



Electrocardiographic Predictors of Early Atrial Fibrillation After Transcatheter Atrial Septal Defect Closure: The Roles of P-Wave Dispersion and P-Terminal Force in V1

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ABSTRACT

Objective: Early atrial fibrillation (AF) can complicate transcatheter atrial septal defect (ASD) closure, yet simple electrocardiographic predictors remain underused. We investigated whether baseline P-wave dispersion (PWDIs) and P-terminal force in V1 (PTFV1) predicted AF within 48 hours post-procedure.

Methods: We conducted a retrospective, two-center cohort study including 555 consecutive adults (18-85 years) who underwent successful percutaneous secundum ASD closure between March 2015 and September 2024. Patients with prior AF, coronary artery disease, failed procedures, emergent surgery, in-hospital death within 48 hours, suboptimal records, or unavailable preprocedural ECGs were excluded. Baseline 12-lead ECGs (25 mm/s, 10 mm/mV; 0.05-100 Hz) were digitized and measured at 4× magnification. PWDIs was calculated as the difference between maximum and minimum P-wave duration across 12 leads; PTFV1 was defined as the product of the duration and amplitude of the terminal negative P-wave component in V1 (μV.ms).

Results: Of 555 patients, 27 (4.9%) developed AF within 48 hours. Patients with AF were older (59.6±13.1 vs. 37.9±14.7 years, $p<0.001$) and more often NYHA class II ($p<0.001$). On ECG, PWDIs and PTFV1 were significantly higher in the AF group (PWDIs 42.8±13.9 vs. 36.4±11.8 ms, $p=0.007$; PTFV1 4325.9±730.4 vs. 3876.5±656.1 μV.ms, $p=0.001$). Both PWDIs [odds ratio (OR) 1.036; 95% confidence interval (CI) 1.004-1.068; $p=0.025$] and PTFV1 (OR 1.086, 95% CI 1.027-1.149; $p=0.004$) independently predicted AF. ROC analysis identified PTFV1 >4,050 μV.ms and PWDIs >36.5 ms as predictors of early AF.

Conclusion: Preprocedural PWDIs and PTFV1 independently predict early AF after ASD device closure. These ubiquitous, low-cost ECG indices may help risk-stratify candidates and tailor peri-procedural monitoring and prophylaxis strategies.

Keywords: Atrial septal defect, atrial fibrillation, P-wave dispersion, P-terminal force, noninvasive electrocardiology, transcatheter closure

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INTRODUCTION

Adults with an unrepaired secundum atrial septal defect (ASD) are predisposed to atrial arrhythmias due to chronic right atrial (RA) volume overload, progressive atrial dilation, and electrical remodeling, all of which contribute to the development of an arrhythmogenic substrate over time.^{1,3} Although transcatheter ASD closure effectively eliminates the left-to-right shunt and reduces right-sided volume overload, early and late atrial tachyarrhythmias continue to occur following the procedure, particularly in older patients, those with delayed defect closure, and those with advanced atrial remodeling prior

to the procedure.⁴⁻⁶ Previous studies have suggested that persistent electrophysiological abnormalities may remain despite successful anatomical correction, indicating that eliminating the hemodynamic burden does not necessarily reverse established atrial remodeling.^{7,9}

Recent evidence has further expanded the concept of atrial remodeling to encompass the broader entity of atrial cardiomyopathy, which includes structural, electrical, contractile, and autonomic abnormalities that predispose patients to atrial fibrillation (AF), thromboembolic events, and adverse



cardiovascular outcomes, even before clinically overt arrhythmia develops.¹⁰ Consequently, identifying simple, reproducible, and widely available markers that reflect the underlying atrial substrate has become an important clinical objective. Surface electrocardiography (ECG) is an inexpensive and universally accessible tool that provides valuable information on atrial conduction abnormalities without requiring advanced imaging techniques.^{11,12}

