

Impact of AV Block Duration and P-Wave Indices on Atrial Fibrillation Development in Patients With Dual-Chamber Pacemakers

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ABSTRACT

In complete atrioventricular (AV) block, atrial contraction against closed AV valves may induce atrial pressure overload and early electrical remodeling. P-wave indices, such as P-wave area and dispersion, reflect atrial electrical heterogeneity, but the joint impact of AV block duration and baseline P-wave abnormalities on new-onset atrial fibrillation (AF) after dual-chamber pacing is unknown.

We retrospectively studied 80 patients who received dual-chamber DDDR pacemakers for complete AV block. AV block duration was defined as the interval from the first documented complete AV block on ECG to implantation and was quantified in hours. Baseline P-wave indices (area, dispersion, and duration) were measured on pre-implant 12-lead ECGs. Patients were followed for 3 years if incident AF was confirmed by device interrogation or surface ECG. Predictors of AF were evaluated using ROC analysis, Kaplan–Meier curves, and Cox regression.

During follow-up, 14 patients (17.5%) developed AF. These patients had longer AV block duration and higher P-wave area and dispersion than those who remained in sinus rhythm (all $p < 0.001$). Optimal cutoffs were 38.5 hours for AV block duration, 5.150 $\mu\text{V}\cdot\text{ms}$ for P-wave area, and 39.5 ms for dispersion. AV block duration and P-wave indices showed excellent discrimination for incident AF and remained independent predictors in multivariable Cox models.

Prolonged exposure to a complete AV block is associated with adverse P-wave remodeling and a higher risk of incident AF. AV block duration may represent a modifiable determinant of AF after pacemaker implantation, suggesting that earlier pacing could reduce atrial stress and arrhythmic risk.

Keywords: Atrial fibrillation, Dual-chamber pacemaker, Atrioventricular block duration, P-wave indices

Introduction

Complete atrioventricular (AV) block leads to a state of AV dissociation in which atrial contraction may occur against closed AV valves, generating abrupt increases in atrial pressure and wall stress. Repetitive exposure to this hemodynamic disturbance triggers mechano-electrical feedback mechanisms that contribute to early atrial electrical remodeling (1–4). Experimental data have demonstrated that acute atrial stretch promotes conduction heterogeneity, ion-channel alterations, and arrhythmogenic substrate formation (5,6).

Surface P-wave indices—including P-wave area, P-wave duration, and P-wave dispersion—serve as non-invasive markers of atrial electrical

heterogeneity. Increased P-wave dispersion reflects inhomogeneous and discontinuous atrial conduction, while elevated P-wave area represents a higher burden of atrial depolarization and may indicate atrial stretch or early fibrotic change (7–9). Numerous studies have shown that abnormal P-wave indices predict incident atrial fibrillation (AF) across a variety of clinical settings, including hypertension, postoperative states, and structural heart disease (10–12). Recent analyses reaffirmed the predictive value of P-wave dispersion and P-wave morphology in identifying individuals at increased risk for AF in general and device-monitored populations (13,14).

AF remains one of the most frequent long-term complications among permanent pacemaker recipients and is associated with heightened risks

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Received: 29.12.2025, Accepted: 02.03.2026

of stroke, heart failure, and hospitalization (15,16). Although pacing mode, atrial pacing burden, and underlying comorbidities have been recognized as contributors to AF in pacemaker patients, the role of pre-implant complete AV block duration in atrial remodeling has not been previously evaluated. Delays in pacemaker implantation—due to referral logistics or clinical prioritization—may prolong exposure to AV dissociation, thereby exacerbating atrial hemodynamic load. Yet no study to date has examined whether the duration of untreated complete AV block adversely affects P-wave indices or predisposes to AF after pacemaker implantation.

Clarifying this relationship is clinically important because P-wave indices are inexpensive, easily obtainable, and widely accessible markers that can be integrated into routine pre-implant evaluation. Furthermore, if prolonged AV block duration is shown to negatively affect atrial electrophysiology, earlier pacemaker implantation may represent a modifiable strategy to reduce atrial stress and lower long-term arrhythmic risk.

Therefore, this study aimed to investigate whether prolonged complete AV block duration is associated with abnormal baseline P-wave indices, and whether these parameters independently predict new-onset AF in patients undergoing dual-chamber pacemaker implantation. We hypothesized that longer exposure to AV dissociation would adversely affect atrial electrical characteristics, thereby increasing susceptibility to AF during follow-up.

