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Left atrial appendage strain and P-wave dispersion: electro-mechanical markers of paroxysmal atrial fibrillation

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ABSTRACT

Aims: Paroxysmal atrial fibrillation (PAF) is a major clinical challenge due to its intermittent nature and the difficulty of early detection. Left atrial appendage (LAA) function plays a crucial role in atrial mechanics, while P-wave dispersion (PWD) reflects electrical inhomogeneity. We hypothesized that both parameters would independently and synergistically predict PAF and aimed to develop an integrative electro-mechanical model to enhance risk stratification.

Methods: We retrospectively analyzed 191 patients, including 91 with PAF and 100 in sinus rhythm (SR). LAA function was assessed using speckle-tracking echocardiography, and PWD was measured digitally from 12-lead electrocardiography. Multivariable logistic regression models were constructed: model 1 included clinical parameters, model 2 incorporated PWD, and Model 3 further added LAA strain reservoir (LAA-Sr)

Results: PAF patients exhibited significantly lower LAA-Sr (14.7 % [12.2-18.0] vs. 21.6% [19.1-25.3], p<0.001) and higher PWD (30.3 [27.6-34.5] ms vs. 20.9 [17.3-26.6] ms, p<0.001). In multivariable analysis, LAA-Sr (OR: 1.315, 95% confidence interval [CI]: 1.201-1.439, p<0.001) and PWD (OR: 1.128, 95% CI: 1.054-1.215, p=0.038) were independent PAF predictors. Model 3, which included both parameters, demonstrated the best predictive performance (AUC: 0.983, sensitivity: 92.8%, specificity: 79.4%) compared to model 1 (AUC: 0.890) and model 2 (AUC: 0.950).

Conclusion: Our study highlights LAA strain and PWD as robust, independent predictors of PAF. The combination of mechanical and electrophysiological markers enhances AF risk stratification and early detection. Future prospective, multi-center studies are warranted to validate these findings and optimize risk assessment strategies for PAF.

Keywords: Atrial fibrillation, left atrial appendage, transesophageal echocardiography, electrocardiography, prediction algorithms

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and is associated with an increased risk of stroke, heart failure, and cardiovascular mortality.[1] Paroxysmal atrial fibrillation (PAF), a subtype of AF characterized by self-terminating episodes, poses a particular diagnostic challenge as many patients remain asymptomatic or experience only transient symptoms.[2] Identifying patients at risk of developing PAF is crucial for timely intervention and stroke prevention.

Left atrial appendage (LAA) function plays a central role in atrial mechanics and thrombogenesis. As an embryological remnant of the primitive left atrium, the LAA is the most common site of thrombus formation in AF and serves as a key marker of atrial mechanical dysfunction.[3] LAA morphology influences its mechanical function, with variations in size and shape potentially impacting contractile performance and predisposing to AF.[4] Beyond structural remodeling, speckle tracking echocardiography (STE)-derived LAA strain analysis has emerged as a novel method to assess LAA function, providing insights into atrial mechanics beyond conventional echocardiographic parameters.[5] A decline in LAA strain may reflect early atrial remodeling, potentially identifying patients at increased risk for AF development.[6]

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Similarly, P-wave dispersion (PWD), defined as the difference between the maximum and minimum P-wave durations on a surface electrocardiogram (ECG), is a marker of atrial conduction heterogeneity.[7] Increased PWD has been associated with atrial electrical remodeling and a higher risk of developing AF.[8] Despite their potential complementary roles, the predictive value of LAA strain and PWD in PAF detection remains largely unexplored.

We hypothesized that impaired LAA mechanical function and increased atrial electrical dispersion may independently predict PAF, and their combination could enhance diagnostic performance in at-risk individuals. To our knowledge, this is among the first studies to evaluate their combined role in PAF prediction.

METHODS

Ethics

The study was conducted in accordance with the Declaration of Helsinki and was initiated with the approval of the Clinical Researches Ethics Committee of Başakşehir Çam and Sakura City Hospital (Date: 27.04.2022, Decision No: 136). Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Study Population

This retrospective, observational study included patients diagnosed with PAF and a control group with sinus rhythm (SR). PAF was defined as AF episodes lasting less than seven days and terminating spontaneously or with medical intervention. Importantly, all patients in the PAF group were in SR at the time of evaluation. The control group consisted of individuals with persistent SR on standard 12-lead ECG at the time of enrollment, without a history of palpitations, documented arrhythmia, or known structural heart disease.

In the SR group, TEE was performed either to evaluate suspected interatrial septal abnormalities (e.g., to differentiate between patent foramen ovale [PFO] and atrial septal defect [ASD]) or due to inadequate transthoracic echocardiographic imaging. Patients with PFO, hemodynamically insignificant small ASD (<10 mm)[9], or suboptimal transthoracic views were included, provided they did not meet other exclusion criteria. LAA strain measurements were retrospectively evaluated in patients with optimal image quality.

