P wave duration and interatrial conduction abnormalities in paroxysmal and persistent typical atrial flutter

Short title: P wave duration in paroxysmal and persistent atrial flutter

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Abstract:

Background: Functional and structural changes, the enlargement of the right atrium is the background for the development of typical atrial flutter (AFL). These changes in ECG are manifested in the morphology of the initial part of the P wave.

Objectives: The aim of the study was to assess the duration and morphology of the P wave in patients with paroxysmal and persistent AFL.

Material and methods: The study population consisted of 131 patients with AFL, 38 women and 93 men aged 66 years (60-72), divided in 62 patients with paroxysmal and 69 with persistent AFL. P wave duration was measured with an electrophysiological system in all leads at a paper speed of 200 mm/s.

Results: The groups did differ in terms of gender (38/24 vs 55/14, (M/F), p = 0.033). Patients with persistent AF had a longer P wave duration - 175 ms ± 26.3 ms versus 159 ms ± 22.6 ms, p =0.01, and higher creatinine concentration - 1.2 mg/dl ± 0.60 mg/dl versus 1.08 mg/dl ± 0.68 mg/dl, p = 0.007. The presence and severity of interatrial conduction block (I-none, II-partial, III-total) was related to age of the patients (60.3+/-12.1 vs. 64.7+/-8.3 vs. 68.9+/-9.5 years, respectively).

Conclusions: Patients with persistent AFL show a longer P wave compared to paroxysmal AFL, regardless of comorbidities and antiarrhythmic drugs. The arrhythmia-related longer P wave duration should encourage the clinicians to restore sinus rhythm earlier in order to more effectively maintain it over the long term.

Key words: P wave duration, chronic kidney disease, Bachmann’s bundle block, typical atrial flutter

Introduction:

Typical atrial flutter (AFL) is primarily a disease of the right atrium of the heart [1]. The enlargement of the atrium dimensions is the most important factor responsible for the onset and persistence of arrhythmias.

Such widening of the right atrial cavity may occur in the course of numerous chronic and acute pathological processes in the right heart and pulmonary circulation, but also in the course of left heart pathology, especially in some valve defects [2,3]. A paroxysm of arrhythmia begins similarly to all other reentrant arrhythmias - with premature beats of various origins [4]. The clinical course of AFL is mainly dependent on the individual variability of an AV conduction. Typically such a conduction is 2:1, giving a ventricular rate of about 130-150/min with quite severe clinical symptoms [5]. In patients with an impaired atrioventricular conduction, 3:1 and 4:1 conduction types cause relatively mild symptoms of palpitations, and sometimes an irregular or a very slow conduction, clinically similar to a complete block [6].

The treatment of a typical AFL with the tricuspid isthmus ablation practically eliminates the problem, as literature data and our own experience indicate a high percentage of early and long-term efficacy, although, of course, arrhythmia recurrences continue to be likely [7, 8]. A prolonged atrial flutter should not be referred to as sustained, because after various periods of time the arrhythmia stops spontaneously or degenerates into an atrial fibrillation (AF), which in this situation often persists like a permanent arrhythmia [9].

Electrocardiographic changes accompanying a typical AFL do not have to be limited to changes in the initial part of the P-wave. A longer-lasting arrhythmia, due to an increase in the left ventricular filling pressure, may generate the left atrial hypertrophy and dilatation, with typical consequences such as a prolonged P-wave duration and an increase of the negative phase of the P-wave in lead V1. Moreover, age and comorbidities, especially hypertension, can lead to an impaired interatrial conduction, contributing to a complete conduction block, with an

image of a Bachmann's bundle block. The aforementioned pathological processes and coexisting diseases may lead to a situation where the primary right atrial pathology becomes a pathology of both atria, increasing the risk of a paroxysmal AF [10]. This increases the risk of thromboembolism, which in the recommendations is described similarly for both arrhythmias [11].

The purpose:

The purpose of our study was to assess the duration of the P-wave as well as the interatrial conduction abnormalities in patients with a typical atrial flutter in different clinical presentations of the arrhythmia.

