P-Wave Morphology, Amplitude, Duration and Dispersion in Atrial Arrhythmias

Osmar Antonio Centurión[1,2,*], Laura Beatriz García[1], Alberto Marecos[1], Judith Torales[2], Karina Scavenius[2], Luis Miño[2] and Orlando Sequeira[2]

[1]*Department of Health Science's Investigation, Sanatorio Metropolitano, Fernando de la Mora, Paraguay*

[2]*Division of Cardiovascular Medicine, Clinic Hospital, Asunción National University-UNA, San Lorenzo, Paraguay*

Abstract: The detailed analysis of the P-wave duration and dispersion by means of conventional electrocardiography with the 12 standard surface leads in the stratification of patients suffering from AF is a recognized universal approach. P-wave dispersion (PWD) has received increasing attention in the field of non-invasive electrophysiology studying atrial arrhythmias and has been examined in a broad range of clinical settings including cardiovascular and non-cardiovascular diseases. It is well accepted that, not only the P-wave duration, but also the P-wave morphology and dispersion have the potential to give information about the anatomical substrate predisposing to AF. Patients with diseased atrial myocardium with fibrotic changes may develop abnormal electrophysiological alterations. Therefore, these atrial anisotropic characteristics may play an important role in creating reentry circuits by causing inhomogeneous and discontinuous propagation of the impulse in the atrial tissue. The altered atrial myocardium may generate unidirectional block, conduction delay and reentrant atrial rhythms. The P-wave of the electrocardiogram may show alterations that can be associated with atrial arrhythmias and AF. PWD is considered a noninvasive electrocardiographic marker for atrial remodeling and predictor for AF. It has been shown that increased P-wave duration and PWD reflect prolongation of intra-atrial and inter-atrial conduction time. In patients prone to develop atrial arrhythmias and AF, PWD reflects prolonged, inhomogeneous and anisotropic distribution of connections between myocardial fibers resulting in discontinuous anisotropic propagation of sinus impulses and atrial conduction. PWD is considered as a sensitive and specific ECG marker and predictor of atrial arrhythmias and paroxysmal AF.

Keywords: P-wave duration and dispersion, Atrial arrhythmias, Atrial fibrillation.

INTRODUCTION

The study and quantitative measurements of the P-wave by means of conventional electrocardiography (ECG) with the 12 standard surface leads in the stratification of patients suffering from atrial fibrillation (AF) is a recognized universal approach. It has been known that increased P-wave duration and P-wave dispersion (PWD) reflect prolongation of intra-atrial, and inter-atrial conduction time, which are well-known electrophysiological characteristics in patients with atrial arrhythmias and especially paroxysmal AF [1-4]. P-wave duration and dispersion has received increasing attention in the field of non-invasive electrophysiology studying atrial arrhythmias and has been examined in a broad range of clinical settings including cardiovascular and non-cardiovascular diseases [5-7]. It is well accepted that, not only the P-wave duration, but also the P-wave morphology and dispersion have the potential to give information about the anatomical substrate predisposing to AF [8-11]. Patients with diseased atrial myocardium with fibrotic changes may develop abnormal electrophysiological alterations [12-17]. Connective tissue surrounding atrial myocytes represents sites where electrical coupling between adjacent cells is altered [18-20]. Therefore, these atrial anisotropic characteristics may play an important role in creating reentry circuits by causing inhomogeneous and discontinuous propagation of the impulse in the atrial tissue [20]. The altered atrial myocardium may generate unidirectional block, conduction delay and reentrant atrial rhythms. The P-wave of the electrocardiogram may show alterations that can be associated with atrial arrhythmias and AF. PWD is considered a noninvasive ECG marker for atrial remodeling and predictor for AF [8-11]. It has been shown that increased P-wave duration and PWD reflect prolongation of atrial conduction time inside the right atrium and between both atria, and the inhomogeneous and discontinuous atrial propagation of sinus impulses [7-12]. Therefore, it is the aim of this manuscript to analyze the relationship of the P-wave characteristics and dispersion and the development of atrial arrhythmias and atrial fibrillation.

