Standard deviation; LVEF: Left ventricular ejection fraction; LA: Left atrium; PAlat: Lateral mitral annulus; PAsep: Septal mitral annulus; PAtricus: Tricuspid annulus; EMD: Electromechanical delay; Pmax: Maximum P-wave duration; Pmin: Minimum P-wave duration; Pd: P-wave dispersion

Table 3. The aortic elasticity parameters and blood pressure of the individuals before and after cardiac rehabilitation

Parameters	Before CR (mean ± SD)	After CR (mean ± SD)	P
Systolic BP (mmHg)	132.00 ± 14.00	123.00 ± 13.00	< 0.001
Diastolic BP (mmHg)	80.00 ± 11.00	75.00 ± 10.00	< 0.001
Aortic strain (%)	4.00 ± 2.00	6.00 ± 3.00	< 0.001
Aortic stiffness index	0.40 ± 0.20	0.30 ± 0.20	< 0.001
Aortic distensibility (cm2/dyne/106)	0.19 ± 0.10	0.28 ± 0.10	< 0.001

CR: Cardiac rehabilitation; SD: Standard deviation; BP: Blood pressure

Discussion

The patients in the present study were asymptomatic and did not have a history of AF. To the best of our knowledge, the relative contribution of arterial stiffness to the P-wave dispersion from standard ECGs in patients after CR as a risk of developing AF has not been evaluated. Increased P-wave dispersion has been reported in various clinical settings, including coronary artery disease, hypertension, rheumatic mitral stenosis, mitral annular calcification and hypertrophic cardiomyopathy.[20-24] The mechanism of P-wave dispersion prolongation in these patients is thought to be due to structural and electrophysiological changes in the atrial myocardium. Chronic elevation of LV filling pressures may cause atrial fibrosis contributing to the prolongation of atrial activation time.[25] Several studies have suggested that increased P-wave duration may be associated with myocardial ischemia, altered autonomic control, LV diastolic dysfunction, enlarged left atrial dimension, elevated left atrial pressure and fibrosis, and aortic elasticity.[26-32] Also, Emiroglu et al. demonstrated that prolonged EMD and Pd found in hypertensive patients could be related with increased incidence of atrial fibrillation.[33] After the CR program with exercise in this study, left atrial volume was improved, and the ECG-derived Pmax and Pd values were also decreased compared to the baseline, which may suggests the decrease in the incidence of long-term AF risk.

An increase in aortic stiffness may increase the risk of stroke through several mechanisms such as an increase in central pulse pressure or an increase in carotid intima-media thickness, promoting the development of atherosclerotic lesions and thus the likelihood of plaque rupture.[34-36] Potential mechanisms include the possibility that increased arterial stiffness predisposes to neurohormonal activation[37] or a generalized cardiovascular inflammatory response,[38] which, in turn may contribute to the development of AF.[39] Cross-sectional studies show strong correlations between elevated C-reactive protein (CRP) and aortic stiffness. Exercise training is associated with reduced CRP levels, which suggests that exercise training has anti-inflammatory effects on atherosclerosis therefore, aortic stiffness. Furthermore, aerobic exercise training regulates the neurohormonal activation by reducing sympathetic and enhancing parasympathetic (vagal) activity, as evidenced by increased heart rate variability and reduced baroreceptor sensitivity which suggests the decrease in aortic stiffness.[40]

As suggested by Gosse and Safar in view of a common embryological origin, the aorta may be considered along with the LA and ventricle as the third chamber of the left sided cardiac pump

transforming the systolic output of the left ventricle into a continuous flow.[41] Our findings support this theory; after CR in patients with AMI as the aortic stiffness and LA volume decreases, therefore EMD decreases as well.

The favorable impact of CR on aortic stiffness may contribute to the reduction of the extent of atherosclerosis, but also it may prevent the risk of the occurrence of AF. This study suggests that improvement in arterial stiffness may contribute to decrease the LA electromechanical dysfunction, namely, the risk of AF and stroke.

Limitations

The number of patients with CR reported in this investigation was small and female subjects were too few. Also, follow-up after CR in terms of the development of AF was lacking. In addition, we did not perform continuous Holter recordings; we could not be sure about clinically silent paroxysmal AF episodes. This study did not directly address the issue of a link between stiffness and AF and rather used P-wave dispersion as a surrogate marker of the risk of AF. Studies with larger sample size with group analysis of CR would be beneficial in further evaluating the role of CR as a protector from the risk of AF and stroke.

Conclusion

The present study demonstrates the decreased arterial stiffness indexes in AMI patient's participated CR, with a significant relationship between the electromechanical properties of the LA. Therefore, this study illustrates the importance of CR and reopens the question of a new potential benefit of CR in the prevention of AF and stroke in patients with AMI beyond increase in physiological well-being of the individuals.

Conflict of Interests

Authors have no conflict of interests.

