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# Relationship between paroxysmal atrial fibrillation and a novel electrocardiographic parameter P wave peak time

Ersin Yıldırım, MD [a,*], Nuran Günay, MD [a], Emrah Bayam, MD [a], Muhammed Keskin, MD [b], Burak Ozturkeri, MD [a], Murat Selcuk, MD [a]

[a] Istanbul Umraniye Education and Research Hospital, University of Health Sciences, Department of Cardiology, Turkey
[b] Istanbul Sultan Abdulhamid Han Education and Research Hospital, University of Health Sciences, Department of Cardiology, Turkey

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ABSTRACT

*Objective:* The aim of this study was to compare the relationship between a novel electrocardiographic (ECG) parameter P wave peak time (PWPT) and classic P wave parameters with atrial fibrillation (AF).
*Methods:* A total of 140 individuals, including 70 patients with AF history and 70 healthy individuals without AF as the control group were included in the study. These groups were compared in terms of demographic characteristics, laboratory findings and ECG parameters. P wave parameters including; PR interval, P wave dispersion (PWDIS), P wave max duration(PWD) abnormal P wave axis, P-wave terminal force in lead V1 and a novel parameter PWPT were calculated from a 12-lead surface ECG recorded in all patients during sinus rhythm.
*Results:* PR duration, PWDIS, PWD and PWPT in lead V1 and D2 were found to be longer in AF group compared to the control group. The presence of a P-terminal force in lead 1(V1TF) > 0.04 mm/s and abnormal P wave axis were shown to be significantly more frequent in the AF group. Univariate and multivariate regression analyses revealed independent relationship between the PWPT in lead V1 and AF(OR: 1.09, CI:1.01–1.17, p:0.024). In ROC curve analysis $PWPT_{V1}$ above a cut-off level of 49.5msc predicted AF with a sensitivity of 79.4% and a specificity of 56.3% (Area Under Curve(AUC): 0.737, $p < 0.001$).
*Conclusion:* In this study, we observed that $PWPT_{V1}$ is longer in patients with paroxysmal AF than in controls.


## Introduction

Atrial fibrillation (AF) is a common arrhythmia managed in cardiology practice whose prevalence increases with concomitant heart disease and age [1]. It is characterized by disorganized atrial electrical activity leading to loss of effective contraction [2]. AF is associated with higher rates of death, thromboembolic and cardiovascular events, and leads to poor quality of life, decreases exercise capacity and aggravation of heart failure [3]. Prevention is also important in the case of the most common sustained arrhythmia-AF. Despite recent advances in understanding the mechanisms of AF, in drug and interventional therapy, AF remains a public health problem, the cost to society is very high and thromboembolic complications related morbidity are often devastating for the individual patient [3]. Prediction of AF is an important issue because the identification of patients at increased risk could support their close monitoring, more aggressive risk factors therapy, or even anticoagulation in patients at high risk without documented AF [4]. Numerous clinical, electrocardiographic, echocardiographic and biological parameters have been tested as predictors of AF in different settings. Due to the accessibility of the standard electrocardiography (ECG) and the mechanisms of AF, it is logical that indices of P-wave have been assiduously studied [5]. The ECG may be valuable in prevention efforts because of its ability to characterize electrophysiological changes as intermediate phenotypes along the pathway to AF. In recent years many P wave parameters have been studied in patients with AF and shown to be associated with and AF development. The ECG provides a wealth of information which is of value in predicting incident AF. The PR interval and P wave indices are discussed with regard to their ability to predict and characterize AF risk in the general population in previous studies [6]. P wave indices (PWIs) reflect underlying atrial remodelling. These measurements have the advantage of characterizing atrial electrical activity during depolarization. Alterations in atrial activation measured through analysis of P wave indices has been associated with atrial remodelling and ischemic stroke [5,7]. PWIs are also associated with increased risk of AF [8]. These PWIs include PR interval, maximum P-wave duration(PWD), P-wave dispersion (PWDIS), P wave terminal force in lead 1 (V1TF), abnormal P-wave axis. Unlike these classic parameters, P wave peak time (PWPT) is a recently defined ECG parameter and studies regarding the relationship between PWPT and cardiovascular events have been published lately [9]. We thought that this ECG parameter, which is closely related to atrial electrical activity, may also be associated with

* Corresponding author at: Atatürk mah. Fırat cad. 71, Ada, Gardenya 6 Sitesi, H blok, Daire 42 Ataşehir, İstanbul, Turkey.
*E-mail address:* ersinyild44@gmail.com (E. Yıldırım).

