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Prolonged P wave dispersion in pre-diabetic patients

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Abstract

Background: It is known that overt diabetes as well as chronic hyperglycaemia can lead to atrial fibrillation. A P wave dispersion (PWD) represents heterogeneity in atrial refractoriness.

Aim: To investigate PWDs in patients with pre-diabetes.

Method: Based on the results of examinations, 84 pre-diabetic patients (the pre-DM group; 50 female, 34 male; mean age 54 ± 8.6 years) who had no overt diabetes, coronary artery disease or hypertension, whose fasting blood glucose was higher than 100 mg/dL and/or whose 2 h glucose concentrations on an oral glucose tolerance test was in the range of 140 to 199 mg/dL, and 48 healthy volunteers (the non-DM group, 30 female, 18 male; mean age 51.7 ± 7.3 years) with no illnesses, were enrolled in this study. Standard 12-lead electrocardiograms of all patients were taken at 50 mm/s and 20 mm/mV standardisation. Maximum (P_{max}) and minimum (P_{min}) P-wave durations were measured. The PWD was defined as the difference between P_{max} and P_{min}.

Results: The P_{max} and PWD values were significantly higher in pre-DM compared to non-DM (104 ± 13 ms *vs* 98 ± 12 ms; $p < 0.05$, 42 ± 13 ms *vs* 34 ± 11 ms; $p < 0.01$ respectively). A positive correlation was found between PWD and fasting blood glucose ($r = 0.32$; $p < 0.01$). There was no correlation between PWD and HbA1c levels ($r = 19$; $p > 0.05$). Multivariate regression analysis showed no relationship between PWD and age, left atrial diameter, E, A, E/A or HbA1c. However, there was a relationship between PWD and fasting blood glucose.

Conclusions: The P_{max} and PWD are increased in pre-diabetic patients who have no coronary artery disease, hypertension or left ventricular hypertrophy.

Key words: pre-diabetes, P wave dispersion, atrial fibrillation

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INTRODUCTION

The P wave dispersion (PWD) is defined as the difference between the maximum (P_{max}) and minimum (P_{min}) P wave duration on a standard 12-lead electrocardiogram (ECG). The PWD is a measure of the heterogeneity of atrial refractoriness [1]. Prolongation of PWD is known to be an independent risk factor for development of atrial fibrillation (AF) and indicates intra-atrial and inter-atrial non-uniform conduction [1–3].

Pre-diabetes defines patients at high risk for diabetes in the future with impaired fasting glucose (IFG; fasting glucose above 100 mg/dL) and/or impaired glucose tolerance (IGT; 2 h oral glucose tolerance test [OGTT] levels of 140––199 mg/dL). The IFG and IGT should not be considered only as clinical symptoms, but should be regarded as risk factors for both diabetes and cardiovascular diseases [4]. Not only diabetes, but also all stages of glucose abnormalities are associated with an increased risk of cardiovascular morbidity and mortality, making it important to identify such conditions as early as possible [5]. Diabetes mellitus (DM) is an independent and strong risk factor for the development

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of AF. The frequency of AF development is 1.4–2.1 times higher in patients with DM than in those without DM [6, 7]. The incidence of DM in patients with AF is known to be as high as the incidence of pre-diabetes. The prediction of risk for AF development is of importance throughout all phases of diabetes [8].

In the present study, we measured PWD in pre-diabetic patients, which is one of the predictive parameters for the development of AF.

METHODS

Based on examinations performed on admission to cardiology and endocrinology departments, 84 patients (the pre-DM group, 50 female, 34 male; mean age 54 ± 8.6 years) who had no overt diabetes, coronary artery disease (CAD) or hypertension, whose fasting blood glucose (FBG) was higher than 100 mg/dL and/or whose two hour glucose concentrations on an oral glucose tolerance test was between 140 and 199 mg/dL, were enrolled in the present study. The control group consisted of 48 healthy volunteers (the non-DM group, 30 female, 18 male; mean age 51.7 ± 7.3 years) with no illnesses, whose examinations revealed no anomalies and whose laboratory values were within normal ranges.