Among ECG-derived parameters, P-wave dispersion (PWD_{is}) defined as the difference between the maximum and minimum P-wave durations across the 12-lead ECG reflects heterogeneous atrial conduction and has consistently been associated with paroxysmal AF, atrial conduction delay, and recurrent arrhythmias in various cardiovascular conditions.¹³⁻¹⁷ Likewise, P-terminal force in lead V1 (PTFV1), originally described by Morris and colleagues, quantifies the duration and amplitude of the terminal negative portion of the P wave in that lead and has emerged as a robust electrocardiographic marker of left atrial electrical and structural remodeling. A growing body of evidence has demonstrated significant associations between abnormal PTFV1 and incident AF, ischemic stroke, atrial cardiomyopathy, and cardiovascular mortality across diverse patient populations.¹⁸⁻²¹

Despite the growing body of evidence supporting ECG-derived atrial biomarkers, data regarding the predictive value of PWD_{is} and PTFV1 specifically in patients undergoing transcatheter ASD closure remain limited. Because early post-procedural AF may prolong hospitalization, increase the risk of thromboembolism, and necessitate additional rhythm management, identifying patients at increased risk prior to the procedure may facilitate individualized peri-procedural monitoring and preventive strategies. Therefore, the present study aimed to evaluate whether baseline PWD_{is} and PTFV1 independently predict early (≤ 48 -hour) AF following successful percutaneous secundum ASD closure.

METHODS

Ethics committee approval for the study was obtained from the Non-Interventional Clinical Research Ethics Committee of İzmir Bakırçay University (decision number: 2105; date: 7 March 2025). Written informed consent was not obtained because of retrospective design.

We conducted a retrospective cohort study at two tertiary centers. We included 555 consecutive adults (18-85 years) who underwent successful percutaneous secundum ASD closure between March 2015 and September 2024 at two cardiology clinics. Exclusion criteria were prior AF, any failed procedure, need for urgent cardiac surgery, known coronary artery disease, death within 48 hours of the procedure, suboptimal records, or unavailable preprocedural ECGs. Demographics, baseline 12-lead ECGs, transthoracic echocardiography, laboratory values, procedural details, and

occluder device characteristics were extracted from hospital records. During hospitalization, all ECGs were acquired in the supine position at 25 mm/s and 10 mm/mV with 0.05-100 Hz filter settings, scanned and digitized, and analyzed at 4× magnification on a high-resolution monitor. All patients were continuously monitored with in-hospital telemetry during the first 24 hours following the procedure. During the subsequent 24 hours, patients were evaluated with standard 12-lead ECG recordings in the presence of symptoms or rhythm irregularities detected by bedside monitoring systems or wearable devices. AF was defined as an irregularly irregular rhythm without discernible P waves lasting ≥ 30 seconds on surface ECG. PWD_{is} was calculated as the difference between the maximum and minimum P-wave durations across all 12 leads. PTFV1 was defined as the product of the duration and amplitude of the terminal negative P-wave component in lead V1 ($\mu\text{V}\cdot\text{ms}$) (Figure 1). AF within the first 48 hours post-procedure was diagnosed by surface ECG or Holter monitoring. Patients without AF were classified as Group 1, and those with AF were classified as Group 2. ECG measurements were performed by two independent cardiologists who were blinded to clinical outcomes.

Statistical Analysis

Normality of continuous variables was assessed using the Kolmogorov-Smirnov test. Continuous variables are reported as mean $\pm$ standard deviation and categorical variables as counts and percentages. Group comparisons were performed using Student's t-test or Mann-Whitney U test for continuous variables, and chi-square or Fisher's exact test for categorical variables. Logistic regression identified independent ECG predictors of AF. Optimal cut-offs were derived by receiver-operating characteristic (ROC) analysis. Statistical significance was set at $p < 0.05$.