DEFINITIONS AND MEASUREMENTS

PWD reflects disturbances of intra-atrial and inter-atrial conduction, and it is defined as the difference between the wider and the narrower P-wave duration recorded from the 12 ECG leads at a paper speed of

*Address correspondence to this author at the Department of Health Science's Investigation, Asuncion National University, Sanatorio Metropolitano, Teniente Ettiene 215 c/ Ruta Mariscal Estigarribia, Fernando de la Mora, Paraguay; Tel: +595021-421423; Fax: +595021-205824;
E-mail: osmarcenturion@hotmail.com

E-ISSN: 2311-052X/18

50 mm/s. The correct measurement of PWD is derived by subtracting the minimum P-wave duration from the maximum in any of the 12 standard surface ECG leads in supine position following 15 min of rest and room temperature and lighting kept constant [8, 9]. The onset of the P-wave is defined as the point of first detectable upward or downward slope from the isoelectric line for positive or negative waveforms, respectively. Return to the isoelectric line is considered as the end of the P-wave. PWD can be calculated by manual measurements with hand-held calipers or computerized methods. Manual measurement with hand-held calipers is performed by increasing the ECG rate to 50 mm/s and the voltage to 1 mV/cm, accompanied by the use of magnification [21]. The normal value of PWD was found to be 29 ± 9 ms, and values greater than 40 ms indicate the presence of heterogeneous electrical activity in different regions of the atrium that might cause AF to develop [8, 9].

P-Wave Duration

Normal value in adults is 60 to 110 ms. The P-wave duration progressively increases through the years. The proper manner to measure the P-wave duration on the electrocardiogram is as follows. The onset is 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 baseline is considered as the end of the P-wave (Figure **1**).

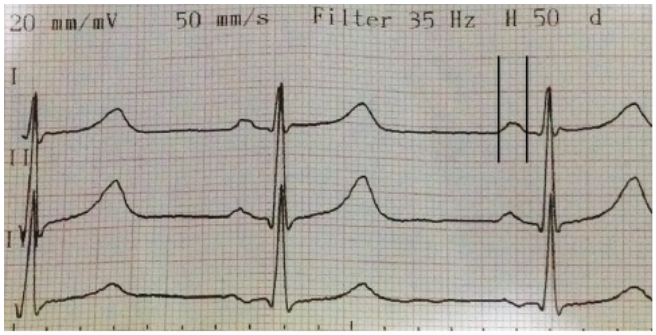

Figure 1: The figure shows measurement of P wave duration on 12-lead surface electrocardiography on lead DI. The paper speed was set at 50 mm/sec and the ECG amplitude at 20 mm/mV. Reproduced with permission from Gudul NE, *et al*. Atrial conduction times and left atrial mechanical functions and their relation with diastolic function in prediabetic patients. Kor J Intern Med. 2016 doi.org/10.3904/kjim.2014. 380.

P-Wave Amplitude

Normal P-wave amplitude is between 5 mm or 0.05 mV to 2.5 mm 0.25 mV. In the precordial leads normal P-wave amplitude is always less than 1.5 mm. The maximal normal value is less than 2.5mm or 0.25 mV.

P-Wave Polarity

The normal P wave polarity is always positive in II, I, aVF and from V3 to V6. It is always negative in aVR and variable in III, aVL and V1eV2. The normal P-wave is typically biphasic in V1, with similar sizes of the positive and negative deflections. Normal P wave axis is considered between 0 and +75 by manually constructing the mean frontal plane electrical P-axis from standard limb leads [22].

P-Wave Morphology

The shape of a P-wave is usually smooth and rounded. It may be notched in the frontal plane in partial interatrial block. It may be broad, greater than 120 ms in left atrial enlargement. It may be bifid in lead II (P mitrale) in marked left atrial dilatation in mitral stenosis [23-26].

Platonov divided the P-wave morphology using orthogonal leads in three types [27]:

Type 1: Upright P-waves in all orthogonal leads which are commonly seen in healthy subjects below 50 years of age.

