

P-WAVE TERMINAL FORCE IN LEAD V1-MORRIS INDEX AFTER CATHETER ABLATION: A STUDY IN ATRIAL FIBRILLATION AND TYPICAL ATRIAL FLUTTER PATIENTS

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ABSTRACT

Introduction: P-wave terminal force in lead V1 (PTFV1, Morris Index) is a marker of atrial remodeling. We evaluated its role three months after atrial fibrillation (AF) or cavotricuspid isthmus (CTI) ablation.

Methods: A total of 120 AF patients (46% persistent, 54% paroxysmal) and 58 CTI patients were analyzed. PTFV1 was measured pre-ablation and at three months. Abnormal PTFV1 was defined as $\geq |0.04|$ mV·s. Patients were followed for 12 months. Endpoints were AF/AT recurrence in the AF cohort and new AF in the CTI cohort.

Results: In the AF cohort, recurrence occurred in 31%. Abnormal 3-month PTFV1 predicted higher recurrence (46% vs. 21%; HR 2.05, 95% CI 1.22–3.46). Addition of PTFV1 improved AUC from 0.67 to 0.76. In the CTI cohort, new AF occurred in 15%, but abnormal PTFV1 was not predictive (OR 1.8, 95% CI 0.8–4.2). Lead II P-wave duration was significant.

Conclusion: Three-month PTFV1 is a robust predictor of AF recurrence after ablation. Its role in CTI ablation is limited.

Keywords: Atrial fibrillation, Atrial Remodelling, Morris index, Catheter ablation, Atrial flutter

INTRODUCTION

The P-wave terminal force in lead V1 (PTFV1), also referred to as the Morris Index, was first described by Morris and colleagues in 1964 as the product of the duration (ms) and amplitude (mm) of the terminal negative deflection of the P wave in lead V1 (1-2). A value more negative than -0.04 mm·s (≥ 40 ms \times ≥ 1 mm) is generally considered abnormal and indicates delayed left atrial activation, increased atrial mass, and interatrial conduction delay. Over subsequent decades, abnormal PTFV1 has been consistently associated with left atrial enlargement, atrial fibrosis, atrial fibrillation (AF), ischemic stroke, heart failure, and increased mortality (3-4). As a simple, non-invasive ECG-derived marker, it provides an accessible estimate of atrial electrical remodeling and has been integrated into both population-based studies and post-ablation risk stratification models (5).

Catheter ablation is an established treatment for AF and typical atrial flutter, aiming to restore and maintain sinus rhythm by modifying the atrial substrate. However, recurrence of arrhythmia remains a significant limitation. Identifying reliable markers of atrial remodeling that predict post-ablation recurrence is therefore clinically important. Although PTFV1 has been widely studied as a risk marker in general populations, its role after catheter ablation particularly whether normalization of

PTFV1 indicates favorable reverse remodeling has not been comprehensively explored.

Recent electrophysiological and imaging studies have highlighted that the degree of atrial remodeling encompassing both structural and electrical alterations plays a critical role in the recurrence of arrhythmia after ablation. Advanced imaging modalities such as cardiac MRI and strain echocardiography have been used to quantify atrial fibrosis and mechanical dysfunction; however, their cost and limited accessibility restrict routine use(6-7). In contrast, PTFV1 offers a simple, inexpensive, and reproducible electrocardiographic parameter that reflects interatrial conduction delay and left atrial fibrosis. Evaluating changes in PTFV1 after ablation may therefore provide a practical, non-invasive window into atrial reverse remodeling and help identify patients at higher risk for recurrence.

In this study, we sought to investigate the prognostic significance of PTFV1 measured three months after ablation in patients undergoing AF or CTI ablation, and to determine whether persistent abnormalities are associated with arrhythmia recurrence during follow-up.

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Table 1. Baseline characteristics

Variable	AF (n=120)	CTI (n=58)	Total (n=178)	p value
Age (years)	61 ± 10	59 ± 11	60 ± 10	0.21
Female (%)	38%	27%	34%	0.18
Hypertension (%)	58%	61%	59%	0.72
Diabetes (%)	21%	19%	20%	0.79
Coronary artery disease (%)	15%	12%	14%	0.64
LAVI (ml/m ²)	32 ± 8	30 ± 6	31 ± 7	0.09
Ejection fraction (%)	58 ± 6	59 ± 5	58 ± 6	0.31

Abb. AF – atrial fibrillation; CTI – cavotricuspid isthmus; LAVI – left atrial volume index; EF – ejection fraction.

