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Effects of different atrial pacing modes evaluated by intracardiac signal-averaged ECG

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Abstract

Background: *Analysis of high gain, signal-averaged (SA) ECG is an accepted method evaluating abnormalities of atrial repolarization — the presence of late potentials (ALP) — predictive for atrial arrhythmias. Recently it has been proven that the location of atrial leads has an influence on atrial activation and modifies the risk of atrial arrhythmias. The aim of our study was to estimate the effect of different modes of atrial pacing on signal-averaged P waves recorded from external (conventional) and from intra-atrial leads.*

Methods: *Recordings were performed in 24 patients during biatrial (BiA) pacing system implantation. A surface SA-ECG was obtained from orthogonal leads, and intra-atrial signals were recorded and averaged separately from the right and left atrium during sinus rhythm (SR) and atrial pacing from the right atrial appendage, coronary sinus or both (BiA pacing). We analyzed standard SA-ECG parameters (P/A wave duration, RMS20 and LAS5) and the presence of atrial late potentials (ALP-Pdur > 125 ms and RMS20 < 2.40 μV).*

Results and conclusions: *Right atrial appendage pacing prolongs the duration of atrial potential in external and intracardiac leads and decreases its homogeneity in comparison to SR. RAA pacing increases the occurrence of ALP both in external and internal SA-ECG. Coronary sinus pacing does not deteriorate atrial activation in comparison to SR. Biatrial pacing shortens atrial potential, increases its homogeneity and eliminates atrial late potential criteria in most of patients in comparison to SR. It can be observed both in external and intra-atrial leads and confirms the beneficial effects of BiA pacing on atrial excitation, explaining its antiarrhythmic effect. Evaluation of signal-averaged intra-atrial electrograms supplies more data about local conduction disturbances with micro-voltage oscillations during final part of atrial excitation (low RMS20 and prolonged LAS5) than conventional techniques and seems to be a valuable tool for the evaluation of new resynchronizing atrial pacing modes.* (Cardiol J 2008; 15: 129–142)

Key words: signal-averaged ECG, biatrial pacing, intra-atrial signal, atrial late potentials, atrial fibrillation

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Introduction

Cardiac pacing is becoming a widely accepted non-pharmacological approach to drug-resistant atrial arrhythmias [1–4]. The main favourable effects of cardiac pacing are: 1) rate control — stable

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cycle length decreases the dispersion of repolarization, leading to suppression of rhythm-dependent arrhythmias (e.g. vagally-mediated atrial fibrillation or bradycardia-dependent atrial fibrillation) by reduction of "escape" atrial ectopic activity [1–5]; 2), overdrive suppression of atrial ectopy and elimination of compensatory pauses and arrhythmogenic effects of altering cycle length (long-short or short-long-short), supported by special antiarrhythmic algorithms [6, 7].

Right atrial appendage (RAA) has become the standard atrial lead location due to its optimal pacing and sensing conditions [8, 9] although its potential proarrhythmic effect has also been described [2, 10]; preliminary studies have linked this effect with mechanical interaction with the atrial wall [11, 12]. Nonetheless, more recent studies have concentrated on atrial conduction disturbance that were increased (or even produced) by RAA pacing [13, 14]. Despite this, RAA is still the standard atrial pacing site [15], also in biatrial [16, 17] or dual right atrial [18, 19] pacing systems.

Coronary sinus (CS) was the first permanent atrial pacing site in the late sixties and at the beginning of the seventies [20–22]. In that pioneering era of atrial pacing it was observed that "in some patients with intermittent atrial fibrillation, CS pacing can provide an atrial impulse that minimizes the tendency to return atrial fibrillation" [22]. The studies on permanent CS pacing in patients with bradytachycardia syndrome conducted in our centre since 1996 [23] have demonstrated its remarkable antiarrhythmic effect compared to standard RAA pacing [24, 25]; therefore, we frequently apply this pacing mode nowadays. In the following studies we have demonstrated the beneficial influence of CS pacing on signal-averaged (SA) ECG parameters (P wave duration, RMS20 and LAS5) in comparison to SA and RAA pacing [26, 27]. Even though CS is today an accepted alternative atrial pacing site [28], its application is generally limited to cases that have technical problems with right atrial (RA) lead location, or as part of biatrial (BiA) pacing systems.

Conduction disturbances within the atria along with the phenomenon of anisotropic conduction and increased dispersion of refractoriness (usually with impaired rate-adaptation) are the main factors maintaining atrial arrhythmias [13, 29]. Biatrial pacing, providing pre-excitation of the infero-posterior (potentially arrhythmogenic) part of the right atrium in patients with recurrent atrial fibrillation (AF) and severe interatrial conduction delay, was proposed by Daubert et al. in 1991 [30]. Biatrial pacing technically provides "restoration" of natural synchrony of atrial activation during both pacing and right- or left-atrial premature beats [14, 16, 19, 30, 31]. The main electrophysiological feature of BiA pacing is the permanent preexcitation of the triangle of Koch region due to CS stimulation, which minimizes the chance of an atrial premature beat to initiate reentry loop within the atria [29, 32–34]. Our previous studies, based on intra-atrial potential recording, demonstrated that CS pacing tremendously improved the synchrony of atrial activation, shortening P wave duration (25 ms) and total atrial activation time (TAAT) by 54 ms [14]; it also shortened the filtered P wave duration and improved RMS20 and LAS5 parameters assessed in SA-ECG [27]. Corresponding results were published by Orr et al. [35], who demonstrated that CS pacing dramatically shortens SA-ECG P wave duration with virtually no influence on frequency-domain parameters.

Considering the fact that CS pacing reduces AF recurrence in 60–70% of patients [24, 25], which is similar to the effects of BiA [14, 16, 31, 36] and dual right-atrial [18, 19] pacing, and the results of electrophysiological studies which proved that that both CS and BiA pacing equally prevents AF induction [13, 29, 32–34], it seemed interesting to compare its electrophysiological effects by means of recently deployed signal-averaged intracardiac electrogram (SA-IEGM) analysis [37–40]. Since the proven key role of local conduction disorders within the right atrium (anisotropic conduction of premature beats) [13, 32, 33, 41, 42], the assessment of termination of right-atrial potential (difficult to evaluate in external ECG) is particularly attractive. The comparative analysis of the influence of different atrial pacing modes upon the duration and homogeneity of termination of atrial potential appeared to be especially important in the group of patients with prominent atrial conduction disturbances (apparent in external ECG at sinus rhythm).