Patients with structural heart disease, prior cardiac interventions involving the left atrium (including ASD or PFO closure), significant valvular heart disease (including prosthetic valves, moderate-to-severe mitral regurgitation, mitral stenosis, tricuspid regurgitation), severe atrial dilation >50 mm, left ventricular dysfunction (LVEF <50%), prior AF ablation, persistent or ongoing AF at enrollment, pacemaker history, QRS duration >120 ms, uncontrolled hypertension (systolic blood pressure >160 mmHg despite medical therapy), or obesity (BMI ≥35 kg/m²) were excluded. Additionally, patients with suboptimal LAA visualization on STE and those with ECGs showing unclear P-wave morphology or poor signal quality were excluded to ensure measurement accuracy.

All participants underwent a comprehensive echocardiographic evaluation and a standard 12-lead ECG at the time of enrollment. Clinical characteristics, including age, sex, cardiovascular risk factors, and medication history, were recorded.

Electrocardiographic Assessment of P-Wave Dispersion

P-wave dispersion was assessed using a digital electrocardiographic measurement software on standard 12-lead ECG recorded at a paper speed of 50 mm/s and an amplitude of 10 mm/mV. P-wave onset was defined as the first positive or negative deflection from the isoelectric line, while P-wave offset was marked as the return to baseline.

Measurements were performed manually with software assistance by two independent observers blinded to the clinical data. PWD was calculated as the difference between the longest and shortest P wave durations measured across the 12 leads. To minimize inter-observer variability, both observers independently repeated the measurements, and significant discrepancies (>5 ms) were resolved by consensus. Additionally, a subset of randomly selected ECGs was reanalyzed after two weeks by the same and different observers to evaluate intra-observer and inter-observer agreement.

Left Atrial Appendage Speckle Tracking Strain Echocardiography

This study retrospectively included patients who had undergone TEE for various clinical indications. TEE imaging was performed using a Philips EPIQ CxT system with an X8-2t transesophageal probe. TEE examinations were performed under conscious sedation and oropharyngeal anesthesia, following standard institutional protocols. Routine LAA functional parameters, including emptying velocity, end-diastolic volume, end-systolic volume, ejection fraction, and volume change, were assessed using two-dimensional TEE. LAA volumes were measured using the biplane area-length method in the long-axis view.

LAA function was further evaluated using speckle tracking echocardiography (STE), as illustrated in **Figure 1**. As no dedicated software currently exists for LAA strain analysis, we adopted a methodology similar to previous studies that adapted standard LV or LA strain software for this purpose.[6] LAA strain analysis was performed via offline post-processing using TomTec Imaging Systems (Unterschleissheim, Germany). LAA strain measurements were derived using an automated LA strain analysis software initially developed for LA strain evaluation. The region of interest (ROI) was manually repositioned to focus on the LAA myocardium, ensuring accurate contouring. The software automatically provided LAA reservoir strain (LAA-Sr), conduit strain (LAA-Scd), and contraction strain (LAA-Sct). These parameters reflect distinct aspects of LAA function: LAA-Sr represents reservoir function during atrial filling, LAA-Scd reflects passive emptying function, and LAA-Sct indicates active contraction capacity. **Figure 1A** demonstrates an example of strain tracking on the LAA, while **Figure 1B** displays the corresponding strain curve with numerical values. To ensure measurement reliability, strain curves with inadequate tracking quality were manually corrected, and poorly tracked segments were excluded from the analysis. The onset of the

QRS complex was used as a reference point for strain curve analysis.

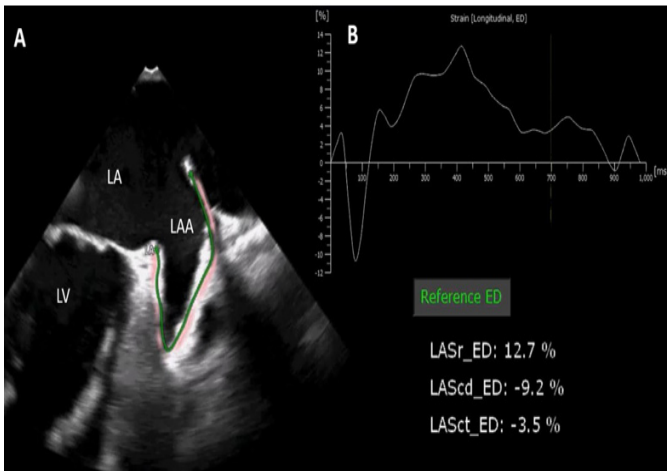

Figure 1. Left atrial appendage (LAA) strain analysis using speckle-tracking echocardiography (STE). (**A**) Mid-esophageal transesophageal echocardiographic (TEE) view at 90°, showing automated speckle-tracking of the LAA myocardium with manual contour adjustments. (**B**) Corresponding strain curve analysis displaying LAA reservoir strain (LAA-Sr), conduit strain (LAA-Scd), and contraction strain (LAA-Sct).