Materials and Methods

Study Design and Population: This retrospective observational study included 80 consecutive patients who underwent dual-chamber DDDR pacemaker implantation for complete AV block at a tertiary cardiology center between January 2022 and January 2025. Patients were eligible if they presented with electrocardiographically confirmed complete AV block and had an analyzable baseline 12-lead ECG recorded prior to implantation. Individuals with a prior diagnosis of AF, structural heart disease, moderate-to-severe valvular disease, congenital heart disease, cardiomyopathies, thyroid dysfunction, significant electrolyte abnormalities, a previous pacemaker implantation, or poor-quality ECGs that impeded accurate P-wave measurements were excluded. A total of 80 patients met these criteria and were included in the final analysis. Demographic characteristics,

baseline laboratory values, echocardiographic measurements, and device interrogation data were obtained from the electronic hospital records. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki and received approval from the local Ethics Committee. Informed consent was obtained from all patients prior to participation.

Electrocardiographic Assessment: Baseline 12-lead ECGs recorded at a paper speed of 25 mm/s and an amplitude of 10 mm/mV were obtained prior to pacemaker implantation. All P-wave measurements were performed manually using digital calipers by two independent cardiologists blinded to clinical outcomes, and the mean of the two measurements was used for analysis. P-wave area ($\mu\text{V}\cdot\text{ms}$) was calculated as the quantitative area under the P-wave curve, obtained by digitally tracing the P-wave contour and integrating the enclosed waveform. P-wave dispersion (ms) was defined as the difference between the maximum and minimum P-wave durations across all leads, while P-wave duration (ms) represented the longest measurable P wave in any derivation. Post-implantation paced-rhythm ECGs were recorded within the first 24 hours to evaluate early changes in atrial depolarization parameters. AV block duration was also derived from ECG-anchored time stamps and defined as the interval between the first documented complete AV block on a 12-lead ECG and the initiation of pacemaker implantation, expressed in hours. All pacemakers were implanted within 72 hours, although a minority of patients experienced untreated AV block exposure exceeding 48 hours.

Echocardiographic Assessment: Transthoracic echocardiography was performed in all patients using standardized imaging protocols and in accordance with contemporary guidelines. Left ventricular ejection fraction (LVEF) was measured using the modified Simpson's biplane method obtained from apical two- and four-chamber views. Left atrial size and morphology were assessed qualitatively to exclude structural abnormalities that could influence atrial electrophysiology. Mitral A-wave velocity (cm/s) was recorded using pulsed-wave Doppler at the level of the mitral leaflet tips during late diastole to evaluate atrial contractile function. All echocardiographic measurements were performed by experienced operators who were blinded to patient outcomes.

Clinical and Laboratory Evaluation: Baseline clinical evaluation included assessment of vital signs, review of medical history, and identification

of cardiovascular risk factors. Laboratory parameters collected at admission included serum creatinine, potassium, C-reactive protein (CRP), and thyroid-stimulating hormone (TSH), all measured using standard automated laboratory methods. All laboratory values were within normal reference ranges, and no metabolic, inflammatory, or endocrine abnormalities were identified that could influence atrial electrophysiology. Laboratory findings were comparable between patients who developed AF and those who remained in sinus rhythm during follow-up.

Follow-up and AF detection: Department Of Cardiology All patients were followed for a total of 3 years through scheduled outpatient clinic visits and routine device interrogations. Pacemaker diagnostics were reviewed at each visit to assess atrial sensing, atrial pacing burden, and the presence of atrial high-rate episodes (AHREs). Incident AF was defined as an AHRE or atrial tachyarrhythmia lasting ≥ 30 seconds, confirmed by intracardiac electrogram or a 12-lead electrocardiogram. The date of the first documented AF episode was recorded as the event time. Patients who did not develop AF during follow-up were considered censored at the end of the 36-month observation period.

Ethical Approval: The present study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki and its later amendments. The study protocol was reviewed and approved by a local ethics committee (approval date: 27 November 2025; protocol number: 2025-141). Given the retrospective observational design of the study, the requirement for obtaining written informed consent from the participants was waived.

Statistical Analysis: Statistical analyses were performed using IBM SPSS Statistics, Version 25.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed data were presented as mean \pm standard deviation (SD), while non-normally distributed variables were expressed as median (interquartile range, IQR). Comparisons between patients who developed AF and those who remained in sinus rhythm were performed using the Mann–Whitney U test for continuous variables and the chi-square or Fisher’s exact test for categorical variables. Pre-implant and post-implant P-wave indices were compared using the Wilcoxon signed-ranks test. Correlations between AV block duration, P-wave parameters, and echocardiographic measurements

were analyzed using Spearman’s correlation coefficient. The predictive performance of AV block duration and baseline P-wave indices for incident AF was evaluated using receiver operating characteristic (ROC) curve analysis, with optimal cut-off values determined by Youden’s index. Kaplan–Meier survival curves with log-rank testing were used to compare AF-free survival among subgroups stratified by ROC-derived thresholds. Independent predictors of incident AF were identified using a Cox proportional hazards regression model, and a two-sided $p < 0.05$ was considered statistically significant.