Type 2: Upright P-waves in leads X and Y, as well as, biphasic in lead Z. These changes are commonly seen in patients with paroxysmal AF, and left atrial enlargement. However, it may also be seen in healthy patients older than 50 years of age.

Type 3: Upright in X but biphasic in leads Y and Z which are seen in advanced, complete or third degree interatrial block, often associated with prolongation of P-wave duration over 120 ms. This P-wave morphology is uncommon in healthy subjects.

P-Wave Dispersion

P-wave dispersion is defined as the difference between the maximum and the minimum P-wave duration recorded from multiple different surfaces ECG leads (Figure **2**). Maximum and minimum P-wave durations are calculated from the standard ECG during sinus rhythm. PWD is derived by subtracting the minimum P-wave duration from the maximum in any of the 12 ECG leads. It has been known that increased P-wave duration and PWD reflect prolongation of intra-atrial and inter-atrial conduction time and the

inhomogeneous atrial propagation of sinus impulses [3], which are well-known electrophysiological characteristics in patients with atrial arrhythmias and especially PAF.

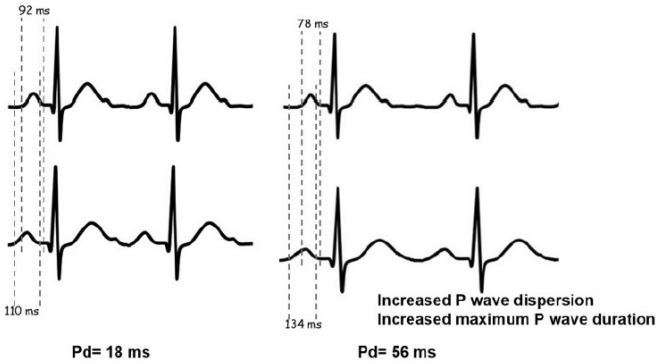

Figure 2: Examples for measurement of P-wave dispersion, minimum and maximum P-wave duration in a normal individual and hypertensive patient. Reprinted with permission from Okutucu S, Aytemir K, Oto A. P-wave dispersion: what we know till now? JRSM Cardiovasc Dis 2016; 5: 1-9.

CLINICAL IMPLICATIONS AND OUTCOME FINDINGS

Several studies evaluated the P-wave alterations especially dispersion changes in the assessment of the risk for atrial fibrillation in patients without organic heart disease, in patients with arterial hypertension, in patients with coronary artery disease, in patients undergoing coronary artery bypass surgery, in patients with congenital heart diseases, as well as in other groups of patients suffering from various cardiac or non-cardiac diseases [28-39]. It was clearly observed that PWD can be utilized to discriminate patients with different kinds of diseases whom are prone to develop paroxysmal AF in the course of their lives [40-50]. Aytemir K *et al.* [8] demonstrated that a P wave maximum duration value of 106 ms differentiated patients with paroxysmal AF from control subjects with a sensitivity of 83%, a specificity of 72%, and a positive predictive accuracy of 79%. In addition, they found that a PWD value greater than 36 ms separated AF patients from control subjects with a sensitivity of 77%, a specificity of 82%, and a positive predictive accuracy of 85% [8]. PWD has shown to have a significant correlation with maximum P-wave duration and a weak, although significant association with age [8]. Very similar findings were reported by Dilaveris *et al.* [40]. They found that PWD was significantly higher in patients with paroxysmal AF than in 40 control subjects. Again, a very similar cutoff value of 40 ms proved to have a sensitivity of 83%, a specificity of 85% and a positive predictive accuracy of 89% for the identification of patients with history of paroxysmal AF [40]. Moreover, during a 12-month follow-up period, the relative risk of an AF recurrence was 2.4 for a PWD value greater than 40 ms [40]. Therefore, PWD was proven to be a sensitive and specific ECG marker for the best separation between patients with history of paroxysmal AF and control subjects [40]. As additional findings of another study, Dilaveris PE and Gialafos JE demonstrated that PWD is a significant predictor of frequent symptomatic AF paroxysms [9]. They also found that PWD has a significantly positive correlation with maximum P-wave duration and negative correlation with minimum P-wave duration [9].