Material and methods:

The study group consisted of 131 patients diagnosed with a typical atrial flutter: 38 females and 93 males, at the average age of 66 (60-72) years old. The essential co-morbidities were reported. 62 patients presented with a paroxysmal AFL (SR group), in the sinus rhythm during the examination and 69 other patients presented with a persistent AFL. In all included patients the successful cavo-tricuspid isthmus ablation was performed, In patients with persistent AFL it was the method of sinus rhythm restoration. An antiarrhythmic medication, including beta-blockers, propafenone and amiodarone, also in combinations, was also recorded. As the exact duration of the arrhythmia episodes was not possible to recollect, we only included the patients with the persistent AFL lasting from 2 to 48 weeks.

The P-wave duration, was measured using LabSystemTM Pro EP Recording System, Boston Scientific, where the ECG tracings allowed to assess the sinus P-waves. The P-wave duration measurement was meticulously in all leads at the paper speed of 200 mm/s and the enhancement of 64-128x after the cavo-tricuspid isthmus ablation. To avoid any influence of accidental measurement inaccuracies The P wave duration was measured in five consecutive

heartbeats, measured in all leads according to the principle earliest P wave signal on any of the 12 leads to latest signal in any lead and the mean value was presented as a result.

The study protocol was approved by the local Bioethical Committee at Wroclaw Medical University.

Statistical analysis

The statistical analysis was performed using the STATISTICA computer program (StatSoft, Inc., Tulsa, USA, version.13.3). P values lower than 0.05 were considered significant.

For quantitative variables, basic descriptive statistics were calculated (M - average, SD - standard deviation, Me - median, Q1 - lower quartile, Q3 - upper quartile, Min - minimum value, Max - maximum value) and the compliance of their distributions with the theoretical normal distribution was checked using the Shapiro-Wilk's W test. Comparisons were performed using either the Students' T test or the Mann-Whitney U test for independent groups, or the Kruskal-Wallis ANOVA for multiple comparisons. Each categorical variables were presented as numbers and percentages. The comparisons were performed using the Chi-square test. The correlations between the studied parameters were performed using Spearman's rank correlation coefficient according to statistical properties of the data.

The receiver operational curve (ROC) was used to assess the ability of the P-wave duration to categorize disease statuses. Based on the results of examination and receiver operating characteristic (ROC) analysis, a cut-off threshold for the P-wave duration was calculated for SR and AFL groups.

Results:

Basic clinical characteristics of the total population and the comparisons of two groups of studied patients are presented in Table 1.

Table 1.

The studied patients did not differ in terms of age, antiarrhythmic medication, use of aldosterone receptor blockers or basic laboratory tests results except for the creatinine concentration. There were no differences in coexisting diseases. The significant difference in the duration of the P-wave was established. Patients with a persistent form of AFL showed a longer P-wave in comparison to patients with a paroxysmal form of the arrhythmia. The creatinine level in patients with a persistent atrial flutter (AFL) was higher than in patients with a paroxysmal atrial flutter (SR) on average by 0.12 mg /dl (1.2 mg /dl vs 1.08 mg/dl; $p = 0.05$) and this difference is significant at the level of $p < 0.05$.

In the studied group of female patients, paroxysmal atrial flutter was more frequent than a persistent atrial flutter (38.7% vs. 20.3%, $p = 0.033$).

The demographic and electrocardiographic parameters in all studied patients according to interatrial conduction disorders are presented in Table 2. The criteria for incomplete Bachmann's bundle block recognition were the P-wave duration above 110 ms (time) and the distance of 40 ms or more between the two peaks in lead II (morphology). The complete Bachmann's bundle block was diagnosed in the presence of P wave prolongation (P>110 ms) and biphasic P-wave morphology in lead II [10].

Table 2.

A ROC curve for the duration of the P-wave as a parameter used to differentiate patients in terms of the atrial flutter type, was created with the cut-off threshold for the P-wave duration of 163 ms showing the categorization accuracy of 70.3%. This was depicted in Figure 1.

Figure 1

The scatter plot (correlation chart) of the mean duration of the P-wave against the creatinine concentration was created but it did not reveal any statistically significant correlations. The chart was presented in Figure 2.