Results

Baseline demographic, clinical, laboratory, echocardiographic, and electrocardiographic characteristics of the study population ($n = 80$) are summarized in Table 1. The cohort had a median age of 47 years (40–54) and was predominantly male (56.3%). Traditional cardiovascular comorbidities were present in a minority of patients (diabetes mellitus 22.5%, hypertension 32.5%, hyperlipidemia 18.8%). Baseline renal function and inflammatory markers were within the normal range (serum creatinine 0.95 mg/dL [0.92–0.98], C-reactive protein 3.60 mg/L [3.10–3.90]), and thyroid function was normal (TSH 1.90 ± 0.30 μ IU/mL). Left ventricular systolic function was preserved (LVEF $58 \pm 6\%$). All patients had narrow QRS complexes at baseline (94 ± 9 ms) and wide paced QRS after implantation (142 ± 15 ms). Median AV block duration before pacemaker implantation was 31.0 hours (22.0–42.5). During 3-year follow-up, 14 patients (17.5%) developed incident AF (Table 1).

In between-group comparisons, patients who developed AF showed significantly more prolonged exposure to complete AV block and worse baseline atrial electrical indices. Specifically, AV block duration, baseline P-wave area, and baseline P-wave dispersion were all significantly higher in the AF group compared with those who remained in sinus rhythm ($p < 0.001$; Table 2). These findings indicate that longer pre-implant AV dissociation and greater baseline atrial electrical burden are associated with later AF occurrence.

In within-patient paired analyses, pacing was associated with a significant increase in atrial electrical indices over time. Both P-wave area and P-wave dispersion decreased after pacemaker implantation compared with baseline.

Table 1: Baseline Demographic, Laboratory, Echocardiographic and Electrocardiographic Characteristics of The Study Population (n = 80)

Parameter	Value
Age (years)	47 (40 – 54)
Male sex, n (%)	45 (56.3 %)
Diabetes mellitus, n (%)	18 (22.5 %)
Hypertension, n (%)	26 (32.5 %)
Hyperlipidemia, n (%)	15 (18.8 %)
Serum creatinine (mg/dL)	0.95 (0.92 – 0.98)
Hemoglobin (g/dL)	13.76 ± 0.28
Sodium (mmol/L)	139.42 ± 0.85
Potassium (mmol/L)	4.20 ± 0.30
CRP (mg/L)	3.60 (3.10 – 3.90)
TSH (µIU/mL)	1.90 ± 0.30
Mitral A-wave velocity (cm/s)	76 ± 10
Left ventricular ejection fraction (%)	58 ± 6
QRS duration (basal, ms)	94 ± 9
QRS duration (post-implant, ms)	142 ± 15
P-wave dispersion (basal, ms)	37.3 ± 5.4
P-wave dispersion (post-implant, ms)	35.4 ± 4.5
P-wave area (basal, µV·ms)	4.8 ± 0.6
P-wave area (post-implant, µV·ms)	4.7 ± 0.5
AV block duration (hours)	31.0 (22.0 – 42.5)
AF development during follow-up, n (%)	14 (17.5 %)

Table 2: Comparison of AV Block Duration and P-wave Indices Between AF and non-AF Groups

Parameter	Mann–Whitney U	Z value	p value
AV block duration (hours)	172.000	–3.673	<0.001
Baseline P-wave area (µV·ms)	170.000	–3.715	<0.001
Baseline P-wave dispersion (ms)	158.000	–3.861	<0.001

Table 3: ROC curve Analysis for Predictors of Incident Atrial Fibrillation

Parameter	AUC (95% CI)	Cut-off value	Sensitivity (%)	Specificity (%)	p value
AV block duration (hours)	0.814 (0.685–0.943)	>38.5	86	77	<0.001
Baseline P-wave area (µV·ms)	0.816 (0.638–0.994)	>5.150	78.6	93.9	<0.001
Baseline P-wave dispersion (ms)	0.829 (0.688–0.970)	>39.5	78.6	89.4	<0.001

*Cut-off values were determined using Youden’s index (J = sensitivity + specificity – 1). AUC: area under the curve