RESULTS

Of 555 patients, 175 (31.5%) were male and 380 (68.5%) female; mean age was 39.1 ± 15.3 years (range 18-85). At baseline, 510 (91.9%) patients were NYHA class I and 45 (8.1%) were class II. All procedures were technically successful; however, 27 patients (4.9%) developed AF within 48 hours. Patients with AF were



Figure 1. P-wave terminal force measurement in lead V1 (PTFV1) = (a) P' duration $\times$ (b) P' amplitude

PTFV1: P wave terminal force in lead V1

significantly older (59.6 ± 13.1 vs. 37.9 ± 14.7 years, $p < 0.001$), and AF was more common in NYHA class II ($p < 0.001$). During the study period, no patients with NYHA class III-IV were deemed suitable for transcatheter closure. Mean ASD diameter was 15.8 ± 5.7 mm; mean occluder size 21.5 ± 6.2 mm. Mean LVEF was $61.7 \pm 3.2\%$, mean sPAP 42.4 ± 16.6 mmHg, and mean Qp/Qs 2.2 ± 0.6 . There were no between-group differences in echocardiographic or laboratory parameters (all $p > 0.05$) (Tables 1 and 2). Mean heart rate was 70.8 ± 9.2 bpm; mean PWDIs 36.7 ± 11.9 ms; mean PTFV1 3904 ± 666 $\mu\text{V}\cdot\text{ms}$. Compared with Group 1, Group

2 had greater PWDIs (42.8 ± 13.9 vs. 36.4 ± 11.8 ms, $p = 0.007$) and higher PTFV1 (4325.9 ± 730.4 vs. 3876.5 ± 656.1 $\mu\text{V}\cdot\text{ms}$, $p = 0.001$) (Table 1). In multivariable models, both PWDIs [odds ratio (OR) 1.036; 95% confidence interval (CI) 1.004-1.068; $p = 0.025$] and PTFV1 (OR 1.086; 95% CI 1.027-1.149; $p = 0.004$) independently predicted early AF (Table 3). ROC analysis yielded cut-offs for PTFV1 ($> 4,050$ $\mu\text{V}\cdot\text{ms}$; AUC 0.681; sensitivity 63%; specificity 62%) and for PWDIs (> 36.5 ms; AUC 0.634; sensitivity 57%; specificity 61%) (Figure 2). ROC analysis showed modest discriminatory performance.

Table 1. Baseline clinical, electrocardiographic and echocardiographic characteristics

Variables (mean ± SD)		Group 1 n=528	Group 2 n=27	p value
Age, years (mean ± SD)		37.9±14.7	59.6±13.1	<0.001
Male gender, n (%)		164 (31.1)	11 (40.7)	0.291
HT, n (%)		13 (2.5)	2 (7.4)	0.162
DM, n (%)		6 (1.1)	1 (3.7)	0.296
NYHA class	1	494 (93.6)	16 (59.2)	<0.001
	2	34 (6.4)	11 (40.8)	
Echocardiographic parameters				
ASD diameter (mm)		15.8±5.5	16.1±8.8	0.759
Device size (mm)		21.4±6.1	23.1±8.2	0.185
LVEDD (mm)		41.9±7.2	44.2±8.1	0.115
LVESD (mm)		28.9±5.9	28.8±6.4	0.897
LA (mm)		36.5±5.1	34.6±2.9	0.233
RA (mm)		32.8±8.6	31.5±3.5	0.619
LVEF (%)		61.7±3.2	61.9±3.1	0.806
RVEDD (mm)		35.6±9.1	36.6±10.5	0.653
SPAB (mmHg)		42.4±16.8	43.9±13.9	0.666
RV/LV ratio		0.85±0.2	0.91±0.3	0.336
RA/LA ratio		0.91±0.2	0.92±0.4	0.924
Qp/Qs ratio		2.2±0.6	2.3±0.4	0.609
Electrocardiographic parameters				
Heart rate (bpm)		71.2±9.3	68.2±8.4	0.309
Maximum P wave duration (ms)		101.5±10.2	105.1±10.7	0.076
Minimum P wave duration (ms)		65.1±7.4	62.2±8.8	0.056
P wave dispersion (ms)		36.4±11.8	42.8±13.9	0.007
PTFV1 (μV.ms)		3876.5±656.1	4325.9±730.4	0.001
SD: Standard deviation, n: Number of patients, ASD: Atrial septal defect, HT: Hypertension, LA: Left atrium, LV: Left ventricle, LVEDD: Left ventricular end-diastolic diameter, LVEF: Left ventricular ejection fraction, LVESD: Left ventricular end-systolic diameter, DM: Diabetes mellitus, NYHA: New York Heart Association classification, PTFV1: P wave terminal force in lead V1, RA: Right atrium, RV: Right ventricle, RVEDD: Right ventricular end-diastolic diameter, SPAB: Systolic pulmonary artery pressure				