There are interesting data pertaining PWD in AF electrical cardioversion. Boriani *et al.* [41] investigated the association of different PWD values and short-term vs late AF recurrence after electrical cardioversion. They reported significantly higher PWD values in patients with short-term AF recurrence. In addition, they demonstrated that values greater than 25 ms of PWD were associated with a higher short-term relapse rate. However, no significant relationship was present in the long-term in their study [41]. On the other hand, Perzanowski *et al.* [42] reported that a PWD value of 80 ms or greater was both a univariate and independent predictor for AF recurrence after cardioversion. In this context of electrical cardioversion, it is very interesting the finding by Ozdemir *et al.* [43] after electrical cardioversion of ventricular tachycardia. They identified 18 patients in whom an episode of AF was induced by urgent or elective cardioversion for a ventricular tachycardia. They observed that the patients whom developed AF had higher maximum P-wave duration and PWD values compared with a control group of patients without AF. They concluded that the patients with higher PWD values had a greater risk for development of AF after an electrical cardioversion of ventricular tachycardia [43]. These results are very interesting, and may have a clinical implication in patients with implantable cardioverter defibrillator. Because it may suggest that those patients with these cardiac devices that have higher PWD and maximum P-wave duration carry a greater risk for development of AF after an appropriate or inappropriate shock [43].

We have previously found that patients with a predisposition to develop AF have significantly higher incidence of atrial conduction defects, and abnormally prolonged and fractionated atrial endocardial electrograms [12-17]. We have reported that an abnormally prolonged and fractionated right atrial electrogram may reflect inhomogeneous local electrical

activity related to a delayed and non-uniform anisotropic conduction through diseased atrial muscle, and were closely related to the vulnerability of the atrial muscle in patients with paroxysmal AF [14-16]. Indeed, we demonstrated that the greater the extent of the compromised atrial muscle, the greater the likelihood that paroxysmal AF would develop [15]. Qualitative and quantitative analysis of atrial endocardial electrograms recorded during sinus rhythm should be an important analysis in evaluating local atrial electrophysiological abnormalities, and acquire particular relevance in the study of patients with paroxysmal AF (Figure **3**). In the evaluation of patients with altered P wave morphology and dispersion in the electrocardiogram, it is very important to keep in mind that patients who have a great susceptibility to develop AF possess abnormally prolonged and fractionated atrial endocardial electrograms, a significantly longer P wave duration and dispersion, a significantly longer intra-atrial and inter-atrial conduction time of sinus impulses; and a significantly greater sinus node dysfunction and higher incidence of induction of sustained atrial fibrillation [12].

