HeartRhythm

The Official Journal of the Heart Rhythm Society, The Cardiac Electrophysiology Society, and The Pediatric & Congenital Electrophysiology Society

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Prevalence and progression of P-wave abnormalities in patients with atrial fibrillation

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ABSTRACT

BACKGROUND Various electrocardiographic P-wave indices are associated with cardiovascular comorbidities, such as atrial fibrillation (AF) and stroke. However, information on their stability is limited.

OBJECTIVE This study explored the prevalence and progression of P-wave abnormalities (PWAs) as well as their risk factors in an AF population.

METHODS PWAs were identified in a series of 3 sinus rhythm electrocardiograms (ECGs) of 1316 individuals undergoing the index cardioversion (CV) for acute AF in the FinCV study. Patients were assigned to the category of extensive PWA if they had P-wave duration $\geq$180 ms, P-terminal force $\geq$80 mm·ms, advanced interatrial block (biphasic P waves in inferior leads and P-wave duration $\geq$120 ms), or deflected P-wave morphology; moderate PWA consisted of P-wave duration of 150–180 ms or P-terminal force of 40–80 mm·ms.

RESULTS Between pre-CV and index CV ECGs, 133 of 342 (38.9%) and 54 of 342 (15.8%) patients progressed from normal P wave to moderate and extensive PWAs, respectively. During the follow-up after index CV, the respective rates were 131 of 407 (32.2%) and 74 of 407 (18.2%). At the end of follow-up, prevalence for normal P wave was 311 of 1121 (27.7%), whereas 434 (38.7%) patients had moderate PWA and 376 (33.5%) had extensive PWA. Increasing age, heart failure, hypertension, vascular disease, history of previous AF episodes, high CV frequency, left ventricular hypertrophy, and wide QRS complex in the ECG were independent risk factors for persistent or progressive PWA status in a Cox regression analysis.

CONCLUSION The prevalence and rate of progression of PWA are high in this cohort of AF patients, with development mainly driven by aging, chronic cardiovascular conditions, and frequent AF recurrences.

KEYWORDS Atrial cardiomyopathy; Atrial fibrillation; Electrocardiogram; Interatrial block; P wave; P-terminal force

(Heart Rhythm 2025;■:1–8)

Introduction

Electromechanical changes in the atria contribute to the manifestation of atrial fibrillation (AF) and vice versa.[1,2] AF-mediated remodeling has been shown to be reversible in the short term, but as episodes become longer and more frequent, changes may become permanent.[1] Several electrocardiographic P-wave abnormalities (PWAs) have been suggested to predict new-onset AF and to signal underlying structural changes. However, information on the occurrence and progression of PWA is lacking.[1–3]

Increased P-wave duration, P-terminal force (PTF), advanced interatrial block (AIAB), and deflected P-wave morphology have previously been studied as indicators for atrial cardiomyopathy (aCM), which by definition refers to deterioration of atrial structure, function, and conduction that could induce clinically relevant manifestations.[1,4] The composite of these electrocardiographic parameters reflects myocardial fibrosis in histologic analysis and predicts stroke, heart failure, and mortality.[4] The prevalences of abnormal P-wave duration, PTF, and AIAB are low in the general

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https://doi.org/10.1016/j.hrthm.2025.03.1981

population, but the presence of other cardiovascular conditions increases the likelihood of these electrocardiographic changes.[5–10] Despite identifying comorbidities of PWA, information about incidence and risk factors is lacking. In a longitudinal analysis, AIAB and PTF were found to be labile, and transient interatrial block has also been described in the literature.[11,12] Elongated P waves have been linked to aging, although a 3-year observation revealed no prolongation of P wave.[6,7,10,13]

In this study, we assessed the prevalence and progression of PWAs in a series of electrocardiograms (ECGs) before and after cardioversion (CV).

Methods

This is a substudy of the Finnish Cardioversion (FinCV) protocol in progress to assess clinical challenges of AF (ClinicalTrials.gov identifier: NCT04001205). This retrospective registry study originally gathered all patients (n = 3143) undergoing electrical CV for an acute (episode duration <48 hours) AF between 2003 and 2010 in 3 Finnish hospitals.[14,15] Patient selection and study protocol have previously been described in detail.[14] The baseline clinical data during the first CV visit were collected from electronic patient records by standardized protocol. Information about peripheral artery disease and coronary artery disease including previous myocardial infarctions was combined into a single variable depicting the presence of vascular disease. The CHA_2DS_2-VA score is calculated according to the 2024 European Society of Cardiology guidelines.[16]

This substudy targeted patients undergoing CV at Turku University Hospital and having sinus rhythm ECG after the first successful CV stored in the ECG database (MUSE Cardiology Information System 9.0; GE HealthCare, Chicago, IL). We have previously shown that post-CV atrial stunning has no significant effect on P-wave duration, PTF, or morphology.[17] To assess the long-term stability and progression of PWAs before and after this index ECG, we collected the earliest (within 5 years) and latest available sinus rhythm ECG from the database. Supplemental Figure 1 summarizes the study flowchart.

