VIA MEDICA

ORIGINAL ARTICLE

Cardiology Journal
2007, Vol. 14, No. 4, pp. 372–383

ISSN 1897–5593

The electrophysiological effects of single-site RAA pacing evaluated by means of high-gain SA-ECG recorded from intra-atrial leads

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Abstract

Background: *Conventional right atrial appendage pacing (RAAp) eliminates the electrophysiological consequences of bradycardia only, leading to suppression of the rhythm-dependent arrhythmias but in some patients RAAp may increase AF recurrences or even promote it in patients without AF history. Relatively rare incidence of AF in patients implanted with single lead VDD pacing system may indicate RAAp influence. Atrial conduction disturbances (ACD) are the known substrate of re-entrant atrial arrhythmias and their detection is important for the selection of proper therapy. Time-domain analysis of P-wave in signal-averaged ECG (SA-ECG) recorded from chest leads is an accepted method evaluating inhomogeneity of atrial excitation, predictive for atrial arrhythmias. The aim of our study was to estimate the effect of RAAp on SA-ECG recorded from conventional external and from intraatrial leads.*

Methods: *Recordings were performed in 24 patients during biatrial pacing system implantation. A surface SA-ECG was obtained from orthogonal leads and intraatrial signals were recorded and averaged separately from the right and left atrium at SR and RAAp (LA pacing was temporary switched). 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: *RAAp significantly prolongs all parameters reflecting atrial activation (P ECG, TAAT, SA-ECG Pdur, SA-IEGM Adur in RA and LA) by 20 to 30 ms in comparison to SR. RAAp decreases RMS20 and prolongs LAS5 values both in external and intraatrial leads, which reflects increased micro-oscillations in the final portion of atrial potential. The lower RMS20 and higher LAS5 values in RA compared to LA suggest less homogenous depolarization in right atrium. This may suggest that atrial activation extinguishes more homogenously in LA. A different explanation may be that the observed sluggish ending of RA signal may be the result of a far-field sensing from the LA. The strong correlations between RAA paced P wave, TAAT, SA-ECG Pdur, SA-IEGM RA and LA Adur confirm that those parameters reproduce mostly the velocity of conduction within the atria. Our findings indicates significant aggravation of ACD (mainly in RAA) and suggests that the search is needed for another RA lead location for permanent single site and biatrial pacing.* (Cardiol J 2007; 14: 372–383)

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

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Received: 5.07.2007 Accepted: 5.07.2007

Introduction

Cardiac pacing has become a widely accepted non-pharmacological approach to atrial arrhythmias, as the value of antiarrhythmic drugs is limited and knowledge on the mechanisms of arrhythmia has broadened [1–4]. The distortions of atrial repolarisation (its shortening, inadequate adaptation to the heart rate and increased dispersion) are the main substrate of the arrhythmias, while atrial premature beats and abrupt changes in heart rate (short-long--short cycle) [4–6] are the recognised triggers. The main favourable effects of atrial pacing are: 1) the rate of the control-stable cycle length decreases the dispersion of repolarisation, leading to the suppression of rhythm-dependent arrhythmias such as vagally-mediated atrial fibrillation (AF) or bradycardia--dependent AF, by reduction of "escape" atrial ectopic activity [2–4, 7]; 2) the overdrive suppression of atrial ectopy (the shortening of paced cycle length after an atrial ectopic beat; 3) the elimination of compensatory pauses; the arrhythmogenic effect of altering cycle length (long-short or short-long-short) has been demonstrated in ventricular arrhythmias, and its role in the initiation of atrial arrhythmias is under consideration [8, 9].

The "classic" right atrial appendage pacing (RAAp) with fixed rhythm eliminates the electrophysiological consequences of bradycardia only, thus having a satisfactory effect in vagally-mediated AF [10] but a less satisfactory one in the brady-tachycardia syndrome [1–4, 7–9]. To enhance its antiarrhythmic efficacy consistent atrial pacing algorithms have been introduced [8, 9]. In some patients right atrial pacing may increase instances of the recurrence of AF or even promote AF in patients without a history of it [1, 11–13]. This phenomenon has more frequently been observed after the implantation of an active-fixation lead to the free wall of the right atrium (RA). The relatively rare incidence of AF in patients implanted with a single-lead VDD pacing system has been explained by the absence of the "irritating" influence of the atrial lead [12, 14]. Since the unfavourable electrophysiological effects of RA pacing and the role of interatrial conduction disturbances have become better known [18, 19], resynchronising atrial pacing has been widely applied [1–3, 7, 11, 20].

Our previous studies on the electrophysiological effects of RAAp based on analysis of IEGM tracings obtained via pacemaker telemetry, revealed that this pacing mode caused prolongation of AV activation (+30 ms), P wave duration (+17 ms), interatrial conduction time (+40 ms) and total atrial conduction time (TAAT) by 25 ms, in comparison to sinus rhythm. The changes were inversely proportional to the pacing cycle length. Despite the increase in data on the unfavourable proarrhythmic effects of RAAp [21–23] and the unequivocal opinion of electrophysiologists, the RAA is still the most frequent pacing site in the RA, even in patients with brady-tachycardia syndrome.