Figure 2

In order to assess the potential effect of anti-arrhythmic treatment on the duration of the P wave, an analysis was performed in relevant patient subgroups. The results of the analysis are presented in Table 3. They coincide with the previously written work on the duration of the P wave in persistent and paroxysmal atrial fibrillation [10]

Table 3.

The results of multivariate analysis to analyze all potential contributors that could have affected the P-wave duration was presented in table 4.

Table 4.

The results of univariate and multivariate logistic regression analysis and odds ratios of exceeding the P-wave duration were then calculated and presented in table 5.

Table 5.

The only independent predictors of long P-wave duration (over 163 ms) were age - 71 years of age and older and the presence of persisting arrhythmia i.e. AFL at the beginning of the study.

Discussion:

Many factors contribute to the development of AFL. Undoubtedly, it is worthwhile to observe that this disease affects men more often than women. In the study by Granada et al. the incidence of AFL in men is 2.5 times higher than in women. The authors also note that the population of people with an atrial flutter is considerably older and more diseased, especially in terms of the heart failure, the chronic obstructive pulmonary disease and diabetes [12]. By contrast, Lelorier et al. compare the contributors of AFL and AF. It is only the patients gender that marks any statistically significant difference between the patients populations. In the study group, 70% of patients with AFL are males, while in the group with AF, males constitute nearly 60% of the population [13]. This is also the result of our study indicating the presence of

interatrial conduction disorders is more common at older age, which according to logistic regression analysis was an independently related. In patients with AF the same phenomenon could be observed [10].

The main finding of our study is the demonstration of a longer duration of the P-wave in patients with a persistent atrial flutter compared to patients with a paroxysmal arrhythmia and a sinus rhythm at the time of the study. The degree of this extension (approximately 15%) of the P-wave duration is not only statistically significant, but also clinically important. It should be emphasized that the P-waves measured by us in both groups significantly exceeded the normal values of 120 ms (150 vs 174 ms respectively). There are not many similar comparisons in the available literature but if they are mainly concern an atrial fibrillation [10]. As one of the few reports the paper Stiles et al. indicated that the patients with persistent AFL had statistically longer conduction within the right atrium, as well as more pronounced sinus node dysfunction (sino-atrial conduction). In electroanatomical visualization the authors showed also more slow conduction zones in AFL patients in comparison to sinus rhythm individuals. On the other hand the study group is relatively small (20 patients) and the study focuses on conduction delay within the right atrium which is responsible for AFL It is obvious that the small even if statistically significant conduction delay within the right atrium can’t be responsible for relatively large P-wave duration prolongation [14]. The authors come to the conclusion that the observed abnormalities could be responsible for AF development but it seems to be going too far. Thus our study concentrates on Bachmann’s bundle conduction indicating its crucial role in P-wave duration prolongation, while the atrial flutter itself as a disease of the right atrium does not cause significant changes in the duration of the P-wave. Nevertheless a frequent coexistence of both types of arrhythmias leads to a deeper reflection and linking of both arrhythmias types with each other by interatrial conduction delay.

Among our patients with the persistent atrial flutter, higher degrees of the block in the Bachmann's bundle were significantly more frequent. Among patients with incomplete block, 35 individuals manifested a persistent AFL vs.25 individuals with the sinus rhythm. Of the patients with a complete block, 26 had AFL vs.18 who were in the sinus rhythm, and these relationships were statistically significant. There are no descriptions of these disorders in the available literature. They are more likely to occur in the descriptions of AF patients [15]. These changes, described in the publications, relate primarily to the risk of AF, however, there are reports indicating interatrial conduction disturbances, in which the presence of the low amplitude of the P-waves contributes to the formation of an atypical atrial flutter, where the tachycardia loop mostly rotates around the left atrium and atrial septum [16]. With a typical AFL and an interatrial block, a lower amplitude of the flutter wave and the development of non-typical AFL morphologies were reported [17].