Abbreviations

aCM: atrial cardiomyopathy

AIAB: advanced interatrial block

AF: atrial fibrillation

CHA_2DS_2-VA: congestive heart failure, hypertension, age ≥75 years (doubled), diabetes, prior stroke or transient ischemic attack or thromboembolism (doubled), vascular disease, age 65–74 years

CV: cardioversion

ECG: electrocardiogram

PTF: P-terminal force

PWA: P-wave abnormality

ECG

The ECG database was searched for standard ECGs in sinus rhythm calibrated to 50 mm/s speed and 10 mm/mV voltage gain. Line thickness was 0.15 mm. Measurements were made manually with the precision set to 0.25 mm (5 ms, 0.025 mV), always rounding down. Digital magnification was used up to 64×, but no other tools were used. ECG analysis was performed by 2 independent observers. Repeated measurements of each patient were carried out in different evaluation sessions, whereas no actual blinding to previous data was performed.

P-wave duration was assessed from all limb leads simultaneously by identifying earliest and latest baseline deviations. Multiple waves were measured from each ECG to acknowledge P-wave dispersion and signal noise. Of the 7 previously described P-wave morphologies (normal, bimodal, biphasic, tail, peak, flat, and deflected) in lead II, here we focused on the deflected and biphasic waveforms.[4] The wave was considered deflected if a distinct peak was followed by a second, flattened peak. A biphasic wave was required to have a positive deflection followed by a 40-ms-long negative deflection with at least 0.025 mV upward deviation. The presence of biphasic P-waves was also noted in leads III and aVF to diagnose AIAB in addition to P-wave elongation (≥120 ms). PTF was calculated as the product of depth and duration of the terminal negative portion of P-wave in lead V_1.

Patients were assigned to normal P wave, moderate PWA, or extensive PWA categories according to ECG findings.[4] The extensive PWA category included participants with P-wave duration ≥180 ms, PTF ≥80 mm·ms, deflected morphology, or AIAB (P-wave duration ≥120 ms and biphasic morphology in all inferior leads). Moderate PWA consisted of individuals with P-wave duration ≥150 ms and <180 ms as well as PTF ≥40 mm·ms and <80 mm·ms. P wave was considered normal if P-wave duration was <150 ms and PTF <40 mm·ms.

In addition to P-wave analysis, the QRS complexes were assessed for left ventricular hypertrophy and conduction abnormalities. QRS amplitudes were measured according to Sokolow-Lyon, Cornell, and modified Cornell criteria. Left ventricular hypertrophy was considered present if any of the criteria were met.[18] Bundle branch blocks and unspecified ventricular conduction defects were diagnosed according to the consensus criteria.[19] For this study, we used a composite variable "wide QRS" that includes all defects with QRS duration ≥120 ms.

Statistical analysis

Statistical analyses were conducted with SPSS version 27.0 (IBM, Armonk, NY) and R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria) using the "survival," "lme4," and "lmerTest" packages. A 2-sided P value < .05 marked statistical significance. To keep data presentation uniform, analyses of prior index CV time period include 797 patients (both pre-CV and index CV ECGs available), and analyses of post-CV time period include 1121 patients (both index CV and follow-up ECGs available), unless otherwise specified in subgroup analyses. Univariate analyses were performed with Pearson χ^2 test, Fisher exact test, McNemar test, analysis of variance with Tukey test, Wilcoxon rank sum test, and Kruskal-Wallis test as appropriate. Table 1 applies the Bonferroni correction in groupwise testing. A linear-by-linear association test was used to assess the trend in PWA progression during follow-up quartiles. Risk factors for PWA evolution were studied with the Cox proportional hazards regression. Multivariable models included all variables with P values <