Baseline demographic and clinical characteristics of patients in the atrial fibrillation (AF) and cavotricuspid isthmus (CTI) ablation cohorts. Continuous variables are presented as mean ± standard deviation, and categorical variables as percentages.

METHODS

Study Population: Between January 2024 and January 2025 a total of 178 consecutive patients undergoing catheter ablation were enrolled. This retrospective study included consecutive patients who underwent catheter ablation. The study population consisted of two cohorts: 120 patients with atrial fibrillation (AF cohort) and 58 patients undergoing cavotricuspid isthmus (CTI) ablation for typical atrial flutter (CTI cohort). The mean age was 61±10 years in the AF cohort and 59±11 years in the CTI cohort. Women comprised 38% and 27% of the AF and CTI cohorts, respectively. Hypertension was present in 58% of AF patients and 61% of CTI patients, while diabetes mellitus was observed in 21% and 19%, respectively. Coronary artery disease was present in 15% of the AF cohort and 12% of the CTI cohort. Patients with prior ablation, permanent pacemaker or CRT, QRS duration >120 ms, Wolff-Parkinson-White syndrome, or significant valvular surgery were excluded.

Ablation Protocol: All AF patients underwent pulmonary vein isolation (PVI) using radiofrequency energy, with additional linear lesions performed at the discretion of the operator. CTI ablation was performed using standard techniques to achieve bidirectional isthmus block. Procedural endpoints included demonstration of entrance and exit block for PVI and bidirectional conduction block for CTI ablation.

Electrocardiographic Measurements: Standard 12-lead ECGs were recorded at baseline and at three months after ablation (paper speed 25 mm/s, calibration 10 mm/mV). PTFV1 was calculated as the product of the duration (ms) and amplitude (mV) of the terminal negative portion of the P wave in lead V1. An abnormal PTFV1 was defined as $\geq |0.04|$ mV·s, consistent with prior studies. Each measurement was averaged over three consecutive beats. Two independent blinded cardiologists analyzed the ECGs, and interobserver reproducibility was assessed using intraclass correlation coefficients (ICC). Interobserver reproducibility of PTFV1 measurements was assessed using intraclass correlation coefficients (ICC), demonstrating excellent agreement between observers.

Table 2. AF cohort – ECG and recurrence

Variable	No Recurrence (n=83)	Recurrence (n=37)	p value
PTFV1 pre (mV·s)	-0.039 ± 0.015	-0.041 ± 0.017	0.44
PTFV1 3-month (mV·s)	-0.022 ± 0.012	-0.034 ± 0.015	0.001
Abnormal 3-month PTFV1 (%)	22%	76%	<0.001

Abb. PTFV1 – P-wave terminal force in lead V1; AF – atrial fibrillation.

Table Legend :Electrocardiographic measurements and arrhythmia recurrence rates in the atrial fibrillation cohort during 12-month follow-up. Comparisons between patients with and without recurrence include PTFV1 values and the prevalence of abnormal three-month PTFV1.

Follow-up: All patients were followed for 12 months after ablation. Clinical visits, 12-lead ECGs, and ambulatory monitoring (24-hour Holter or 7–14 day patch monitoring) were performed at 3, 6, and 12 months. Additional monitoring was performed if symptoms occurred. The primary endpoint for the AF cohort was recurrence of atrial fibrillation or atrial tachyarrhythmia lasting ≥ 30 seconds after the three-month blanking period. The primary endpoint for the CTI cohort was new-onset AF documented within 12 months.

Statistical Analysis: Continuous variables were expressed as mean ± SD and compared using Student's t-test. Categorical variables were expressed as frequencies (%) and compared using chi-square or Fisher's exact test. Cox proportional hazards regression was used to evaluate predictors of recurrence in the AF cohort, while logistic regression was applied to the CTI cohort for new-onset AF. Incremental prognostic value of PTFV1 was assessed using receiver operating characteristic (ROC) curves, changes in the area under the curve (Δ AUC), net reclassification improvement (NRI), and integrated discrimination improvement (IDI). A two-tailed p value <0.05 was considered statistically significant. Interobserver agreement for ECG-derived parameters was evaluated using intraclass correlation coefficients.

The study protocol was approved by Gazi Yaşargil Training and Research Hospital Ethics Committee on September 5, 2025 (Approval No: 650). All procedures were conducted in accordance with the Declaration of Helsinki.