Subjects with a history of myocardial infarction, angina pectoris or other symptoms of CAD, those with right or left bundle branch block, Wolff-Parkinson-White syndrome, intraventricular conduction defect on resting ECG, and those with ischaemic changes on the ECG and stress test, were excluded from the study. Moreover, patients with a history of palpitations, AF, permanent pacemaker implantation, antiarrhythmic drug use and those with thyroid disease, left ventricular (LV) hypertrophy or LV dysfunction were also excluded from the study. All patients underwent 12-lead ECG and exercise stress test (according to Bruce protocol) and echocardiography. Renal and hepatic functions of the study group were within normal ranges. A signed consent form was obtained from each volunteer enrolled. The study was approved by the Local Ethics Committee.

Laboratory measurements

Fasting blood glucose, total cholesterol, triglyceride, low density lipoprotein, high density lipoprotein and HbA1c values were measured from venous blood collected from the pre-DM and non-DM groups after fasting for eight hours. A simplified protocol was used for OGTT. Fasting blood glucose was determined immediately before ingestion of 75 g of solubilised glucose, followed 2 h later by determination of 2 h plasma glucose (2 h-PG).

Patients with FBG > 100 mg/dL fulfilled the criteria for IFG and those with 2 h OGTT level of 140–199 mg/dL fulfilled the criteria for IGT based on the American Diabetes Association criteria. Patients with IFG and IGT were both included in the pre-DM group [4].

Electrocardiographic measurements

All standard 12-lead ECGs were obtained using a recorder (Agilent, Andover, MA, USA) set at a 50 mm/s paper speed and 20 mm/mV standardisation. Recordings were performed during spontaneous breathing in the supine position. All measurements of P-wave duration were calculated blindly by two observers. The onset of the P wave was defined as the point of first visible upward slope from the baseline for positive waveforms, and as the point of first downward slope from the baseline for negative waveforms. The return to the baseline was considered as the end of the P wave. Biphasic P waves were measured to the time of final return to the baseline.

The P_{max} in any of the 12-lead surface ECGs was measured as the longest atrial conduction time. The P_{min} was measured as the shortest atrial conduction time. Mean P-wave durations for at least three complexes were calculated in each lead. Patients whose measurements could be performed in at least eight derivations were included in the study. The P wave measurements could not be performed because of low amplitude in approximately 1–2 of 12 ECG leads in each case. In all patients, measurements were excluded if the beginning or the end of the P wave could not be clearly identified. If the baseline noise was over 1.0 mV and/or if the peak of the P wave amplitude from isoelectric line was below 1.5 mV, the lead was excluded from the analysis. The difference between the P_{max} and the P_{min} was calculated and this was defined as PWD.

Echocardiographic measurements

All echocardiographic examinations were performed with a System Five (GE Vingmed Ultrasound, Horten, Norway) cardiac ultrasound scanner and 2.5–3.5 MHz transducers. All patients were examined in the left lateral and supine positions by precordial M-mode, two-dimensional (2D) echocardiography. The LV and left atrial (LA) diameters, as well as wall thickness, were measured from the 2D targeted M-mode echocardiographic tracings in the parasternal long axis, according to the criteria of the American Society of Echocardiography [9]. The LV ejection fraction was measured according to the Simpson method [10]. Pulse wave mitral flow velocities were measured from the apical four-chamber view by inserting a sample volume to mitral leaflet tips. Mitral early diastolic velocity (E), late diastolic velocity (A), E/A ratio, and E wave deceleration time (EDT) were measured.

Statistical analysis

All statistical analyses were carried out using SPSS statistical software (version 11.0, SPSS, Chicago, IL, USA). Data are presented as mean ± SD. Statistical comparisons of quantitative data concerning P_{max}, P_{min} and PWD were performed by independent sample t test. The association of HbA1c and glucose levels with PWD was assessed using Pearson's correlation test. The relationship between PWD and clinical and

echocardiographic variables in patients with pre-diabetes were evaluated by multivariate regression analysis. A p value < 0.05 was considered statistically significant.

RESULTS

Table 1 demonstrates the clinical characteristics and Table 2 the echocardiographic measurements in both groups. Controls had a significantly better lipid profile and diastolic function compared to the pre-DM group.