Table 1 Clinical characteristics of patients at index cardioversion

	P-wave abnormality					
	Normal (n = 407)	Moderate (n = 453)	Extensive (n = 261)	P*	P†	P‡
Age, y	61 (53–71)	63 (55–71)	66 (58–73)	<.001	.180	<.001
≥65 y	161 (39.6)	206 (45.5)	147 (56.3)	<.001	.255	<.001
≥75 y	59 (14.5)	67 (14.8)	56 (21.5)	.033	.999	.064
Male sex	227 (55.8)	300 (66.2)	165 (63.2)	.006	.006	.192
Heart failure	11 (2.7)	24 (5.3)	14 (5.4)	.119	.012	.031
Hypertension	156 (38.3)	218 (48.1)	127 (48.7)	.005	.177	.282
Diabetes	37 (9.1)	62 (13.7)	33 (12.6)	.1	.126	.465
Prior stroke or TIA	19 (4.7)	31 (6.8)	18 (6.9)	.334	.573	.687
Vascular disease	66 (16.2)	97 (21.4)	77 (29.5)	<.001	.168	<.001
CHA_2DS_2-VA score	1 (0–2)	1 (0–3)	2 (1–3)	<.001	.001	<.001
≥2	142 (34.9)	205 (45.3)	144 (55.2)	<.001	.007	<.001
Previous AF episodes	248 (60.9)	291 (64.2)	175 (67.0)	.263	.972	.354
Any AAD	217 (53.8)	295 (66.1)	191 (74.3)	<.001	<.001	<.001
Class I or III AAD	58 (12.6)	63 (12.0)	65 (20.1)	.002	.999	.015
Wide QRS	19 (4.7)	33 (7.3)	28 (10.7)	.012	.351	.014
LVH	48 (12.0)	89 (20.0)	56 (22.3)	<.001	.006	.002

Total number of patients amounts to 1121 to ensure uniform presentation of the data with further analyses. Data are reported as median (interquartile range) or count (percentage), as appropriate.

AAD = antiarrhythmic drug; AF = atrial fibrillation; CHA_2DS_2-VA = congestive heart failure, hypertension, age ≥75 years (doubled), diabetes, prior stroke or transient ischemic attack or thromboembolism (doubled), vascular disease, age 65–74 years; LVH = left ventricular hypertrophy; TIA = transient ischemic attack.

*Difference between all groups.

†Bonferroni corrected comparison of normal P wave and moderate P-wave abnormality.

‡Bonferroni corrected comparison of normal P wave and extensive P-wave abnormality.

.10 in univariable testing, except for the CHA_2DS_2-VA score, which was omitted because of obvious correlations with other covariates. Regression analyses were initially performed with a composite group whereby patients with normal P-wave or PWA improvement were compared with those with progressed PWA or persistent PWA status. Further comparisons were made between normal P-wave and new moderate PWA or extensive PWA groups. Regression analyses were performed only to assess post-CV PWA evolution as clinical information was collected concurrent to index CV. A linear mixed effect model was used to estimate mean P-wave duration and its rate of change before and after index CV as ECG collection intervals were not fixed. Patient ID was treated as a random effect. Model performances were compared stepwise after each covariate alteration by Akaike and Bayesian information criteria, the Bayes factor, and analysis of variance test (data not shown); only the final multivariate models are presented.

Study ethics

The FinCV study has received approvals from the Medical Ethics Committee of the Hospital District of Southwest Finland and the ethics committee of the National Institute for Health and Welfare. The study conforms to the Declaration of Helsinki. Informed consent was not required because of the retrospective nature of the study.

Results

The sinus rhythm ECG at index CV was available in 1316 patients; 797 (60.5%) of them had a pre-CV ECG and 1121 (85.2%) had follow-up recordings. The median time was 2.6 (1.0–4.1) years (n = 797) between pre-CV and index ECGs and 6.1 (2.5–10.3) years (n = 1121) between the index and follow-up ECGs.

Changes in PWAs before index CV

In the pre-CV ECG, moderate PWA was observed in 310 (38.9%) patients and extensive PWA in 145 (18.2%) patients. At index ECG after CV, normal P-wave had progressed to moderate PWA in 133 (38.9%) patients and to extensive PWA in 54 (15.8%) patients, and the PWA changes had regressed to normal P-wave morphology in 95 (20.9%) patients. The median time between ECGs was similar between patients with progressed, persistent, or improved PWA (data not shown).

Changes in PWAs after index CV

In the ECGs immediately after index CV, 453 (40.4%) patients had moderate PWA and 261 (23.3%) had extensive PWA. Participants with extensive PWA were more likely to be older and suffered from cardiovascular conditions (Table 1). AF recurred within 30 days of the index CV in 47 (11.5%), 62 (13.7%), and 52 (20.0%) of cases with normal P-wave, moderate PWA, and extensive PWA, respectively ($P = .009$). In the same groups, high frequency of CVs (time between procedures <180 days) was observed in 42 (11.4%), 59 (14.8%), and 52 (21.6%) patients during the follow-up ($P = .003$). Overall, 69 (7.1%) patients had AF ablation during the follow-up period, and the rates were similar between PWA groups (data not shown).

In the follow-up ECG, 131 (32.2%) patients with previously normal P-wave in the index ECG had a moderate PWA and 74 (18.2%) had extensive PWA. Conversely, PWA had regressed to normal P-wave in 83 (18.3%) patients with previously moderate PWA and 26 (10.0%) with previously extensive PWA. Thus, 545 (38.6%) patients had moderate PWA and 470 (33.3%) had extensive PWA in the follow-up ECG. Complete groupwise patient flow is presented in Figure 1. PWA progression was more often observed in ECGs analyzed in the quartile with longest follow-up time compared with those analyzed in the shortest quartile of follow-up (P = .002, for trend; Figure 2). Example ECGs from a single patient with PWA progression are provided in Figure 3.