At this point, it is also worth mentioning the correct measurement of the P wave. Normal P waves are easy to measure because they have a high amplitude and you can clearly see their beginning and end, while in patients with a Bachmann bundle block, the duration of the P wave is extended and the amplitude decreases. which makes it difficult to find the exact end of the P wave [15] This led to the erroneous theory of P-wave dispersion [18], however, with modern technological possibilities, it is possible to accurately measure the P wave as the beginning and end in all 12- leads.

Even though diabetic patients were not often represented in our study group, the influence of diabetes and of elevated blood glucose levels on the duration of the P-wave and on the occurrence of AFL was noticeable. There is little literature on diabetes mellitus (DM) that leads to electrical changes in the atrial substrate, and it mainly concerns an atrial fibrillation [8, 19]. In the study by Movahed et.al, the authors suggested diabetes be a potent independent contributor to the development of AF and AFL. The atrial flutter occurred in 4 percent of

diabetic patients compared to 2.5 percent of non-diabetic controls [20]. In an experimental DM model, there was an association between an increased atrial fibrosis, an interatrial conduction delay, and a greater arrhythmia inducibility [21]. An equally interesting animal study confirmed these results, complementing them with additional observations of the P-wave duration extension in diabetic rats without the left atrial enlargement. The authors related these abnormalities with diabetic changes in the Cx junction protein [22]. Similar results were obtained in patients with an impaired fasting glycaemia leading to a significant increase in interatrial conduction time and a consequent reduction in the left atrial emptying volume and fraction [23].

In a subgroup of our patients, it was found that a chronic kidney disease and elevated creatinine levels were associated with AFL. In the literature there is a relationship described between the maximum P-wave duration and worsening of a renal disease to specific hemodialysis endpoints, death, or a specific decrease in the estimated glomerular filtration rate [24, 25]. With regards to the patients included in our study, only a small number of 11 (8.4%) patients presented with CKD as a comorbidity, but it was found that the creatinine level was not statistically significantly associated with the duration of the P-wave. This area requires further studies that will be unaffected by a small number of CKD patients. AF and AFL are often described together with a renal dysfunction, but mainly as a comorbidity. So far, no association has been found that CKD might be causing AF and AFL. However, it seems that there is such a possibility and this topic is regard a better exploring, because at least one of possible explanations could be taken into account. The fluid overload, present in CKD patients between the hemodialysis sessions could contribute to atria dilation and AFL paroxysms. This kind of a trigger was already described in patients with acute pulmonary embolisms [26, 27].

Among the patients undergoing ablation of the cavotricuspid isthmus-related AFL who had not previously reported episodes of atrial fibrillation, 1/3 had paroxysms of AF within 2

years after the procedure, while among patients who had AF paroxysms before the procedure, 50% had AF paroxysms reported within 2 years [28]. In the analysis of the factors associated with the occurrence of AF in patients undergoing cavotricuspid isthmus ablation Lee et al. found that the only independent predictor of AF was the size of the left ventricle [29]. By contrast to this finding Ellis et al. suggested that left atrium dimension and the left ventricle ejection fraction and size, the presence of mitral regurgitation, and an easy induction of AF during an electrophysiological study were associated with the frequent occurrence of AF, but this did not translate into a significant statistical relationship [30].

Our results indicate that the presence of persistent form of arrhythmia was independently related to the longer duration of the P-wave. This fact emphasize the clinical well recognized importance of shortening of the duration of arrhythmia when trying to preserve the sinus rhythm This was obvious in AF, now there is the evidence for AFL.

Another important finding of our study is the relative frequent presence of disturbances in the interatrial conduction, not previously reported in an unselected typical AFL patients population. In the study by Enriquez et al. the authors showed an association between an advanced interatrial block (described as a combination of the P wave duration > 120 ms and a biphasic P-wave in inferior leads) and the risk of a recurrent AF in patients undergoing the cavotricuspid isthmus ablation. Interestingly, the researchers suggest that the prolonged duration of the P-wave alone was insufficient to predict the risk of an atrial fibrillation recurrence [31].

Study limitations:

Our study is subjected to some limitations. The assessment was observational in nature ,for this reason the causal relationship cannot be directly derived from the results. Our study population was moderate in number of participants. We did not have the precise atria dimensions measurements. The exact AFL duration was also not possible to obtain so the

persistent AFL criterion was estimated by 2 weeks. Nevertheless, this particular subject matter has never been extensively research. With that in mind, our study adds a substantial piece of data regarding the issue.