References

1. Laurent S, Boutouyrie P, Lacolley P. Structural and genetic bases of arterial stiffness. Hypertension 2005; 45(6): 1050-5.
2. Lloyd-Jones D, Adams R, Carnethon M, De SG, Ferguson TB, Flegal K, et al. Heart disease and stroke statistics-2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009; 119(3): 480-6.
3. Daubert JC, Pavin D, Jauvert G, Mabo P. Intra- and interatrial conduction delay: implications for cardiac pacing. Pacing Clin Electrophysiol 2004; 27(4): 507-25.
4. Agarwal YK, Aronow WS, Levy JA, Spodick DH. Association of interatrial block with development of atrial fibrillation. Am J Cardiol 2003; 91(7): 882.
5. Goyal SB, Spodick DH. Electromechanical dysfunction of the left atrium associated with interatrial block. Am Heart J 2001; 142(5): 823-7.
6. Ramsaran EK, Spodick DH. Electromechanical delay in the left atrium as a consequence of interatrial block. Am J Cardiol 1996; 77(12): 1132-4.
7. 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.
8. Porciani MC, Sabini A, Colella A, Michelucci A, Musilli N, Pieragnoli P, et al. Interatrial septum pacing avoids the adverse effect of interatrial delay in biventricular pacing: an echo-Doppler evaluation. Europace 2002; 4(3): 317-24.
9. Rein AJ, O'Donnell CP, Colan SD, Marx GR. Tissue velocity Doppler assessment of atrial and ventricular electromechanical coupling and atrioventricular time intervals in normal subjects. Am J Cardiol 2003; 92(11): 1347-50.
10. Merckx KL, De Vos CB, Palmans A, Habets J, Cheriex EC, Crijns HJ, et al. Atrial activation time determined by transthoracic Doppler tissue imaging can be used as an estimate of the total duration of atrial electrical activation. J Am Soc Echocardiogr 2005; 18(9): 940-4.
11. Van Beeumen K, Duytschaever M, Tavernier R, Van de V, de Sutter J. Intra- and interatrial asynchrony in patients with heart failure. Am J Cardiol 2007; 99(1): 79-83.
12. Deniz A, Sahiner L, Aytemir K, Kaya B, Kabakci G, Tokgozoglu L, et al. Tissue Doppler echocardiography can be a useful technique to evaluate atrial conduction time. Cardiol J 2012; 19(5): 487-93.
13. Tentolouris N, Liatis S, Moyssakis I, Tsapogas P, Psallas M, Diakoumopoulou E, et al. Aortic distensibility is reduced in subjects with type 2 diabetes and cardiac autonomic neuropathy. Eur J Clin Invest 2003; 33(12): 1075-83.
14. Cruickshank K, Riste L, Anderson SG, Wright JS, Dunn G, Gosling RG. Aortic pulse-wave velocity and its relationship to mortality in diabetes and glucose intolerance: an integrated index of vascular function? Circulation 2002; 106(16): 2085-90.
15. Kingwell BA, Waddell TK, Medley TL, Cameron JD, Dart AM. Large artery stiffness predicts ischemic threshold in patients with coronary artery disease. J Am Coll Cardiol 2002; 40(4): 773-9.
16. Leon AS, Franklin BA, Costa F, Balady GJ, Berra KA, Stewart KJ, et al. Cardiac rehabilitation and

secondary prevention of coronary heart disease: an American Heart Association scientific statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity), in collaboration with the American association of Cardiovascular and Pulmonary Rehabilitation. Circulation 2005; 111(3): 369-76.

17. Witt BJ, Jacobsen SJ, Weston SA, Killian JM, Meverden RA, Allison TG, et al. Cardiac rehabilitation after myocardial infarction in the community. J Am Coll Cardiol 2004; 44(5): 988-96.

18. Brown RA. Rehabilitation of patients with cardiovascular diseases. Report of a who expert committee. World Health Organ Tech Rep Ser 1964; 270: 3-46.

19. Taylor RS, Brown A, Ebrahim S, Jolliffe J, Noorani H, Rees K, et al. Exercise-based rehabilitation for patients with coronary heart disease: systematic review and meta-analysis of randomized controlled trials. Am J Med 2004; 116(10): 682-92.

20. Dilaveris PE, Andrikopoulos GK, Metaxas G, Richter DJ, Avgeropoulou CK, Androulakis AM, et al. Effects of ischemia on P wave dispersion and maximum P wave duration during spontaneous anginal episodes. Pacing Clin Electrophysiol 1999; 22(11): 1640-7.

21. Dilaveris PE, Gialafos EJ, Chrissos D, Andrikopoulos GK, Richter DJ, Lazaki E, et al. Detection of hypertensive patients at risk for paroxysmal atrial fibrillation during sinus rhythm by computer-assisted P wave analysis. J Hypertens 1999; 17(10): 1463-70.

22. Ozer N, Yavuz B, Can I, Atalar E, Aksoyek S, Ovunc K, et al. Doppler tissue evaluation of intra-atrial and interatrial electromechanical delay and comparison with P-wave dispersion in patients with mitral stenosis. J Am Soc Echocardiogr 2005; 18(9): 945-8.