Risk factors for PWA evolution were assessed with a composite variable comparing progressed or persistent PWA with improved PWA or persistently normal P wave. Results of Cox regression analyses are listed in Table 2. Subsequently, progression from normal P wave to moderate PWA and extensive PWA was studied in subgroup analyses presented in Supplemental Table 1. In a multivariable Cox regression analysis, age (hazard ratio [HR], 1.04; 95% confidence interval [CI], 1.01–1.06; P = .001), heart failure (HR, 1.15; 95% CI, 1.03–9.77; P = .045), AF recurrence within 30 days of index CV (HR, 1.92; 95% CI, 1.06–3.49; P = .033), and left ventricular hypertrophy (HR, 2.86; 95% CI, 1.69–4.84; P < .001) were the significant predictors for new moderate PWA, but only age (HR, 1.06; 95% CI, 1.03–1.09; P < .001) predicted development of new extensive PWA.

Evolution of individual PWAs

The P-wave parameters underlying composite PWA at each time point are listed in Supplemental Table 2. In a linear mixed effect model without covariates, P-wave duration was estimated to 136.45 ms (standard error [SE], 1.23 ms) 5 years before index CV, and its rate of change was 1.12 ms/y (SE, 0.28). Similarly, at the index CV, the univariate estimate for P-wave duration was 143.45 ms (SE, 0.76), and the duration

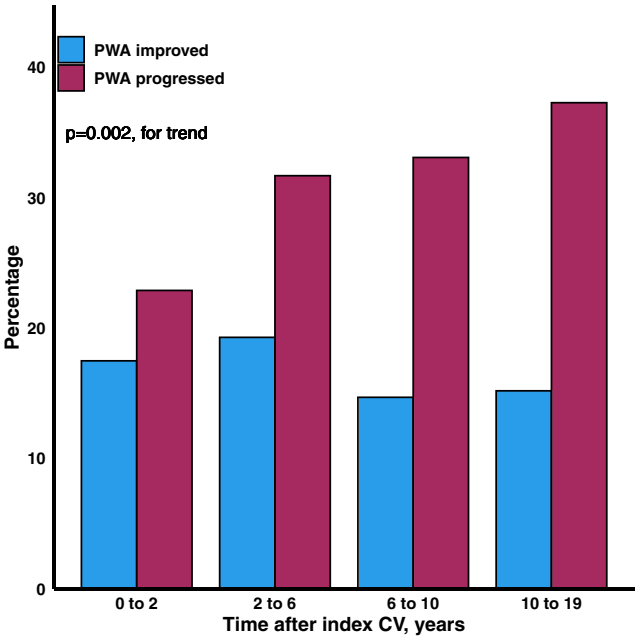

Figure 2
Distribution of follow-up electrocardiograms (ECGs) with improvement or progression in P-wave abnormality (PWA) categories. The evolution of PWA between index and follow-up ECGs was assessed in follow-up time quartiles. ECGs analyzed later in the follow-up had more likely PWA progression. CV = cardioversion.