The aim of the study was to compare the electrophysiological effects of right atrial appendage, proximal coronary sinus and BiA pacing upon atrial activation synchrony, assessed by means of the time domain parameters of signal-averaged intracardiac electrograms.

Methods

Patients

The study was conducted on a group of 24 patients (15 female, 9 male, mean age 68.8 ± 9.97 years) eligible for permanent BiA pacing. Sporadic AF was identified in 7 patients (29.2%), recurrent

AF in 8 patients (33.3%) and frequent AF (according to the Kingma et al. paradigm [43]) in 9 patients (37.5%). Due to high arrhythmia burden the ongoing medication was not modified — discontinuation of treatment could provoke AF episodes, thus impeding the measurement of pacing and sensing conditions. During the pacemaker implantation procedures, 5 patients were treated with 1 drug (amiodarone or propafenone), 8 were on 2 drugs (propafenone and sotalol/amiodarone) and 11 patients (46%) had no ongoing antiarrhythmic medication.

Procedures

The following measurements were taken at sinus rhythm and RAA, CS and BiA pacing. 1) 12-lead ECG with 100 mm/s speed 80 mm/1 mV gain; 2) IEGM recording from RAA and CS, simultaneously with ECG lead II; 3) SA-ECG recording from external orthogonal leads; 4) SA-IEGM recording from the right and left atrium separately. Intra-atrial signals were obtained with standard bipolar pacing leads introduced during the pacemaker implantation procedure, subsequently employed for permanent pacing.

External SA-ECG recording and processing

Equipment constructed in the National Institute of Cardiology (Warsaw) was applied for signal recording and processing. It consisted of a micropotential amplifier (noise < 1.5 μV in 0.1–300 Hz bandwidth, CMRR > 130 Db), 12-bit A/D converter onboard PC and software designed for signal-averaging and subsequent analysis of data. Standard Ag/AgCl electrodes were used on cleansed chest skin. The P wave was derived from three bipolar orthogonal (Frank) leads. Signals (from each lead) were amplified (×1000), passed through a band-pass filter (cut-off frequency 0.1–300 Hz) and digitized by the A/D converter with a 12-beat accuracy. The signal-averaging process was triggered by the R wave at sinus rhythm and the pacing spike during pacing. Ectopic beats, if present, were identified and rejected. Approximately 50 beats were averaged and stored on PC HD. The procedure was described before [26, 27].

High-gain SA-ECG P wave parameters time-domain analysis

The first stage was to combine filtered (Butterworth bidirectional filter) and averaged signals from three leads X, Y and Z to a spatial vector magnitude $(X^2 = Y^2 + Z^2)^{1/2}$. The onset and offset of P wave were defined as the points at which the atrial signal exceeded and returned to the 1.5 μV level,

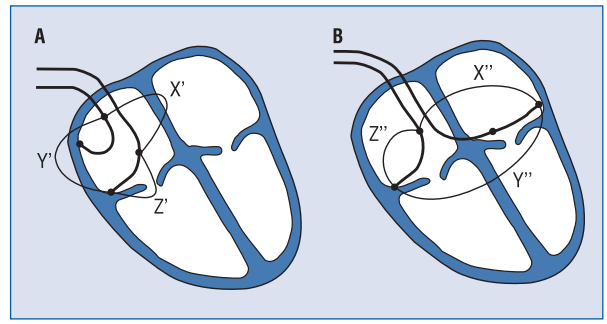

Figure 1. Scheme showing the connections of intracardiac leads to obtain right atrial (X′, Y′, Z′) (**A**) and left atrial (X″, Y″, Z″) (**B**) signals.

respectively. The following parameters were measured and calculated automatically: 1) filtered P wave duration (Pdur); 2) root mean square voltage of the final 20 ms of filtered P wave (RMS20); 3) duration of low amplitude signal < 5 μV (LAS5). Atrial late potentials (ALP) were considered positive with Pdur > 125 ms and RMS20 < 2.4 μV [25, 27].

Intracardiac SA-IEGM recording and processing

Three bipolar pacing leads were used: a standard "J" shaped lead implanted into the RAA, the second lead was introduced into the coronary sinus and the third lead (for permanent ventricular pacing) was temporarily placed in the LRA position. The same equipment, as described above, was employed for signal recording and processing. To obtain right- and left-atrial signals intracardiac leads were attached to the micro-potential amplifier via sterile connectors according to scheme in Figure 1. Right atrial electrogram was recorded from three combined intra-atrial leads X', Y' and Z', and left atrial electrogram from leads X'', Y'' and Z''. The signal from each lead was augmented and filtered in the same mode as during external signal recording. The averaging process was triggered and obtained parameters analyzed in the same way as the external SA-ECG. The employed technique was described before [37, 38].

Interatrial conduction evaluation with IEGM recordings

Internal electrogram (IEGM) was recorded from the RAA and CS leads connected to a dual-chamber pacemaker via telemetry (Fig. 2), simultaneously with lead II ECG. The following timing parameters were determined: 1) P wave duration in lead II or III in high resolution ECG (P_{II});

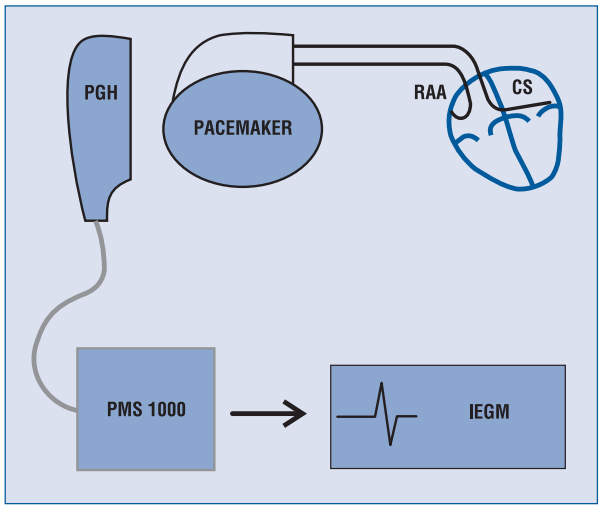

Figure 2. Internal electrogram (IEGM) recorded from the right atrial appendage (RAA) and coronary sinus (CS) leads connected to a dual-chamber pacemaker via telemetry.