In patients with recurrent drug-resistant atrial arrhythmias, in whom inter- and intra-atrial conduction disturbances can be detected, the presence of micropotentials at the final phase of atrial activation should be anticipated [24]. Conduction distortions within the atria resulting from RAAp can occur on a "macro" scale and be assessed by P wave duration and TAAT [21–23] or on a "micro" scale, reflected by the root mean square voltage of the last 20 ms of atrial activation (RMS20) and the duration of the low-amplitude signal < 5 mV (LAS5) [24–27]. The literature indicates that RAAp should increase conduction disturbances [15–17] and the inhomogeneity of depolarisation, which may be reflected by abnormal RMS20 and LAS5 values, resulting in positive atrial late potentials (ALP) criteria [28, 29].

The aim of the study was to estimate the electrophysiological effects of conventional RAAp upon conduction disturbances within the atria and the homogeneity of atrial depolarisation, assessed by the values of RMS20 and LAS5, as well as the presence of ALP criteria. Moreover, we intended to assess the influence of RAAp upon the homogeneity of activation measured separately in the left atrium (LA) and RA, since there are no published studies concerning this problem.

Methods

Patients

The study was conducted on a group of 24 patients (15 female, 9 male, with a mean age of 68.8 ± 9.97 years) eligible for permanent biatrial pacing. In 7 patients (29.2%) sporadic AF, in 8 patients (33.3%) recurrent AF and in 9 patients (37.5%) frequent AF (according to the Kingma and Suttorp paradigm [30]) was identified. Owing to the considerable 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 one drug (amiodarone or propafenone), 8 were on two drugs (propafenone and sotalol/amiodarone) and 11 patients (46%) had no ongoing antiarrhythmic medication.

Procedures

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

External SA-ECG recording and processing

Equipment constructed in the National Institute of Cardiology (Warsaw) was employed for signal recording and processing. It consisted of a micropotentials amplifier (noise < 1.5 μV in 0.1–300 Hz bandwidth, CMRR > 130 Db), 12-bit A/D converter, 486 CPU PC and software designed for signal-averaging processing and subsequent analysis of data. Standard Ag/AgCl electrodes were applied 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 digitised by the A/D converter with a 12-beat accuracy. The signal-averaging process was triggered by the R wave during sinus rhythm and the pacing spike during RAAp. Ectopic beats, if present, were identified and rejected. Approximately 50 beats were averaged and stored on the PC HD. The process has been described previously [11].

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 the P wave were defined as the point at which the atrial signal exceeded and returned to the 1.5 μV level 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). The ALP were considered positive with Pdur > 125 ms and RMS20 < 2.4 μV [11].

Intracardiac SA-IEGM recording and processing

Three bipolar pacing leads were used: a standard J-shaped lead implanted into the RAA, a second lead introduced into the CS and a third lead (for permanent ventricular pacing), which was temporarily placed in the LRA position. The equipment described above was employed for signal recording and processing. To obtain RA and LA signals, intracardiac leads were attached to the micropotentials amplifier via sterile connectors, according to the scheme in Figure 1. The right atrial electrogram was recorded from three combined intra-atrial leads X' Y' Z' and the left atrial electrogram from the X'' Y'' Z'' leads. The signal from each lead was augmented and filtered in the same mode as during external signal recording. The averaging process was triggered by the A wave during sinus rhythm and the pacing spike during RAAp, and the parameters obtained were analysed in the same way as the external SA-ECG. The technique employed has been described previously [30–32].

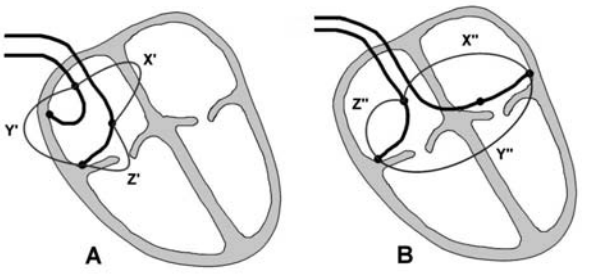

Figure 1. The scheme presents the connections of intracardiac leads to obtain right atrial (X' Y' Z') (**A**) and left atrial (X'' Y'' Z'') (**B**) signals.

Interatrial conduction evaluation with internal electrogram recordings

The 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 leads II or III at high resolution ECG; 2) interatrial conduction time (IACT) measured from the onset of the A wave in the RAA signal to the onset of the A wave in the CS signal; 3) total atrial activation time (TAAT) measured from the onset of the P wave in the lead II ECG to the end of atrial activation in the CS signal.