Spontaneous echocardiographic contrast (SEC) and thrombus assessment was performed by optimizing gain settings to enhance contrast resolution while minimizing noise and artifacts, reducing the risk of misinterpretation. SEC in the LA and LAA was evaluated based on echogenic swirling patterns and classified as grade 0 (absent), grade 1 (minimal), grade 2 (mild), grade 3 (moderate), or grade 4 (severe), as previously described.[10]

To assess intra-observer and inter-observer variability, a subset of randomly selected measurements was repeated by the same and different observers after a two-week interval.

Statistical Analysis

The study population was divided into two groups: SR and PAF, and all statistical comparisons were performed between these groups. The distribution of continuous variables was assessed using the Shapiro-Wilk test and visual inspection of histograms. Continuous variables were expressed as mean±standard deviation (SD) or median (interquartile range, IQR), depending on data distribution, and compared using the independent samples t-test or Mann-Whitney U test. Categorical variables were presented as counts and percentages [n (%)] and analyzed using the χ^2 test or Fisher's exact test, where appropriate.

To determine independent predictors of PAF, variables demonstrating statistical significance in univariate analyses ($p<0.05$) were incorporated into multivariable logistic regression models. Model performance was evaluated using Nagelkerke R^2 values, Akaike information criterion (AIC), and C-index analysis. The incremental predictive value of LAA strain parameters was assessed using integrated discrimination improvement (IDI) and net reclassification improvement (NRI) indices. Model accuracy was further assessed by receiver operating characteristic (ROC) curve analysis, and decision-making utility was evaluated using decision curve analysis (DCA). Model calibration was verified with calibration plots.

Post-hoc power analyses were performed to assess the adequacy of the sample size. Intra-observer and inter-observer variability for PWD and LAA strain measurements were assessed using intraclass correlation coefficients (ICCs). All statistical analyses were performed using IBM SPSS Statistics for Windows, version 30.0 (IBM Corp., Armonk, NY, USA) and R software (R Foundation for Statistical Computing, Vienna, Austria). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

A total of 191 patients were included, with 91 in the PAF group and 100 in the SR group. Patients with PAF had a higher burden of comorbidities, including hypertension and coronary artery disease (p=0.001 for both) (Table 1) and had significantly larger LA dimensions and volume index (LAVI) (p<0.001).

Echocardiographic assessments revealed that LAA function was significantly impaired in the PAF group. LAA ejection fraction was lower in PAF patients compared to the SR group. LAA end-diastolic and end-systolic volumes were also larger in the PAF group (p=0.005 and p<0.001, respectively). LAA-Sr was significantly lower in the PAF group (21.6 [19.1-25.3]% vs. 14.7 [12.2-18.0]%, p<0.001), indicating impaired LAA mechanics (Table 2). The optimal cut-off value for LAA-Sr to predict PAF was 19.21%, with a sensitivity of 74% and a specificity of 72%, yielding an AUC of 0.832 (95% confidence interval [CI]: 0.775–0.890).

PWD was significantly higher in the PAF group compared to the SR group (30.3 [27.6–34.5] ms vs. 20.9 [17.3–26.6] ms, p<0.001). The optimal cut-off value for PWD in predicting PAF was determined as 23.79 ms, with a sensitivity of 84% and a specificity of 69%, yielding an AUC of 0.829 (95% CI 0.767–0.891).

To identify predictors of PAF, multivariable logistic regression analysis was performed, incorporating variables that were significant in univariate analysis and clinically relevant (Table 3). Given the collinearity among LA parameters, only LA strain was included in the final models. Model 1 included general clinical parameters such as age, hypertension, coronary artery disease, CHA_2DS_2-VASc score, and LA strain. Model 2 extended model 1 by adding PWD. Model 3 further extended model 2 by incorporating LAA- Sr (Table 4). The predictive performance of the models, as assessed by ROC analysis, including AUC, sensitivity, and specificity values, is summarized in Figure 2. Model 3 demonstrated the highest AUC (0.983, 95% CI 0.970–0.996), with improved sensitivity (92.8%) and specificity (79.4%), indicating its superior predictive ability. Further comparisons of model indices confirmed the superiority of model 3. The final model exhibited the strongest predictive capability, as evidenced by a higher Nagelkerke R^2 (0.575), a lower AIC: (162.9), an improved C-index (0.890), and significant improvements in IDI (0.152). These findings are summarized in Figure 3. DCA demonstrated the highest net clinical benefit with