2) P-Q or S-Q (from onset of P wave or spike to Q) interval in the same leads; 3) interatrial conduction time (IACT) measured from the onset of A wave (or spike) in RAA signal to the onset of A wave in CS at sinus rhythm and RAA pacing, respectively (during CS pacing it was measured from the pacing spike to the onset of A wave in CS; during BiA pacing the value of IACT was 0); 4) total atrial activation time measured from the onset of P wave (or pacing spike) in lead II ECG to the end of atrial activation in CS or RAA signal, depending upon which was later. The method was described before [14, 44].

Statistical analysis

The significance of the differences between all groups was evaluated by the F variance test, and specific differences between the groups were analyzed by LSD test. The results are presented as mean ± standard deviation. Statistical significance of differences between the groups was evaluated by the Student's t-test. Correlation between parametric values was estimated using Pearson's r-test. A P level of < 0.05 was accepted as statistically significant.

The study was approved by the Bioethical Committee of the Medical University of Lublin (approval KE-0254/70/2003).

Results

The results are presented in Table 1.

The initial analysis of the data confirms that the shift in atrial pacing mode brings about notable changes in the duration of atrial potential (assessed in external ECG, SA-ECG and intra-atrial signal-averaged electrograms from both atria); it also influences the homogeneity of atrial potential termination (reflected by the root mean square voltages of the last 20 ms of filtered P wave (RMS20) and the duration of low-amplitude signal (LAS5) in SA-IEGM, both in the right and left atria.

The significance of the differences in ECG P wave duration, TAAT and PQ/SQ (spike-Q) interval during SR and different atrial pacing modes is presented in Table 2.

Right atrial appendage pacing significantly prolonged ECG P wave duration (+23 ms), while BiA pacing shortened it significantly (–40 ms). Total atrial

Table 1. ECG, IEGM, SA-ECG and SA RA and LA IEGM atrial potential parameters during sinus rhythm (SR), right atrial appendage pacing (RAA), coronary sinus pacing (CS) and biatrial pacing (BiA)

Parameters	SR			RAA			CS			BiA		
	N	Aver.	SD	N	Aver.	SD	N	Aver.	SD	N	Aver.	SD
ECG Pdur	24	157.7	16.0	24	180.9	26.8	24	151.7	25.68	24	117.8	18.2
TAAT	24	181.5	22.5	24	201.1	29.0	24	179.2	35.7	24	108.0	13.7
PII-Q	24	208.7	36.5	24	241.1	63.9	24	202.5	36.3	24	191.8	37.1
Ext. Pdur	24	156.2	16.8	24	188.9	30.5	24	160.7	32.05	24	120.5	13.9
Ext. RMS20	20	2.17	0.76	24	1.82	0.71	24	2.15	0.86	24	2.95	0.86
Ext. LAS5	24	6.68	7.48	24	14.59	12.81	24	10.21	8.02	24	5.17	3.13
Int. RA Adur	24	174.8	24.1	24	199.1	29.1	24	183.3	29.7	24	136.8	19.8
Int. RMS20	23	1.77	0.72	22	1.36	0.64	16	1.95	0.69	23	2.72	1.33
Int. RA LAS5	23	12.91	7.91	22	24.62	19.99	16	10.36	4.94	23	9.83	7.522
Int. LA Adur	23	175.3	26.7	24	201.3	31.2	24	143.5	26.1	24	127.8	18.4
Int. LA RMS20	15	2.33	1.19	16	1.96	0.84	22	4.35	4.03	23	3.43	1.24
Int. LA LAS5	15	9.01	6.17	16	13.45	14.10	21	6.37	4.09	23	6.21	3.81

Table 2. The comparison of values of ECG P wave duration, total atrial activation time (TAAT) and P-Q interval during sinus rhythm (SR), right atrial appendage pacing (RAA), coronary sinus pacing (CS) and biatrial pacing (BiA)

Examined parameters	N	Rhythm	Average	SE	LSD test Groups	LSD test Comparison	LSD test p
High resol.	24	**SR**	157.7	3.3	**1**	1 *vs.* 2	0.000
ECG P wave	24	**RAA**	180.9	5.5	**2**	1 *vs.* 3	0.133
duration	24	**CS**	151.7	5.2	**3**	1 *vs.* 4	0.000
	24	**BiA**	117.8	3.7	**4**	2 *vs.* 3	0.000
		Analysis of variance	F = 1581.3 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.000
TAAT	24	**SR**	181.5	4.6	**1**	1 *vs.* 2	0.000
	24	**RAA**	201.1	5.9	**2**	1 *vs.* 3	0.671
	24	**CS**	179.2	7.3	**3**	1 *vs.* 4	0.000
	24	**BiA**	108.0	2.8	**4**	2 *vs.* 3	0.000
		Analysis of variance	F = 1562.1 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.000
P-Q (spike-Q)	24	**SR**	208.7	7.5	**1**	1 *vs.* 2	0.000
interval	24	**RAA**	241.1	13.0	**2**	1 *vs.* 3	0.385
	24	**CS**	202.5	7.4	**3**	2 *vs.* 4	0.019
	24	**BiA**	191.8	7.6	**4**	2 *vs.* 3	0.000
		Analysis of variance	F = 679.9 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.135

activation time, abnormal at sinus rhythm (181 ms, which is comprehensible in this selected group), was even more prolonged (+19 ms) by RAA pacing and did not change with CS pacing, while BiA pacing caused significant shortening of TAAT (–73 ms). The average PR interval normal (181 ms) at sinus rhythm was significantly prolonged by RAA pacing (+32 ms), and slightly (–6 ms) and significantly (–17 ms) shortened by CS and BiA pacing, respectively. The difference in atrioventricular conduction (reflected by SQ interval) between RAA and BiA pacing (49 ms) is remarkable (Fig. 3).

The significance of differences of external SA-ECG parameters (P duration, RMS20, LAS5) during sinus rhythm and different atrial pacing modes is illustrated in Table 3.

The filtered P wave duration in SA-ECG, abnormal at sinus rhythm (156 ms), was additionally prolonged by RAA pacing (+33 ms) and significantly shortened (–36 ms) by BiA pacing, with no apparent influence of CS pacing (+4 ms). What is remarkable is the significant difference in filtered P wave duration between RAA and BiA pacing (69 ms). The RMS20 value, low at sinus rhythm (2.17 μV), was even lower with RAA pacing (–0,3 μV) and significantly increased (+0.8 μV) with BiA pacing, with no apparent shift with CS pacing (+0.1 μV). Of note is the significant difference between RAA and BiA pacing (1.1 μV). The low amplitude signal duration (LAS5) was significantly prolonged by RAA pacing (+8 ms), with insignificant shifts during CS (+3.5 ms) and BiA (–1.5 ms) pacing. Again, the contrast between the influence of RAA (prolonging) and BiA pacing (shortening, which is favourable) was marked (Fig. 4).