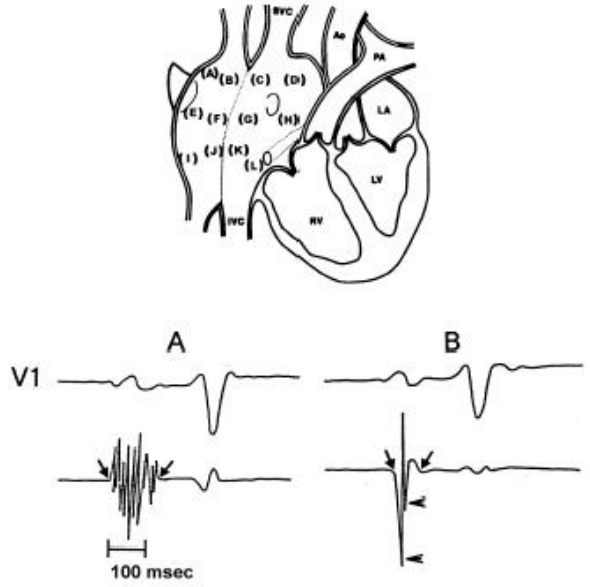

Figure 3: Atrial endocardial mapping sites. The upper part of the figure shows 12 endocardial mapping sites in the right atrium. The atrial endocardial electrograms were recorded in each patient from the anterior, lateral, posterior, and medial aspects of the high right atrium (a,b,c,d), mid right atrium (e,f,g,h), and low right atrium (i,j,k,l). SVC, superior vena cava; IVC, inferior vena cava; Ao, aorta; PA, pulmonary artery; LA, left atrium; RV, right ventricle; LV, left ventricle. The lower part of the figure shows two atrial endocardial electrograms to distinguish an abnormal atrial electrogram (**A**) with 10 fragmented deflections and 130 ms in duration, from a normal atrial electrogram (**B**) with two deflections and 80 ms in duration. Reprinted with permission from Centurion OA *et al*. Influence of advancing age on fractionated right atrial endocardial electrograms. Am J Cardiol 2005; 96: 239-242.

CONCLUSION

In conclusion, in patients prone to develop atrial arrhythmias and AF, PWD reflects prolonged, inhomogeneous and anisotropic distribution of connections between myocardial fibers resulting in discontinuous anisotropic propagation of sinus impulses, as well as, inhomogeneous and discontinuous atrial conduction. PWD is considered as a sensitive and specific ECG marker and predictor of atrial arrhythmias and paroxysmal AF.

REFERENCES

[1] Okutucu S, Aytemir K, Oto A. P-wave dispersion: what we know till now? JRSM Cardiovasc Dis 2016; 5: 1-9. https://doi.org/10.1177/2048004016639443

[2] Justo F, Fuller H, Nearing BD, Rajamani S, Belardinelli L, Verrier RL. Inhibition of the cardiac late Na+ current with eleclazine protects against ischemia induced vulnerability to atrial fibrillation and reduces atrial and ventricular repolarization abnormalities in the absence and presence of concurrent adrenergic stimulation. Heart Rhythm 2016; 13(9): 1860-1867. https://doi.org/10.1016/j.hrthm.2016.06.020

[3] Geng HH, Li R, Su YM, Pan HY, Pan M, Ji XP. A functional single-nucleotide polymorphism in interleukin-6 promoter is associated with p wave dispersion in hypertensive subjects with atrial fibrillation. Int J Clin Exp Med 2014; 7(11): 4434-4440.

[4] Sovari AA. Cellular and molecular mechanisms of arrhythmia by oxidative stress. Cardiol Res Pract 2016; 2016: 9656078. https://doi.org/10.1155/2016/9656078

[5] Magnani JW, Mazzini MJ, Sullivan LM, *et al*. P-wave indices, distribution and quality control assessment (from the Framingham Heart Study). Ann Noninvasive Electrocardiol 2010; 15: 77-84. https://doi.org/10.1111/j.1542-474X.2009.00343.x

[6] Aytemir K, Amasyali B, Kose S, *et al*. Maximum P-wave duration and P-wave dispersion predict recurrence of paroxysmal atrial fibrillation in patients with Wolff- Parkinson-White syndrome after successful radiofrequency catheter ablation. J Interv Card Electrophysiol 2004; 11: 21-27. https://doi.org/10.1023/B:JICE.0000035925.90831.80

[7] Magnani JW, Williamson MA, Ellinor PT, *et al*. P wave indices: current status and future directions in epidemiology, clinical, and research applications. Circ Arrhythm Electrophysiol 2009; 2: 72-79. https://doi.org/10.1161/CIRCEP.108.806828

[8] Aytemir K, Ozer N, Atalar E, *et al*. P wave dispersion on 12-lead electrocardiography in patients with paroxysmal atrial fibrillation. Pacing Clin Electrophysiol 2000; 23(7): 1109-1112. https://doi.org/10.1111/j.1540-8159.2000.tb00910.x

[9] Dilaveris PE, Gialafos JE. P-wave dispersion: a novel predictor of paroxysmal atrial fibrillation. Ann Noninvasive Electrocardiol 2001; 6: 159-165. https://doi.org/10.1111/j.1542-474X.2001.tb00101.x