23. Pekdemir H, Cansel M, Yagmur J, Acikgoz N, Ermis N, Kurtoglu E, et al. Assessment of atrial conduction time by tissue Doppler echocardiography and P-wave dispersion in patients with mitral annulus calcification. J Electrocardiol 2010; 43(4): 339-43.

24. Ozdemir O, Soylu M, Demir AD, Topaloglu S, Alyan O, Turhan H, et al. P-wave durations as a predictor for atrial fibrillation development in patients with hypertrophic cardiomyopathy. Int J Cardiol 2004; 94(2-3): 163-6.

25. Korantzopoulos P, Kolettis T, Siogas K, Goudevenos J. Atrial fibrillation and electrical remodeling: the potential role of inflammation and oxidative stress. Med Sci Monit 2003; 9(9): RA225-RA229.

26. Myrianthefs MM, Shandling AH, Startt-Selvester RH, Bernstein SB, Crump R, Lorenz LM, et al. Analysis of the signal-averaged P-wave duration in patients with percutaneous coronary angioplasty-induced myocardial ischemia. Am J Cardiol 1992; 70(7): 728-32.

27. Cheema AN, Ahmed MW, Kadish AH, Goldberger JJ. Effects of autonomic stimulation and blockade on signal-averaged P wave duration. J Am Coll Cardiol 1995; 26(2): 497-502.

28. Celik T, Yuksel UC, Bugan B, Celik M, Fici F, Iyisoy A, et al. P-wave dispersion and its relationship to aortic elasticity in young prehypertensive patients. Am J Hypertens 2009; 22(12): 1270-5.

29. Gunduz H, Binak E, Arinc H, Akdemir R, Ozhan H, Tamer A, et al. The relationship between P wave dispersion and diastolic dysfunction. Tex Heart Inst J 2005; 32(2): 163-7.

30. Shettigar UR, Barry WH, Hultgren HN. P wave analysis in ischaemic heart disease. An echocardiographic, haemodynamic, and angiographic assessment. Br Heart J 1977; 39(8): 894-9.

31. Waggoner AD, Adyanthaya AV, Quinones MA, Alexander JK. Left atrial enlargement. Echocardiographic assessment of electrocardiographic criteria. Circulation 1976; 54(4): 553-7.

32. Cha YM, Dzeja PP, Shen WK, Jahangir A, Hart CY, Terzic A, et al. Failing atrial myocardium: energetic deficits accompany structural remodeling and electrical instability. Am J Physiol Heart Circ Physiol 2003; 284(4): H1313-H1320.

33. Emiroglu MY, Bulut M, Sahin M, Acar G, Akcakoyun M, Kargin R, et al. Assessment of atrial conduction time in patients with essential hypertension. J Electrocardiol 2011; 44(2): 251-6.

34. Boutouyrie P, Bussy C, Lacolley P, Girerd X, Laloux B, Laurent S. Association between local pulse pressure, mean blood pressure, and large-artery remodeling. Circulation 1999; 100(13): 1387-93.

35. Tomonori T, Keiko S, Shinkichi H, Yoji N, Akira T. Carotid atherosclerosis and arterial peripheral pulse wave velocity in cerebral thrombosis. J Clin Neurosci 2006; 13(1): 45-9.

36. Lovett JK, Howard SC, Rothwell PM. Pulse pressure is independently associated with carotid plaque ulceration. J Hypertens 2003; 21(9): 1669-76.

37. Kato J, Kitamura K, Uemura T, Kuwasako K, Kita T, Kangawa K, et al. Plasma levels of adrenomedullin and atrial and brain natriuretic peptides in the general population: their relations to age and pulse pressure. Hypertens Res 2002; 25(6): 887-92.

38. Yasmin, McEniery CM, Wallace S, Mackenzie IS,

Cockcroft JR, Wilkinson IB. C-reactive protein is associated with arterial stiffness in apparently healthy individuals. Arterioscler Thromb Vasc Biol 2004; 24(5): 969-74.

39. Aviles RJ, Martin DO, Apperson-Hansen C, Houghtaling PL, Rautaharju P, Kronmal RA, et al. Inflammation as a risk factor for atrial fibrillation. Circulation 2003; 108(24): 3006-10.

40. Murry CE, Jennings RB, Reimer KA. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation 1986; 74(5): 1124-36.

41. Gosse Ph, Safar M. The Heart and the Macro- and Microcirculation in Hypertension. In: Macro- and Microcirculation in Hypertension. In: Safar M, Editor. Macro- and Microcirculation in Hypertension. Philadelphia, PA: Lippincott Williams & Wilkins; 2005. p. 107-17.

How to cite this article: Deniz Acar R, Bulut M, Ergün S, Yesin M, Boztosun B, Akçakoyun M. **P-wave dispersion and its relationship to aortic stiffness in patients with acute myocardial infarction after cardiac rehabilitation.** ARYA Atheroscler 2014; 10(4): 185-91.