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AF. In the present study, we aimed to assess the potential relationship between a novel ECG parameter P wave PWPT and classic P wave parameters with AF.

## Methods

### *Study design and population*

Following ethical committee approval, a total of 140 individuals, which included 70 patients with AF attack and 70 healthy individuals with no history of AF as the control group from January 2018 to February 2019 were retrospectively were included in the study. The AF group included consecutive patients with paroxysmal atrial fibrillation (PAF) episodes during their follow-up in our hospital. Only patients with AF documented by ECG were included in the study. Patients in whom sinus rhythm ECGs were not reached were excluded from the study. Control group was selected consecutively from the patients that visit the cardiology clinic retrospectively with 1:1 matched cohort. PAF episodes, defined by irregular ventricular response and absence of P-waves, were annotated by expert cardiologists. Sinus rhythm ECG evaluation of AF group was performed and compared with those of the control group. The clinical and demographic characteristics of the patients were collected from the patients and hospital records. Complete blood count (CBC) and biochemical tests were performed using a Beckman Coulter LH-750 and a Beckman Coulter L × 20 respectively, and the results of each patient were recorded. Echocardiographic evaluations of all patients were made at the first admission to the hospital. All participants underwent 2D and Doppler echocardiographic evaluation (VIVID 3, General Electric, United States of America) and the left ventricular ejection fraction was calculated by using the modified Simpson rules. Those who with metabolic or electrolyte disorders, acute or chronic infections or the patients take antiarrhythmic or other medications which can effect on P wave, PR segment, QT and QTc interval were excluded from the study.

### *ECG analysis*

ECG records of the patients were obtained via the Schiller Cardiovit AT-102 plus using the standard 12 derivation (10 mm/mV calibration and 25 mm/s sliding rate). All ECG papers were scanned, loaded to a computer, magnified sufficiently and analyzed with digital image processing software (imagej.nih.gov/ij). All measurements were calculated by two cardiologists. Average of two measurements was used for comparison. Magnified on-screen measurements were generally consistent and this provided to us more stable results. Inter-observer agreement for PWPT was evaluated by calculating the Pearson's correlation coefficient ($r = 0.93$). On the surface ECG, QT interval is calculated as the interval from the beginning of the QRS to the end of the T wave. QTc intervals were calculated by using the Bazett formula.

### *P wave indices*

The P wave indices are quantitative measures of atrial electrical function derived from the surface ECG. In our study P wave indices included, observed in previous studies parameters (PR interval, maximum P-wave duration, P-wave dispersion, P wave terminal force, abnormal P-wave axis) and a novel ECG parameter PWPT. P wave indices were evaluated from surface ECG, the results were recorded and these are defined as follows.

PR interval was defined as the period of time between the onset of the P wave (atrial depolarization) and the onset of the QRS complex. P wave peak time (PWPT) was defined as the duration between beginning and peak of the P wave and measured from D2 (PWPTD2) and V1 (PWPTV1) leads. In lead V1 with negative and biphasic P waves, we measured the duration from the starting point of the P wave to the top of the negative P wave. Negative waves which are ≥0.1 mv were used for measurement and only these patients were accepted as biphasic. Negative waves smaller than these limit values did not taken into account in the measurement. Measurement examples of PWPT are shown in Fig. 1.