[10] Lazzeroni D, Parati G, Bini M, *et al*. P-wave dispersion predicts atrial fibrillation following cardiac surgery. Int J Cardiol 2016; 203: 131-133. https://doi.org/10.1016/j.ijcard.2015.10.143

[11] Yoshizawa T, Niwano S, Niwano H, *et al*. Prediction of new onset atrial fibrillation through P wave analysis in 12 lead ECG. Int Heart J 2014; 55(5): 422-427. https://doi.org/10.1536/ihj.14-052

[12] Centurión OA. Clinical implications of the P wave duration and dispersion: Relationship between atrial conduction defects and abnormally prolonged atrial endocardial electrograms. Int J Cardiol 2009; 134: 6-8. https://doi.org/10.1016/j.ijcard.2008.12.072

[13] Centurión OA, Isomoto S, Shimizu A, Konoe A, Kaibara M, Hirata T, Hano O, Sakamoto R, Hayano M, Yano K. The effects of aging on atrial endocardial electrograms in patients with paroxysmal atrial fibrillation. Clin Cardiol 2003; 26: 435-438. https://doi.org/10.1002/clc.4960260911

[14] Centurión OA, Shimizu A, Isomoto S, *et al*. Influence of advancing age on fractionated right atrial endocardial electrograms. Am J Cardiol 2005; 96: 239-242. https://doi.org/10.1016/j.amjcard.2005.03.052

[15] Centurión OA, Fukatani M, Konoe A, Tanigawa M, Shimizu A, Isomoto S, Kaibara M, Hashiba K. Different distribution of abnormal endocardial electrograms within the right atrium in patients with sick sinus syndrome. Br Heart J 1992; 68: 596-600. https://doi.org/10.1136/hrt.68.12.596

[16] Centurión OA, Isomoto S, Fukatani M, Shimizu A, Konoe A, Tanigawa M, Kaibara M, Sakamoto R, Hano O, Hirata T, Yano K. Relationship between atrial conduction defects and fractionated atrial endocardial electrograms in patients with sick sinus syndrome. PACE 1993: 16: 2022-2033. https://doi.org/10.1111/j.1540-8159.1993.tb00996.x

[17] Centurión OA, Shimizu A, Isomoto S, Konoe A, Hirata T, Hano O, Kaibara M, Yano K. Repetitive atrial firing and fragmented atrial activity elicited by extrastimuli in the sick sinus syndrome with and without abnormal atrial electrograms. Am J Med Sci 1994; 307: 247-254. https://doi.org/10.1097/00000441-199404000-00001

[18] Spach MS, Dober PC, Anderson PAW. Multiple regional differences in cellular properties that regulate repolarization and contraction in the right atrium of adult and newborn dogs. Circ Res 1989; 65: 1594-1611. https://doi.org/10.1161/01.RES.65.6.1594

[19] Spach MS, Miller WT, Dolber PC, Kootsey JM, Sommer JR, Mosher Jr CE. The functional role of structural complexities in the propagation of depolarization in the atrium of the dog: cardiac conduction disturbances due to discontinuities of effective axial resistivity. Circ Res 1952; 50: 175-191. https://doi.org/10.1161/01.RES.50.2.175

[20] Spach MS, Dober PC. Relating extracellular potentials and their derivatives to anisotropic propagation at microscopic level in human cardiac muscle. Evidence for electrical uncoupling of side-to-side fiber connections with increasing age. Circ Res 1986; 58: 356-371. https://doi.org/10.1161/01.RES.58.3.356

[21] Oylumlu M, Dogan A, Ozer O, Yuce M, Ercan S, Davutoglu V. Effects of lying position on P-wave dispersion in patients with heart failure. Med Princ Pract 2014; 23(6): 556-560. https://doi.org/10.1159/000365510

[22] Prajapat L, Ariyarajah V, Frisella ME, Apiyasawat S, Spodick DH. Association of P-wave duration, dispersion, and terminal force in relation to P-wave axis among outpatients. Ann Noninvasive Electrocardiol 2007; 12(3): 210-215. https://doi.org/10.1111/j.1542-474X.2007.00163.x