Table 4: Kaplan–Meier survival Analysis Based On Optimal ROC Cut-Off Values

Parameter	Cut-off value	Total N	AF events (n, %)	Mean AF-free survival (months, 95% CI)	Log-rank χ^2	p value
AV block duration (hours)	> 38.5	80	12 (44.4%) vs 2 (3.8%)	31.0 ± 1.4 (28.3–33.7) vs 35.7 ± 0.2 (35.3–36.2)	22.49	< 0.001
Baseline P-wave area ($\mu\text{V}\cdot\text{ms}$)	> 5.150	80	11 (73.3%) vs 3 (4.6%)	28.3 ± 1.6 (25.2–31.5) vs 35.5 ± 0.5 (34.6–36.4)	52.07	< 0.001
Baseline P-wave dispersion (ms)	> 39.5	80	11 (61.1%) vs 3 (4.8%)	29.1 ± 1.6 (25.9–32.3) vs 35.6 ± 0.4 (34.9–36.3)	38.03	< 0.001

*Cut-off values were determined using Youden’s index from ROC analysis. AF-free survival estimates are expressed as mean ± standard error (95% confidence interval)

Table 5: Cox Proportional Hazards Regression Analysis

Variable	B	SE	Wald	p value	Exp(B)	95% CI Lower	95% CI Upper
AV block duration (hours)	0.065	0.023	8.037	0.005	1.067	1.020	1.116
Baseline P-wave area ($\mu\text{V}\cdot\text{ms}$)	0.669	0.320	4.377	0.036	1.952	1.043	3.652
Baseline P-wave dispersion (ms)	0.121	0.043	7.923	0.005	1.129	1.037	1.228

Z = -6.387, $p < 0.001$; P-wave dispersion Z = -3.518, $p < 0.001$), suggesting progressive atrial electrical remodeling during follow-up.

Spearman correlation analysis demonstrated a significant inverse relationship between mitral A-wave velocity and both AV block duration ($r = -0.265$, $p = 0.017$) and baseline P-wave area ($r = -0.250$, $p = 0.025$), indicating that prolonged AV dissociation and higher atrial electrical load were associated with reduced atrial mechanical function. Baseline P-wave area correlated positively with P-wave dispersion ($r = 0.346$, $p = 0.002$), supporting a close link between atrial depolarization burden and conduction heterogeneity.

ROC analysis showed that all three primary predictors had strong discriminative ability for incident AF (Table 3; Figures 1–3). AV block duration yielded an AUC of 0.814 (95% CI 0.685–0.943, $p < 0.001$), with an optimal Youden-derived threshold of >38.5 hours (sensitivity 86%, specificity 77%). Baseline P-wave area also demonstrated strong prediction (AUC 0.816, 95% CI 0.638–0.994, $p < 0.001$), with a cut-off of >5.150 $\mu\text{V}\cdot\text{ms}$ (sensitivity 78.6%, specificity 93.9%). Baseline P-wave dispersion provided

similarly strong discrimination (AUC 0.829, 95% CI 0.688–0.970, $p < 0.001$), with a cut-off of >39.5 ms (sensitivity 78.6%, specificity 89.4%).

Collectively, these results confirm that both the duration of complete AV block and baseline atrial electrical indices are robust predictors of new-onset AF.

Kaplan–Meier survival curves demonstrated significantly lower AF-free survival for patients above each ROC-derived cut-off (Table 4; Figures 4–6). For AV block duration, AF incidence was 44.4% in the prolonged group (>38.5 h) versus 3.8% in the shorter group, with mean AF-free survival of 31.0 ± 1.4 vs 35.7 ± 0.2 months (log-rank $\chi^2 = 22.49$, $p < 0.001$). For baseline P-wave area, AF developed in 73.3% of patients with high P-wave area versus 4.6% with low area, with mean AF-free survival 28.3 ± 1.6 vs 35.5 ± 0.5 months ($\chi^2 = 52.07$, $p < 0.001$). Similarly, high baseline P-wave dispersion was associated with AF rates of 61.1% vs 4.8% and reduced AF-free survival 29.1 ± 1.6 vs 35.6 ± 0.4 months ($\chi^2 = 38.03$, $p < 0.001$). These survival analyses highlight a clear time-dependent increase in AF risk linked to both prolonged AV block exposure and abnormal baseline P-wave indices.