The analysis of intracardiac SA-IEGM parameters (A duration, RMS20, LAS5) recorded from three right-atrial leads during sinus rhythm and different atrial pacing modes is illustrated in Table 4.

The filtered A wave duration in right-atrial SA-IEGM was 175 ms (certainly a high value, although the normal range in healthy subjects is yet undetermined). It was additionally prolonged by RAA pacing (+24 ms) and significantly shortened (–38 ms) by BiA pacing, with no significant influence of CS pacing (+8 ms). Of note once more is the significant difference in filtered A wave duration between RAA and BiA pacing (62 ms). The RMS20 value, low at sinus rhythm (1.5 μV), was even lower with RAA pacing (–0.3 μV) and increased with CS and BiA pacing (+0.5 μV and +1.2 μV, respectively); the latter shift, as well as the difference between RAA and BiA pacing (1.4 μV), was statistically significant. The LAS5 value in right-atrial SA-IEGM was 15.5 ms at sinus rhythm (interestingly, much longer than in the left atrium) and was prolonged by RAA pacing (+12.2 ms), with

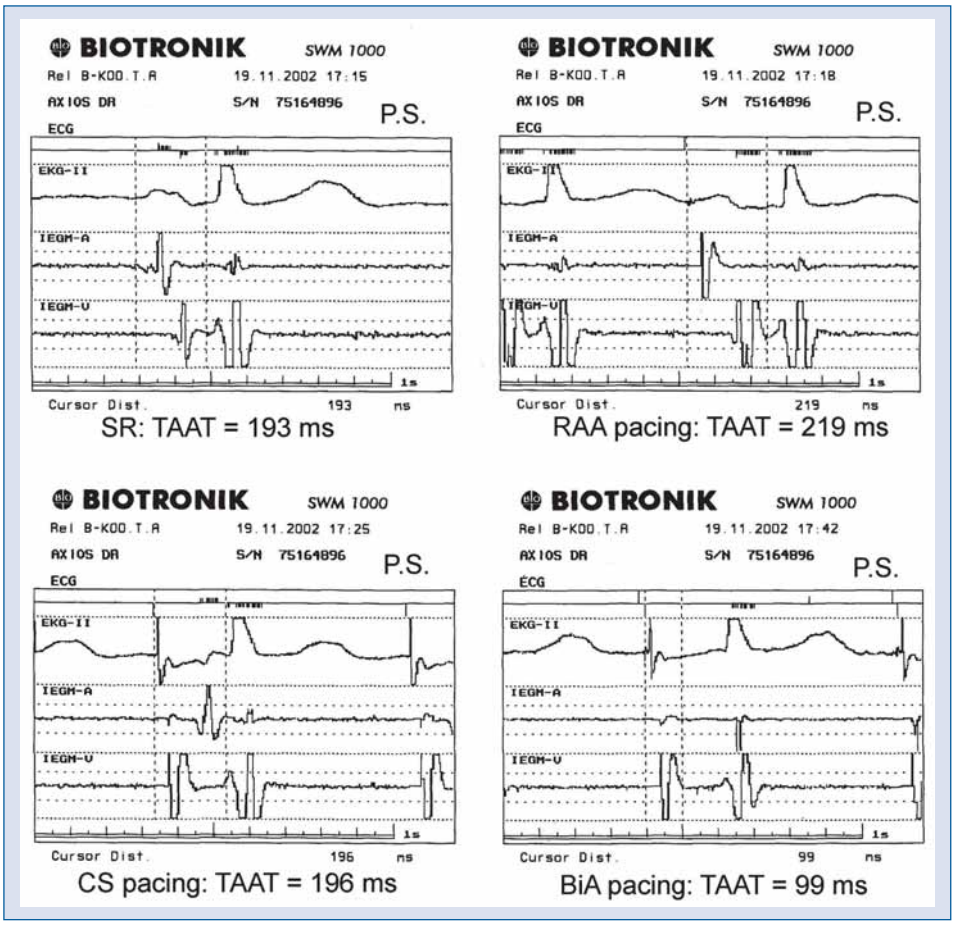

Figure 3. The effect of different atrial pacing modes compared with sinus rhythm upon total atrial activation time (TAAT) in the same patient.

Table 3. Conventional (external) lead SA-ECG P wave duration and its RMS20 and LAS5 values during sinus rhythm (SR) and right atrial appendage pacing (RAA), coronary sinus pacing (CS) and biatrial pacing (BiA)

Examined parameters	N	Rhythm	Average	SE	LSD test		
					Groups	Comparison	p
Conventional	24	**SR**	156.2	3.4	**1**	1 *vs.* 2	0.000
(external) leads	24	**RAA**	188.9	6.2	**2**	1 *vs.* 3	0.370
SA-ECG P wave	24	**CS**	160.7	6.5	**3**	1 *vs.* 4	0.000
duration	24	**BiA**	120.5	2.8	**4**	2 *vs.* 3	0.000
		Analysis of variance	F = 1534.8 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.000
Conventional	20	**SR**	2.17	0.17	**1**	1 *vs.* 2	0.177
(external) leads	20	**RAA**	1.85	0.15	**2**	1 *vs.* 3	0.635
SA-ECG RMS20	20	**CS**	2.28	0.18	**3**	1 *vs.* 4	0.000
	20	**BiA**	2.99	0.20	**4**	2 *vs.* 3	0.070
		Analysis of variance	F = 423.56 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.003
Conventional	24	**SR**	6.68	1.53	**1**	1 *vs.* 2	0.001
(external) leads	24	**RAA**	14.60	2.61	**2**	1 *vs.* 3	0.135
SA-ECG LAS5	24	**CS**	10.21	1.64	**3**	2 *vs.* 4	0.520
	24	**BiA**	5.17	0.64	**4**	2 *vs.* 3	0.064
		Analysis of variance	F = 82.61 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.034