[23] Morris Jr JJ, Estes Jr EH, Whalen RE, Thompson Jr HK, Mcintosh HD. P-wave analysis in valvular heart disease. Circulation 1964; 29: 242-252. https://doi.org/10.1161/01.CIR.29.2.242

[24] Ariyarajah V, Spodick DH. Advanced interatrial block: a classic electrocardiogram. Cardiology 2005; 104(1): 33-34. https://doi.org/10.1159/000086052

[25] Spodick DH, Ariyarajah V, Apiyasawat S. Higher prevalence of cardiovascular events among patients with abnormal atrial depolarization and coronary artery disease at 18 months' post-exercise tolerance testing. Am Heart Hosp J 2007; 5(4): 236-240. https://doi.org/10.1111/j.1541-9215.2007.07361.x

[26] Ozyigit T, Kocas O, Karadag B, Ozben B. Three dimensional left atrial volume index is correlated with P wave dispersion in elderly patients with sinus rhythm. Wien Klin Wochenschr 2016; 128(5e6): 182-186.

[27] Platonov PG. Atrial conduction and atrial fibrillation: what can we learn from surface ECG? Cardiol J 2008; 15(5): 402-407.

[28] Puerta RC, Aliz EL, Lopez-Calleja MA, Ramirez RR, Pena GP. Increased p wave dispersion in elite athletes. Indian Pacing Electrophysiol J 2011; 11(3): 73-80.

[29] Ertem AG, Erdo_gan M, Keles¸ T, Durmaz T, Bozkurt E. P-wave dispersion and left ventricular diastolic dysfunction in hypertension. Anatol J Cardiol 2015; 15(1): 78-79. https://doi.org/10.5152/akd.2014.5748

[30] Suner A, Cetin M. The effect of trimetazidine on ventricular repolarization indexes and left ventricular diastolic function in patients with coronary slow flow. Coron Artery Dis 2016; 27(5): 398-404. https://doi.org/10.1097/MCA.0000000000000373

[31] Kim DH, Kim GC, Kim SH, *et al*. The relationship between the left atrial volume and the maximum P-wave and P-wave dispersion in patients with congestive heart failure. Yonsei Med J 2007; 48(5): 810-817. https://doi.org/10.3349/ymj.2007.48.5.810

[32] Dursun H, Tanriverdi Z, Colluoglu T, Kaya D. Effect of transcatheter aortic valve replacement on P-wave duration, P-wave dispersion and left atrial size. J Geriatr Cardiol 2015; 12(6): 613-617.

[33] Beig JR, Tramboo NA, Rather HA, *et al*. Immediate effect of percutaneous transvenous mitral commissurotomy on atrial electromechanical delay and P wave dispersion in patients with severe mitral stenosis. Indian Heart J 2015; 67(Suppl 2): S46-S54. https://doi.org/10.1016/j.ihj.2015.10.375

[34] Kizilirmak F, Demir GG, Gokdeniz T, *et al*. Changes in electrocardiographic P wave parameters after Cryoballoon ablation and their association with atrial fibrillation recurrence. Ann Noninvasive Electrocardiol 2016; 21(6): 580-587. https://doi.org/10.1111/anec.12364

[35] Ding L, Hua W, Zhang S, *et al*. Improvement of P wave dispersion after cardiac resynchronization therapy for heart failure. J Electrocardiol 2009; 42(4): 334-338. https://doi.org/10.1016/j.jelectrocard.2009.02.005

[36] Kawamura M, Scheinman MM, Lee RJ, Badhwar N. Left atrial appendage ligation in patients with atrial fibrillation leads to a decrease in atrial dispersion. J Am Heart Assoc 2015; 4(5): e001581. https://doi.org/10.1161/JAHA.114.001581

[37] Mugnai G, Chierchia GB, de Asmundis C, *et al*. P-wave indices as predictors of atrial fibrillation recurrence after pulmonary vein isolation in normal left atrial size. J Cardiovasc Med Hagerst 2016; 17(3): 194-200. https://doi.org/10.2459/JCM.0000000000000220