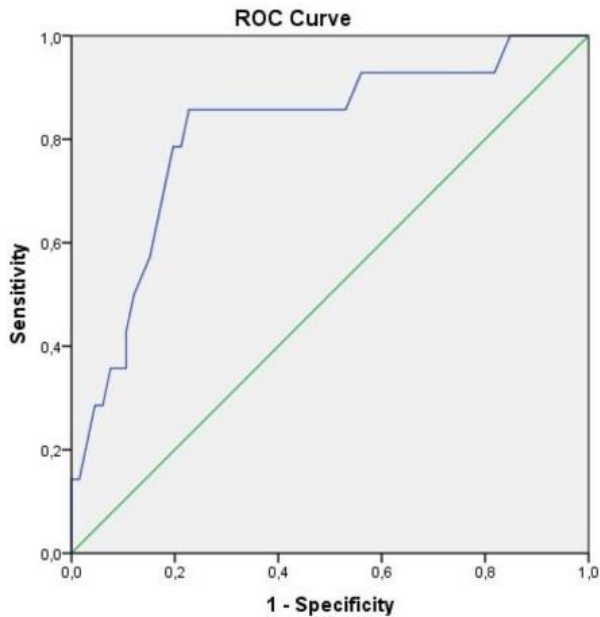


Fig. 1. ROC curve for AV block duration predicting incident atrial fibrillation. Receiver operating characteristic (ROC) curve demonstrating the predictive performance of AV block duration for incident atrial fibrillation (AF). The area under the curve (AUC) was 0.814 (95% confidence interval [CI]: 0.685–0.943, $p < 0.001$). The optimal cut-off value determined by Youden's index was >38.5 hours, yielding 86% sensitivity and 77% specificity for AF prediction

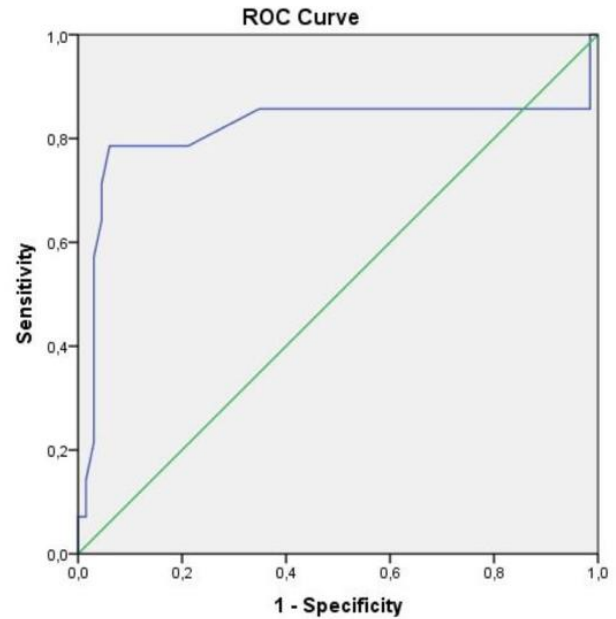


Fig. 2. ROC curve for baseline P-wave area predicting incident atrial fibrillation. ROC curve showing the discriminative ability of baseline P-wave area for predicting incident AF. AUC was 0.816 (95% CI: 0.638–0.994, $p < 0.001$). The ROC-derived optimal threshold was >5.150 $\mu\text{V}\cdot\text{ms}$, providing 78.6% sensitivity and 93.9% specificity

In multivariable Cox regression including AV block duration, baseline P-wave area, and baseline P-wave dispersion as continuous covariates, the overall model was highly significant (model $\chi^2 = 48.04$, $df = 3$, $p < 0.001$). All three variables remained independent predictors of incident AF (Table 5). Each additional hour of AV block increased AF hazard by 6.7% ($\beta = 0.065$, HR 1.067, 95% CI 1.020–1.116, $p = 0.005$). Higher baseline P-wave area conferred an almost two-fold higher risk ($\beta = 0.669$, HR 1.952, 95% CI 1.043–3.652, $p = 0.036$), and greater baseline P-wave dispersion also independently predicted AF ($\beta = 0.121$, HR 1.129, 95% CI 1.037–1.228, $p = 0.005$).

These findings support a model in which prolonged pre-implant complete AV block drives adverse atrial electrical remodeling, captured by P-wave indices, ultimately translating into higher time-to-AF risk.

Discussion

In this study, we demonstrated that prolonged exposure to untreated complete AV block is

significantly associated with marked alterations in atrial electrophysiology, reflected by increases in P-wave area, P-wave dispersion, and P-wave duration. These atrial electrical abnormalities were powerful predictors of incident AF during long-term follow-up. While the role of P-wave indices as markers of atrial conduction heterogeneity and AF susceptibility has been well established across diverse clinical settings (7–12), our findings expand this concept by suggesting that the duration of untreated AV block itself functions as an upstream, previously underrecognized driver of atrial remodeling, even before pacemaker implantation.