The P_{max} and PWD were significantly greater in the pre-DM group compared to the non-DM group (Table 3). However, there was no significant difference in the P_{min} duration (Table 3). A positive correlation between PWD and FBG ($r = 0.32$; $p < 0.01$) was found. No correlation was found between PWD

Table 1. Clinical characteristics of the groups

	Pre-DM (n = 84)	Non-DM (n = 48)	P
Age [years]	54.1 ± 8.6	51.7 ± 7.3	NS
Gender (female)	50	30	NS
SBP [mm Hg]	127 ± 12	118 ± 10	NS
DBP [mm Hg]	82 ± 11	78 ± 8	NS
Heart rate [beat/min]	78 ± 21	69 ± 9	NS
BMI [kg/m^2]	27.4 ± 3.5	25.9 ± 4.1	NS
FBG [mg/dL]	117 ± 5.5	89 ± 7.7	< 0.001
HbA1c [%]	6 ± 1.5	4.9 ± 0.7	NS
TC [mg/dL]	198 ± 40	168 ± 33	< 0.001
TG [mg/dL]	164 ± 89	103 ± 37	< 0.05
LDL-C [mg/dL]	120 ± 31	98 ± 30	< 0.05
HDL-C [mg/dL]	45 ± 11	52 ± 13	< 0.05

BMI — body mass index; DBP — diastolic blood pressure; DM — diabetes mellitus; FBG — fasting plasma glucose; HbA1c — glycosylated haemoglobin; HDL-C — high density lipoprotein cholesterol; LDL-C — low density lipoprotein cholesterol; SBP — systolic blood pressure; TC — total cholesterol; TG — triglycerides

Table 2. Echocardiographic characteristics of the groups

	Pre-DM (n = 84)	Non-DM (n = 48)	P
LVEDD [mm]	46.7 ± 2.7	45.1 ± 3.1	NS
LVESD [mm]	28.4 ± 2.3	27.5 ± 1.7	NS
LA [mm]	35.5 ± 3.9	33.6 ± 2.1	NS
AOD [mm]	30.1 ± 3.2	28.8 ± 2.1	NS
IVS [mm]	9.4 ± 1.5	8.7 ± 1.2	NS
PW [mm]	9.0 ± 1.5	8.6 ± 1.2	NS
EF [%]	63.3 ± 2.9	64.8 ± 2.1	NS
E [cm/s]	0.66 ± 0.14	0.82 ± 0.1	< 0.05
A [cm/s]	0.79 ± 0.19	0.59 ± 0.1	< 0.05
E/A	0.88 ± 0.29	1.40 ± 0.23	< 0.05
EDT [ms]	228 ± 40	178 ± 17	< 0.05

A — mitral diastolic A wave; AOD — aortic diameter; DM — diabetes mellitus; E — mitral diastolic E wave; EDT — E wave deceleration time; EF — ejection fraction; IVS — interventricular septum; LA — left atrium; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; PW — posterior wall thickness

Table 3. Comparison of P wave measurements in the groups

	Pre-DM (n = 84)	Non-DM (n = 48)	P
P_{max} [ms]	104 ± 13	98 ± 12	< 0.05
P_{min} [ms]	62 ± 11	64 ± 13	NS
PWD [ms]	42 ± 13	34 ± 11	< 0.01

DM — diabetes mellitus; PWD — P wave dispersion

Table 4. Relationship between P wave dispersion and clinical, echocardiographic and laboratory characteristics

	P wave dispersion		
	β	*t*	P
Age [years]	0.267	1.883	NS
LA diameter [mm]	0.091	0.677	NS
E [cm/s]	0.108	0.221	NS
A [cm/s]	–0.308	–0.636	NS
E/A ratio	–0.055	–0.084	NS
HbA1c [%]	0.285	1.645	NS
FBG [gr/dL]	0.434	2.373	0.029

A — mitral diastolic A wave; E — mitral diastolic E wave; FBG — fasting blood glucose; HbA1c — glycosylated haemoglobin; LA — left atrial

and HbA1c levels ($r = 19$; $p > 0.05$). Although there were significant differences with regard to mitral E, A and E/A values between the pre-DM and non-DM groups (Table 2), no correlation between PWD and E, A or E/A was observed ($r = –0.12$; $r = 0.09$ and $r = –0.13$; NS respectively).