Table 1. Baseline clinical, demographic, and electrocardiographic characteristics compared between the sinus rhythm and PAF groups

Clinical parameters	Sinus rhythm, n=100	PAF, n=91	Total, n=191	p-value
Male, n (%)	45 (45%)	42 (46%)	87 (46%)	0.885
Age, years	49 (37-58)	59 (50-66)	54 (44-63)	<0.001
Hypertension, n (%)	15 (15%)	34 (37%)	49 (26%)	0.001
Coronary artery disease, n (%)	14 (14%)	48 (53%)	62 (32%)	0.001
Diabetes mellitus, n (%)	22 (22%)	32 (35%)	54 (28%)	0.076
Chronic kidney disease, n (%)	9 (9%)	11 (12%)	20 (10%)	0.638
Chronic obstructive pulmonary disease, n (%)	3 (3%)	7 (8%)	10 (5%)	0.202
History of stroke, n (%)	26 (26%)	10 (11%)	36 (19%)	0.009
BMI, kg/m²	25.9 (23.0-30.2)	26.6 (24.4- 29.7)	26.1 (23.8-30.0)	0.145
HR, bpm	71 (61-75)	79 (67-89)	73 (65-83)	<0.001
CHADS2-VASc score	2 (1-3)	3 (2-4)	3 (1-4)	0.001
Anticoagulation, n (%)				0.001
None, n (%)		11 (12%)	11 (6%)	
Apixaban, n (%)		16 (18%)	16 (8%)	
Rivaroxaban, n (%)		28 (31%)	28 (15%)	
Edoxaban, n (%)		22 (24%)	22 (12%)	
Dabigatran, n (%)		2 (2%)	2 (1%)	
Warfarin, n (%)		11 (12%)	11 (6%)	
Pmax, ms	90 (80-100)	100 (80-120)	92.5 (80-110)	0.266
Pmin, ms	40 (40-50)	50 (40-58.75)	45 (40-55)	0.002
P dispersion, ms	20.9 (17.3-26.6	30.3 (27.6-34.5)	26.9 (20.1-32.0)	<0.001

Data are presented as median (IQR) for continuous variables and n (%) for categorical variables. A p-value <0.05 was considered statistically significant. BMI: Body-mass index, CHADS2-VASc: Congestive heart failure, hypertension, age, diabetes, stroke, vascular disease, sex, EDV: End-diastolic volume, EF: Ejection fraction, E/e': Mitral E-wave to early diastolic annular velocity, HR: Heart rate, LAA: Left atrial appendage, LAA EF: Left atrial appendage ejection fraction, LAA emptying velocity: Left atrial appendage emptying velocity, LAA volume ED: Left atrial appendage volume at end-diastole, LAA volume ES: Left atrial appendage volume at end-systole, LAA volume change: Left atrial appendage volume change, LA: Left atrium, LAAScd: Left atrial appendage strain conduit phase, LAASct: Left atrial appendage strain contraction phase, LAASr: Left atrial appendage strain reservoir phase, LAVI: Left atrial volume index, P dispersion: P-wave dispersion, Pmax: Maximum P-wave duration, Pmin: Minimum P-wave duration

model 3, and calibration analysis confirmed the reliability of predictions. These findings are summarized in **Figure 4**.

Post-hoc power analyses based on group differences in LAA-Sr and PWD demonstrated large effect sizes (Cohen's d≈1.55) and statistical power >0.99, confirming the adequacy of the sample size, which is also comparable to or larger than those reported in similar observational studies[6], in line with current STROBE recommendations for observational studies.[11] Intra-observer and inter-observer variability analyses showed good reproducibility for both PWD and LAA strain measurements. The ICC values were 0.92 and 0.89 for intra- and inter-observer agreement of PWD, respectively, and 0.91 and 0.87 for intra- and inter-observer agreement of LAA strain.

DISCUSSION

This study demonstrated that LAA- Sr and PWD are independent predictors of PAF. The addition of these parameters to a conventional clinical model significantly enhanced the ability to predict PAF, improving both model discrimination and classification performance. To the best of our knowledge, this is one of the first studies to evaluate the combined role of LAA-Sr and PWD in PAF prediction. These findings suggest that a combined assessment of LAA mechanical function and atrial conduction properties may provide additional value in identifying patients at risk for PAF.