Conclusions:

1. Patients with a persistent AFL manifest a longer P-wave duration compared to a paroxysmal AFL, regardless of comorbidities and antiarrhythmic drugs, which therefore seems to be arrhythmia-related.

2. The population of patients with a typical AFL exhibit a high percentage of interatrial conduction disorders in the form of a partial or a complete Bachmann’s bundle block. This last finding seems to be at least partially age-related.

3. The arrhythmia-related longer P-wave duration should encourage the clinicians to earlier restore the sinus rhythm in patients with a persistent AFL in order to more effectively maintain it in the long term treatment.

Figure legend:

Figure 1. ROC curve for the duration of the P wave as a parameter differentiating patients in terms of the type of atrial flutter. For "mean P wave duration> 163 ms" the classification accuracy (Accuracy) is 70.3%.

Figure 2. The scatter plot (correlation diagram) of the mean duration of the P wave against creatinine concentration and the value of the Spearman's rank correlation coefficient (rho). There was no statistically significant correlation ($p > 0.05$).

Table 1. Clinical characteristics of the total population and the comparisons of two groups of studied patients.

Table 2. Demographic and electrocardiographic parameters in all studies patient according to interatrial conduction.

Table 3. P wave duration in patients group according to antyarytmic treatment

Table 4. The number (percentage) of patients in the groups differing in the duration of the P wave and the analyzed demographic and clinical factors as well as the results of independence tests and odds ratios

Table 5. Results of univariate and multivariate logistic regression analysis and odds ratios of exceeding the P-wave duration

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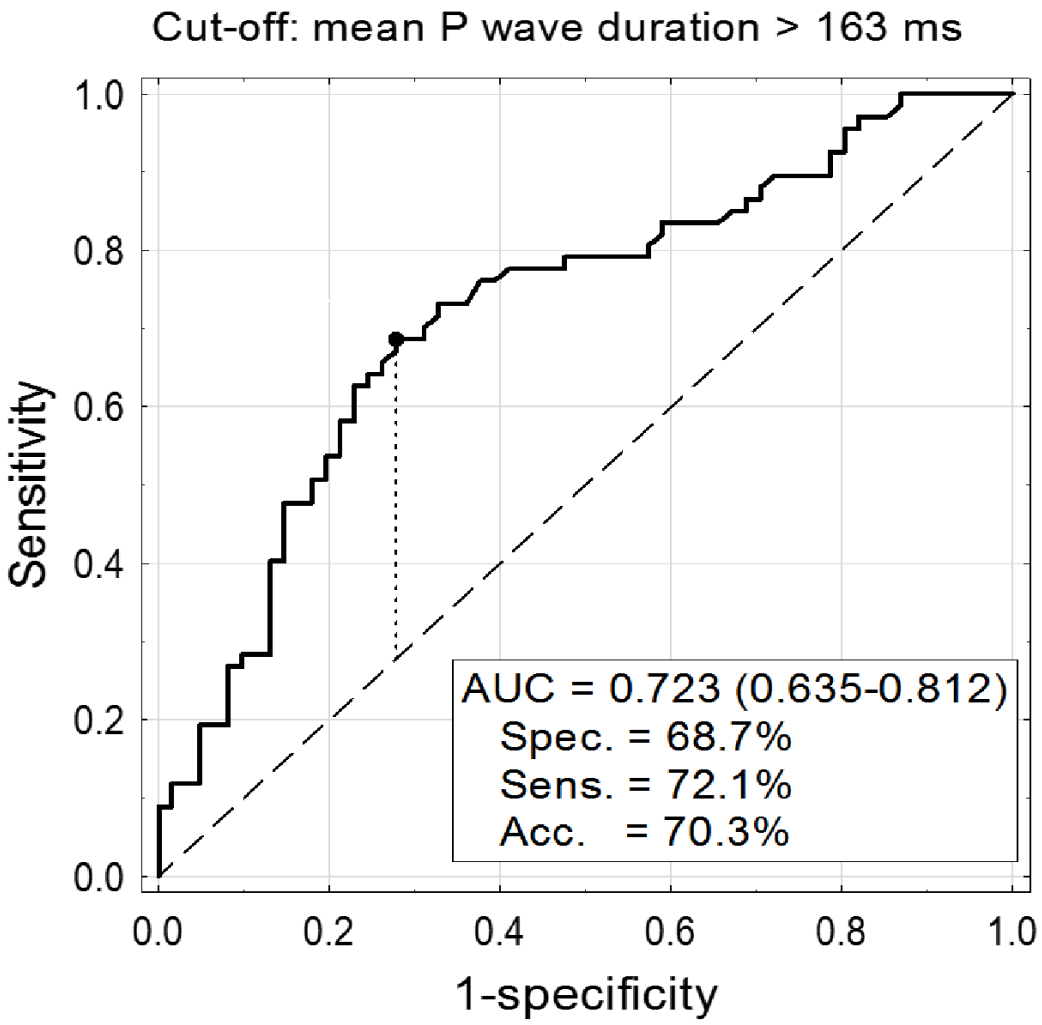