The mechanistic basis for this relationship is compelling. During complete AV block, atrial contractions frequently occur against closed atrioventricular valves, generating abrupt increases in atrial pressure and intermittent wall stretch. Experimental electrophysiology research has demonstrated that atrial stretch produces rapid changes in action potential duration, conduction velocity, anisotropy, and wavefront stability—core components of mechano-electric remodeling that predispose to AF (3,4,6). These mechano-electric interactions have long been recognized as critical modulators of atrial electrophysiology (1,2), and our findings provide clinical evidence in support

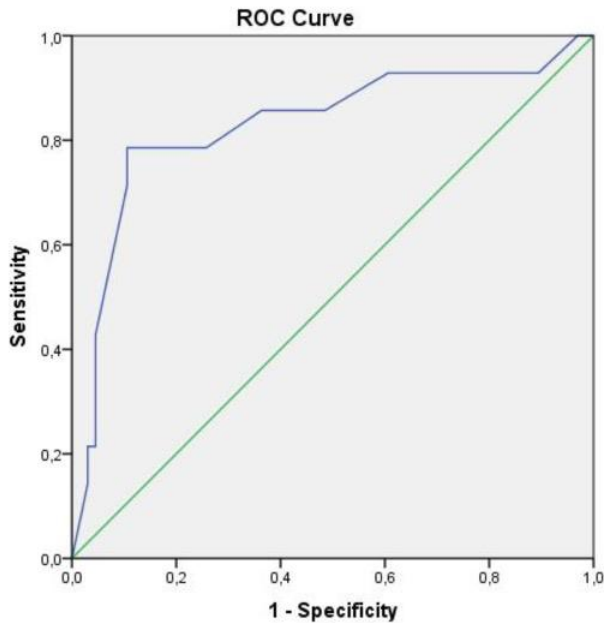


Fig. 3. ROC curve for baseline P-wave dispersion predicting incident atrial fibrillation. ROC curve demonstrating the predictive performance of baseline P-wave dispersion for incident AF. AUC was 0.829 (95% CI: 0.688–0.970, $p < 0.001$). The optimal cut-off value was >39.5 ms, with 78.6% sensitivity and 89.4% specificity

of this mechanism. The positive correlations observed between AV block duration and both P-wave area and P-wave dispersion suggest that sustained atrial stretch and pressure fluctuations during prolonged AV block contribute to conduction delay and interatrial conduction heterogeneity.

P-wave area is a global marker of prolonged atrial activation, integrating spatial conduction delay and potential interatrial conduction block. Increased values may reflect delayed activation of the posterior left atrium, septal regions, or Bachmann’s bundle—sites where fibrosis or conduction slowing is known to emerge early during atrial remodeling (13). Similarly, P-wave dispersion quantifies the difference between maximum and minimum P-wave duration across the 12-lead ECG. Larger dispersion values have been consistently associated with increased electrical heterogeneity and AF vulnerability in conditions such as hypertension, heart failure, and early atrial cardiomyopathy (10–12,14). Our data indicate that this electrical heterogeneity may begin developing during the period of untreated AV block itself, rather than being solely attributable to intrinsic atrial disease.

The negative correlation between AV block duration and mitral A-wave velocity further

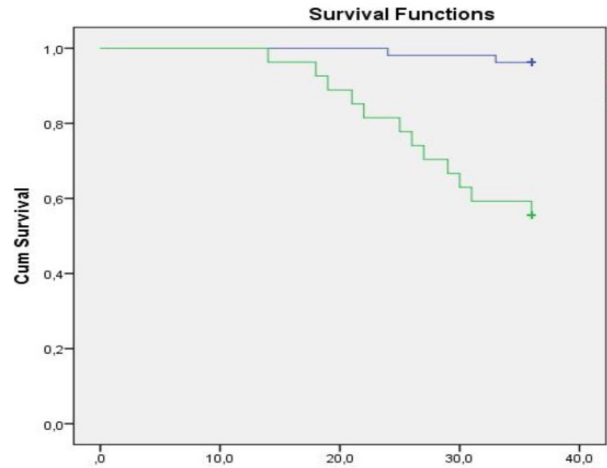


Fig. 4. Kaplan–Meier AF-free survival curves stratified by AV block duration. Kaplan–Meier curves comparing AF-free survival between patients with shorter AV block duration (<38.5 h) and prolonged AV block duration (>38.5 h). AF-free survival was significantly lower in the prolonged AV block group (log-rank $\chi^2 = 22.49$, $df = 1$, $p < 0.001$)

supports the concept that atrial mechanical dysfunction develops in parallel with electrical remodeling. Atrial contractile impairment (“atrial stunning”) has been reported in settings of acute atrial dilation, pressure overload, and stretch-induced afterdepolarizations (4,21). Together, these findings suggest that untreated AV block may lead to a form of mechano-electrical atrial injury, thereby creating a transient but clinically relevant substrate for AF shortly after pacemaker implantation.

