

# The electrocardiographic characteristics of the P-wave with incomplete Bachmann's bundle block in patients with atrial fibrillation

Charakterystyka elektrokardiograficzna załamka P u pacjentów z niepełnym blokiem pęczka Bachmanna i migotaniem przedsionków

Jakub Szymon Mercik<sup>1</sup>, Malte Unkell<sup>2</sup>, Maxim Marinov<sup>2</sup>, Peter Stephan Wolff<sup>2</sup>,  
Jadwiga Radziejewska<sup>3</sup>, Jacek Gajek<sup>1</sup>, Agnieszka Sławuta<sup>4</sup>

<sup>1</sup>Department of Emergency Medicine, Wrocław Medical University, Wrocław, Poland

<sup>2</sup>Students' Scientific Association, Department of Emergency Medical Service, Wrocław Medical University, Wrocław, Poland

<sup>3</sup>Kłodzko County Hospital, Kłodzko, Poland

<sup>4</sup>Department of Internal and Occupational Diseases, Hypertension and Clinical Oncology,  
Wrocław Medical University, Wrocław, Poland

## Abstract

**Introduction.** The structural and functional changes in atrial myocardium create a substrate for atrial fibrillation (AF). The pathophysiology includes enlargement of the cavities and replacing the cardiomyocytes with connective tissue. That slows down the conduction velocity and creates focal conduction block zones and slow conduction areas promoting the re-entrant circuits. The described changes influence the duration and amplitude of the P-wave in the electrocardiography.

**Materials and methods.** The study group consisted of 54 patients diagnosed with AF. There were 19 women and 35 men, aged  $65.8 \pm 10.0$  years. 22 patients had paroxysmal AF, in sinus rhythm during the examination and 32 had persistent AF, in whom the direct current cardioversion was performed to achieve sinus rhythm.

**Results.** In patients with persistent atrial fibrillation the P-wave duration after the restoration, the sinus rhythm was significantly longer in comparison to patients with paroxysmal atrial fibrillation ( $159.2 \pm 14.3$  vs.  $171.2 \pm 16.6$  ms,  $p = 0.006$ ). The patients with persistent AF showed higher positive amplitude in lead V1 as well as higher negative amplitude than patients with paroxysmal AF; positive amplitude:  $0.053 \pm 0.023$  vs.  $0.084 \pm 0.040$  mV,  $p = 0.002$ , negative amplitude:  $0.045 \pm 0.018$  vs.  $0.075 \pm 0.037$  mV,  $p = 0.001$ .

**Conclusions.** In patients with incomplete Bachmann's bundle block and atrial fibrillation, the duration of the P-wave is prolonged, more in the persistent arrhythmia group. The P-wave duration and morphology correlate with the interatrial conduction disturbances. The P-wave morphology changes indicate the causal relationship of AF rather with interatrial conduction delay than with left atrial hypertrophy.

Key words: P-wave duration, P-wave morphology, incomplete Bachmann's bundle block, atrial fibrillation

Folia Cardiologica 2020; 15, 5: 343–348

## Introduction

The structural and functional changes in atrial musculature create a substrate for atrial fibrillation [1]. Those pathologies result from numerous diseases such as ischaemic heart disease, hypertension or heart defects, in particular mitral valve disease [2]. Over time and the ongoing course of pathophysiological changes, the atria enlarge and cardiomyocytes are replaced by connective tissue [3, 4]. The slowing down the activation wave of conduction velocity creates focal conduction block zones and slow conduction areas promoting the formation of re-entrant circuits. Those changes promote arrhythmia maintenance. The influence for described disturbances contributes to the functional isolation of left atrium and pulmonary veins junctions leading ultimately to the creation of trigger zones for atrial fibrillation [5].

The structural changes described above contribute to the formation of arrhythmogenic foci perpetuating premature beats and atrial rhythms. Rapid arrhythmias induce local changes in the refractory period of the atrial muscle, which increases the possibility of re-entrant rhythms and leads to the persistence of atrial fibrillation [6]. The impact of arrhythmic paroxysms on further enlargement of the left atrium by increasing left ventricle filling pressure leads to consecutive structural changes.