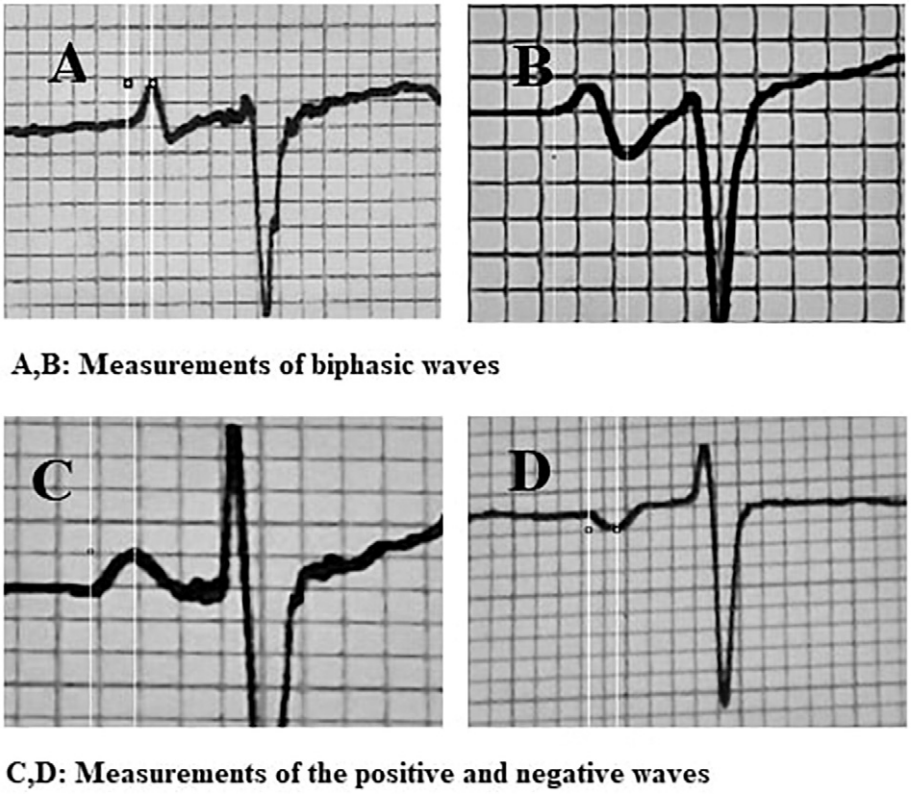


**Fig. 1.** Measurement examples of PWPT.

Maximum P-wave duration(PWD) was measured from the beginning of the P-wave deflection on the isoelectric line to the end of the deflection returning to isoelectric line in all simultaneous 12 leads and longest P-wave duration was saved. P-wave dispersion (PWDISP) was defined as the difference between the longest (Pmax) and the shortest (Pmin) P-wave duration measured in any of the standard ECG leads. P wave terminal force (PWTF) was calculated by multiplying the depth and the duration of the terminal negative component of the P wave in lead V1 and abnormal PWTF was defined as PWTF ≥40 mm X ms. Abnormal P-wave axis was determined as P-wave axis less than $0^o$ or more than $75^o$. In the case that the P wave morphology consisted of a positive or negative deflection only, this deflection was used for the calculation of the average value, and in the case of a biphasic deflection, the absolute sum of the deflections were added and used for the analysis.

### *Statistical analysis*

Statistical analysis was performed using SPSS 22 for Windows Evaluation Version statistical package. The normality distribution was evaluated using the Kolmogorov-Smirnov test. Continuous variables were presented as mean ± standard deviation. Categorical variables were summarized as frequencies. Differences between the two groups according to continuous variables were determined by the independent samples *t*-test or Mann–Whitney *U* test. Categorical variables were compared by, chi-square or Fisher's exact test. The logistic regression analysis was used for determining the effect of potential prognostic factors on the presence of AF and the independent predictors were determined through inclusion of significant risk factors in the logistic regression model and then Receiver operating characteristics (ROC) curve analysis was performed for the parameters determined significantly in regression analysis. Comparison of ROC curves was done by using the De Long test. (MedCalc Software, Mariakerke, Belgium). A p level of <0.05 was accepted as statistically significant with 95% confidence interval and 5% margin of error.

## Results

The distribution of the study population ($n = 140$, mean age 58.59 ± 15.25) was as follows: 70 patients (50.0%) in AF(+) group and 70 patients (50.0%) in AF(−) group. The rate of females was 57.85%. The mean age (65.87 ± 10.80 vs 51.41 ± 15.58; $p < 0{,}001$) was significantly higher in the AF(+) group. The rate of cerebrovascular disease was higher in the AF(+) group compared to the AF(−) group. There was no statistically significant difference between the groups in terms of gender, presence of coronary artery disease, hypertension, diabetes mellitus and smoking. The mean blood urea nitrogen (38.52 ± 11,36 vs 30.51 ± 11.51, $p < 0.001$) and creatinine (0.93 ± 0.27 vs 0.80 ± 0.16, $p = 0.002$) values in the AF group were observed to be significantly higher compared to the control group. Mean left atrium anteroposterior diameter (38.72 ± 5.26 vs 33.17 ± 3.50; $p < 0{,}001$) and mean ascending aorta diameter (38.82 ± 4.13 vs 32.95 ± 4.14; $p < 0{,}008$) were detected significantly higher in the AF(+) group. No difference was determined in the other demographic, laboratory and echocardiographic characteristics between the groups (Table 1).