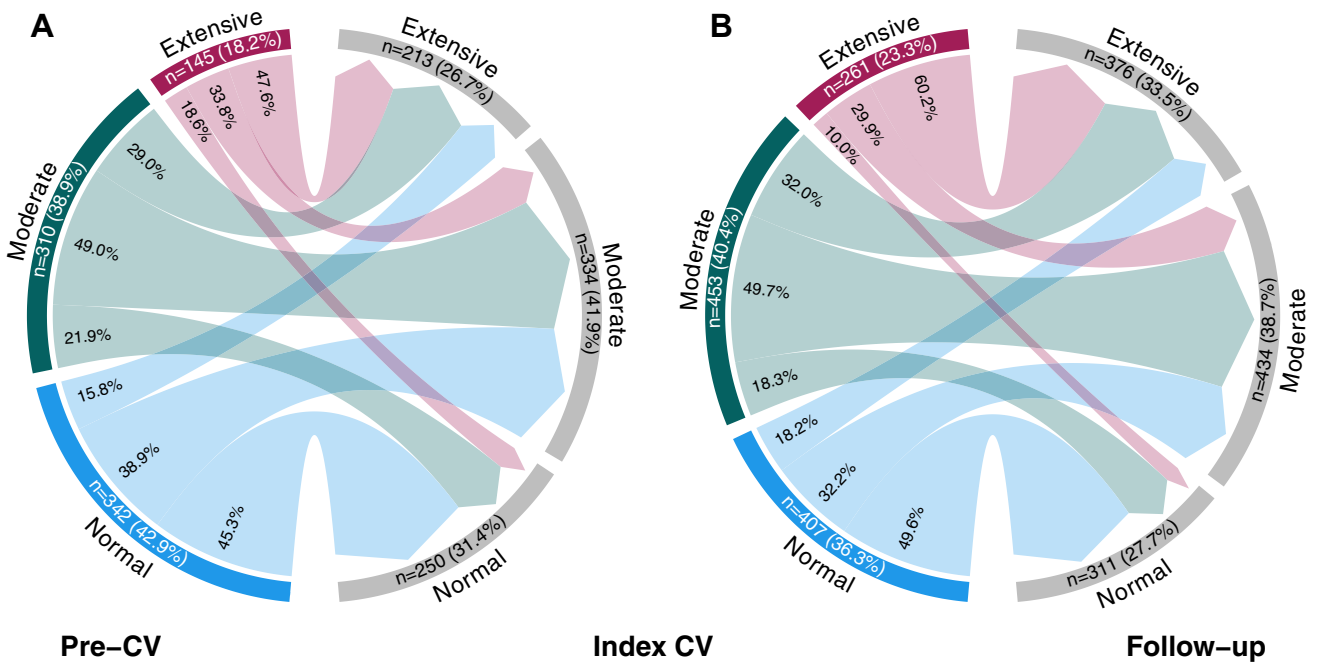

Figure 1
Temporal change in P-wave abnormality categories. **A:** Changes in P-wave abnormality category between pre-cardioversion (CV) and index CV (n = 797). **B:** Change between index CV and follow-up (n = 1121).

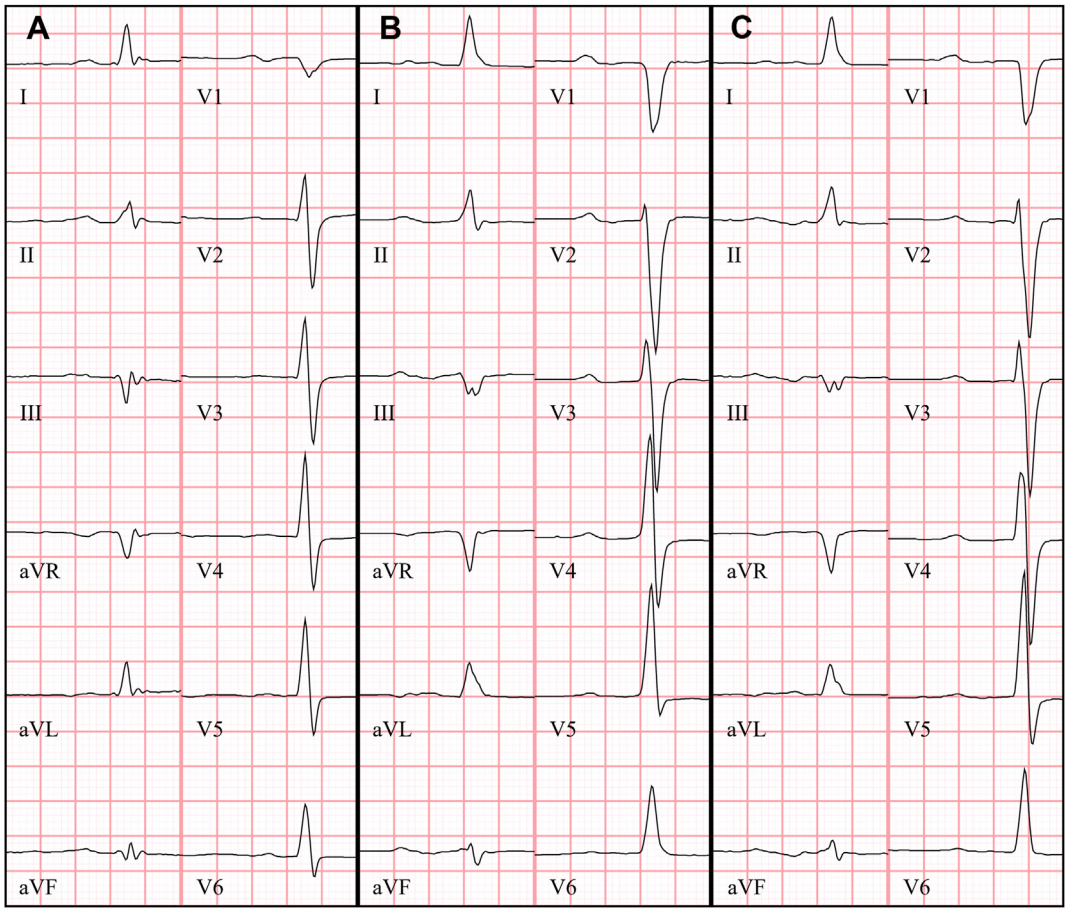

Figure 3
Sample electrocardiograms from a single patient. **A:** Pre-cardioversion recording with no P-wave abnormalities: P-wave duration 110 ms, normal morphology in inferior leads, P-terminal force 16.25 mm·ms. **B:** Moderate P-wave abnormality at index cardioversion: P-wave duration 160 ms, tail morphology in lead II and biphasic in leads III and aVF, P-terminal force 37.5 mm·ms. **C:** Extensive P-wave abnormality in the follow-up electrocardiogram: P-wave duration 200 ms, biphasic morphology in inferior leads (advanced interatrial block), P-terminal force 23.75 mm·ms. Recordings are calibrated to 50 mm/s speed and 10 mm/mV voltage gain.