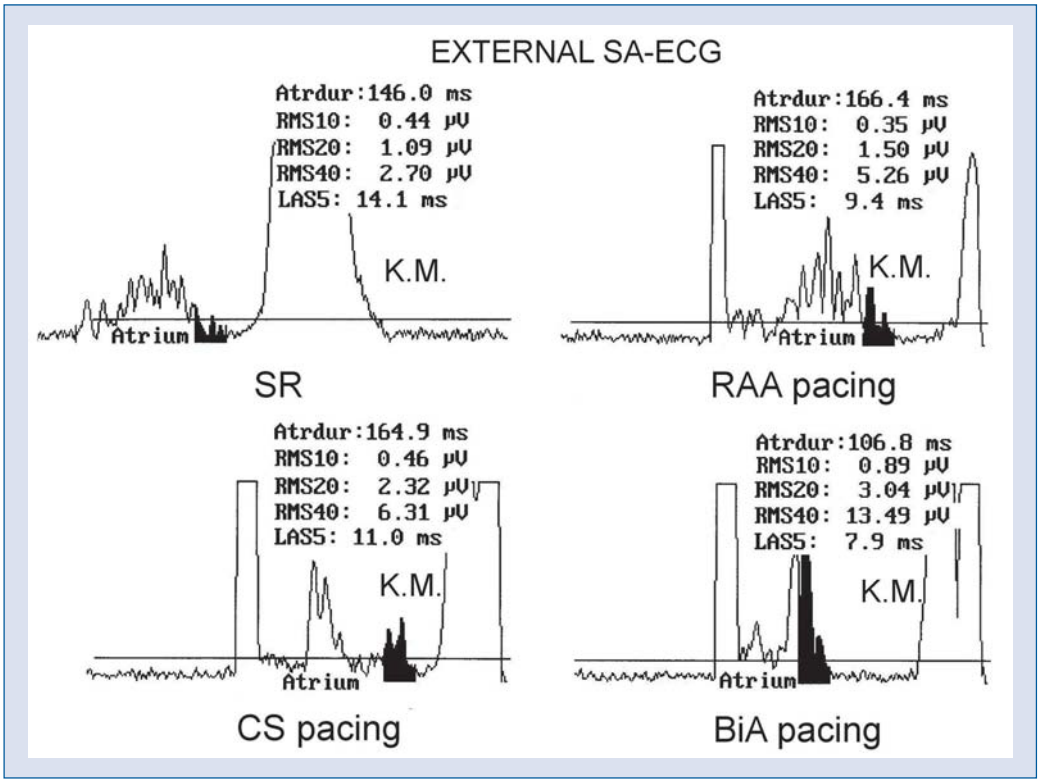

Figure 4. The effect of different atrial pacing modes compared with sinus rhythm upon SA-ECG parameters obtained from external leads in the same patient.

Table 4. Right atrial (internal) lead SA-ECG A wave duration and its RMS20 and LAS5 values during sinus rhythm (SR) and right atrial appendage pacing (RAA), coronary sinus pacing (CS) and biatrial pacing (BiA)

Examined parameters	N	Rhythm	Average	SE	LSD test		
					Groups	Comparison	p
Right atrial	24	**SR**	174.8	4.9	**1**	1 *vs.* 2	0.000
(internal) leads	24	**RAA**	199.1	5.9	**2**	1 *vs.* 3	0.103
SA-ECG A wave	24	**CS**	183.3	6.1	**3**	1 *vs.* 4	0.000
duration	24	**BiA**	136.8	4.0	**4**	2 *vs.* 3	0.003
		Analysis of variance	F = 1659.2 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.000
Right atrial	16	**SR**	1.48	0.11	**1**	1 *vs.* 2	0.400
(internal) leads	16	**RAA**	1.22	0.12	**2**	1 *vs.* 3	0.094
SA-ECG RMS20	16	**CS**	1.95	0.17	**3**	1 *vs.* 4	0.000
	16	**BiA**	2.60	0.36	**4**	2 *vs.* 3	0.013
		Analysis of variance	F = 205.67 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.027
Right atrial	16	**SR**	15.42	1.92	**1**	1 *vs.* 2	0.003
(internal) leads	16	**RAA**	27.58	5.27	**2**	1 *vs.* 3	0.199
SA-ECG LAS5	16	**CS**	10.36	1.23	**3**	2 *vs.* 4	0.293
	16	**BiA**	11.29	2.05	**4**	2 *vs.* 3	0.000
		Analysis of variance	F = 71.83 P = 0.000		**Comparison**	2 *vs.* 4	0.000
						3 *vs.* 4	0.812

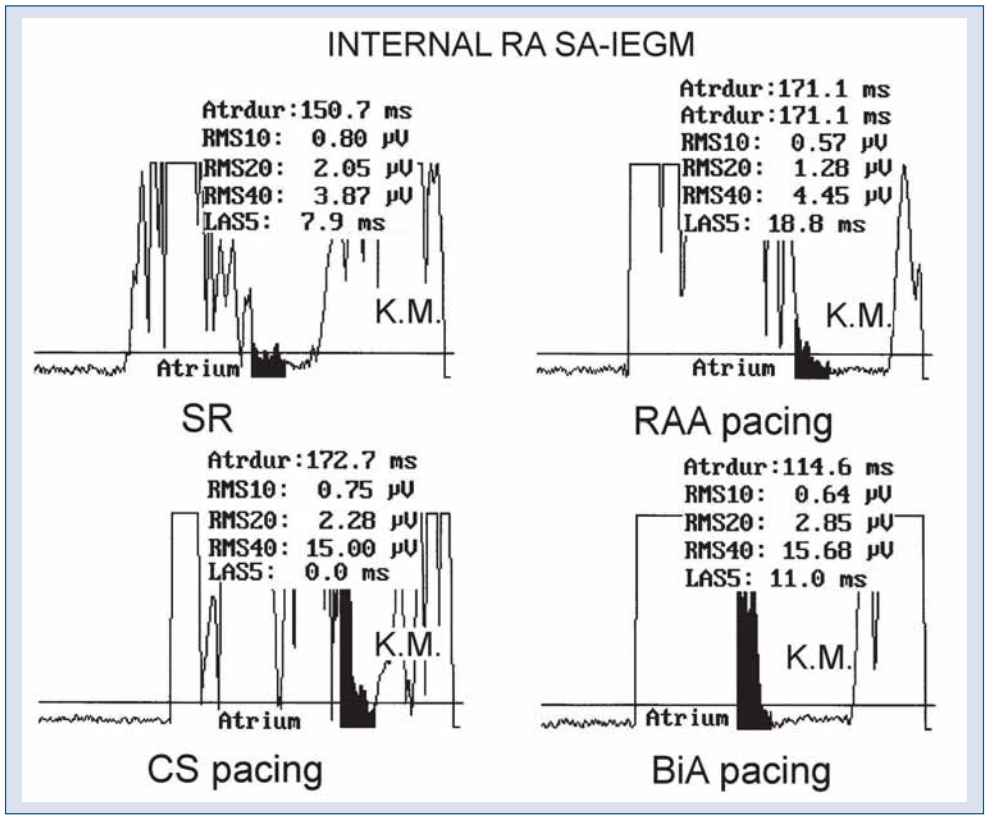

Figure 5. The effect of different atrial pacing modes compared with sinus rhythm upon SA-IEGM parameters obtained from right-atrial leads in the same patient.

insignificant shortening during CS (–5.1 ms) and BiA (–4.1 ms) pacing (Fig. 5).