Statistical analysis

Significance of difference of the means in all subgroups was assessed using variance F test and between-group significance of difference of the means was tested using lowest significant difference (LSD) test. For significance of difference of the

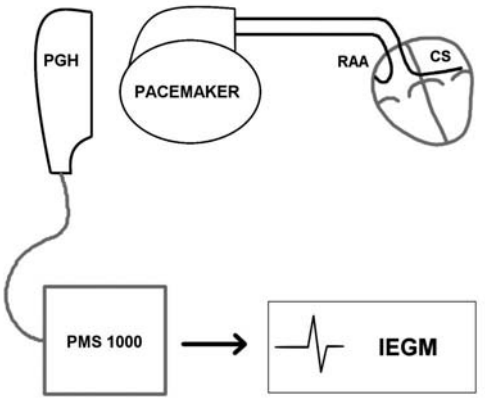

Figure 2. The internal electrogram (IEGM) was recorded from the right atrial appendage (RAA) and coronary sinus (CS) leads connected to a dual-chamber pacemaker via telemetry.

means between two subgroups t-test for unpaired data was used. Correlation between parametric values was assessed by Pearson correlation coefficient.

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

Results

The results are presented in Table 1. The average P wave duration was 158 ms at sinus rhythm and increased to 181 ms at RAAp. A significant influence was exerted by RAA on atrial activation, assessed by the interatrial conduction time (104 *vs*. 120 ms), TAAT (182 *vs*. 201 ms), SA-ECG P wave duration (156 *vs*. 199 ms) and SA-IEGM A wave duration from the RA (175 *vs*. 201 ms) and LA (175 *vs*. 201 ms). There was also a significant influence upon the duration of low-amplitude potential (LAS5), which was markedly prolonged in external SA-ECG (6.7 *vs*. 14.5 ms) and intra-atrial SA-IEGM in the RA (12.9 *vs*. 24.6 ms) and the LA (9.1 *vs*. 13.5 ms) during RAAp. There was no clear influence of this pacing mode upon the RMS20 values. Table 2 illustrates the statistical evaluation of the above data (Fig. 3).

The results imply that RAAp not only significantly prolongs atrial activation, but also increases the inhomogeneity of the final part of atrial potential, worsening the asynchrony of atrial activation. This is consistent with clinical observations.

The conduction of atrial activation and the homogeneity of its last part can be illustrated by three basic SA-ECG parameters: the P wave duration (Pdur), the root mean square voltage of the final 20 ms of the P wave (RMS20) and the duration of the low-amplitude signal $< 5\ \mu$V (LAS5). The homogenous atrial activation is characterised by a short Pdur and LAS5, with a high RMS20 [24–29]. The coincident abnormal values of Pdur and RMS20 lead to the detection of the ALP, a widely accepted predictor of atrial arrhythmias. Table 3 presents the rate of ALP criteria occurrence in the external and intra-atrial leads at SR and RAAp.

Atrial late potentials criteria were present in 79% of patients in external SA-ECG and in 96% and 86% of patients in RA and LA SA-IEGM respectively. RAAp yielded positive ALP criteria in all but one (in RA SA-IEGM) patients, which additionally con-

Table 1. Comparison of interatrial conduction parameters and high-gain SA P&A wave time domain during sinus rhythm and right atrium appendage pacing.

Rhythm parameters	Sinus rhythm						Right atrium appendage pacing					
	N	Aver.	Med.	Min.	Max.	SD	N	Aver.	Med.	Min.	Max.	SD
Pdur ECG	24	157.7	159.0	125.0	199.0	16.0	24	180.9	180.0	140.0	245.0	26.8
IACT	24	103.7	99.5	55.0	148.0	22.6	24	119.9	123.5	51.0	165.0	24.8
TAAT	24	181.5	182.0	144.0	225.0	22.4	24	201.1	199.0	160.0	265.0	28.9
Ext. SA Pdur	24	156.2	155.4	119.3	186.8	16.8	24	188.9	185.1	150.7	253.8	30.5
Ext. SA RMS20	20	2.17	1.96	1.09	3.69	0.76	24	1.82	1.78	0.68	3.14	0.71
Ext. SA LAS5	24	6.68	5.50	0.00	26.7	7.48	24	14.59	13.35	1.60	64.40	12.81
Int. SA RA Adur	24	174.8	168.8	144.4	224.5	24.1	24	199.1	187.1	158.0	256.9	29.1
Int. SA RA RMS20	23	1.77	1.69	0.75	3.46	0.72	22	1.35	1.12	0.64	3.35	0.64
Int. SA RA LAS5	23	12.91	11.00	0.00	33.00	7.92	22	24.62	18.80	3.10	84.80	19.99
Int. SA LA Adur	23	175.3	169.6	138.2	237.1	26.7	24	201.3	189.1	151.7	270.0	31.2
Int. SA LA RMS20	15	2.33	1.78	0.98	4.81	1.19	16	1.96	1.93	0.84	3.64	0.84
Int. SA LA LAS5	15	9.01	9.40	0.00	18.80	6.17	16	13.45	9.45	0.00	61.20	14.10

Table 2. Interatrial conduction and high-gain SA P&A wave parameters during SR and RAAp. Comparison and statistical evaluation differences of average values.