Several studies have explored LAA strain in AF, specifically focusing on its role in predicting stroke risk, arrhythmia occurrence and AF recurrence. Saberniak et al.[6] investigated LAA-Sr in stroke patients and found that lower LAA-Sr was associated with subclinical AF, with a cut-off of 22.2%. Similarly, studies assessing AF recurrence after catheter ablation reported even lower LAA-Sr values, with one study identifying a cut-off of 10.2%, indicating that LAA function deteriorates significantly in persistent AF or post-ablation settings.[12] Another study demonstrated that LAA-Sr is associated with thromboembolic risk, showing an inverse correlation between strain values and SEC or thrombus formation.[13] In a direct comparison of persistent vs. paroxysmal AF patients, LAA strain was significantly lower in persistent AF, reinforcing its role in AF progression.[14] Additionally, a study evaluating AF burden in non-valvular AF patients demonstrated that LAA strain was significantly reduced in those with higher arrhythmic burden, further supporting its predictive value.[15]

Our study differs from previous research on LAA strain in AF, particularly in patient selection and methodology. While prior studies mainly focused on persistent AF, stroke populations, or post-ablation recurrence, our cohort consisted of PAF patients evaluated in SR. This distinction is important, as AF itself impairs LAA contractility and remodeling, leading to lower strain values in persistent AF. Consequently, while previous studies reported LAA-Sr cut-offs as low as 10.2% in post-ablation settings and 22.2% in stroke patients, we identified a cut-off of 19.21% for PAF prediction. This

Table 2. Echocardiographic and strain parameters compared between the sinus rhythm and PAF groups

Parameters	Sinus rhythm, n=100	PAF, n=91	Total, n=191	p-value
EDV, ml	45 (41-47)	46 (43-50)	45 (43-49)	0.111
EF, %	60 (69-65)	61 (57-63)	60 (58-62)	0.060
E/e' ratio	12.8 (11.7-13.4)	13.3 (12- 14.8)	13.2 (11.7-14.6)	0.078
Mitral stenosis, n (%)				0.321
None	89 (89%)	84 (92%)	173 (91%)	
Mild	8 (8%)	3 (3%)	11 (6%)	
Mitral regurgitation, n (%)				0.001
None	17 (17%)	7 (8%)	24 (13%)	
Mild	83 (83%)	65 (71%)	148 (77%)	
Tricuspid regurgitation, n (%)				0.003
None	29 (29%)	25 (27%)	54 (28%)	
Mild	71 (71%)	54 (59%)	125 (65%)	
LAA spontaneous echo contrast, n (%)				0.008
Grade 0, n (%)	97 (97%)	77 (84%)	174 (91%)	
Grade 1, n (%)	3 (3%)	10 (10%)	13 (7%)	
Grade 2, n (%)	0	4 (4%)	4 (2%)	
LAA thrombus, n (%)	0	3 (3%)	3 (2%)	0.001
LA diameter, mm	35 (32-41.75)	40 (37-45)	39 (34-44)	<0.001
LAVI, ml/m²	32.1 (29.5-35.4)	42.6 (40.1-46.3)	37.0 (31.4-42.6)	<0.001
LAA emptying velocity, cm/s	73.6 (71.2-76.9)	74.6 (72.1-78.3)	74.2 (71.3-77.6)	0.124
LAA volume ED, ml	3.7 (2.7-5.7)	4.6 (3.6-6.1)	4.2 (2.9-5.6)	0.005
LAA volume ES, ml	0.8 (0.5-1.1)	1.5 (1.2-1.8)	1.2 (0.7-1.6	<0.001
LAA EF, %	78.3 (62.1-87.0)	66.9 (64.4-68.6)	68.2 (64.0-79.8)	<0.001
LAA volume change, ml	2.7 (1.6-4.4)	3.09 (2.33-4.21)	2.9 (1.9-4.3)	0.279
LA Sr, %	26.7 (24.3-30.0)	13.5 (11.0-17.2)	21.2 (13.9-27.0)	<0.001
LA Scd, %	-15.0 (-17.5--11.7)	-7.2 (-9.7--3.5)	-11.2 (-15.9--6.8)	<0.001
LA Sct, %	-12.1 (-14.6--8.8)	-5.2 (-7.7--1.5)	-8.6 (-13.0--4.3)	<0.001
LAA Sr, %	14.7 (12.2-18.0)	21.6 (19.1-25.3)	18.2 (13.8-22.6)	<0.001
LAA Scd, %	-10.1 (-12.5--6.8)	-13.3 (-15.8--9.6)	-11.3 (-14.8--8.1)	<0.001
LAA Sct, %	-6.9 (-9.4--4.0)	-9.3 (-11.8--5.6)	-7.8 (-10.8--4.7)	0.004

EDV: End-diastolic volume, EF: Ejection fraction, LAA: Left atrial appendage, LAA EF: Left atrial appendage ejection fraction, LAA emptying velocity: Left atrial appendage emptying velocity, LAA volume ED: Left atrial appendage volume at end-diastole, LAA volume ES: Left atrial appendage volume at end-systole, LAA volume change: Left atrial appendage volume change, LA: Left atrium, LAAScd: Left atrial appendage strain conduit phase, LAASct: Left atrial appendage strain contraction phase, LAASr: Left atrial appendage strain reservoir phase, LAVI: Left atrial volume index