There were no significant differences in the P_{max}, P_{min} and PWD values between the IFG and IGT patients (103 ± 14 *vs* 108 ± 9 ms; $p > 0.05$, 62 ± 11 *vs* 61 ± 8 ms; $p > 0.05$; 41 ± 13 *vs* 46 ± 11 ms; NS respectively). Multivariate regression analysis showed no relationship between PWD and age, LA diameter, E, A, E/A, or HbA1c values. However, there was a relationship between PWD and FBG (Table 4).

DISCUSSION

Atrial fibrillation is the commonest arrhythmia encountered in clinical practice, even in the absence of antecedent congestive heart failure or myocardial infarction, and is associated with an increased risk of ischaemic stroke, heart failure, and overall mortality [1–4]. Thus, identifying risk factors is important for the development of therapeutic approaches to AF. Diabetes has been reported to be one of the independent risk factors in AF development [11–14]. However, previous studies have reported that chronic hyperglycaemia, through some mechanisms, can trigger AF even before the development of overt diabetes [8]. In an epidemiologic study by Johansen et al. [8] increased glycaemic burden was reported in ≥ 75 year old patients with AF. The authors concluded that in patients with a ≥ five-year history of AF, pre-diabetes or diabetes should be actively investigated [8].

Although it remains to be elucidated why a hyperglycaemic state leads to AF, several mechanisms have been proposed. Chronic hyperglycaemia may contribute to the AF burden [6, 15–17] in several ways. One recently described is through the activation of the advanced glycosylation endproduct (AGE) — receptors for AGE system and the up-regulation of circulating tissue growth factors (CTGF) that may promote atrial structural remodelling [18]. Even though long-term exposure to high glucose concentrations increases eNOS expression in endothelial cells, an increase in superoxide anions (probably NADH/NADPH oxidase) results in NO inactivation. Additionally, prolonged hyperglycaemic stress leads to accumulation of advanced glycosylation end products (AGES), which are capable of inactivating NO [19, 20]. One potential reason for the increased prevalence of long-standing AF in the presence of abnormal glucose metabolism could be related to an activation of inflammation, an early manifestation of AF as well as of DM, hence potentially also being a common pathway for these two conditions [6].

Another possible mechanism is that chronic hyperglycaemia causes structural and functional disorders by changing the chemical composition of the proteins present in cell membrane structure. An experimental study has reported that extracellular protein deposition and interstitial myocardial fibrosis might be other mechanisms responsible for the prolongation of PWD, by influencing heterogeneity of atrial conduction time and atrial refractoriness in diabetic patients [21].

In the present study, we were unable to investigate whether there was atrial cell damage at the tissue level. However, the reason for the prolongation of PWD may be a structural defect in the atrium, which is likely to be caused by damage due to chronic hyperglycaemia. In a study investigating PWD in type 1 and 2 diabetic patients without CAD or hypertension, PWD was reported to be prolonged [22].

Stahrenberg et al. [23] investigated the association between glucose metabolism and diastolic function along the whole spectrum of glucose metabolism states in a subgroup of patients classified as normal, OGTT, pre-diabetic, non-insulin treated or insulin-treated type 2 diabetic. They found impaired diastolic functions in all groups, and concluded that glucose metabolism is associated with diastolic dysfunction across the whole spectrum.

In another study, Gunduz et al. [24] found enhanced PWD in patients with diastolic dysfunction, with PWD values increasing as the degree of diastolic dysfunction worsened. They found no relationship between PWD and E, A, E/A ratio, isovolumetric relaxation time or LA diameter. Similar to Gunduz et al. [24], we found impaired diastolic functions in pre-diabetic patients compared to the control group. How-

ever, there was no relationship between PWD and E, A, E/A or LA diameters.

Our study demonstrated that the prolongation of PWD might occur before the development of overt diabetes. It may be that the non-invasive demonstration of the prolongation of PWD before the development of overt diabetes could assist in predicting the occurence of AF. Echocardiographic examination of volunteers enrolled in this study revealed no dilatation of the cardiac cavities and no valvular pathology. This indicates that the prolongation of PWD is independent of structural defects in the cardiac cavities.