RESULTS

Baseline Characteristics: A total of 178 patients were included (120 AF, 58 CTI). The mean age was 61±10 years in the AF cohort and 59±11 years in the CTI cohort. Women comprised 38% and 27% of the AF and CTI cohorts, respectively. Hypertension was present in 58% of AF and 61% of CTI patients, and diabetes in 21% and 19%. Coronary artery disease was present in 15% of the AF cohort and 12% of the CTI cohort. Left atrial volume index (LAVI) averaged 32±8 ml/m² in the AF cohort and 30±6 ml/m² in the CTI cohort. Baseline demographic and clinical characteristics of the study population are summarized in Table 1.

AF Cohort Outcomes: At 12 months, recurrence of atrial fibrillation or atrial tachyarrhythmia was observed in 37 of 120 patients (31%). Patients with abnormal three-month PTFV1 experienced significantly higher recurrence (46%) compared with those whose PTFV1 normalized (21%). In multivariable Cox regression, abnormal 3-month PTFV1 remained an independent

predictor of recurrence (HR 2.05, 95% CI 1.22–3.46, $p=0.007$), after adjustment for age, sex, hypertension, diabetes, AF type, and LAVI. Addition of 3-month PTFV1 improved discrimination from an AUC of 0.67 to 0.76 ($\Delta\text{AUC}=0.09$, $p=0.02$), with net reclassification improvement (NRI=0.19). ECG findings and recurrence outcomes in the atrial fibrillation cohort are presented in Table 2.

CTI Cohort Outcomes: During 12 months of follow-up, new-onset AF developed in 9 of 58 patients (15%). Abnormal three-month PTFV1 was not independently predictive (OR 1.8, 95% CI 0.8–4.2). In logistic regression, lead II P-wave duration was a significant predictor (OR 1.34 per 10 ms, 95% CI 1.05–1.74).

DISCUSSION

This study demonstrated that persistence of abnormal PTFV1 three months after AF ablation is an independent predictor of arrhythmia recurrence and provides incremental prognostic value beyond clinical and echocardiographic parameters. These findings are consistent with prior reports by Sudo et al. and Li et al., which highlighted the predictive role of PTFV1 in AF populations. Wang et al. further demonstrated its prognostic value in paroxysmal AF with normal left atrial size(8-9).

Pathophysiological Insights: PTFV1 reflects delayed and amplified activation of the left atrium due to electrical and structural remodeling. Cardiac magnetic resonance imaging (CMR) with late gadolinium enhancement has shown associations between abnormal PTFV1 and atrial fibrosis burden. Echocardiographic studies similarly linked abnormal PTFV1 with impaired left atrial strain, supporting its role as a marker of atrial myopathy. Similar findings were also demonstrated in stroke patients in sinus rhythm, where an elevated Macruz index was shown to reflect underlying atrial electrical remodeling (10).

Comparison with Other ECG Indices: In this cohort, PTFV1 outperformed traditional clinical predictors, but other ECG markers such as P-wave duration, dispersion, and axis have also been associated with recurrence. Notably, in CTI patients, lead II P-wave duration was more predictive of new AF, consistent with Liu et al. (11).

Clinical Implications: Routine measurement of PTFV1 at three months post-ablation offers a practical tool for risk stratification. Patients with persistently abnormal PTFV1 may require more intensive rhythm surveillance, extended anticoagulation, or earlier consideration of repeat ablation. Conversely, normalization of PTFV1 may indicate favorable reverse remodeling and better long-term outcomes. The observed improvement in discrimination and reclassification metrics (ΔAUC , NRI, and IDI) suggests that the inclusion of three-month PTFV1 enhances the identification of patients at higher risk of post-ablation arrhythmia recurrence. From a clinical perspective, this improvement may translate into more accurate risk stratification, allowing clinicians to selectively intensify rhythm monitoring, extend follow-up, or individualize anticoagulation strategies in patients with persistently abnormal PTFV1.

Rather than replacing established clinical risk scores such as CHA₂DS₂-VASc, three-month PTFV1 may serve as a complementary ECG-based marker that reflects atrial remodeling and refines post-ablation risk stratification when integrated with clinical and echocardiographic parameters.