In AF(+) group, PR (168.40 ± 40.06 vs 151.26 ± 20.80, $p = 0.003$), P wave dispersion (51.47 ± 6.83 vs 47.44 ± 7.15, $p = 0.001$) and P wave maximum durations (121.20 ± 22.15 vs 109.77 ± 15.53, $p = 0{,}001$) were significantly longer than the control group. There was statistically significant difference between the groups in terms of $PWPT_{V1}$ (56.32 ± 8.26 vs 48.60 ± 6.45, $p < 0{,}001$), $PWPT_{D2}$ (57.85 ± 11.41vs 51.07 ± 7.39, $p < 0{,}001$) and the rate of abnormal P wave axis (34.3% vs 12.9%, $p = 0.003$). The presence of a P-terminal force >0.04 mm/s, was shown to be significantly more frequent in the AF group (46.8% vs 23.4%, $p = 0.003$). There was no statistically difference between the groups in terms of average P wave axis. Other electrocardiographic features were shown in the table (Table 2). In addition PWPT was significantly longer

**Table 1**
Baseline characteristics, laboratory and echocardiographic findings of the groups.

| Variables | Atrial fibrillation(+) (n: 70) | Atrial fibrillation(−) (n: 70) | p |
|---|---|---|---|
| Baseline characteristics | | | |
| Age (years), mean (SD) | 65.87 ± 10.80 | 51.41 ± 15.58 | <**0.001** |
| Gender (female), n (%) | 39(55.7%) | 42(60%) | 0.608 |
| Current Smoker, n (%) | 19(27.1%) | 18(25.7%) | 0.848 |
| Coronary Artery disease, n(%) | 19(27.1%) | 11(15.7%) | 0.099 |
| Hypertension, n (%) | 40(57.1%) | 31(44.3%) | 0.128 |
| Diabetes mellitus, n (%) | 15(21.4%) | 21(30%) | 0.246 |
| Cerebrovascular disease, n (%) | 15(21.4%) | 2(2.9%) | **0.001** |
| Laboratory findings | | | |
| Glucose (mg/dl; SD) | 114.35 ± 42.09 | 108.03 ± 33.35 | 0.355 |
| Sodium (mmol/dl; SD) | 139.27 ± 3.11 | 139.03 ± 1.96 | 0.612 |
| Potassium (mmol/dl; SD) | 4.45 ± 0.48 | 4.41 ± 0.36 | 0.584 |
| Calcium (mg/dl; SD) | 9.39 ± 0.51 | 9.44 ± 0.47 | 0.617 |
| Magnesium (mg/dl; SD) | 1.97 ± 0.27 | 2.03 ± 0.31 | 0.873 |
| BUN (mg/dl; SD) | 38.52 ± 11,36 | 30.51 ± 11.51 | <**0.001** |
| Creatinine (mg/dl; SD) | 0.93 ± 0.27 | 0.80 ± 0.16 | **0.002** |
| HDL-C (mg/dl; SD) | 48.63 ± 16.92 | 49.06 ± 13.54 | 0.877 |
| LDL-C (mg/dl; SD) | 126.80 ± 35.74 | 134.28 ± 31.51 | 0.226 |
| Triglyceride (mg/dl; SD) | 133.86 ± 67.61 | 138.93 ± 54.33 | 0.656 |
| WBC ($x10^3$/μL; SD) | 7.36 ± 2.45 | 7.71 ± 2.09 | 0.380 |
| Hemoglobin (g/dL; SD) | 13.01 ± 1.53 | 13.16 ± 1.66 | 0.608 |
| Hematocrit, n (%;SD) | 39.57 ± 4.49 | 39.39 ± 4.29 | 0.815 |
| Platelets ($x10^3$/μL; SD) | 247.50 ± 70.77 | 282.58 ± 71.93 | **0.006** |
| RDW% | 14.43 ± 2.45 | 13.95 ± 1.51 | 0.185 |
| TSH(uIU/mL) | 1.84 ± 1.86 | 1.65 ± 0.93 | 0.483 |
| Echocardiographic findings | | | |
| Left ventricular ejection fraction (%) | 57.61 ± 10.19 | 60.60 ± 4.68 | 0.051 |
| Moderate or severe heart valve disorder, n(%) | 8(14%) | 3(4.9%) | 0.089 |
| Left ventricular hypertrophy, n(%) | 16(22.9%) | 9(13%) | 0.132 |
| Interventricular septum thickness, mm | 11.62 ± 5.10 | 10.45 ± 1.38 | 0.067 |
| LAAPD, mm | 38.72 ± 5.26 | 33.17 ± 3.50 | <**0.001** |
| LVEDD, mm | 48.01 ± 5.15 | 46.93 ± 3.19 | 0.139 |
| Ascending aorta, mm | 34.82 ± 4.13 | 32.95 ± 4.14 | **0.008** |