Table 3. Univariate regression analysis for predictors of paroxysmal atrial fibrillation

Univariate regression	OR (95% CI)	p value
Age (years)	1.063 (1.038-1.091)	<0.001
HT	3.198 (1.617-6.57)	<0.001
DM	1.831 (0.967-3.515)	0.070
CAD	6.612 (3.349-13.742)	<0.001
CHA_2DS_2-VASc score (points)	1.611 (1.322-1.997)	<0.001
LA (mm)	1.121 (1.065-1.188)	<0.001
LAVI (ml/m²)	1.477 (1.342-1.659)	<0.001
LAA emptying velocity (cm/s)	1.034 (0.982-1.091)	0.210
LAA EF (%)	1.002 (0.997-1.008)	0.460
LA Sr (%)	0.612 (0.522-0.692)	<0.001
LAA Sr (%)	1.298 (1.208-1.411)	<0.001
P-wave dispersion (ms)	1.075 (1.042-1.116)	<0.001

Odds ratios (OR) with 95% confidence intervals (CI) are presented. A p-value <0.05 was considered statistically significant. CAD: Coronary artery disease, CHA_2DS_2-VASc score (points): Congestive heart failure, hypertension, age ≥75 (2 points), diabetes mellitus, stroke/TIA (2 points), vascular disease, age 65-74, DM: Diabetes mellitus, HT: Hypertension, LAA Sr (%): Left atrial appendage strain reservoir, LA Sr (%): Left atrial strain reservoir

difference likely reflects variations in study populations, as we excluded patients with prior LAA interventions, significant atrial fibrosis, or advanced structural disease, making our findings more relevant for early-stage AF detection.

Methodological differences may have also contributed to these discrepancies. Unlike prior studies that often used LV strain software, we utilized LA strain software, providing a more physiologically accurate assessment of reservoir, conduit, and contraction phases. Moreover, whereas most previous studies performed strain analysis using GE ultrasound systems with EchoPAC software, our study utilized Philips ultrasound systems with TomTec software. These vendor-related differences in imaging platforms and post-processing algorithms may have influenced contouring precision and strain quantification.

The role of PWD in AF prediction has also been extensively studied. Aytemir et al.[16] identified a PWD cut-off of 36 ms for PAF detection, with 77% sensitivity and 82% specificity.

Table 4. Multivariate regression analysis for predictors of paroxysmal atrial fibrillation across different models

	Model 1		Model 2		Model 3	
Parameter	**p-value**	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**	**OR (95% CI)**
Age (years)	0.007	1.045 (1.012-1.078)	0.006	1.048 (1.014-1.084)	0.004	1.057 (1.018-1.098)
HT	0.170	0.257 (0.037-1.786)	0.769	0.649 (0.036-11.636)	0.137	2.262 (0.772 - 6.627)
CAD	<0.001	4.430 (1.871-10.491)	0.003	3.814 (1.562-9.315)	0.015	3.532 (1.276-9.772)
DM	0.117	0.300 (0.067-1.352)	0.188	0.218 (0.023-2.105)	0.448	1.505 (0.523-4.330)
CHA_2DS_2-VASc score	0.355	1.357 (0.710-2.594)	0.742	0.855 (0.335-2.179)	0.476	0.851 (0.546-1.326)
LA Sr (%)	<0.001	0.555 (0.451-0.683)	<0.001	0.465 (0.330-0.655)	0.027	0.685 (0.512-0.849)
P-wave dispersion (ms)	-	-	<0.001	1.174 (1.086-1.270)	0.038	1.128 (1.054-1.215)
LAA Sr (%)	-	-	-	-	<0.001	1.315 (1.201-1.439)

Odds ratios (OR) with 95% confidence intervals (CI) are presented for three models. A p-value <0.05 was considered statistically significant. CAD: Coronary artery disease, CHA_2DS_2-VASc score (points): Congestive heart failure, hypertension, age ≥75 (2 points), diabetes mellitus, stroke/TIA (2 points), vascular disease, age 65-74, DM: Diabetes mellitus, HT: Hypertension, LAA Sr (%): Left atrial appendage strain reservoir, LA Sr (%): Left atrial strain reservoir