increased 1.24 ms/y (SE, 0.08). Simple projections of pre-CV and post-CV models are presented in Figure 4, and the results of multivariable models are shown in Table 3.

Discussion

Our large study with repeated ECG recordings of 1316 AF patients shows that PWAs are a slowly progressive phenomenon. Two of 3 patients with an existing PWA in the pre-CV ECG had stable or progressed status in the index ECG at CV, and only 1 in 5 had reverted to normal P wave. The respective percentages were 74% and 15% in the follow-up after the index CV. The progression in PWA categories was more prominent after longer follow-up and resulted mainly from gradual increase in P-wave duration over time. As expected, age and chronic cardiovascular diseases predicted progression of PWAs.

PWA, as a composite variable, was constructed for a prior study and therefore lacks information about prevalence or incidence. For individual PWAs, the prevalence varies significantly. In the general population, the prevalence of AIAB is around 1%, whereas PTF (≥40 mm·ms) ranges from 2% to 16%.[5,10,11] For patients with AF, the respective percentages increase to >10% and 20%.[17,20] In this study, the prevalence of AIAB and PTF varied between 3%–12% and 36%–42%, respectively. The overall prevalence of PWA increased at each time point, with 7 of 10 patients exhibiting some degree of PWA at the end of the follow-up, although some normalization was also observed. Whereas shifts between categories may reflect actual physiologic changes, some could also be explained by patients being initially borderline between categories. A meta-analysis outlined that effective treatment of hypertension reduces P-wave duration, and in some cases AIAB has been described as a transient phenomenon comparable to transient ventricular block.[8,12]

Previous research regarding the progression of individual PWAs is scarce but mainly in line with our results. In longitudinal studies, new elongated P wave (≥120 ms), AIAB, and PTF were associated with aging and other cardiac risk factors such as hypertension and vascular disease.[10,11] In line with these observations, these factors together with heart failure were the most significant risk factors for progressive or persistent PWA, and their combination in the higher CHA_2DS_2-VA scores predicted the same outcome. The results were aligned in subgroup analysis assessing progression from normal P

Table 2 Risk factors for persistent or progressed P-wave abnormality after index cardioversion

	Univariable HR (95% CI)	*P*	Multivariable HR (95% CI)	*P*
Age, per year	1.04 (1.03–1.05)	<.001	1.03 (1.03–1.04)	<.001
Male sex	0.88 (0.76–1.02)	.095	1.15 (0.98–1.37)	.096
Heart failure	1.84 (1.30–2.61)	<.001	1.56 (1.05–2.33)	.027
Hypertension	1.75 (1.51–2.02)	<.001	1.37 (1.15–1.63)	<.001
Diabetes	1.42 (1.14–1.77)	.002	1.05 (0.81–1.36)	.701
Previous stroke or TIA	1.77 (1.33–2.36)	<.001	1.03 (0.73–1.46)	.852
Vascular disease	1.88 (1.58–2.24)	<.001	1.28 (1.04–1.46)	.020
CHA_2DS_2-VA	1.38 (1.32–1.45)	<.001		
Previous AF episodes	1.32 (1.13–1.54)	<.001	1.20 (1.01–1.43)	.043
Early AF recurrence*	1.35 (1.11–1.65)	.003	1.20 (0.95–1.53)	.130
High CV frequency†	1.88 (1.52–2.32)	<.001	1.30 (1.03–1.64)	.026
Any AAD	1.54 (1.32–1.81)	<.001	0.99 (0.83–1.20)	.943
Class I or III AAD	0.95 (0.77–1.18)	.639		
Wide QRS	1.78 (1.37–2.31)	<.001	1.74 (1.27–2.38)	<.001
LVH	1.59 (1.32–1.92)	<.001	1.33 (1.08–1.65)	.007

Cox regression analysis comparing the composite of progressed or persistent P-wave abnormality status (n = 732) with improved P-wave abnormality or consistently normal P wave (n = 389).
CI = confidence interval; CV = cardioversion; HR = hazard ratio; other abbreviations as in Table 1.
*Recurrence within 30 days after the index CV.
†High CV frequency: an interval between procedures <180 days.

wave to either new moderate PWA or extensive PWA. Similarly, cross-sectional studies have shown longer P-waves in older people, but no significant P-wave elongation was observed during a 3-year follow-up.[6,7,13] Here, the linear mixed effect models demonstrate modest yearly prolongation in P-wave duration; however, the cumulation seems to account for clinically significant progression during longer follow-up. Whereas other typical cardiovascular risk factors promoted PWA progression, diabetes remained insignificant, suggesting a difference in underlying mechanisms.