*Independent samples *t*-test, chi-square test, Fisher's exact test *$p < 0.05$ statistically significant. Continues variables are reported mean ± SD. Categorical variables are reported n(%).
Abbreviations: BUN; blood urea nitrogen, HDL-C; high density lipoprotein cholesterol, LDL-C; low density lipoprotein cholesterol, WBC; white blood cell, RDW; red cell distribution width, TSH; thyroid stimulating hormone, LAAPD; left atrium anteroposterior diameter, LVEDD; left ventricular end diastolic diameter.

**Table 2**
ECG findings of the groups.

| ECG findings | Atrial fibrillation (+) (n: 70) | Atrial fibrillation (−) (n: 70) | p |
|---|---|---|---|
| P wave parameters | | | |
| PR, msc | 168.40 ± 40.06 | 151.26 ± 20.80 | **0.003** |
| P wave dispersion, msc | 51.47 ± 6.83 | 47.44 ± 7.15 | **0.001** |
| P wave max time, msc | 121.20 ± 22.15 | 109.77 ± 15.53 | **0.001** |
| P wave peak time D2, msc | 57.85 ± 11.41 | 51.07 ± 7.39 | <**0.001** |
| P wave peak time V1, msc | 56.32 ± 8.26 | 48.60 ± 6.45 | <**0.001** |
| Abnormal P wave axis, n(%) | 23(34.3%) | 9(12.9%) | **0.003** |
| Biphasic P wave (+/−), n(%) | 11(15.7%) | 8(11.4%) | 0.459 |
| Average P wave axis° | 43.68 ± 34.47 | 43.75 ± 26.45 | 0.989 |
| V1TF >40, n(%) | 34(46.8%) | 17(23.4%) | **0.003** |
| Other basic ECG parameters | | | |
| Heart rate, beat/min | 73.30 ± 11.45 | 75.24 ± 13.81 | 0.367 |
| QRS, msc | 92.22 ± 12.05 | 89.28 ± 9.48 | 0.111 |
| QT, msc | 397.05 ± 31.13 | 365.71 ± 31.49 | <**0.001** |
| QTc, msc | 427.82 ± 30.99 | 416.11 ± 27.76 | **0.020** |
| LAH, n(%) | 15(21.4%) | 7(10.1%) | 0.068 |
| LPH, n(%) | 5(7.1%) | 7(10.1%) | 0.529 |
| ST segment-T wave changes, n (%) | 10(14.2%) | 5(7.1%) | 0.181 |

Independent samples *t*-test, continues variables are reported mean ± SD, $p < 0.05$ statistically significant.
Abbreviations: V1TF; P-wave terminal force in lead V1, LAH; Left anterior hemiblock, LPH; Left posterior hemiblock.

**Table 3**
Comparison of PWPT in subgroups formed according to the presence of biphasic P waves.

| | Atrial fibrillation (+) (n: 59) | Atrial fibrillation (−) (n: 62) | p |
|---|---|---|---|
| $PWPT_{V1}$, msc (except biphasic) | 54.57 ± 7.13 | 47.59 ± 5.90 | <**0.001** |
| | Atrial fibrillation (+) (n: 11) | Atrial fibrillation (−) (n: 8) | p |
| $PWPT_{V1}$, msc (only biphasic) | 65.72 ± 7.79 | 56.37 ± 5.31 | **0.009** |

Independent samples *t*-test, continues variables are reported mean ± SD, p < 0.05 statistically significant.
Abbreviations: $PWPT_{V1}$; P-wave peak time in lead V1.

in AF(+) patients, in both subgroups with only biphasic P waves and no biphasic P waves (Table 3).

Among the P wave parameters to be different between groups were evaluated with the logistic regression analysis. Multivariable regression analysis was performed to determine independent predictors of AF found to be significant in univariate analyses. PR duration(OR:1.01, $p = 0.038$), P wave dispersion(OR:1.07, $p = 0.037$), $PWPT_{V1}$ (OR:1.09, $p = 0.024$), abnormal P wave axis(OR:5.96, $p = 0.002$) and V1TF >40 (OR:2.94, $p = 0.027$) were found to be independent predictors for AF. Independent predictors of AF were shown in the table (Table 4).