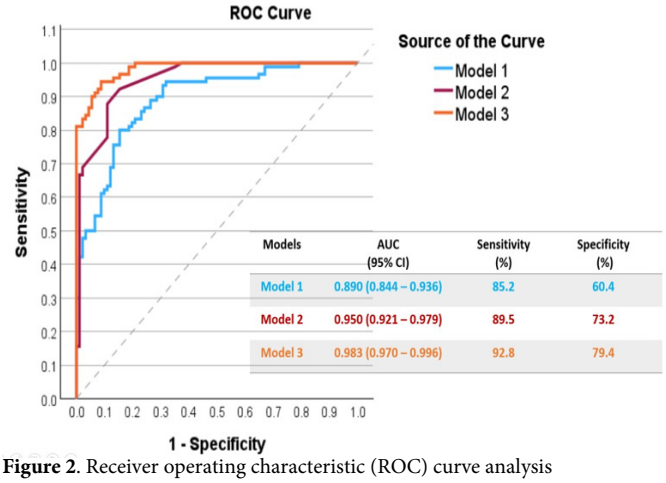

Figure 2. Receiver operating characteristic (ROC) curve analysis

The ROC curves illustrate the discriminatory performance of the three models. The table below presents the AUC, sensitivity, and specificity values for each model, highlighting the superior predictive accuracy of model 3

A meta-analysis further confirmed the association between PWD and AF risk, demonstrating significantly higher values in patients with recurrent AF. Additionally, increased PWD has been linked to greater AF burden, reinforcing its role in atrial conduction abnormalities.[17] However, many of these studies primarily included patients with persistent AF, stroke, or advanced atrial remodeling, which may account for differences in reported findings.[8]

In contrast, our study determined a PWD cut-off of 23.79 ms for PAF prediction. This discrepancy is likely due to differences in study populations, as our cohort consisted of PAF patients evaluated while in SR, making our findings particularly relevant for early-stage AF detection. Furthermore, we excluded patients with severe atrial fibrosis or structural disease, further distinguishing our cohort from prior studies. Methodological variations may have also contributed—while previous studies often relied on manual ECG measurements,

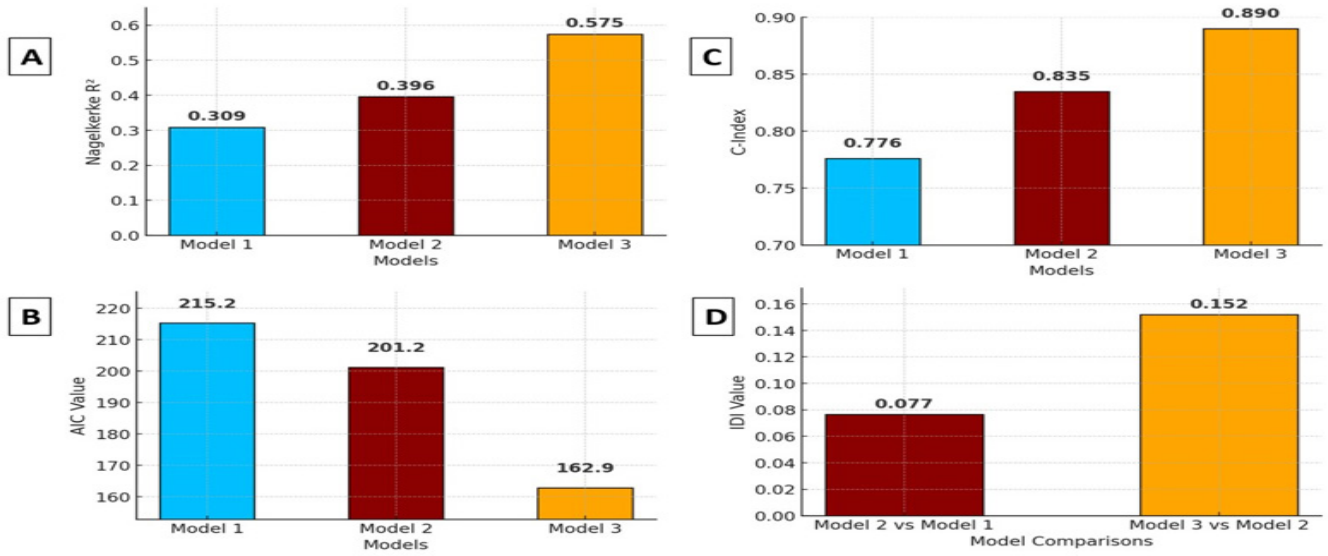

Figure 3. Model performance metrics. Panel (**A**) shows Nagelkerke R^2 values, demonstrating the improved explanatory power of model 3. Panel (**B**) presents Akaike information criterion (AIC) values, indicating better model fit with lower AIC. Panel (**C**) displays the C-index, highlighting the enhanced discriminative ability of model 3. Panel (**D**) illustrates the integrated discrimination improvement (IDI) values, confirming the incremental predictive value gained with each model refinement