Parameters	Rhythm	N	Average	SD	p
P duration	SR	24	157.67	15.99	0.000
High-resolution ECG	RAAp		180.92	26.82	
TAAT	SR	24	181.50	22.46	0.000
(IEGM)	RAAp		201.12	28.96	
SA P duration	SR	24	156.19	16.84	0.000
Ext. leads	RAAp		188.91	30.49	
RMS20	SR	20	2.17	0.76	0.177
Ext. leads	RAAp		1.85	0.71	
LAS5	SR	24	6.68	7.48	0.001
Ext. leads	RAAp		14.59	12.81	
SA A duration	SR	24	174.77	24.10	0.000
Int. RA leads	RAAp		199.13	29.10	
RMS20	SR	16	1.48	0.44	0.400
Int. RA leads	RAAp		1.22	0.47	
LAS5	SR	16	15.42	27.58	0.003
Int. RA leads	RAAp		27.58	21.08	
SA A duration	SR	23	175.29	26.66	0.000
Int. LA leads	RAAp		200.26	31.51	
RMS20	SR	12	2.09	0.93	0.179
Int. LA leads	RAAp		1.65	0.60	
LAS5	SR	12	9.82	5.60	0.135
Int. LA leads	RAAp		15.18	15.95	

firms the potential proarrhythmic effect of this pacing mode.

Subsequently the differences between the parameters obtained from the external and (separately) intra-atrial leads were analysed (Table 4). It could be anticipated that the higher amplitude of the intra-atrial signals would give the opportunity to assess more extensive trial activation (omitted in the external leads).

The RAA paced SA atrial potential recorded from the intra-atrial leads was significantly longer (approx. 10 ms) than the external SA P wave recorded from Frank's leads. There was no substantial difference between SA-IEGM A wave duration recorded from the RA and the LA, although it must be recognised that different timings were measured; the onset and the ending of the LA A wave was recorded noticeably later than the RA A wave timing. The LAS5 values were significantly longer in the RA than in the LA and external leads. There were no significant differences between RMS20 values recorded from external and RA leads; in contrast, the RMS20 value was significantly higher in the LA than in the RA. This may suggest that atrial activation extinguishes more homogenously in the LA. A different explanation may be that the sluggish ending of the RA signal observed may be the result of far-field sensing from the LA (Fig. 4).

The correlations between RAA paced ECG P wave duration, IACT, TAAT and the SA-ECG/ /IEGM parameters analysed are presented in Tables 5, 6 and 7. The results indicate that RAA paced P wave duration assessed in external ECG does not reflect the whole atrial activation, since it is noticeably shorter than TAAT, which implies that a considerable portion of atrial potential is omitted in external ECG. Although the SA-IEGM Adur values were virtually equal in the Ra and LA, it is essential that the onset of the A wave was apparently recorded later in the LA than in the RA SA-IEGM, which indicates that in both electrograms the activation of different atrial regions was evaluated.

Discussion

A biatrial pacing system gives the opportunity of simultaneous, non-invasive registration of signals from the RA and LA. We implemented this technique in our previous studies [28, 29], demonstrating an unfavourable prolongation of atrial potential by