Beyond age and comorbidities, previous AF history and high CV frequency predicted PWA progression. Patients with extensive PWA were more likely to experience early AF recurrence (within 30 days) after the index CV. Although this study lacks precise AF burden data, these factors could serve as indirect markers of increased episode frequency, which

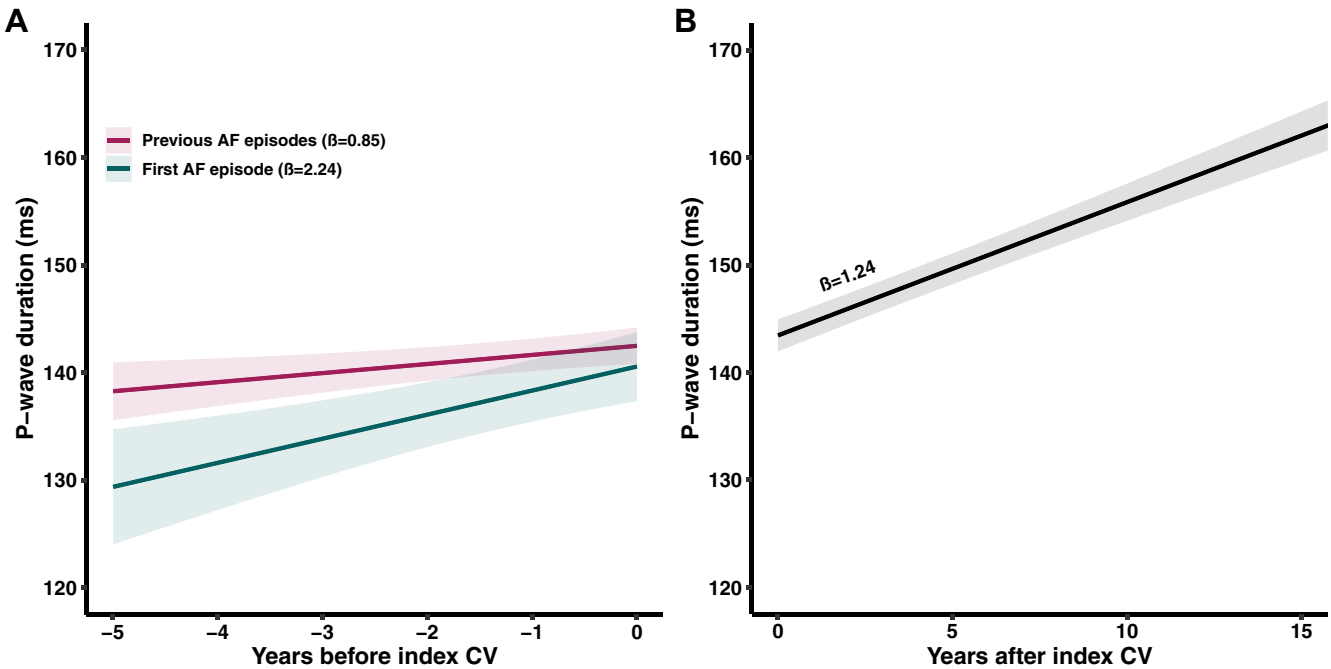

Figure 4
Estimated progression of P-wave duration over time. (**A**) Change between pre-cardioversion (CV) and index CV and (**B**) change after index CV in linear mixed effect models. The models represent the general trend; only **A** is adjusted for previous atrial fibrillation (AF) history to highlight the difference in slopes (β). The shaded area represents 95% confidence intervals. Note the x-axis scaling.

Table 3 Covariates affecting P-wave duration in linear mixed effect models

	Intercept (SE)	Slope, per year (SE)
P-wave duration, ms (pre-CV)	116.94 (3.71)	2.32 (0.61)
Age ≥65 y	+6.66 (1.36)	
Male sex	+5.61 (1.41)	
Previous AF episodes	+8.87 (3.03)	−1.48 (0.68)
P-wave duration, ms (index CV)	124.21 (3.03)	1.29 (0.08)
Age ≥65 y	+6.39 (1.64)	
Male sex	+5.76 (1.60)	
Hypertension	+4.55 (1.59)	
Vascular disease	+4.86 (1.99)	
High CV frequency	+5.42 (2.12)	
Wide QRS	+9.50 (3.28)	
LVH	+3.43 (2.03)	