The analysis of intracardiac SA-IEGM parameters (A duration, RMS20, LAS5) recorded from three left-atrial leads during sinus rhythm and different atrial pacing modes is illustrated in Table 5.

The filtered A wave duration in left-atrial SA-IEGM was 175 ms (a value comparable to that of the right atrium). It was additionally prolonged by RAA pacing (+25 ms) and significantly shortened by both CS and BiA pacing (–32 ms and –47 ms, respectively). Again there was highly significant difference in filtered A wave duration between RAA and BiA pacing (72 ms). The RMS20 value was significantly increased (which is favourable) with BiA pacing, with non-significant shifts (±0.4 μV) with RAA or CS pacing. Again, the difference between RAA and BiA pacing (1.6 μV) was statistically significant. The LAS5 value in right-atrial SA-IEGM was 15.5 ms at sinus rhythm (interestingly, much longer than in the left atrium) and was prolonged by RAA pacing (+12.2 ms), with insignificant shortening during CS (–5.1 ms) and BiA (–4.1 ms) pacing. The changes in left-atrial SA-IEGM LAS5 values, although not significant, were apparently consistent with the trends observed in external and right-atrial leads (Fig. 6).

Homogenous termination of atrial potential evaluated by means of a signal-averaging technique is reflected by high RMS20 and low LAS5 values and by filtered P wave duration not exceeding 125 ms. The combination of two of the above parameters, in our studies Pdur > 125 ms and RMS20 < 2.40 μV [37–40], indicates the presence of atrial late potentials (ALP), which have predictive value for atrial arrhythmias. Therefore, the next stage was to estimate the occurrence of ALP criteria in signal-averaged electrograms, both from external and intra-atrial leads during sinus rhythm and different atrial pacing modes (Table 6).

Atrial late potential criteria in external SA-ECG were present in 79% of patients at sinus rhythm (which is understandable in a group of patients with recurrent atrial arrhythmias) and in 87% of patients at CS pacing. During RAA pacing, ALP criteria were present in all of the patients, while BiA pacing reduced the ALP occurrence to only 8%. In the right-atrial SA-IEGM, atrial late potentials were present in 96% of patients both at sinus rhythm and RAA pacing, in 92% with CS pacing and in only 46% of patients during BiA pacing. In the left-atrial SA-IEGM, ALP criteria were positive in 86% of patients at sinus rhythm and in all of them during RAA

Table 5. Left atrial (internal) lead SA-ECG A wave duration and its RMS20 and LAS5 values during sinus rhythm (SR) and right atrial appendage pacing (RAA), coronary sinus pacing (CS) and biatrial pacing (BiA)

Examined parameters	N	Rhythm	Average	SE	LSD test		
					Groups	Comparison	p
Left atrial (internal)	23	**SR**	175.3	5.6	**1**	1 *vs*. 2	0.000
leads SA-ECG	23	**RAA**	200.3	6.6	**2**	1 *vs*. 3	0.000
A wave duration	23	**CS**	142.9	5.5	**3**	1 *vs*. 4	0.000
	23	**BiA**	127.8	3.9	**4**	2 *vs*. 3	0.000
		Analysis of variance	F = 1181.5 P = 0.000		**Comparison**	2 *vs*. 4	0.000
						3 *vs*. 4	0.001
Left atrial (internal)	12	**SR**	2.09	0.27	1	1 *vs*. 2	0.179
leads SA-ECG	12	**RAA**	1.65	0.17	2	1 *vs*. 3	0.201
RMS20	12	**CS**	2.51	0.21	**3**	1 *vs*. 4	0.000
	12	**BiA**	3.29	0.25	**4**	2 *vs*. 3	0.011
		Analysis of variance	F = 416.90 P = 0.000		**Comparison**	2 *vs*. 4	0.000
						3 *vs*. 4	0.020
Left atrial (internal)	12	**SR**	9.82	1.61	**1**	1 *vs*. 2	0.135
leads SA-ECG LAS5	12	**RAA**	15.18	4.60	**2**	1 *vs*. 3	0.683
	12	**CS**	8.37	1.01	**3**	2 *vs*. 4	0.436
	12	**BiA**	7.06	0.99	**4**	2 *vs*. 3	0.060
		Analysis of variance	F = 55.28 P = 0.000		**Comparison**	2 *vs*. 4	0.026
						3 *vs*. 4	0.709

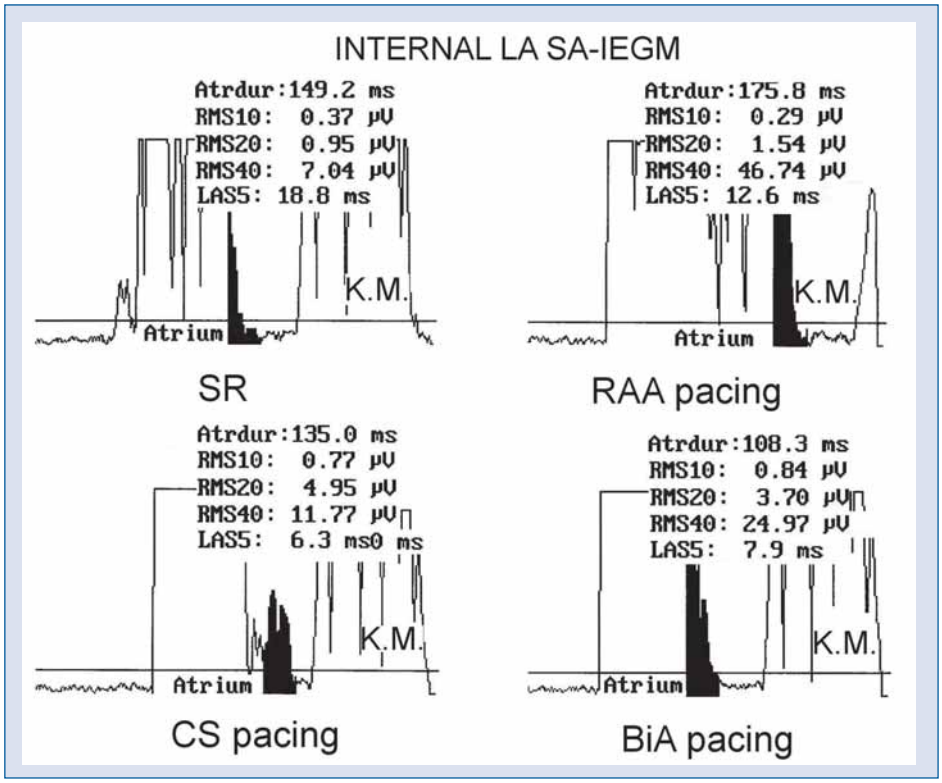

Figure 6. The effect of different atrial pacing modes compared with sinus rhythm upon SA-IEGM parameters obtained from left-atrial leads in the same patient.