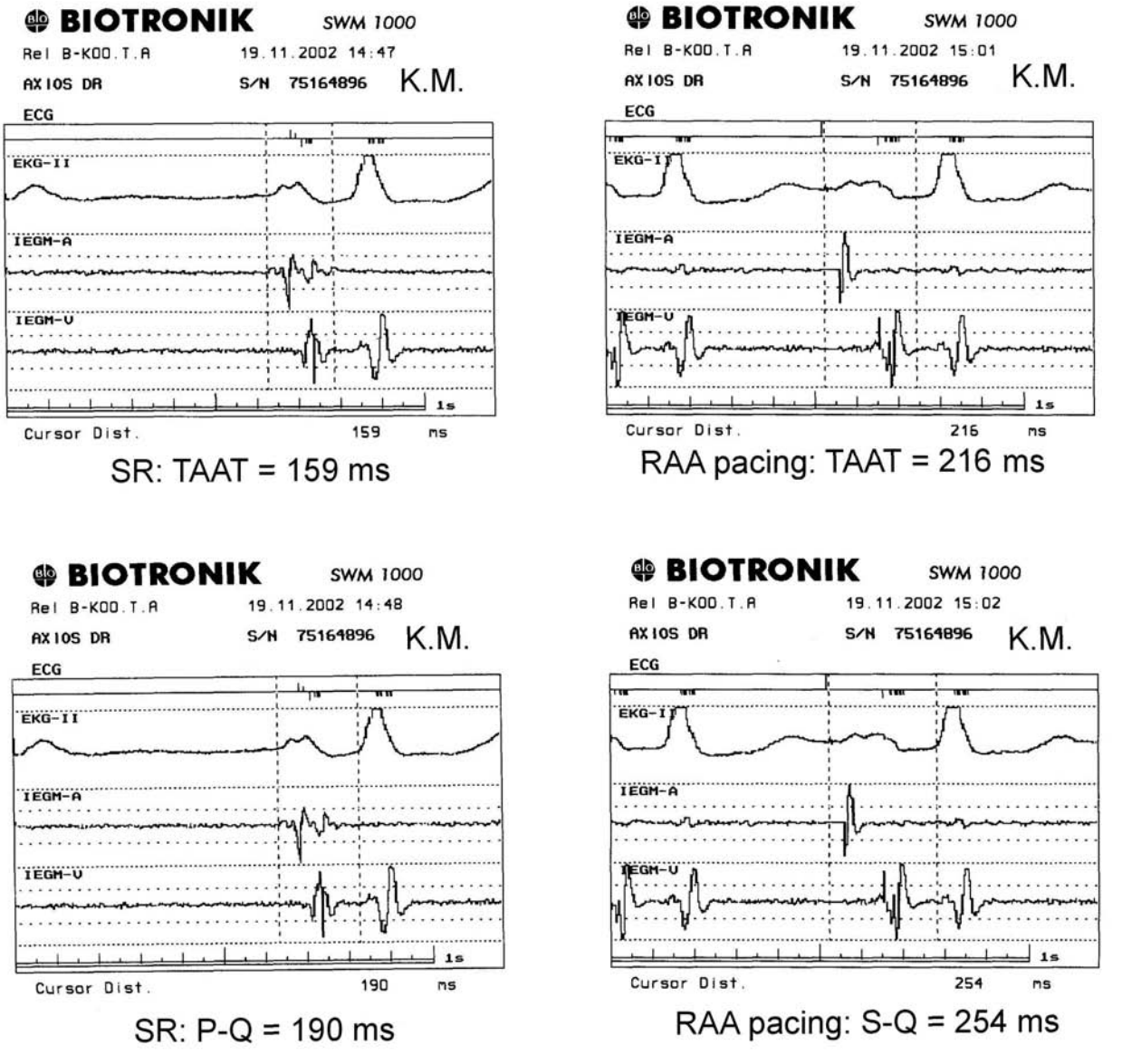

Figure 3. Right atrial appendage (RAA) pacing prolongs total atrial activation time (TAAT) and P-Q/S-Q interval.

Table 3. Presence of atrial late potentials (ALP) criteria during sinus rhythm and right atrium appendage (RAA) pacing in conventional and intra-atrial SA ECG/IEGM.

Rhythm	Leads	Ext.	Int. RA	Int. LA
Sinus rhythm	ALP — YES	19/24 (79.2%)	23/24 (95.8%)	19/22 (86.4%)
	ALP — NO	5/24 (20.8)	1/24 (4.2%)	3/22 (14.6%)
	Lack*	0/24 (0%)	0/24 (0%)	2/24 (8.3%)
RAA pacing	ALP — YES	24/24 (100.0%)	22/23 (95.6%)	24/24 (100.0%)
	ALP — NO	0/24 (0%)	1/23 (4.3%)	0/24 (0%)
	Lack*	0/24 (0%)	1/24 (4.2%)	0/24 (0%)

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

RAAp; this is consistent with the data obtained by invasive methods by other authors [15–18].

Analysis of the time- and frequency-domain parameters of the signal-averaged P wave detects

Table 4. Comparison of right atrium appendage paced atrial potential duration and its RMS20 and LAS5 in recordings obtained from external (Frank's) and intra-atrial leads.

Examined parameters	N	Leads	Ave-rage	SE	Analysis of variance	NIR test				
						Groups	Compa-rison	Aver. diff.	SD of diff.	p
SA ECG/IEGM	24	Extern.	188.9	6.22	F = 1054.2 P = 0.000	1	1 *vs*. 2	–10.2	2.20	0.000
P&A wave	24	Int. RA	199.1	5.94		2	1 *vs*. 3	–12.4	2.20	0.000
duration	24	Int. LA	201.3	6.37		3	2 *vs*. 3	–2.18	2.20	0.333
SA ECG/IEGM	16	Extern.	1.59	0.13	F = 201.7 P = 0.000	1	1 *vs*. 2	0.32	0.19	0.096
P&A RMS20	16	Int. RA	1.17	0.10		2	1 *vs*. 3	–0.47	0.19	0.016
	16	Int. LA	1.96	0.21		3	2 *vs*. 3	–0.79	0.19	0.000
SA ECG/IEGM	16	Extern.	18.75	3.41	F = 62.2 P = 0.000	1	1 *vs*. 2	–9.32	5.62	0.107
P&A LAS5	16	Int. RA	28.07	5.19		2	1 *vs*. 3	5.30	5.62	0.353
	16	Int. LA	13.45	3.52		3	2 *vs*. 3	14.62	5.62	0.014