All of this affects the duration and amplitude of the P-wave in the electrocardiography (ECG). The slow muscle conduction and the left atrium enlargement causes the prolongation of the P-wave duration [7]. In hypertension patients, the pressure overload can induce atrial musculature hypertrophy with an increase in P-wave amplitude (increase in negative deflection in lead V1) [8]. However, a reduction in the number of cardiomyocytes and an increase in the amount of connective tissue can decrease

the generated potential and thus decrease the P-wave amplitude. The profound changes in intra- and interatrial conduction are related to the Bachmann's bundle block. Changes in activation time significantly affect the P-wave duration and morphology [9]. The detailed assessment of those parameters has not been extensively studied in the literature so far.

The authors aimed at the assessment of the P-wave characteristics in patients with atrial fibrillation and incomplete Bachmann's bundle block.

## Material and methods

The study group consisted of 54 patients with atrial fibrillation. There were 19 women and 35 men, aged  $65.8 \pm 10.0$  years. The presence of essential co-morbidities and coexisting anti-arrhythmic treatment was also assessed. The patients' population was divided into 22 patients with paroxysmal atrial fibrillation and 32 patients with a persistent form of arrhythmia, in whom the electrical cardioversion was performed to re-establish sinus rhythm. All the patients were treated with beta-blockers and some of them received propafenone or amiodarone. In patients with persistent AF, the precise estimation of the arrhythmia was not possible, but to narrow the time span included only the patients with episodes lasting from 1 to 6 months. The clinical characteristics of the study group were presented in Table 1.

The P-wave parameters: duration and amplitude in particular leads were measured using LabSystem™ Pro EP Recording System from Boston Scientific. The P-wave duration was measured in all leads at a paper speed of 200 mm/s and enhancement 64–128. The P-wave morphology was assessed in lead II to recognize the typical pattern of the incomplete Bachmann's bundle block. The amplitudes

**Table 1.** The clinical characteristics of the study groups

Parameter	Patients with paroxysmal AF	Patients with persistent AF	p
Number	22/54	32/54	0.382
Sex	8 F, 14 M	9 F, 23 M	0.390
Age	$67.8 \pm 7.2$	$64.8 \pm 10.2$	0.321
HT	20/22	30/32	0.874
IHD	6/22	4/32	0.197
HF	2/22	3/32	0.924
DM	4/22	7/32	0.668
CKD	4/22	2/32	0.192
Metoprolol	7/22	14/32	0.290
Amiodarone	3/22	3/32	0.458
Propafenone	12/22	25/32	0.457

AF – atrial fibrillation; F – female; M – male; HT – hypertension, IHD – ischaemic heart disease, HF – heart failure; DM – diabetes mellitus; CKD – chronic kidney disease

**Table 2.** The P-wave parameters in patients with paroxysmal and persistent atrial fibrillation (AF)

Parameter	Patients with paroxysmal AF	Patients with persistent AF	p
Number of patients	22	32	-
P-wave duration [ms]	<b>159.2 ± 14.3</b>	<b>171.2 ± 16.6</b>	<b>0.006</b>
Peak separation [ms]	58.3 ± 13.9	62.1 ± 18.7	0.56
Amplitude II lead [ms]	0.137 ± 0.164	0.109 ± 0.040	0.613
Amplitude V1 lead positive [ms]	<b>0.053 ± 0.023*</b>	<b>0.084 ± 0.040#</b>	<b>0.002</b>
Amplitude V1 lead negative [ms]	<b>0.045 ± 0.018*</b>	<b>0.075 ± 0.037#</b>	<b>0.001</b>

Differences between positive and negative phases in lead V1: in patients with paroxysmal AF: \*p = 0.16, in patients with persistent AF: #p = 0.062

of the P-wave were measured in lead II. The incomplete Bachmann's bundle block was recognized if the separation of the right and left atrial P-wave peaks was significant (more than 40 ms). The positive and negative phases in lead V1 were also measured. To avoid the measurement inaccuracies, all the measurements were repeated 5 times and presented as the mean value.

In the group of patients with persistent AF, the direct current cardioversion was done (in general anaesthesia under propofol 1 mg/kg and fentanyl 50 µg given intravenously) to restore the sinus rhythm. A single 300 J shock was successful in all patients.

The study protocol was accepted by the local Bioethical Committee at Wrocław Medical University.