Table 6. Presence of atrial late potentials (ALP) during sinus rhythm (SR) and right atrial (RA), coronary sinus (CS) and biatrial pacing (BiA) in recordings from conventional (external) and intra-atrial (Int. RA, Int. LA) leads

Leads	External		Int. RA			Int. LA	
ALP	Yes	No	Yes	No	Lack*	Yes	No
SR	19 79.2%	5 20.8%	23 95.8%	1 4.2%	0 0%	19 86.4%	3 13.6%
RA	24 100.0%	0 0%	22 95.6%	1 4.3%	1 4.3%	24 100.0%	0 0%
CS	21 87.5%	3 12.5%	22 91.7%	2 8.3%	0 0%	9 37.5%	15 62.5%
BiA	2 8.3%	22 91.7%	11 45.8%	13 54.2%	0 0%	3 12.5%	21 87.5%

*Impossible to evaluate (terminal part of A wave cancelled in V wave)

pacing, while CS and BiA pacing reduced the ALP occurrence to 38% and 12%, respectively.

The obtained results confirm that BiA pacing remarkably shortens the duration of atrial potential, significantly decreasing the occurrence of atrial late potentials in both external and right-atrial leads, although it fails to eliminate ALP in almost half of the patients in the right atrium. This observation, coherent with previous studies, seems to be principal. It confirms the existence of "purely right-atrial" late potentials. In the spotlight of the obtained data, the late micropotentials recorded at sinus rhythm could not just be the effect of far-field sensing of delayed activation of the left atrial lateral wall, since during BiA pacing this region is activated at the very beginning of atrial potential. This corresponds to the common opinion of electrophysiologists that local conduction disturbances in the right atrium are "the key to atrial arrhythmias".

In the spotlight of the demonstrated data, standard BiA pacing (from RAA and distal CS) does not restore the synchrony of activation in the right atrium, where regions of delayed activation can be still present. The discovered phenomenon can elucidate the lack of antiarrhythmic effectiveness of BiA pacing in many patients. The presented data advocate the search for another site for the right-atrial lead in BiA pacing systems or the potential utility of trifocal pacing, particularly in patients with severe conduction disturbances within the atria, with recurrent AF, in whom BiA pacing fails to restore the synchrony of atrial activation.

Discussion

Local conduction disturbances (delayed and anisotropic conduction) increase inhomogeneity of depolarization and repolarization within the atria [13, 29, 32–34, 41, 42]. Inconsistent extinction of atrial depolarization manifests with low-amplitude micropotentials omitted in standard ECG [45–48]. The signal-averaging technique allows those signals to be augmented, appearing in the form of micro-oscillations in the final part of the atrial potential, which can identify those patients having high risk of atrial arrhythmia [45–48]. In the past few years our group has applied this technique for the estimation of electrophysiological consequences of different atrial pacing modes [26, 27]. Electrophysiological study, yielding precise data concerning interatrial conduction, effective refractory period, maximal conduction delay after extrastimuli or the zones of delayed activation are standard tools for the assessment of the effects of cardiac pacing in the acute experiment [13, 29, 32–34, 41, 42] but unsuitable for patients with implanted resynchronizing pacing systems. Therefore, in recent years, we have applied recordings obtained via pacemaker telemetry from atrial leads to evaluate the conduction parameters at sinus rhythm, as well as during single- or dual-site atrial pacing [14, 24, 44]. In previous studies we demonstrated that RAA pacing prolonged atrioventricular conduction (+30 ms in comparison to sinus rhythm), P wave duration (+17 ms), interatrial conduction time (+40 ms) and total atrial activation time (+25 ms). Bipolar CS pacing had no apparent influence upon atrioventricular conduction and P wave duration, while BiA pacing dramatically improved atrial synchrony, shortening P wave duration (–25 ms) and TAAT (–54 ms). Furthermore, a moderate increase in pacing rate from RAA resulted in substantial prolongation of TAAT (+26 ms), with only a slight effect when pacing from CS (+14 ms). These results indicate that standard RAA pacing

increases the asynchrony of atrial activation, suggesting that CS and BiA pacing could be favourable [14, 25, 44]. This method, however, did not allow assess local conduction disturbances. Consequently, we applied the analysis of time-domain parameters of signal-averaged P wave [26, 27]. With this method, BiA pacing remarkably improved the synchrony of atrial excitation (reflected in a shortening of filtered P wave duration and normalization of RMS20 and LAS5 parameters) compared to sinus rhythm and RAA pacing in the same patients. Single-site (unipolar) coronary sinus pacing did not influence P wave duration and raised the RMS20 value only insignificantly [26, 27]. Since the leads in that group of patients were connected in a row, it was not possible to evaluate the previously described effect of high-energy bipolar CS pacing [24, 25]. In 1999 Orr at al. [35] confirmed that BiA pacing significantly shortens SA-ECG P wave duration, with no influence on its frequency-domain parameters; he did not compare, however, different modes of atrial pacing. In 2001 Yamada et al. [48] confirmed the unfavourable effects of RAA pacing, which prolonged filtered P wave duration (147 *vs*. 167 ms) with no apparent effect on its dispersion and RMS20 value; on the contrary, pacing of Bachmann's bundle region resulted in significantly shortened filtered P wave (147 *vs*. 126 ms), decreased dispersion of P wave and doubled RMS20 value (2.0 μV *vs*. 4.3 μV). Yamada et al. [48] also emphasized the arrhythmogenic consequences of RAA pacing.