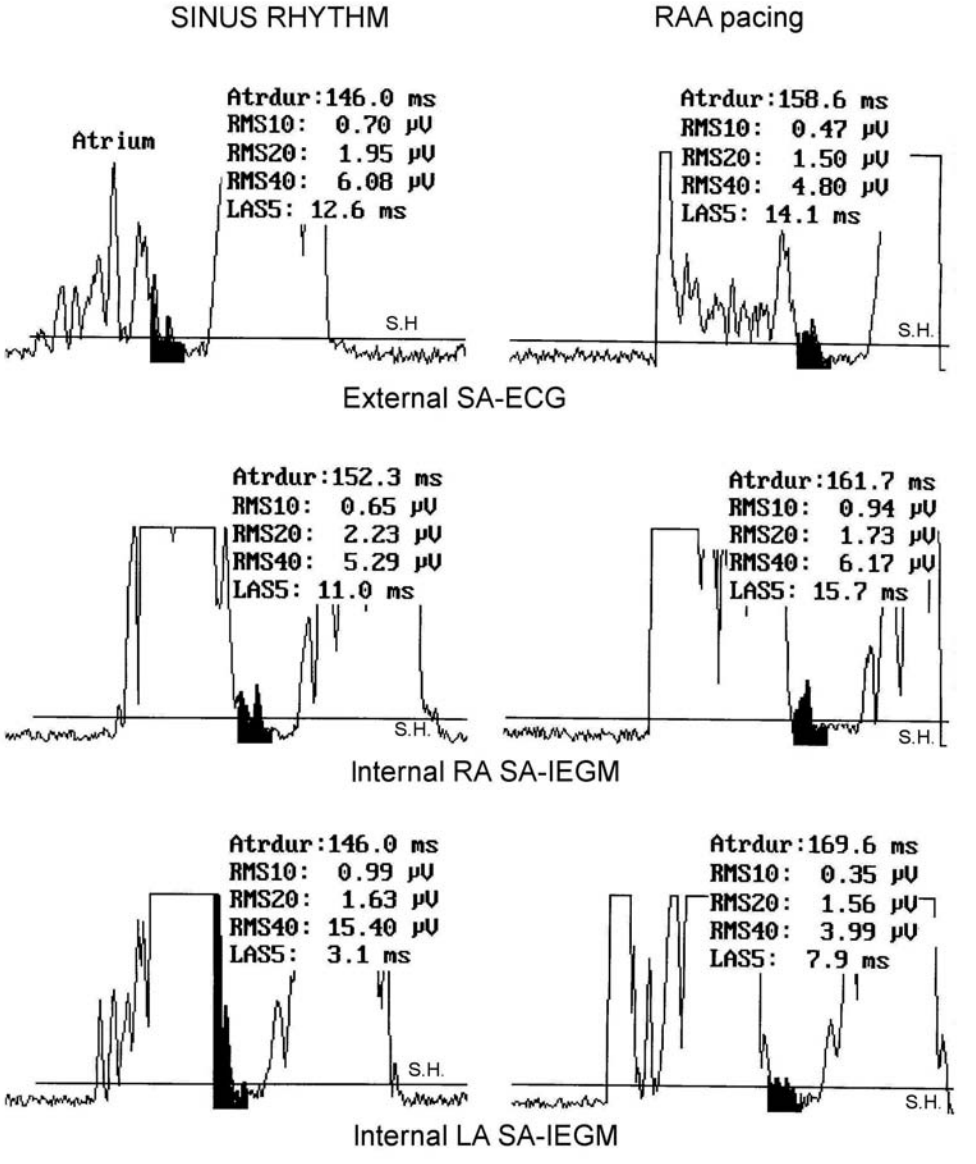

Figure 4. Signal-averaged electrogram obtained from external and intra-atrial leads in the same patient during sinus rhythm and right atrial appendage (RAA) pacing reveals its unfavourable effect on atrial activation synchrony.

Table 5. Examinations of mutual correlation (*r* Pearson's) between RAA paced P wave duration (ECG) and SA atrial potential duration, its RMS20 and LAS5 examined in external and intra-atrial recordings.

Parameters	N	Average	SD	r (X,Y)	t	p
High res. ECG Pdur Ext. SA ECG Pdur	24	180.9 188.9	26.8 30.4	0.91	10.170	0.000
High res. ECG Pdur Ext. SA ECG RMS20	24	180.9 1.82	26.8 0.71	0.02	0.097	0.923
High res. ECG Pdur Ext. SA ECG LAS5	24	180.9 14.59	26.8 12.81	0.24	1.171	0.254
High res. ECG Pdur Int. SA IEGM RA Adur	24	180.9 199.1	26.8 29.1	0.90	9.481	0.000
High res. ECG Pdur Int. SA IEGM RA RMS20	22	181.0 1.35	28.1 0.64	–0.32	–1.495	0.150
High res. ECG Pdur Int. SA IEGM RA LAS5	22	181.0 24.62	28.1 19.99	0.60	3.351	0.003
High res. ECG Pdur Int. SA IEGM LA Adur	24	180.9 201.3	26.8 31.2	0.86	7.795	0.000
High res. ECG Pdur Int. SA IEGM LA RMS20	16	184.6 1.96	30.4 0.84	0.20	0.75206	0.464
High res. ECG Pdur Int. SA IEGM LA LAS5	16	184.6 13.45	30.4 14.10	0.06	0.227	0.823

Table 6. Examination of mutual correlation (*r* Pearson's) between interatrial conduction time (IACT) values and SA atrial potential duration, its RMS20 and LAS5 examined in external and intra-atrial recordings during RAA pacing.