### Statistical analysis

The continuous variables are presented as a mean and standard deviation. The comparisons were performed with the parametrical Students *t*-test or non-parametrical U Mann-Whitney test for independent variables. The dependent variables comparisons were performed using the Students *t*-test for dependent variables or Wilcoxon paired test. Each categorical variables were presented as numbers and/or percentages. The comparisons were performed with the chi-square test. The correlations between the studied parameters were performed using Pearson's correlations coefficient or Spearman's rank correlation according to the statistical properties of the data.

P values less than 0.05 were considered significant.

### Results

In patients with persistent atrial fibrillation, the P-wave duration after the restoration of sinus rhythm was significantly longer in comparison to patients with paroxysmal atrial fibrillation. The parameters and morphology of the P-waves were shown in Table 2.

The persistent arrhythmia group had more pronounced interatrial conduction abnormalities even if it didn't reach the statistical significance. The P-wave amplitude in lead II was not significantly different between the groups as was

the separation of the peaks in this lead. The patients with persistent AF showed higher positive amplitude in lead V1 as well as higher negative amplitude than patients with paroxysmal AF. The comparison of positive and negative V1 deflection in both groups according to atrial fibrillation form indicated the borderline statistical difference in persistent AF (p = 0.061).

The P-wave duration correlated with the separation of the right and left atrial peaks in lead II slightly differently in patients with paroxysmal (A) and persistent (B) atrial fibrillation, which was depicted in Figure 1.

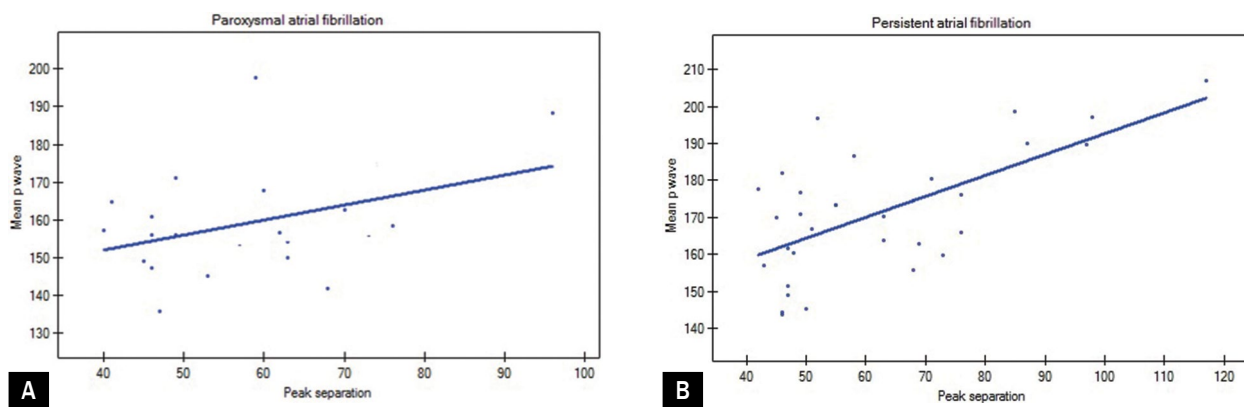
There was a strong positive correlation between the positive and the negative amplitudes in lead V1 in patients with persistent atrial fibrillation. This correlation was shown in Figure 2. This coherence did not reach statistical significance in patients with paroxysmal AF.

After the combination of the parameters for the whole group studied the strong positive interrelation between the positive and negative deflections in lead VI was maintained what was presented in Figure 3.

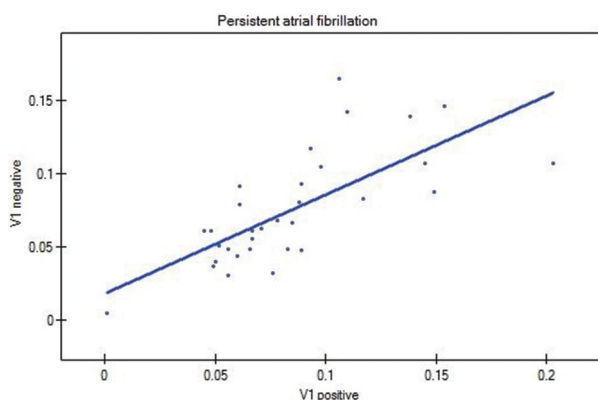
To illustrate the complexity of P-wave morphology in a patient with incomplete Bachmann's bundle block the typical example of the ECG tracings was presented in Figure 4.