Figure 1. ROC curve for the duration of the P wave as a parameter differentiating patients in terms of the type of atrial flutter. For "mean P wave duration> 163 ms" the classification accuracy (Accuracy) is 70.3%.

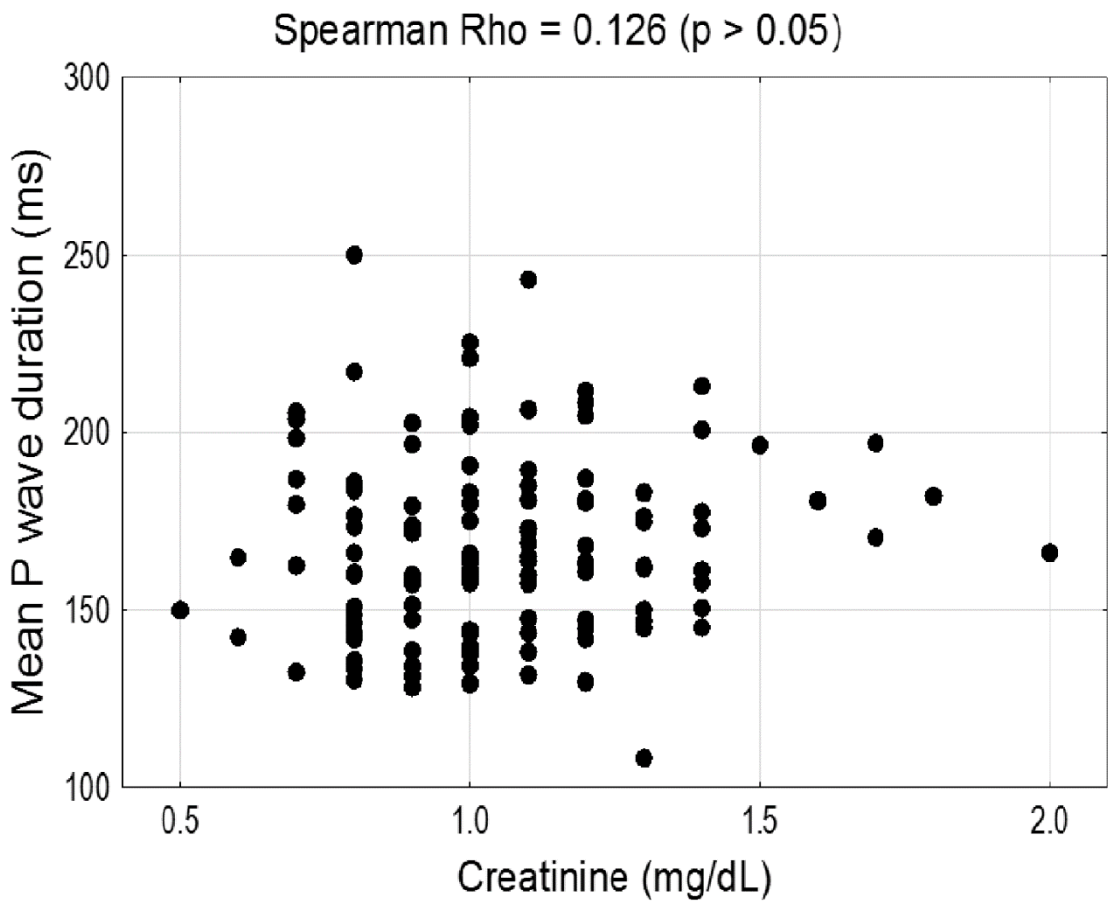

Figure 2. The scatter plot (correlation diagram) of the mean duration of the P wave against creatinine concentration and the value of the Spearman's rank correlation coefficient (rho). There was no statistically significant correlation (p> 0.05).

Table 1. Clinical characteristics of the total population and the comparisons of two groups of studied patients.

Parameter	Total N = 131	SR N = 62	AFL N = 69	P-value
Age (years), mean ± SD	65.3 ±10.1	63.4±10.4	66.9±9.4	0.057
Male vs female	93 vs 38	38 vs 24	55 vs 14	**0.033**
Propafenone, n (%)	19 (30.6%)	19 (30.6%)	16 (23.2%)	0.444
Amiodarone, n (%)	15 (11.5%)	5 (8.1%)	10 (14.5%)	0.379
Metoprolol, n (%)	70 (53.4%)	34 (54.8%)	36 (52.2%)	0.897
Bisoprolol, n (%)	44 (33.6%)	23 (37.1%)	21 (30.4%)	0.535
MRA, n (%)	50 (38.2%)	26 (41.9%)	24 (34.8%)	0.508
HB(g/dl), mean ± SD,	14.1 ± 1.5	14.0 ± 1.5	14.2 ± 1.5	0.381
K+(mmol/l), mean ± SD, Me (IQR)	4.5 ± 0.4, 4.4 (4.2-4.7)	4.4 ± 0.4, 4.4 (4.1-4.6)	4.6 ± 0.5, 4.5 (4.3-4.7)	0.062
Glucose(mg/dl),meanSD, Me (IQR)	120± 42.8, 110 (98-124)	118± 49.6, 105 (96-118)	122 ± 35.7, 113 (100-131)	0.145