Table 2. Laboratory characteristics of patients according to groups			
Variables (mean ± SD)	Group 1 n=528	Group 2 n=27	p value
Urea, mg/dL	27.5±10.8	30.3±6.4	0.426
Creatinine, mg/dL	0.7±0.2	0.65±0.1	0.212
Sodium, mEq/L	139.4±2.5	139.4±2.9	0.951
Potassium, mEq/L	4.3±0.4	4.2±0.4	0.686
Calcium, mg/dL	9.2±0.4	8.9±0.6	0.088
WBC, ×10 ⁹ /L	7.76±2.1	8.21±1.1	0.685
Neutrophil count, ×10 ⁹ /L	4.75±1.8	4.9±0.8	0.879
Lymphocyte count, ×10 ⁹ /L	2.14±0.7	2.37±0.4	0.506
Hemoglobin, g/dL	13.3±1.7	13.6±1.9	0.419
Hematocrit, %	40.1±5.5	40.5±7.1	0.777
Platelet count, ×10 ⁹ /L	239.18±89.2	282.55±48.4	0.159
SD: Standard deviation, n: Number of patients, WBC: White blood cell count			

Table 3. Effects of ECG indicators on the development of atrial fibrillation in univariate and multivariate logistic regression analysis				
Variables	Univariate logistic regression		Multivariate logistic regression	
	OR (95% CI)	p value	OR (95% CI)	p value
Maximum P wave duration	1.034 (0.996-1.074)	0.078		
Minimum P wave duration	0.951 (0.904-1.001)	0.056		
P wave dispersion	1.042 (1.011-1.074)	0.008	1.036 (1.004-1.068)	0.025
PTFV1	1.096 (1.038-1.157)	0.001	1.086 (1.027-1.149)	0.004
PTFV1: P wave terminal force in lead V1, CI: Confidence interval, OR: Odds ratio				

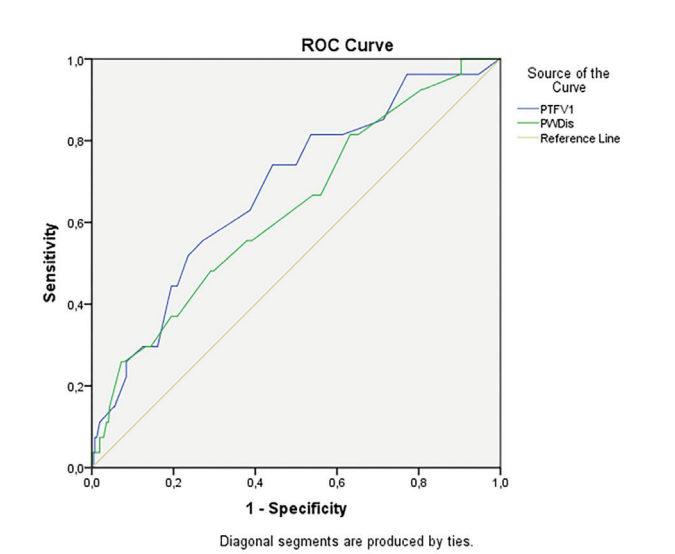


Figure 2. Receiver operating characteristic (ROC) curve analysis of the optimal cut-off values of P-wave dispersion and P-wave terminal force in lead V1