Limitations of the study

Dilaveris et al. [25] concluded that manual measurement of P wave duration in standard 12-lead ECGs is not only feasible, but also more stable and reliable when performed on a high resolution screen of a digital ECG system than more conventional methods involving paper printed ECGs. We measured P wave dispersion on paper printed ECGs. Another limitation to our study was the use of non-invasive imaging techniques to exclude those with CAD in the study group. Pre-diabetic patients were enrolled in the study after being evaluated in detail for the presence of AF and symptoms that may be associated with other arrhythmias. However, we did not follow up our patients, thus the value of PWD in predicting AF development is unknown.

CONCLUSIONS

The PWD may be increased in pre-diabetic patients without CAD, hypertension or structural anomalies in the cardiac cavities, and it may be a non-invasive marker of prolonged and inhomogenous atrial conduction. Even though the mechanism has not yet been identified, it could be that hyperglycaemia may be responsible for the prolongation of atrial conduction time in patients with pre-diabetes and hence predisposed to AF.

Conflict of interest: *none declared*

References

1. Perzanowski C, Ho AT, Jacobson AK. Increased P-wave dispersion predicts recurrent atrial fibrillation after cardioversion. J Electrocardiol, 2005; 38: 43–46.
2. Dogan SM, Buyukates M, Kandemir O et al. Predictors of atrial fibrillation after coronary artery bypass surgery. Coron Artery Dis, 2007; 18: 327–331.
3. Li Z, Hertervig E, Yuan S, Yang Y, Lin Z, Olsson SB. Dispersion of atrial repolarization in patients with paroxysmal atrial fibrillation. Europace, 2001; 3: 285–291.
4. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care, 2010; 33 (suppl. 1): 62–69.
5. Laakso M, Kuusisto J. Understanding patient needs: diabetology for cardiologists. Eur Heart J, 2003; 5 (suppl. B): 5–13.
6. Movahed MR, Hashemzadeh M, Jamal MM. Diabetes mellitus is a strong, independent risk for atrial fibrillation and flutter in addition to other cardiovascular disease. Int J Cardiol, 2005; 105: 315–318.
7. Ostgren CJ, Merlo J, Rastam L, Lindblad U. Atrial fibrillation and its association with type 2 diabetes and hypertension in Swedish community. Diabetes Obes Metab, 2004; 6: 367–374.
8. Johansen OE, Brustad E, Enger S, Tveit A. Prevalence of abnormal glucose metabolism in atrial fibrillation: a case control study in 75-year old subjects. Cardiovasc Diab, 2008, 7: 28.
9. Lang RM, Bierig M, Devereux RB et al. American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. 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, a branch of the European Society of Cardiology. J Am Soc Echocardiogr, 2005; 18: 1440–1463.
10. Feigenbaum H, Armstrong WF, Ryan T. Evaluation of systolic and diastolic function of the left ventricle. In: Feigenbaum H ed. Feigenbaum's echocardiography. 6th Ed. Lippincott Williams and Wilkins 2005: 142–145.
11. Lloyd-Jones DM, Wang TJ, Leip EP et al. Lifetime risk for development of atrial fibrillation: the Framingham Heart Study. Circulation, 2004; 110: 1042–1046.
12. Benjamin EJ, Wolf PA, D'Agostino RB, Silbershatz H, Kannel WB, Levy D. Impact of atrial fibrillation on the risk of death: the Framingham Heart Study. Circulation, 1998; 98: 946–952.
13. Stewart S, Hart CL, Hole DJ, McMurray JJ. A population-based study of the long-term risks associated with atrial fibrillation: 20-year follow-up of the Renfrew/Paisley study. Am J Med, 2002; 113: 359–364.
14. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham study. Stroke, 1991; 22: 983–988.
15. Kannel WB, Wolf PA, Benjamin EJ, Levy D. Prevalence, incidence, prognosis, and predisposing conditions for atrial fibrillation: population-based estimates. Am J Cardiol, 1998; 82: 2N–9N.
16. Aksnes TA, Schmieder RE, Kjeldsen SE et al. Impact of new-onset diabetes mellitus on development of atrial fibrillation and heart failure in high-risk hypertension (from the VALUE Trial). Am J Cardiol, 2008; 101: 634–638.
17. Gandhi GY, Nuttall GA, Abel MD et al. Intraoperative hyperglycemia and perioperative outcomes in cardiac surgery patients. Mayo Clin Proc, 2005; 80: 862–866.
18. Grundy SM, Cleeman JI, Daniels SR et al.; American Heart Association; National Heart, Lung, and Blood Institute. Diagnosis and Management of the Metabolic Syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation, 2005; 112: 2735–5272.
19. Nakagami H, Kaneda Y, Ogihara T, Morishita R. Endothelial dysfunction in hyperglycemia as a trigger of atherosclerosis. Curr Diabetes Rev, 2005; 1: 59–63.
20. Rask-Madsen C, King GL. Mechanisms of disease: endothelial dysfunction in insulin resistance and diabetes. Nature Clin Pract Endocrinol Metab, 2007; 3: 46–56.
21. Kato T, Yamashita T, Sekiguchi A et al. What are arrhythmogenic substrates in diabetic rat atria? J Cardiovasc Electrophysiol, 2006; 17: 890–894.
22. Yazici M, Ozdemir K, Altunkeser BB et al. The effect of diabetes mellitus on the P-wave dispersion. Circ J 2007; 71: 880–883.
23. Stahrenberg R, Edelmann F, Mende M, Kockskämper A, Düngen HD, Scherer M. Association of glucose metabolism with diastolic function along the diabetic continuum. Diabetologia, 2010; 53: 331–1340.
24. Gunduz H, Binak E, Arinc H, Akdemir R, Ozhan H, Tamer A. The relationship between P wave dispersion and diastolic dysfunction. Tex Heart Inst J, 2005; 32: 163–167.
25. Dilaveris P, Batchvarov V, Gialafos J, Malik M. Comparison of different methods for manual P wave duration measurement in 12-lead electrocardiograms. PACE, 1999; 22: 1532–1538.