Our study also provides insight into pacing management strategies. Prolonged AV block has traditionally been conceptualized primarily as a symptomatic bradyarrhythmia requiring pacing; however, its potential to induce atrial remodeling has been under appreciated. Recent evidence indicates that conduction system pacing—via His bundle or left bundle branch pacing—reduces long-term AF risk compared with conventional right ventricular pacing, likely due to superior atrioventricular synchrony and reduced atrial stretch (18,19,22). Nevertheless, our findings indicate that delays occurring even before the pacing modality is selected may already predispose patients to atrial remodeling and AF. These results reinforce emerging recommendations emphasizing timely recognition of complete AV block and minimizing diagnosis-to-implantation delays (17,23).

The predictive performance of AV block duration in our study (AUC 0.814) is comparable to, and in some cases exceeds, established

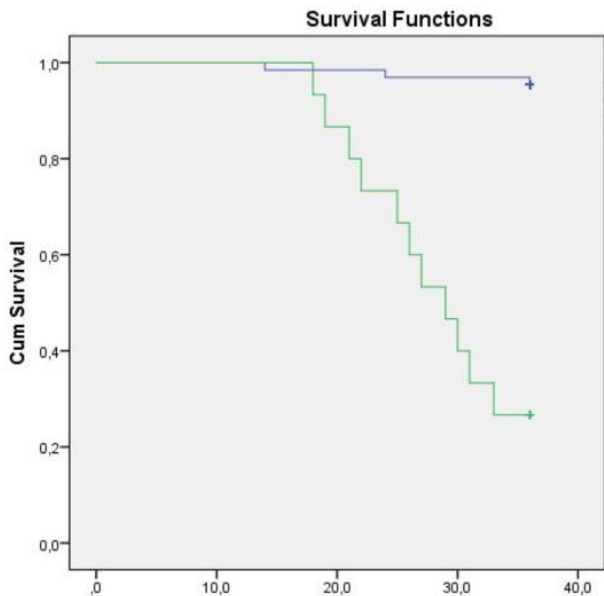


Fig. 5. Kaplan–Meier AF-free survival curves stratified by baseline P-wave area. Kaplan–Meier curves stratified by baseline P-wave area using the ROC-derived cut-off (>5.150 vs <5.150 $\mu\text{V}\cdot\text{ms}$). Patients with higher baseline P-wave area exhibited markedly reduced AF-free survival (log-rank $\chi^2 = 52.07$, $df = 1$, $p < 0.001$)

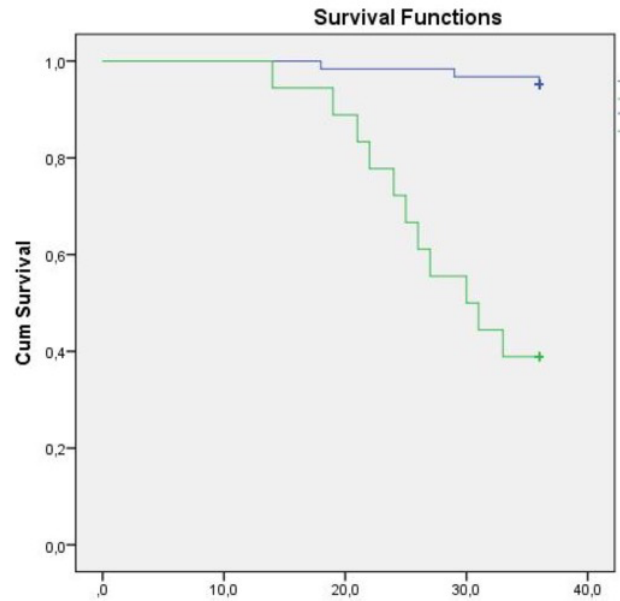


Fig. 6. Kaplan–Meier AF-free survival curves stratified by baseline P-wave dispersion. Kaplan–Meier curves stratified by baseline P-wave dispersion (>39.5 vs <39.5 ms). Higher baseline dispersion was associated with significantly earlier and more frequent AF development (log-rank $\chi^2 = 38.03$, $df = 1$, $p < 0.001$)

electrocardiographic AF risk markers. The identified cut-off value of >38.5 hours suggests that even modest delays in pacemaker implantation may have clinically meaningful electrophysiological consequences. Meanwhile, P-wave area and P-wave dispersion demonstrated complementary prognostic value, with all three parameters serving as independent predictors in multivariable Cox regression. The absence of multicollinearity among P-wave indices highlights that each parameter captures a distinct—but interconnected—aspect of atrial remodeling: P-wave area reflects conduction time and atrial enlargement, while P-wave dispersion captures spatial conduction heterogeneity.