PWDIs: P-wave dispersion, PTFV1: P wave terminal force in lead V1

DISCUSSION

In this two-center cohort of adults undergoing transcatheter secundum ASD closure, we demonstrated that two readily obtainable ECG-derived parameters, PWDIs and PTFV1, independently predicted early AF within 48 hours after the procedure. Although the incidence of early AF was relatively low, patients who developed AF exhibited significantly more pronounced abnormalities in both ECG markers measured before intervention, suggesting that pre-existing atrial electrical remodeling, rather than the procedure itself, is the principal determinant of early arrhythmogenesis. These findings support the growing concept that conventional surface ECG may provide clinically meaningful information regarding atrial substrate before structural heart interventions.

The biological plausibility of our findings is supported by the well-established pathophysiology of ASD. Chronic left-to-right shunting exposes the right atrium to prolonged volume overload, resulting in atrial dilatation, myocyte stretch, neurohormonal activation, inflammation, and progressive fibrosis. These changes alter conduction velocity and increase conduction heterogeneity, facilitating re-entry and AF. Importantly, several experimental and clinical studies have demonstrated that electrical remodeling may persist despite successful defect closure, particularly in adults undergoing late intervention.²²⁻²⁶ Consequently, elimination

of the hemodynamic burden does not necessarily normalize the pre-existing arrhythmogenic substrate.

PWD_{is} reflects heterogeneous atrial conduction and has consistently been associated with AF in several cardiovascular disorders.²⁷⁻³¹ Increased dispersion indicates delayed and non-uniform atrial activation, thereby facilitating wavefront fragmentation and re-entry. Our results extend previous observations by demonstrating that PWD_{is} remains clinically relevant in patients undergoing transcatheter ASD closure, indicating that atrial conduction abnormalities persist despite correction of the underlying congenital lesion.

PTFV1 has emerged as one of the most robust ECG markers of atrial cardiomyopathy. Unlike simple P-wave duration, PTFV1 reflects both delayed atrial activation and increased left atrial electrical load, and correlates with atrial fibrosis, AF, stroke, and cardiovascular outcomes.³²⁻³⁵ Although ASD primarily affects the right heart, long-standing interatrial interactions and biatrial remodeling may explain why an index reflecting left atrial abnormalities independently predicted early AF in our cohort. These findings further support the concept that atrial electrical remodeling in ASD represents a biatrial, rather than an exclusively RA, process.

The incidence of early AF in our cohort (4.9%) is comparable to that reported in previous studies evaluating transcatheter ASD closure, in which post-procedural AF rates have generally ranged between approximately 2% and 7%, depending on patient age, timing of rhythm assessment, and duration of follow-up. Older age has consistently emerged as one of the strongest determinants of post-closure atrial arrhythmias, supporting the concept that the burden of pre-existing atrial remodeling, rather than the procedure itself, primarily drives arrhythmia occurrence.^{4,5,22,23} The present findings extend these observations by demonstrating that ECG-derived markers of atrial electrical remodeling provide additional prognostic information beyond age alone.

The modest ROC performance observed in our analysis should not diminish the clinical value of these markers. AF is a multifactorial disease, and excellent discrimination based on a single ECG variable is not expected. Instead, PWD_{is} and PTFV1 should be regarded as complementary biomarkers that may improve prediction when integrated with age, atrial dimensions, clinical variables and procedural characteristics.