[38] Badhwar N, Lakkireddy D, Kawamura M, *et al*. Sequential percutaneous LAA ligation and pulmonary vein isolation in patients with persistent AF: initial results of a feasibility study. J Cardiovasc Electrophysiol 2015; 26(6): 608-614. https://doi.org/10.1111/jce.12655

[39] Kose MD, Bag O Güven B, Mese T, Oztürk A, Tavlı V. P-wave dispersion: an indicator of cardiac autonomic dysfunction in children with neurocardiogenic syncope. Pediatr Cardiol 2014; 35(4): 596-600. https://doi.org/10.1007/s00246-013-0825-y

[40] Dilaveris PE, Gialafos EJ, Sideris SK, *et al*. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J 1998; 135: 733-738. https://doi.org/10.1016/S0002-8703(98)70030-4

[41] Boriani G, Diemberger I, Biffi M, *et al*. P wave dispersion and short-term vs. late atrial fibrillation recurrences after cardioversion. Int J Cardiol 2005; 101: 355-361. https://doi.org/10.1016/j.ijcard.2004.03.039

[42] Perzanowski C, Ho AT and Jacobson AK. Increased P wave dispersion predicts recurrent atrial fibrillation after cardioversion. J Electrocardiol 2005; 38: 43-46. https://doi.org/10.1016/j.jelectrocard.2004.09.008

[43] Ozdemir O, Soylu M, Demir AD, *et al*. Does p-wave dispersion predict the atrial fibrillation occurrence after direct-current shock therapy? Angiology 2006; 57: 93-98. https://doi.org/10.1177/000331970605700113

[44] Amasyali B, Kose S, Aytemir K, *et al*. P wave dispersion predicts recurrence of paroxysmal atrial fibrillation in patients with atrioventricular nodal reentrant tachycardia treated with radiofrequency catheter ablation. Ann Noninvasive Electrocardiol 2006; 11: 263-270. https://doi.org/10.1111/j.1542-474X.2006.00114.x

[45] Gudul NE, Karabag T, Sayin MR, Bayraktaroglu T, Aydin M. Atrial conduction times and left atrial mechanical functions and their relation with diastolic function in prediabetic patients. Kor J Intern Med 2016. https://doi.org/10.3904/kjim.2014.380

[46] Tsioufis C, Konstantinidis D, Nikolakopoulos E, Vemou E, Kalos T, Georgiopoulos G, *et al*. Biomarkers of atrial fibrillation in hypertension. Curr Med Chem 2017. https://doi.org/10.2174/0929867324666171006155516

[47] Okutucu S, Oto A. P-wave dispersion in different clinical situations: Expanding list with resembling mechanisms. JRSM Cardiovasc Dis 2017; 6. https://doi.org/10.1177/2048004017720369

[48] Fujimoto Y, Yodogawa K, Takahashi K, Tsuboi I, Hayashi H, Uetake S, *et al*. Noninvasive evaluation of reverse atrial remodeling after catheter ablation of atrial fibrillation by P wave dispersion. Heart Vessels 2017; 32(11): 1375-1381. https://doi.org/10.1007/s00380-017-1008-1

[49] Abou R, Leung M, Tonsbeek AM, Podlesnikar T, Maan AC, Schalij MJ, *et al*. Effect of Aging on Left Atrial Compliance and Electromechanical Properties in Subjects Without Structural Heart Disease. Am J Cardiol 2017; 120(1): 140-147. https://doi.org/10.1016/j.amjcard.2017.03.243

[50] Acampa M, Lazzerini PE, Martini G. How to Identify Patients at Risk of Silent Atrial Fibrillation after Cryptogenic Stroke: Potential Role of P Wave Dispersion. J Stroke 2017; 19(2): 239-241. https://doi.org/10.5853/jos.2016.01620

Received on 15-01-2018 Accepted on 02-02-2018 Published on 11-04-2018

DOI: https://doi.org/10.12970/2311-052X.2018.06.01