Future Directions: Three-month PTFV1 measurement provides a simple, inexpensive, and reproducible method to assess atrial remodeling after AF ablation. Persistently abnormal values were

independently associated with arrhythmia recurrence, while normalization suggested favorable atrial reverse remodeling. Incorporation of PTFV1 into routine post-ablation ECG assessment may allow for early identification of high-risk patients who warrant intensive rhythm monitoring, extended anticoagulation strategies, or closer clinical follow-up. The three-month time point for PTFV1 assessment was intentionally selected as it corresponds to the end of the standard post-ablation blanking period, during which early arrhythmia episodes may reflect transient inflammation rather than durable recurrence. Nevertheless, atrial electrical and structural remodeling may continue beyond this period, and serial or longer-term PTFV1 measurements may provide additional prognostic information. Future studies should evaluate the temporal evolution of PTFV1 after ablation.

From a practical perspective, we propose a simple algorithm: all patients should undergo a 12-lead ECG at three months post-ablation. If PTFV1 remains abnormal ($\geq|0.04|$ mV-s), patients should be considered for prolonged ambulatory monitoring (e.g., 14–30 day Holter or implantable loop recorder) and reassessment of anticoagulation irrespective of CHA₂DS₂-VASc score. If PTFV1 normalizes, standard follow-up protocols may be sufficient. Integration with echocardiographic parameters such as left atrial strain or MRI-based fibrosis assessment could further refine this strategy.

Although our results indicate limited prognostic value of PTFV1 after CTI ablation, combining this index with other ECG markers such as P-wave duration may provide incremental benefit in this population. Future multicenter studies are warranted to validate these findings and to establish standardized cut-off values for clinical implementation

PTFV1 is a simple, inexpensive, and powerful marker of atrial remodeling. Its robust predictive role after AF ablation supports its integration into post-procedural follow-up, while its limited value after CTI ablation underscores the heterogeneous pathophysiology of atrial arrhythmias. Post-ablation antiarrhythmic drug therapy and rhythm control strategies were applied according to contemporary guideline-directed clinical practice and were relatively homogeneous across the study population. However, these variables were not systematically quantified in a manner suitable for inclusion in multivariable regression models. In addition, advanced atrial functional parameters such as left atrial strain were not routinely available for all patients. Therefore, residual confounding related to these factors cannot be fully excluded.

Limitations: Future multicenter and prospective studies are required to validate these findings across diverse populations. Integration of PTFV1 with advanced imaging modalities such as cardiac magnetic resonance (CMR) with late gadolinium enhancement and speckle-tracking echocardiographic strain could help refine risk stratification. First, this was a single-center retrospective study with a modest sample size, particularly in the CTI ablation cohort, which may limit the external validity and generalizability of the findings. Moreover, biomarker studies (e.g., NT-proBNP, hs-CRP, troponin) in conjunction with PTFV1 may provide deeper insights into the atrial substrate and remodeling processes. Finally, incorporating PTFV1 into composite risk scores that include clinical, imaging, and ECG-derived indices may improve clinical decision-making.

CONCLUSION

Three-month morris index (PTFV1) measurement provides a simple and reproducible method to evaluate atrial remodeling after atrial fibrillation ablation. Persistently abnormal values ($\geq|0.04|$ mV·s) were independently associated with arrhythmia recurrence, whereas normalization suggested favorable reverse remodeling. Incorporating PTFV1 into routine post-ablation ECG follow-up may facilitate identification of patients at higher risk who require intensified rhythm monitoring, prolonged anticoagulation, or early repeat ablation consideration.

From a clinical perspective, we propose a practical algorithm: all patients should undergo a 12-lead ECG at three months after ablation. If PTFV1 remains abnormal, these patients may benefit from extended ambulatory monitoring (14–30 day Holter or implantable loop recorder) and individualized anticoagulation strategies regardless of CHA₂DS₂-VASc score. If PTFV1 normalizes, standard follow-up may suffice. Combining PTFV1 assessment with echocardiographic parameters such as left atrial strain or MRI fibrosis burden could further refine stratification.

Although its predictive value after CTI ablation was limited, integration with other ECG indices such as P-wave duration may enhance prognostication in this subgroup. Future multicenter trials are needed to validate these findings and standardize PTFV1 thresholds for routine clinical use.

In summary, PTFV1 is a low-cost, accessible, and clinically meaningful ECG marker of atrial remodeling that holds strong prognostic significance after AF ablation and should be considered in post-ablation follow-up strategies.

Ethics Committee Approval: The study protocol was approved Gazi Yaşargil Training and Research Hospital Ethics Committee on September 5, 2025 (Approval No: 650). All procedures were conducted in accordance with the ethical standards of the institutional and national research committees and with the 1964 Declaration of Helsinki and its later amendments.

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Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Given the retrospective design, the requirement for written informed consent was waived by the local ethics committee. No animal experiments or use of animal-derived materials were involved in this research.

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