Parameters	N	Average	SD	*r* (X, Y)	t	p
IACT (IEGM) Ext. SA ECG Pdur	24	119.9 188.9	24.8 30.5	0.65	3.99	0.000
IACT (IEGM) Ext. SA ECG RMS20	24	119.9 1.82	24.8 0.71	–0.07	0.336	0.739
IACT (IEGM) Ext. SA IECG LAS5	24	119.9 14.59	24.8 12.81	0.32	1.570	0.130
IACT (IEGM) Int. SA IEGM RA Adur	24	119.9 199.1	24.8 29.1	0.69	4.480	0.000
IACT (IEGM) Int. SA IEGM RA RMS20	22	119.8 1.35	25.8 0.64	–0.16	0.726	0.476
IACT (IEGM) Int. SA IEGM RA LAS5	22	119.7 24.62	25.8 19.99	0.27	1.243	0.227
IACT (IEGM) Int. SA IEGM LA Adur	24	119.9 201.3	24.8 31.2	0.69	4.510	0.000
IACT (IEGM) Int. SA IEGM LA RMS20	16	123.4 1.96	22.3 0.84	–0.19	0.727	0.478
IACT (IEGM) Int. SA IEGM LA LAS5	16	123.4 13.45	22.3 14.10	0.25	0.965	0.350

Table 7. Examination of mutual correlation (*r* Pearson's) between total atrial activation time (TAAT) values and SA atrial potential duration, its RMS20 and LAS5 examined in external and intra-atrial recordings during RAA pacing.

Parameters	N	Average	SD	*r* (X, Y)	t	p
TAAT (IEGM) Ext. SA ECG Pdur	24	201.1 188.9	28.9 30.5	0.87	8.491	0.000
TAAT (IEGM) Ext. SA ECG RMS20	24	201.1 1.82	28.9 0.71	–0.15	0.722	0.477
TAAT (IEGM) Ext. SA ECG LAS5	24	201.1 14.59	28.9 12.81	0.48	2.538	0.018
TAAT (IEGM) Int. SA IEGM RA Adur	24	201.1 199.1	28.9 29.1	0.90	9.916	0.000
TAAT (IEGM) Int. SA IEGM RA RMS20	22	201.2 1.35	30.2 0.64	–0.45	2.230	0.037
TAAT (IEGM) Int. SA IEGM RA LAS5	22	201.2 24.62	30.2 19.99	0.42	2.075	0.0515
TAAT (IEGM) Int. SA IEGM LA Adur	24	201.1 201.3	28.9 31.2	0.91	10.444	0.000
TAAT (IEGM) Int. SA IEGM LA RMS20	16	206.0 1.96	30.9 0.84	–0.08	0.295	0.772
TAAT (IEGM) Int. SA IEGM LA LAS5	16	206.0 13.45	30.9 14.10	0.34	1.376	0.190

the decreased homogeneity of atrial depolarisation and helps to predict the risk of recurrent atrial arrhythmias [24–27]. In previous papers we described the prolongation of signal-averaged P wave duration by RAAp [28, 29].

The delayed activation of the LA (particularly in patients with interatrial conduction disturbances) is generally recognised. Thus the final part of the SA-ECG P wave reflects the LA potential and the RMS20 and LAS5 parameters reveal micro-oscillations originating in the LA. On the other hand, the key role of RA conduction disturbances in the maintenance of atrial arrhythmias is known [13–16, 20].

The techniques described were not capable of evaluating the late potentials in the RA alone and subsequently of judging the effect of RAAp upon RA micro-oscillations. These questions lead us to a hypothesis that the potentials recorded from three intra-atrial leads, processed with a standard signal-averaging technique, will possibly be a valuable instrument for the evaluation of the electrophysiological effects of atrial pacing. The results demonstrated that RAAp prolongs all parameters reflecting atrial activation (P ECG, TAAT, SA-ECG Pdur, SA-IEGM RA and LA Adur) by 20 to 30 ms. Additionally, RAAp significantly prolonged LAS5 in the external and RA leads, and to some extent (but not significantly) decreased the RMS20 values in all leads. Both changes in RMS20 and LAS5 values reflect increased micro-oscillations in the final portion of the atrial potential at RAAp. The lower RMS20 and higher LAS5 values in the RA compared to the LA suggest less homogenous depolarisation in the RA. The strong correlations between the RAA paced P wave, TAAT, SA-ECG Pdur, SA-IEGM RA and LA Adur confirm that these parameters mostly reproduce the velocity of conduction within the atria. The lack of correlation between ECG P wave duration, TAAT or IACT and the values of the RMS20 and LAS5 parameters indicates that the interatrial conduction disturbances are not clearly related to the presence and duration of micro-oscillations in the final part of RAA paced atrial potential.