In ROC curve analysis; Area Under Curve (AUC) was 0.737 for $PWPT_{V1,}$ 0.661 for P wave dispersion, 0.638 for PR duration, 0.605 for Abnormal P wave axis and 0.639 for V1TF (Fig. 2). In the comparison of ROC Curve analysis, the $PWPT_{V1}$ was found superior to the PWTF for predicting AF risk among whole study population ($p = 0.006$) (Fig. 3). $PWPT_{V1,}$ above a cut-off level of 49.5msc. predicted AF with a sensitivity of 79.4% and a specificity of 56.3% (Table 5).

## Discussion

AF is the most common arrhythmia in adults and is associated with significant morbidity and mortality. Substantial interest has developed in the primary prevention and the identification of individuals at risk for developing AF. The ECG provides a great deal of information on AF risk and has the potential to contribute substantially to AF risk estimation, but more research is needed [9]. To our best knowledge, the present study is the first in the literature about the PWPT and AF. Our study demonstrated that prolonged PWPT which was obtained from V1 lead

**Table 4**
Regression analysis of potential prognostic factors on the presence of atrial fibrillation.

| | Univariate analysis | | Multivariable analysis | |
|---|---|---|---|---|
| Variables | OR (95% CI) | *p* value | OR (95% CI) | p value |
| Atrial fibrillation | | | | |
| PR, msc | 1.01(1.00–1.03) | **0.005** | 1.01(1.00–1.002) | **0.038** |
| P wave dispersion, msc | 1.08(1.03–1.14) | **0.001** | 1.07(1.00–1.14) | **0.037** |
| P wave max time, msc | 1.03(1.01–1.05) | **0.001** | 1.01(0.99–1.09) | 0.156 |
| P wave peak time, msc D2 | 1.07(1.03–1.12) | <**0.001** | 1.03(0.97–1.09) | 0.267 |
| P wave peak time, msc V1 | 1.14(1.08–1.21) | <**0.001** | 1.09(1.01–1.17) | **0.024** |
| Abnormal P wave axis, n(%) | 3.54(1.49–8.39) | **0.004** | 5.96(1.89–18.78) | **0.002** |
| V1TF >40, n(%) | 2.94(1.43–6.04) | **0.003** | 2.94(1.31–7.67) | **0.027** |

Abbreviations: OR; odds ratio, ci; confidence interval, V1TF; P-wave terminal force in lead V1.