### Discussion

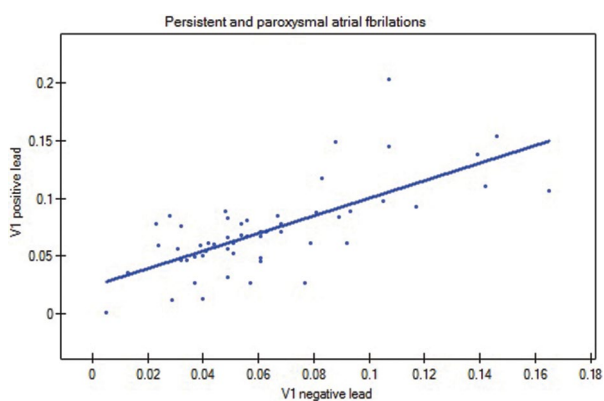
The main result of this study in patients with incomplete Bachmann's bundle block is the effect of sustained arrhythmia episodes on the prolongation of the P-wave duration. This indicates strongly the direct influence of atrial fibrillation-induced intra-atrial and inter-atrial conduction disorders and, most probably, atrial enlargement. The phenomenon should be associated with an increased left ventricle filling pressure and could be dependent on the ventricular rate [10]. Data on the effect of AF on muscle conduction are more scarce and non-equivocal, but if the prolongation of the P-wave is taken into account in combination with the peak separation of the lead II P-wave, one can draw the obvious conclusion that the prolongation is related to the interatrial conduction delay. As the subgroups



**Figure 1.** The mean P-wave duration correlation with peak separation in lead II in patients with paroxysmal (A) ( $r = 0.39$ ,  $p = 0.07$ ) and persistent (B) ( $r = 0.59$ ,  $p = 0.00207$ ) atrial fibrillation



**Figure 2.** A strong correlation ( $r = 0.766$ ,  $p < 0.000001$ ) between positive and negative amplitude in lead V1 in patients with persistent atrial fibrillation



**Figure 3.** A strong positive correlation ( $r = 0.663$ ,  $p < 0.000001$ ) between positive and negative amplitude in lead VI in the whole group of patients



**Figure 4.** The complexity of P-wave morphology in a patient with incomplete Bachmann’s bundle block (paper speed 200 mm/s, enhancement 64×)

of patients with both paroxysmal and persistent atrial fibrillation are similar according to age, comorbidities, gender distribution and anti-arrhythmic treatment, all of which

could affect the duration of the P-wave, it indicates that this study’s results are related only to the absence or presence to the prolonged episode of arrhythmia [1]. This observation seems to be directly in line with the clinical observation, contained in the term “AF begets AF” created by Wijffels et al. [11]. The direct current cardioversion did not affect by itself on the P-wave duration in one study, indicating the stable values of the P-wave parameter immediately after the procedure and on the next day [12].

Another important result from this study is the statistically significant relationship between the duration of the P-wave and the distance of the right and left atrium peaks in lead II. In both studied patients group, the positive correlation was maintained, in the persistent AF patients the correlations coefficient was higher. Although the difference in the separation of the peaks in both groups was statistically insignificant, the separation itself influenced the P-wave duration. It seems justified to conclude, that the prolongation of the P-wave is mainly due to conduction delay in the Bachmann’s bundle and not to the atrial muscle dimensions [13]. While there are no similar papers

yet, and the only one pointed to similar conclusions [14], this is consistent with the literature on Bachman's bundle conduction disorders [8, 9, 13].

The precise methodology for the measurement of the P-wave duration used in this study is the same one the authors adopted a few years ago to show the lack of P-wave dispersion — a parameter which is related to the inaccuracy of the measurement [15]. This approach is qualitatively different from the other researchers' methodology [16]. It is nevertheless in line with the suggestions of Bayes de Luna contained in the paper "Diagnosis of interatrial block", where it was explicitly pointed out to precisely measure the P-wave from the earliest beginning to the latest end [17].