There are a few published papers concerning the application of time- or frequency-domain analysis of SA-ECG parameters for the evaluation of different atrial pacing modes [33–35]. Gribbin et al. [33] compared the effects of long-term physiological (AAI, DDD) and non-physiological (VVI) pacing on the time- and frequency-domain SA-ECG parameters at sinus rhythm (30/min pacing rate) in patients with sick sinus syndrome. Although there was no

significant change in LA dimensions, there was a significant prolongation of the signal-averaged P wave duration in the group with non-physiological pacing. This unfavourable electrophysiological remodelling may explain the higher incidence of atrial arrhythmias in patients with VVI pacing. Keane et al. [34] analysed signal-averaged P wave time- and frequency-domain parameters in sick sinus syndrome patients with and without paroxysmal AF, both at sinus rhythm and RAAp. The SA P wave duration at sinus rhythm was shortest in the control group (134 ms), longer in the SSS group (140 ms) and significantly prolonged in the group with PAF (156 ms); there were no significant differences in the RMS20 values. RAAp additionally prolonged SA P wave duration in patients with SSS (140 *vs*. 167 ms) and PAF (156 *vs*. 170 ms). The authors concluded that RAAp discloses conduction disorders within the atria and can thus be a useful instrument for the selection of the optimal pacing mode [34].

Yamada et al. [35] in 2001 published the results of RAA and Bachmann's bundle (BB) pacing, assessed with the use of SA-ECG time-domain parameters. In the study referred to BB pacing reduced (compared to SR) SA P wave duration (126 *vs*. 122 ms) and increased RMS20 (2.9 *vs*. 2.0 μV). The RAAp resulted in the opposite effects. The authors considered ALP criteria as P > 132 ms and RMS20 < 2.5 μV. They found ALP positive in 80% of patients in the subgroup with PAF and in 20% of the subgroup without arrhythmias. BB pacing changed the ratio to 10% and 13%, and RAAp to 70% and 27% respectively. In the discussion the authors emphasise the arrhythmogenic effect of RAAp [4, 35].

The data presented lends support to the choice of an alternative atrial pacing site in patients with interatrial conduction disturbances and recurrent atrial arrhythmias.

Limitations of the study

The signal-averaging process was triggered by the R wave, since P wave synchronisation was inadequate in patients with a fragmented low-amplitude P wave, which was frequent in the group studied. The "left atrial" lead system applied is not purely left atrial; the introduction of a multipolar catheter to the LA via the trans-septal approach and selection of three bipolar leads would be a better option. It is a routine procedure in pulmonary vein ectopy mapping and ablation, but not during pacemaker implantation. In the study group there were no indications for left heart catheterisation, and therefore we applied a pacing electrode, introduced to the CS, connected to the tip and ring of the electrode placed in the low RA. Consequently, in the "left atrial" leads the activation of the lower part of the RA was additionally recorded. Since the final part of the LA excitation has superior importance, we consider this solution satisfactory.

At the beginning of the study there was some doubt as to whether the fragmented termination of the potential recorded from the RA truly reflected RA activation or just far-field sensing from the LA. Parallel recordings performed during LA and biatrial pacing, however, confirmed the existence of clearly RA late potentials, since the LA was activated earlier during pacing [36, 37]. The study was performed in the selected group of patients with recurrent atrial arrhythmia and considerable conduction disturbances within the atria, and thus our conclusions must not be applied to the general population of patients with paroxysmal atrial fibrillation.

Conclusions

1. RAA pacing significantly prolongs all parameters reflecting atrial activation (P ECG, TAAT, SA-ECG Pdur, SA-IEGM Adur in RA and LA) by 20 to 30 ms.
2. RAA pacing decreases RMS20 and prolongs LAS5 values both in external and intra-atrial leads, which reflects increased micro-oscillations in the final portion of atrial potential.
3. The lower RMS20 and higher LAS5 values in the RA than in the LA suggest less homogenous depolarisation in the RA. This may suggest that atrial activation extinguishes more homogenously in the LA. A different explanation may be that the sluggish ending of the RA signal observed may be the result of far-field sensing from the LA.
4. The strong correlations between the RAA paced P wave, TAAT, SA-ECG Pdur, SA-IEGM RA and LA Adur confirm that these parameters mostly reproduce the velocity of conduction within the atria.
5. The lack of correlation between ECG P wave duration, TAAT or IACT and the values of the RMS20 and LAS5 parameters indicates that interatrial conduction disturbances are not clearly related to the presence and duration of micro-oscillations in the final part of RAA paced atrial potential.
6. The data presented encourage the search for an alternative atrial pacing site in patients with interatrial conduction disturbances and recurrent atrial arrhythmias.

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