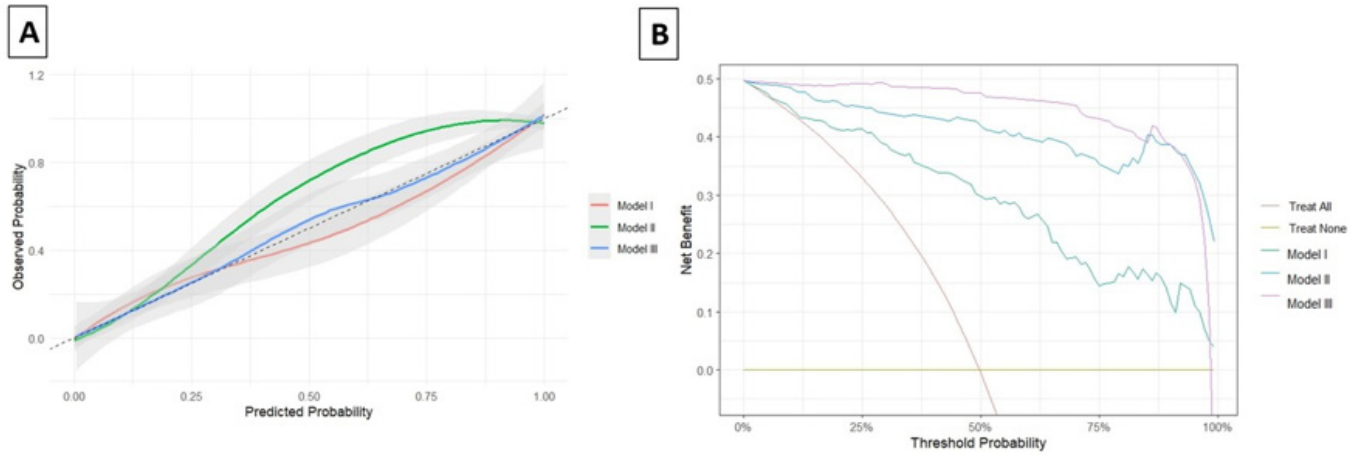

Figure 4. Model calibration and clinical utility**.** Panel (**A**) displays the calibration curves for the three models, illustrating the agreement between predicted and observed probabilities. Model 3 (blue) shows the best calibration. Panel (**B**) presents the decision curve analysis (DCA), demonstrating the net clinical benefit across different threshold probabilities, with model 3 providing the highest benefit

we employed automated digital ECG analysis, ensuring greater precision and reproducibility. These factors collectively highlight the importance of population-specific cut-off values and reinforce PWD's potential utility in PAF prediction.

AF accounts for 15–25% of all ischemic strokes and increases stroke risk by three to five times, highlighting the need for early detection and effective prevention strategies.[18] Given the episodic nature of PAF, its timely identification is crucial, as even short-lived AF episodes can contribute to thromboembolic risk.[19] In our cohort, LAA thrombus was detected in 3% of PAF patients, reinforcing that PAF is not a benign condition and that its early recognition may help prevent stroke-related complications.

In this context, the combined use of LAA strain and PWD offers a novel electro-mechanical approach to PAF prediction. While PWD reflects atrial conduction abnormalities, LAA strain provides insights into atrial mechanical dysfunction, making them complementary markers in AF risk assessment. Our findings suggest that incorporating both parameters improves PAF detection beyond traditional risk factors, potentially refining risk stratification and guiding early intervention strategies in clinical practice.

Limitations

Despite its strengths, our study has several limitations. First, the retrospective design may introduce selection bias. Second, this was a single-center study, which may limit the generalizability of our results to broader populations. Third, long-term follow-up data were not available, preventing an assessment of whether patients with impaired LAA strain and increased PWD eventually developed AF over time. Additionally, since continuous ECG monitoring (e.g., Holter) was not performed, subclinical AF episodes may have been missed, potentially affecting the predictive accuracy of our findings. Finally, we used Philips software for speckle-tracking analysis, and variations in strain values across different imaging vendors should be considered when comparing results across studies.

CONCLUSION

Our study demonstrates that LAA strain and PWD are independent and complementary predictors of PAF. The integration of mechanical and electrophysiological markers enhances AF risk stratification beyond conventional parameters, offering a novel electro-mechanical approach to early AF detection. The final predictive model, incorporating both parameters, exhibited the highest diagnostic performance, emphasizing the clinical relevance of this combined approach. Although not suitable for general screening, TEE-based LAA strain analysis may have clinical value in selected patients at high risk for paroxysmal AF. Future prospective, multi-center studies with long-term follow-up are needed to validate these findings and determine their impact on AF screening, risk stratification, and stroke prevention strategies.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was initiated with the approval of the Clinical Researches Ethics Committee of Başakşehir Çam and Sakura City Hospital (Date: 27.04.2022, Decision No: 136).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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