Creatinine(mg/dl),mean±SD,Me (IQR)	1.12± 0.64, 1.0 (0.8-1.2)	1.08± 0.68, 1.0 (0.8-1.2)	1.2± 0.60, 1.1 (0.9-1.2)	**0.05**
HT, n (%)	126 (96.2%)	58 (93.6%)	68 (98.6%)	0.189
DM, n (%)	45 (34.4%)	17 (27.4%)	28 (40.6%)	0.162
CKD*, n (%)	11 (8.4%)	4 (6.5%)	7 (10.1%)	0.538
IHD, n (%)	35 (26.7%)	13 (21.0%)	22 (31.9%)	0.225
HF, n (%)	15 (11.5%)	6 (9.7%)	9 (13.0%)	0.742
Mean P wave duration (ms)	162 (145-181)	150 (142-165)	174 (160-189)	**<0.001**

Me - median, IQR - quartile range (Q1-Q3), SD- standard deviation, n – frequency, % - percentage , CKD- GFR < 60 ml/min/1,73 m2

Table 2. Demographic and electrocardiographic parameters in all studies patient according to interatrial conduction.

	Patients without Bachmann's Bundle Block N=27	Patients with incomplete Bachmann's Bundle Block N=61	Patients with complete Bachmann's Bundle Block N=43	P
Age (years)	60.3+/-12.1	64.7+/-8.3	68.9+/-9.5	**0.002**
Sex (F/M)	8/19	26/35	17/26	0.623
P wave duration (ms)	142.0+/-13.0	161.3+/-18.8	188.8+/-22.7	**<0.001**
Sinus Rhythm (N=62)	19	25	18	**0.0186**
Atrial Flutter (N=69)	8	35	26	
Glucose (mg/dl)	114.1+/- 6.4	117.4+/-29.0	127.56 +/-59.2	0.274
Creatinine (mg/dl)	0.94+/-0.17	1.17+/-0.68	1.08 +/-0.40	**0.039**
K+ (mmol/l)	4.36 +/-0.42	4.55+/-0.42	4.45+/-0.48	0.456
Hemoglobin (g/dl)	14.8+/-1.1	13.9+/-1.7	13.9+/-1.1	0.411
DM	7	27	13	**0.047**
CKD	1	7	4	0.5147
HT	25	58	42	0.976
IHD	5	17	15	0.395
HF	1	11	4	0.0712