The linear mixed effect model was set to estimate the mean P-wave duration (intercept) at 5 years before cardioversion (CV) and at index CV as well as the rate of yearly change (slope). The presence of each covariate adds to the estimated intercept and slope accordingly. For example, if all covariates at pre-CV are present, the P-wave duration estimate adds to 138.08 ms and the slope to 0.84 ms/y. Figure 4A visualizes the difference in slopes. Covariates mainly had a significant effect on the intercept, and slope was affected only by previous AF status in the pre-CV model.
SE = standard error; other abbreviations as in Table 1.

may promote atrial remodeling.[2] In contrast, effective rhythm control and sinus rhythm maintenance appear to reverse electromechanical alterations.[1] Despite that more than two-thirds of patients were using antiarrhythmic drugs, these did not influence PWA evolution in the multivariable analyses, even though class I and class III agents, in particular, might alter P-wave morphology and duration.[21]

The exact pathophysiologic process underlying the PWA is unknown. Whereas fibrosis may be the final outcome in the continuum, indicated by extensive PWA being characterized by highest areas of fibrotic tissue, the atria may undergo other alterations during the process.[1,3,4,22] AIAB has been described as a specific impairment of interatrial Bachmann bundle, and along with markedly increased P-wave duration and PTF, it has been associated with poor contractility of the atrial tissue and low-voltage areas in intracardiac measurements.[3,12,23,24] AIAB and PTF share a strong association with left atrial enlargement, which may precede conduction defects.[3,12,22] In a recent study, however, PTF was not associated with atrial enlargement; instead, it was found to correlate with electrical remodeling.[25] Interestingly, the histologic samples showed less fibrosis for larger PTF values, explained by the fact that viable cardiomyocytes generate greater terminal amplitudes.[25]

Various PWAs have been proposed to complement aCM diagnosis, which relies on biomarkers, imaging, and electrophysiology to evaluate atrial size and function.[1,2] Although the composite PWA variable has not been validated as a marker for aCM, notable similarities exist; aCM is defined as an electromechanical entity capable of causing clinically relevant manifestations, primarily driven by aging and accelerated by comorbidities.[1] Most importantly, the connection of aCM with AF has been established, although the exact nature of this relationship remains unclear.[1,2] PWA has been associated with atrial fibrosis, stroke, heart failure, and death in AF patients.[4] Its progressive nature is influenced by aging and chronic cardiovascular conditions, with AF history or high CV frequency increasing the risk of PWA progression. Further research is needed to better understand the pathophysiologic properties of PWA.

Clinical implications

The persistent and progressive nature of PWA supports the view that the classification might be useful in clinical practice as an indicator for AF recurrences and complications. We previously showed a gradual increase in stroke risk with PWA categories regardless of the CHA_2DS_2-VASc score in nonanticoagulated AF patients.[4] Similar findings have been demonstrated with individual P-wave parameters, but information on the stability of these markers has been minimal.[26–28] In addition, these results show that old age, chronic cardiovascular diseases, and frequent AF recurrences are the crucial driving forces for PWA progression. Although our study lacks statistical power, our findings indirectly support the current recommendations on careful treatment of comorbidities and early rhythm control that may also prevent PWA progression.[16]

Limitations

The limitations of a retrospective study setting apply. In addition, pre-CV and follow-up ECGs were not acquired at standardized time points, which may introduce heterogeneity in the temporal assessment of PWA progression. Despite this, the large sample size allowed sufficient distribution of ECGs over time. Furthermore, the lack of imaging and biomarker data limits the ability to correlate PWA progression with atrial remodeling, which could provide a more comprehensive understanding of the underlying pathophysiologic process and aCM.

Conclusion

This study highlights the progressive nature of PWA in patients with paroxysmal AF, driven primarily by aging, chronic cardiovascular diseases, and frequent AF recurrences. The findings emphasize the need for further research to clarify the pathophysiologic mechanisms underlying PWA and its potential role as a marker for aCM.

Funding Sources: This work was supported by grants from the Aarne Koskelo Foundation, the Alfred Kordelin Foundation, the Finnish Foundation for Cardiovascular Research, the Finnish Medical Foundation, the Orion Research Foundation, the Paavo Nurmi Foundation, the Paulo Foundation, and the State Research Funding by the Hospital District of Southwest Finland. The funding sources had no role regarding study design and conduct including collection, management, analysis, and interpretation of the data; preparation, review, and approval of the manuscript; or decision to submit the manuscript for publication.

Disclosures: K.E. Juhani Airaksinen has received speaker fees from Bayer, Pfizer, and Boehringer Ingelheim. The remaining authors have no conflicts of interest to disclose.

Authorship: All authors attest they meet the current ICMJE criteria for authorship.

Data Availability: Data underlying this article are available on reasonable request to the corresponding author.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2025.03.1981.

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