Another novel issue resulting from this research is the increase in the amplitudes of the positive and negative phase of the V1 lead P-wave in patients with persistent AF. The direct comparison of the deflections in the studied groups indicate the more advanced disease in the patients with a persistent form of arrhythmia and most probably reflects the more enlarged left atria during ongoing arrhythmia. But this reflects also the known electrocardiographic pattern. In physiology, the overlap of the negative phase (left atrium) with the positive phase (right atrium) influences both amplitudes. The significant extension of the left atrium muscle mass increases the negative deflection in V1, with a negative predominance in this lead [8]. In both groups, in lead V1, the positive deflection is greater than the negative one, even if only barely visible. It gives rise to a hypothesis that among the patients with incomplete Bachmann's bundle block the interatrial conduction delay could be the cause of atrial fibrillation and not hypertrophy of the muscle [3, 18]. The analysis of our entire population indicates this electrocardiographic phenomenon to be true cause it's difficult to assume

the atrial fibrillation results in or is a result of right atrial hypertrophy/enlargement.

### Study limitations

An essential limitation of this study is a relatively small population of patients. The already mentioned very precise measurement method used in the study including the electrophysiological recording system could affect the comparability with other researcher's results. As the duration of persistent AF was not possible to be precisely established, the hypothesis, that the prolongation of the P-wave duration is caused mainly by the arrhythmia itself, should be assessed with caution. This constitutes the main limitation of this research, even if the available data contradict such correlation [12].

### Conclusions

In patients with incomplete Bachmann's bundle block and atrial fibrillation, the duration of the P-wave is prolonged, significantly more in the persistent arrhythmia group.

The P-wave duration and morphology correlate with the interatrial conduction disturbances.

The P-wave morphology changes indicate the causal relationship of AF rather with interatrial conduction delay than with left atrial hypertrophy.

### Funding

All authors none declared.

### Conflict of interest

The authors declare no conflict of interest.

### Streszczenie

**Wstęp.** Zmiany strukturalne i czynnościowe mięśnia przedsionków tworzą podłoże dla migotania przedsionków (AF). Patofizjologia obejmuje powiększanie jam przedsionków i zastępowanie kardiomiocytów tkanką łączną. Spowalnia to prędkość przewodzenia, tworzy strefy lokalnego bloku oraz obszary zwolnionego przewodzenia sprzyjające rytmom nawrotnym. Opisane zmiany wpływają na czas trwania i amplitudę załamka P w elektrokardiografii.

**Materiał i metody.** Badana grupa składała się z 54 pacjentów ze zdiagnozowanym AF. Stanowiło ją 19 kobiet i 35 mężczyzn w wieku  $65,8 \pm 10,0$  lat. U 22 pacjentów stwierdzono napadowe AF, a u 32 pacjentów występowała przetrwała forma arytmii i wykonano u nich kardiowersję prądem stałym w celu uzyskania rytmu zatokowego.

**Wyniki.** U pacjentów z przetrwałym AF czas trwania załamka P po przywróceniu rytmu zatokowego był znacznie dłuższy niż u pacjentów z napadowym AF ( $159,2 \pm 14,3$  vs.  $171,2 \pm 16,6$  ms;  $p = 0,006$ ). Pacjenci z przetrwałym AF wykazywali wyższą amplitudę dodatnią w odprowadzeniu V1, a także bardziej ujemną amplitudę w tym odprowadzeniu niż pacjenci z napadowym AF; amplituda dodatnia:  $0,053 \pm 0,023$  vs.  $0,084 \pm 0,040$  mV;  $p = 0,002$ , amplituda ujemna:  $0,045 \pm 0,018$  vs.  $0,075 \pm 0,037$  mV;  $p = 0,001$ .



**Wnioski.** U pacjentów z niepełnym blokiem wiązki Bachmanna i AF czas trwania załamka P jest wydłużony, bardziej w grupie przetrwałej arytmii. Czas trwania załamka P i morfologia korelują z zaburzeniami przewodzenia międzypredsiolkowego. Zmiany morfologii załamka P wskazują na związek przyczynowy AF raczej z opóźnieniem przewodzenia międzypredsiolkowego niż z przerostem lewego przedsionka.

Słowa kluczowe: czas trwania załamka P, morfologia załamka P, niepełny blok pęczka Bachmanna, migotanie przedsionków

Folia Cardiologica 2020; 15, 343–348

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