DM- diabetes melitus, CKD- Chronic kidney disease, HT- hipertension, IHD- Ischemic heart disease, HF- heart failure

Table 3. P wave duration in patients group according to antyarytmic treatment

Drugs	Sinus Rhythm	Atrial Flutter	P
Propafenon n=33, M, SD, R	n= 20: 167.8 ± 23.8 (129.3-205.7)	n= 13: 169.2 ± 31.1 (134.3-243.0)	0.87
Amiodaron n= 15, M, SD, R	n= 5: 177.6 ± 23.2 (163.4-212.7)	n= 10: 174.0 ± 15.4 (144.2-195.3)	0.504
Without AAD n= 81, M, SD, R	**n= 37: 152.5 ± 21.1 (128.3-204.7)**	**n= 44: 173.3 ± 27.1 (133.3-225.3)**	**0.002**

n- number, M- mean(ms), SD- standard deviation(ms), R- range(ms) , AAD- antyarytmic drugs

Table 4. The number (percentage) of patients in the groups differing in the duration of the P wave and the analyzed demographic and clinical factors as well as the results of independence tests and odds ratios

Parameters	The duration of the P wave				*p*-value	OR (95% CI)
	>163 ms *N* = 62		≤163 ms *N* = 66			
	n	%	*n*	%		
Male, n (%)	44	71.0	46	69.7	0.971	1.06 (0.50-2.27)
Age ≥71 years	29	46.8	12	18.2	0.001	3.95 (1.78-8.80)
AFL	45	72.6	22	33.3	<0.001	5.29 (2.48-11.3)
Propafenone	16	25.8	19	28.8	0.705	0.86 (0.39-1.88)
Amiodarone	10	16.1	4	6.1	0.090	2.98 (0.88-10.1)
Metoprolol	30	48.4	39	59.1	0.225	0.65 (0.32-1.31)
Bisoprolol	22	35.5	21	31.8	0.661	1.18 (0.57-2.46)
MRA	26	41.9	22	33.3	0.315	1.44 (0.70-2.96)
HB < 15	49	79.0	40	60.6	0.024	2.45 (1.12-5.38)
K^+ ≥4.54	32	51.6	22	33.3	0.036	2.13 (1.04-4.36)
Glucose ≥98	50	80.7	46	69.7	0.153	1.81 (0.80-4.11)
Creatinine ≥1.1	31	50.0	26	39.4	0.228	1.54 (0.76-3.10)
HT	62	100.0	61	92.4	0.058	-
DM	23	37.1	21	31.8	0.530	1.26 (0.61-2.62)
CKD	8	12.9	3	4.6	0.119	3.11 (0.79-12.3)
IHD	22	35.5	12	18.2	0.027	2.48 (1.10-5.58)
HF	8	12.9	6	9.1	0.634	1.48 (0.48-4.54)

Table 5. Results of univariate and multivariate logistic regression analysis and odds ratios of exceeding the P-wave duration

Parameters	Univariate analysis		Multivariate analysis		
	b	*p*	*beta*	*p*	OR (95% CI)
Age ≥71 years	1.375	0.001	1.069	0.023	2.91 (1.16-7.32)
AFL	1.667	<0.001	1.591	>0.001	4.91 (2.11-11.4)
HB < 15	1.919	0.025	0.853	0.069	2.35 (0.93-5.89)
K^+ ≥4.54	0.758	0.038	0.586	0.170	1.80 (0.78-4.16)
IHD	0.906	0.029	0.740	0.135	2.10 (0.79-5.55)