Another noteworthy finding of the present study is that neither echocardiographic parameters nor routine laboratory parameters differed significantly between patients who developed early AF and those who remained in sinus rhythm, whereas both ECG-derived indices retained independent predictive value. This observation suggests that subtle electrical remodeling may precede overt structural abnormalities detectable by conventional imaging. Similar findings have been reported in previous studies, which demonstrate that electrocardiographic markers often identify atrial vulnerability before measurable

changes in atrial size or function become evident.^{10,18,20} Accordingly, PWD_{is} and PTFV1 should not be regarded merely as surrogate ECG measurements, but rather as noninvasive indicators of the underlying atrial substrate. This concept is particularly relevant in adults with ASD, in whom chronic atrial remodeling develops gradually over many years and may persist despite successful defect closure. Therefore, the incorporation of ECG-derived atrial biomarkers into routine preprocedural assessment may complement conventional echocardiographic evaluation and improve the identification of patients at increased risk of early post-procedural AF.

From a practical perspective, both PWD_{is} and PTFV1 are inexpensive, universally available, and require only a standard 12-lead ECG. Identification of high-risk patients before ASD closure may justify closer telemetry surveillance, aggressive correction of reversible peri-procedural triggers, and consideration of prolonged ambulatory rhythm monitoring after discharge. Such individualized management may facilitate earlier AF detection and timely initiation of appropriate therapy.

Although early AF may be clinically relevant in the peri-procedural setting, it likely reflects a combination of pre-existing atrial substrate and transient triggers such as procedural inflammation, autonomic imbalance, and acute hemodynamic shifts. Therefore, our results do not necessarily translate to medium- or long-term arrhythmia burden.

Study Limitations

Some limitations of this study should be acknowledged. First, the retrospective design has limitations. Second, rhythm monitoring was performed continuously via telemetry during the first 24 hours after the procedure, while AF detection during the subsequent 24-hour period relied on bedside monitoring systems and symptom-triggered ECG recordings. Although this monitoring strategy reflects routine clinical practice in many institutions, it may have failed to detect short-term or asymptomatic AF episodes. Third, PWD_{is} and PTFV1 measurements were obtained from digitized ECG recordings by manually defining the onset and end of the P wave. Although all measurements were performed at high magnification by two independent cardiologists blinded to clinical outcomes, manual ECG analysis may be subject to measurement variability. Finally, the relatively small number of AF events limited the complexity of multivariate modeling and may have increased the risk of model instability or overfitting. Therefore, the number of predictors included in the regression model was intentionally limited. Accordingly, the findings should be interpreted with caution and primarily considered hypothesis-generating. Furthermore, ROC analyses demonstrated only modest discriminatory performance, suggesting that these ECG indices alone are insufficient for clinical decision-making but may contribute as complementary markers within a broader risk assessment framework. Taken

together, our findings should be considered hypothesis-generating and should be confirmed in larger prospective studies with extended rhythm follow-up.

The present findings also emphasize that ECG-derived atrial biomarkers should be interpreted as markers of the underlying atrial substrate rather than isolated predictors of arrhythmia. Although the predictive performance of PWDIs and PTFV1 was modest, their widespread availability, low cost, and ease of measurement make them attractive adjunctive tools for identifying patients who may benefit from intensified rhythm surveillance after transcatheter ASD closure.

CONCLUSION

Baseline PWDIs and PTFV1 independently predict early AF after percutaneous ASD closure. These readily obtainable ECG indices may enhance risk stratification and inform peri-procedural management in contemporary practice.

Ethics

Ethics Committee Approval: Ethics committee approval for the study was obtained from the Non-Interventional Clinical Research Ethics Committee of İzmir Bakırçay University (decision number: 2105; date: 7 March 2025).

Informed Consent: Written informed consent was not obtained because of retrospective design.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Y.D., S.S., O.Ş., Concept: A.A.B., Design: Y.D., O.Ş., Data Collection or Processing: Y.D., A.A.B., S.S., Analysis or Interpretation: O.Ş., Literature Search: A.A.B., Writing: A.A.B.

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