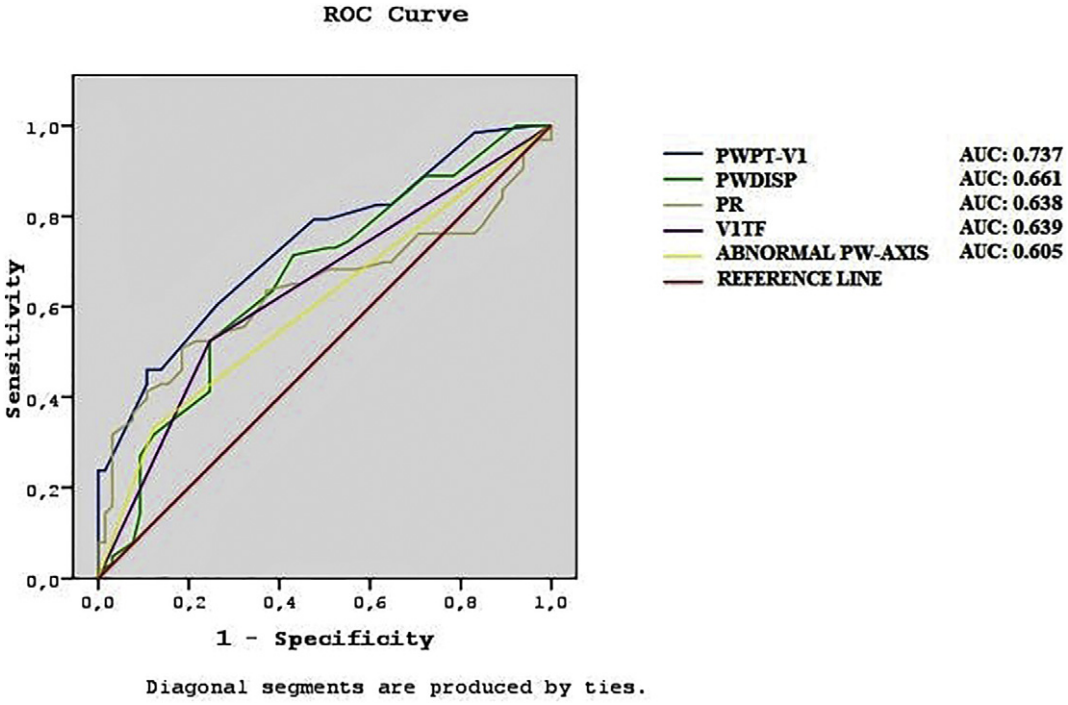


**Fig. 2.** ROC curves of the statistically significant parameters in regression analysis.

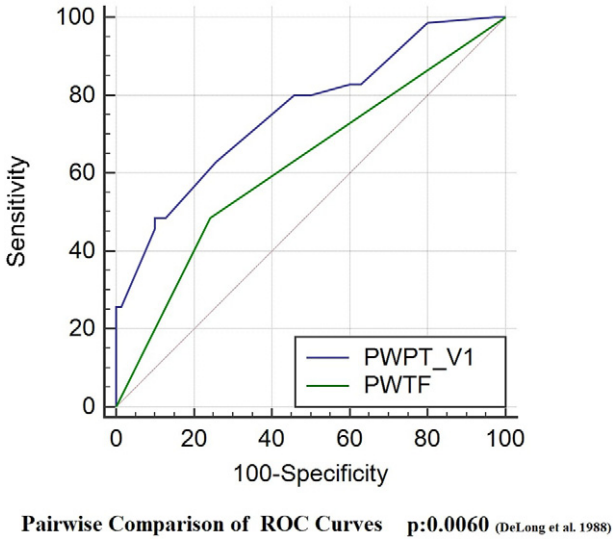


**Fig. 3.** Comparison of ROC Curves of the $PWPT_{V1}$ and $PWTF_{V1}$.

was significantly associated with AF and it was found to be an independent predictor of AF.

ECG remains the inexpensive, readily accessible and most routinely used test for the evaluation of cardiovascular disease. As opposed to manual measurements, the computerized ECG provides quantifiable and easily reproducible measurements [2]. A normal cardiac impulse starts in the sinoatrial node and then spreads throughout the atrial myocardium, thus defining the P wave in the ECG. The P wave indices are quantitative measures of atrial electrical function derived from the surface ECG. These measurements have the advantage of characterizing atrial electrical activity during depolarization without assessing other features of cardiac electrophysiology such as conduction delay through the atrioventricular (AV) node [10]. Studies showing the relationship between P wave and AF are increasing and within this context, P-wave analysis is becoming more and more useful for gathering information about the predisposition of patients to AF, whom could benefit from preventive tailored treatments [11].

The previous studies showed that a P-wave terminal force >0.04 mm/s was an independent predictor of AF [12]. The P-wave terminal force is the algebraic product of the duration and depth of the negative terminal part of the P wave in lead V1. In our study, we confirmed the relative strength of association between P-wave terminal force and AF. However in another study, among patients without atrial fibrillation history, P-wave terminal force was not predictive of atrial fibrillation detected after stroke [13]. On the other hand, P-wave duration is generally accepted as the most reliable noninvasive marker of atrial conduction, and its prolongation has been associated with a history of AF. But in some reports, patients with lone paroxysmal AF failed to demonstrate any impressive P-wave prolongation [13–15]. Platonov et al. found that the unfiltered signal-averaged P wave ECG revealed significant differences in P wave duration in patients with lone PAF, but surprisingly no differences in filtered P wave duration [16]. Therefore, new ECG parameters needed to be examine. Also, the relationship between AF and P wave dispersion [1,3,17], abnormal P wave axis [9,17,18], PR interval [9,19], QT and QTc [20,21] intervals have been shown in previous studies. All these noninvasive markers of AF may also help monitor response to antiarrhythmic therapy or catheter-based interventions and identify patients who are likely to have progression of atrial disease. Our study also showed similar ECG results in accordance with the literature and the incidence of AF increased with age in our patients, as has been shown in previous studies [22]. Only opposite to Nguyen KT et al.'s study, we did not observe any relationship between AF and the left anterior fascicular block [21]. However, in addition to all other studies, this is the first report to reveal the relationship between PWPT and AF in the literature.