The Kaplan–Meier curves further illustrate the clinical relevance of these findings: patients with prolonged AV block duration or abnormal P-wave indices experienced earlier AF onset and significantly reduced AF-free survival. This temporal pattern underscores the possibility that timely pacing—potentially within 24 hours of AV block diagnosis—may reduce atrial stress and mitigate long-term AF risk. Additionally, patients presenting with prolonged AV block exposure and abnormal P-wave indices may benefit from intensified rhythm monitoring, tailored pacing strategies, or early preventive interventions.

The persistence of abnormal P-wave indices after pacing initiation suggests that atrial remodeling occurring during untreated AV block is not immediately reversible. Animal and human studies alike have demonstrated that fibrosis and electrical remodeling may persist for weeks to months after the precipitating stressor is eliminated (1,6,21,24). Furthermore, recent work indicates that atrial mechanical dysfunction may precede electrical instability, serving as an early substrate for AF development and progression (25). This aligns with our observation that pacing restores AV synchrony but does not acutely normalize atrial depolarization metrics—highlighting the importance of preventing remodeling rather than attempting to reverse it once established.

Taken together, our findings support a conceptual framework in which prolonged untreated AV block acts as an upstream, modifiable trigger for atrial remodeling, mediated through repetitive atrial stretch, mechanical dysfunction, and conduction heterogeneity. These changes are detectable using simple, non-invasive ECG markers that provide powerful prognostic information regarding future AF risk. As cardiac pacing strategies evolve toward more physiological modalities and precision-based programming, early recognition and timely intervention in patients with AV block may represent promising approaches to preserving atrial integrity and

reducing the long-term burden of device-detected AF.

Study Limitations: Several limitations of this study should be acknowledged. First, the retrospective single-center design and lower sample size may introduce selection bias and limit the generalizability of the findings, although the uniformity of clinical practice and device programming at our institution reduces heterogeneity. Second, the duration of complete AV block was derived from the time elapsed between the first documented ECG showing complete block and pacemaker implantation; therefore, the true onset of AV block may have preceded clinical detection. This may have led to underestimation of AV block exposure in some patients. Third, P-wave indices were measured from surface ECGs rather than high-resolution signal-averaged recordings, which may have reduced precision, although all measurements were performed using standardized digital calipers and blinded evaluation to minimize variability. Fourth, device-detected AF episodes were used as the primary endpoint; while contemporary pacemakers provide reliable atrial arrhythmia detection, subclinical episodes below the detection threshold could not be fully excluded. Fifth, echocardiographic parameters such as left atrial strain or volumetric indices, which might provide deeper insight into atrial mechanical remodeling, were not systematically available. Lastly, although the sample size was adequate for the analyses performed, larger prospective multicenter studies are needed to confirm these findings and better define the temporal relationship between AV block duration, atrial remodeling, and AF development.

In patients with complete atrioventricular block, the duration of untreated AV block is not a benign interval but a critical period during which atrial electrical and mechanical injury begins to develop. In this study, prolonged AV block exposure was strongly associated with marked deterioration in atrial conduction—reflected by increased P-wave area, P-wave dispersion, and P-wave duration—and these abnormalities were powerful, independent predictors of incident AF after pacemaker implantation. Our findings challenge the traditional view of complete AV block as a purely bradyarrhythmic condition and identify AV block duration as a modifiable, clinically actionable upstream determinant of atrial remodeling and long-term arrhythmic risk. Early diagnosis and prompt pacing may therefore represent an opportunity to protect atrial electrical

integrity and reduce the future burden of AF. Incorporating both AV block duration and baseline P-wave indices into routine evaluation may enhance risk stratification, guide surveillance intensity, and support a more personalized management strategy for patients requiring dual-chamber pacemakers.

Clinical Implications: Our findings have several important clinical implications. First, the duration of untreated complete AV block should not be considered a benign waiting period; even short delays may initiate atrial electrical and mechanical remodeling. Early recognition of AV block and prompt pacemaker implantation may therefore reduce atrial stress and lower the long-term risk of AF. Second, baseline P-wave area and P-wave dispersion—simple, readily obtainable ECG markers—provide powerful prognostic information and may help clinicians identify patients at elevated arrhythmic risk prior to device implantation. Incorporating these indices into routine clinical assessment may improve risk stratification and guide decisions regarding rhythm surveillance intensity. Finally, patients with prolonged AV block exposure or abnormal P-wave indices may benefit from closer follow-up, early rhythm monitoring, and pacing strategies that minimize atrial loading conditions, such as optimized AV delay or conduction system pacing.

Acknowledgments: The authors thank the electrophysiology laboratory staff and the pacemaker clinic team at Kocaeli City Hospital for their support in patient follow-up and data retrieval. The authors also acknowledge the contribution of the hospital medical records department for assistance with accessing archived ECG and device interrogation data.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest: The authors declare no conflicts of interest related to this study.

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