The main disadvantages of conventional SA-ECG technique are low signal amplitude, and the notched and relatively sluggish onset of the P wave recorded from orthogonal Frank's leads (particularly in patients with recurrent atrial arrhythmias), which impedes its use as a synchronization trigger in the signal-averaging process. Alternatively, use of the R wave for synchronization is limited by the variant P-R segment [45–48]. Recently we proposed a solution to this problem by applying a signal-averaging technique to process the intracardiac potentials recorded from atrial pacing leads during BiA pacing system implantation procedure [37–40]. The study demonstrated that the intracardiac signals can be processed in the same mode, and analysis of intra-atrial filtered A wave duration and its RMS20 and LAS5 parameters provides more precise information on atrial electrophysiology than external SA-ECG [37–40].

In the present study, we demonstrated that RAA pacing, in comparison to sinus rhythm, significantly prolongs P wave duration, TAAT, S-Q interval, external SA-ECG P wave duration, intracardiac SA-IEGM A wave duration in both atria and LAS5 values in external and right-atrial leads; it also produces unfavourable, although not significant, changes in RMS20 values. Pacing from distal CS (in comparison to sinus rhythm) significantly shortens filtered A wave duration in the left atrium with no significant shift in other parameters. Biatrial pacing brings about the most dramatic, yet positive, changes: it shortens P wave duration, TAAT, S-Q interval, external SA-ECG P wave duration, intracardiac SA-IEGM A wave duration in both atria and LAS5 values (although not significantly) in external and intra-atrial leads; moreover, BiA pacing significantly increases RMS20 values in external and both intra-atrial leads. During RAA pacing, atrial late potential criteria were positive in virtually all patients in external, right- and left-atrial leads; during CS pacing ALP occurrence decreased (external: 87%, RA: 92%, LA: 38%) — this effect is even stronger with BiA pacing (8%, 46% and 12%, respectively).

The switch from standard atrial (RAA) to BiA pacing significantly reduced ECG P wave duration (–63 ms), TAAT (–74 ms), S-Q (–49 ms), external SA-ECG P wave duration (–48 ms) and intracardiac SA-IEGM A wave duration in the right (–62 ms) and left (–72 ms) atria. This switch also shortened LAS5 values in external (–9 ms), right-atrial (–16 ms) and left-atrial (–8 ms) leads. The switch from RAA to BiA pacing favourably increased RMS20 values in external (+1.0 μV), right-atrial (+1.4 μV) and left-atrial (+1.6 μV) leads. These results support the consideration of the location of atrial leads in sites other than RAA in patients with bradytachycardia syndrome and coexisting conduction disturbances within the atria.

The type of atrial pacing has a strong influence on atrial electrophysiology since RAA pacing greatly prolongs atrial potential and increases the micro-oscillations in its terminal part. Distal CS pacing, compared to sinus rhythm, does not influence atrial potential duration or its terminal fraction. Simultaneous pacing from RAA and distal CS (BiA pacing) remarkably decreases the duration of atrial potential and its terminal micro-oscillations. The occurrence of atrial late potential criteria in the right atrium suggests the presence of local conduction disturbances that are not eliminated by BiA pacing.

These findings correspond with clinical data regarding the surprisingly good antiarrhythmic effect of permanent CS pacing in some patients [20–25, 28], as well as with some observations on limited antiarrhythmic effectiveness of BiA pacing

in patients with severe conduction disturbances within the atria [14, 16, 17, 31, 36]. This confirms the common opinion that the local conduction disturbances in the right atrium are "the key to atrial arrhythmias" [29, 32–34, 42]. Our study demonstrated that, in spite of BiA pacing, there are regions of delayed activation in the right atrium; incomplete resynchronisation could explain the ineffectiveness of arrhythmia suppression in some patients. These findings advocate the search for another site for the right-atrial lead in the BiA pacing system (region of Bachmann's bundle or sinus node?) and the potential usefulness of bifocal right-atrial pacing, particularly in patients with severe conduction disturbances within the right atrium, in whom BiA pacing fails to restore the synchrony of atrial activation.

Limitations of the study

The signal-averaging process was triggered by the R wave, since P wave synchronization was inadequate in patients with fragmented, low-amplitude P wave - numerous in the studied group. The applied "left atrial" lead system is not purely left atrial — introduction of a multipolar catheter to the left atrium via transseptal approach and the selection of three bipolar leads would be a better option. It is a routine procedure during pulmonary vein ectopy mapping and ablation but not during pacemaker implantation. In the study group there were no indications for left heart catheterization; therefore, we applied a pacing electrode introduced to the coronary sinus connected to the tip and ring of the electrode placed in the low right atrium. Consequently, in the "left atrial" leads, the activation of the lower part of the right atrium was additionally recorded. Since the final part of the left atrial excitation has superior importance, we consider this solution satisfactory. The study was performed in a selected group of patients with recurrent atrial arrhythmia and substantial conduction disturbances within the atria; thus, our conclusions must not be applied to the general population of patients with paroxysmal atrial fibrillation.

Conclusions

1. Right atrial appendage pacing, in comparison to sinus rhythm, significantly prolongs P wave duration, TAAT, S-Q interval, external SA-ECG P wave duration, intracardiac SA-IEGM A wave duration in both atria and LAS5 values in external and right-atrial leads; it also produces unfavourable, though not significant, changes in RMS20 values.
2. Pacing from distal CS (in comparison to sinus rhythm) significantly shortens filtered A wave duration in the left atrium with no significant shift in other parameters.
3. Biatrial pacing brings about the most significant changes: it shortens P wave duration, TAAT, S-Q interval, external SA-ECG P wave duration, intracardiac SA-IEGM A wave duration in both atria and LAS5 values (although not significantly) in external and intra-atrial leads; moreover, BiA pacing significantly increases RMS20 values in external and both intra-atrial leads.
4. During RAA pacing, atrial late potential criteria were positive in nearly all patients in external and right- and left-atrial leads; during CS pacing, ALP occurrence decreases (external: 87%, RA: 92%, LA: 38%); this effect is even stronger with BiA pacing (8%, 46% and 12%, respectively).
5. The site of atrial pacing has a profound influence upon atrial electrophysiology; RAA pacing distinctly prolongs atrial potential and increases the micro-oscillations in its terminal part. Distal CS pacing, compared to sinus rhythm, does not influence atrial potential duration or its terminal fraction. Simultaneous pacing from RAA and distal CS (BiA pacing) remarkably decreases the duration of atrial potential and its terminal micro-oscillations.
6. The occurrence of atrial late potential criteria in the right atrium suggests the presence of local conduction disturbances which are not eliminated by BiA pacing.

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