**Table 5**
ROC analysis.

| Variables | AUC | p | Sensitivity | Specificity | Cut of |
|---|---|---|---|---|---|
| P wave peak time, msc V1 | 0.737 | <**0.001** | 79.4% | 56.3% | 49.5 |
| P wave dispersion, msc | 0.661 | **0.002** | 71.4% | 56.9% | 48.5 |
| PR, msc | 0.638 | **0.007** | 63.5% | 63.1% | 154.5 |
| Abnormal P wave axis, n(%) | 0.605 | **0.040** | – | – | – |
| V1TF, n(%) | 0.639 | **0.007** | – | – | – |

Abbreviations: ROC; Receiver operating characteristics, AUC; Area under the curve.

Structural remodelling, including left atrial (LA) enlargement, is a well known predictor of AF recurrence. Sgrigna V. et al. showed that the correlation between P wave duration and LA dimensions in patients with mitral valve stenosis who affected by episodes of AF. Also in this study, the researchers showed that P-wave duration and the amplitudes were the most distinctive parameters to predict who are at risk of developing paroxysmal AF [23]. In this study, the demonstration of P wave reaches its maximum amplitude significantly later in patients with paroxysmal AF is an important finding and supports to our study. On the other hand AF has also been reported to be associated with slow LA conduction, even without detectable structural remodelling. Fukushima K et al. reported that, prolonged peak A'-wave on the tissue Doppler imaging associated with an increased risk of new-onset AF and of AF recurrence [24]. Also, the relationship with peak atrial contraction strain and AF was demonstrated [25]. In fact, we thought it would be appropriate to evaluate a similar parameter in ECG might be more practically than tissue doppler and strain echocardiography. Short time ago, the relationship between PWPT and impaired left atrial function due to coronary artery disease has recently been demonstrated by Cagdas M et al. [9]. Also in another study, it was found that reduction of the P wave peak (amplitude of the p wave) was associated with decrease of the AF recurrence after cryoballoon [26]. Although the parameter is not the same as our study, it is important in terms of revealing AF and p wave peak point relation. As a result of all these studies, we thought that PWPT could be a useful parameter to predict AF.

Prolongation of the PWD in the case of left atrial enlargement is well known for a long time and also it was known that, not only in the setting of chronic process but also in acute atrial stretch, P wave duration could be prolonged due to slowing in atrial conduction [27]. Our study revealed that PWPT is more sensitive than this classical and well known parameter for predicting AF. One of the most important points of this study is, it evaluate P wave peak time instead of P wave maximum time and shows that it is a more sensitive parameter. Another important point is that PWPT is more practical and easy to calculate than parameters such as PWDISP, abnormal P wave axis and P-wave terminal force. Therefore, we consider that the PWPT may be helpful as it may be detected rapidly and easily. This parameter help physicians to be much more careful with regard to AF development and preventive measures could start early. We think this study is important in terms of bringing a new perspective on this issue.

## Study limitations

Our study had some limitations; these were the single-center design, and the relatively lower number of patients are the most important limitations. Also we performed some of the ECG measurements using computer software even though computerized measurements may differ from the measurements made by human eye. We assessed P-terminal force by the Morris index (P-terminal force >0.04 mm/s), we could not reveal the cut off value of P-terminal force. In ROC curve analysis $PWPT_{V1}$ predicted AF with a moderate sensitivity (79.4%) and low specificity (56%). It is a serious limitation to include this parameter for every case in clinical practice. Another important limitation of the study that, P wave morphology varies in lead V1. Biphasic morphologies in this lead may affect the measurement and may be responsible for low specificity.

## Conclusion

In this study, we observed that $PWPT_{V1}$ is longer in patients with paroxysmal AF than in controls. PWPT is practical, easy for calculating and implementation in clinical practice and can help us to predict AF. This novel parameter can be used to identify patients at high risk of developing AF who may benefit from prophylactic anticoagulant or antiarrhythmic therapy to prevent strokes and hospitalizations. The most important limitation of the study that, P wave morphology varies in lead V1 and biphasic morphologies may affect the measurements. Therefore PWPT as a marker should not be applied indiscriminately to P waves without assessing their morphology in lead V1. Nonetheless, further studies with larger sample size are needed for a consensus and clear results.

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## Declaration of competing interest

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