Wydłużona dyspersja załamka P u chorych ze stanem przedcukrzycowym

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Streszczenie

Wstęp: Wiadomo, że jawna cukrzyca i jawna hiperglikemia mogą sprzyjać wystąpieniu migotania przedsionków.

Cel: Celem pracy była zbadanie dyspersji załamka P (PWD) jako metody oceny heterogenności opornosci przedsionków u chorych ze stanem przedcukrzycowym.

Metody: Na podstawie poniższych wyników do badania włączono 84 chorych ze stanem przedcukrzycowym (grupa pre-DM: 50 kobiet, 34 mężczyzn; średni wiek 54 ± 8,6 roku) bez jawnej cukrzycy, choroby wieńcowej czy nadciśnienia, u których stężenie glukozy na czczo wynosiło ponad 100 mg/dl i/lub u których stężenia glukozy po 2 h podczas testu doustnego obciążenia glukozą znajdowały się w zakresie 140–199 mg/dl, oraz 48 zdrowych ochotników (grupa non-DM: 30 kobiet, 18 mężczyzn, średni wiek 51,7 ± 7,3 roku) bez rozpoznanych chorób. Standardowy 12-odprowadzeniowy elektrokardiogram wykonano u wszystkich badanych przy standardowym przesuwie taśmy 50 mm/s z cechą 20 mm/mV. Zmierzono maksymalne (P_{max}) i minimalne (P_{min}) czasy trwania załamków P, a PWD zdefiniowano jako różnicę między P_{max} i P_{min}.

Wyniki: Wartości P_{max} oraz PWD były istotnie wyższe w grupie pre-DM w porównaniu z non-DM (odpowiednio 104 ± 13 ms *v.* 98 ± 12 ms; $p < 0,05$; 42 ± 13 ms *v.* 34 ± 11 ms; $p < 0,01$). Stwierdzono dodatnią korelację między PWD i stężeniem glukozy na czczo ($r = 0,32$; $p < 0,01$), nie zaobserwowano zależności między PWD i HbA_{1c} ($r = 19$; $p > 0,05$). Wieloczynnikowa analiza regresji nie wykazała związku między PWD a wiekiem, wymiarem lewego przedsionka, wartościami E, A, E/A oraz HbA_{1c}. Stwierdzono jednak zależność między PWD i stężeniem glukozy na czczo.

Wnioski: Wartości P_{max} oraz PWD mogą być zwiększone u osób ze stanem przedcukrzycowym bez rozpoznanej choroby wieńcowej, nadciśnienia czy przerostu lewej komory.

Słowa kluczowe: stan przedcukrzycowy, dyspersja załamka P, migotanie przedsionków

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