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Coronary sinus pacing: Its influence on external and intraatrial signal-averaged P wave time domain parameters

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Abstract

Background: *The coronary sinus (CS) was, for 10 years, the standard place for permanent atrial pacing, and the antiarrhythmic properties of CS pacing were described by Moss in the early 70's. These observations were confirmed during EP studies, although currently permanent CS pacing is infrequently applied. Signal averaged (SA) P wave analysis has established values for the examination of EP properties of atrial myocardium. The aim of our study was to estimate the effect of CS pacing on the signal-averaged P wave recorded from external and 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 processed separately from the right and left atrium at SR and CS 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: *Coronary sinus pacing favourably modifies SA P wave parameters of the left atrium; it significantly shortens Pdur, distinctly increases RMS20, decreases LAS5 and eliminates ALP in most patients in comparison to SR. It indicates beneficial effects of CS pacing on left atrial excitation and may explain its antiarrhythmic effect. Coronary sinus pacing does not deteriorate right atrium activation; it even slightly increases RMS20 and shortens the duration of LAS5 in RA in comparison to SR. Our findings suggest that CS is still an attractive site for permanent atrial pacing in patients with atrial arrhythmias and atrial conduction disturbances.* (Cardiol J 2007; 14: 470–481)

Key words: signal-averaged ECG, coronary sinus pacing, intraatrial signal, atrial late potentials, atrial fibrillation

Introduction

The coronary sinus (CS) was the first permanent atrial pacing site in the late 60's and at the beginning of the 70's [1–4] although a few years later the right atrial appendage (RAA) became the standard location [5, 6]. In that pioneering era of atrial pacing several important clinical and electrophysiological observations were underestimated [1–4]. Moss et al. described the shortening of the A-V conduction by CS pacing [1–3]. When Greenberg et al. [4] confirmed the phenomenon several years later he additionally suggested that: *"in some patients with intermittent atrial fibrillation CS pacing can provide an atrial impulse that minimizes the tendency to return atrial fibrillation"*. Another

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important, but forgotten, observation by Greenberg [4] was that "*failure to sense atrial electrogram was not significant in our series (...) we did not find an increased incidence of supraventricular tachycardia in five patients with failure to sense the atrium*".

Permanent RAA pacing together with ongoing antiarrhythmic therapy can suppress arrhythmia in most patients with brady-tachycardia syndrome [6, 7] although in some patients this mode of pacing may be unsuccessful or even proarrhythmic [1, 8–10]. A better recognition of interatrial conduction disturbances as the substrate for atrial arrhythmias [11, 12] lead to the idea of atrial resynchronization [13, 14]. It was proved that in patients with conduction disturbances within the atria the phenomenon is increased by RA pacing, which is reflected by prolonged P wave duration in comparison to SR [15, 16]. To restore the synchrony of atrial activation, Daubert [17] proposed biatrial, and Saksena [14] dual-site, right atrial pacing. In the following years Sopher et al. [18] and Spencer et al. [19] demonstrated the resynchronizing effect of Bachmann's bundle pacing. Padeletti [20] achieved outstanding antiarrhythmic effect by infero-posterior interatrial septum lead location combined with consistent atrial pacing algorithm.

In 1996 we analyzed the data from our first 20 patients with permanent CS pacing, demonstrating that bipolar (BP) CS pacing produced significantly shorter P wave compared to unipolar (UP) pacing (117 *vs*. 138 ms), with different morphology [21, 22]. Our further studies [23] confirmed this observation and supported our hypothesis that high-energy, bipolar mid-CS pacing results in atrial resynchronization [24, 25]. In the following studies we 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, its application is generally limited to cases of technical problems with RA lead location [28, 29], or as part of a biatrial pacing system; the electrophysiological and antiarrhythmic aspects of CS pacing [15, 16, 30–41] are barely considered.

The key role of the conduction disorders within the right atrium in the perpetuation of atrial arrhythmias is widely accepted [11, 12, 15, 16]. Recently we demonstrated the usefulness of intraatrial signals processed with a standard (external) SA-ECG technique for the evaluation of atrial potential [42, 43]. The described method allows the assessment of the additional 15–20 ms of atrial activation omitted in external SA-ECG, allowing the possibility to obtain comprehensive data concerning the effects of atrial pacing [44].

The aim of the study was to estimate the electrophysiological effects of CS pacing upon conduction disturbances within the atria and the homogeneity of atrial depolarization assessed by the SA-ECG technique in external and intraatrial leads.

We assumed that CS pacing, due to inverted direction of atrial activation, should have an influence upon interatrial conduction disturbances; the assessment of homogeneity of right atrial potential during CS pacing seemed to be particularly interesting.

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 biatrial pacing. Seven patients (29.2%) presented sporadic AF, 8 patients (33.3%) recurrent AF and 9 patients (37.5%) frequent AF (according to the Kingma and Suttorp paradigm [45]). Due to the 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 CS 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. Intraatrial 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, 486 CPU PC and software designed for signal-averaging process 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 digitized by an 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 at CS pacing. Ectopic beats, if present, were identified and rejected. Approximately 50 beats were averaged and stored on PC HD. The procedure was described before [27].

High-gain SA-ECG P wave parameter 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 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). Atrial late potentials (ALP) were considered positive with Pdur > 125 ms and RMS20 < 2.4 μV [27].

Intracardiac SA-IEGM recording and processing

Three bipolar pacing leads were used: a standard "J" shaped lead implanted into the right atrial appendage (RAA), and the second lead was introduced into CS. The third lead (for permanent ventricular pacing) was temporarily placed in the LRA position. The same, described above, equipment 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 and cables according to scheme on Figure 1. Right atrial electrogram was recorded from three combined intraatrial leads X' Y' Z' and left atrial electrogram — from leads X'' Y'' 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 [42–44, 51].

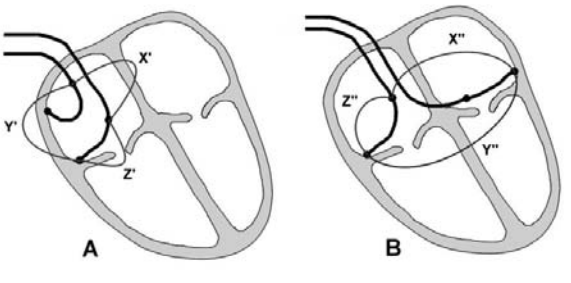

Figure 1. The scheme presents the connections of intracardiac leads to obtain right atrial (X'Y'Z') and left atrial (X''Y''Z'') signals.

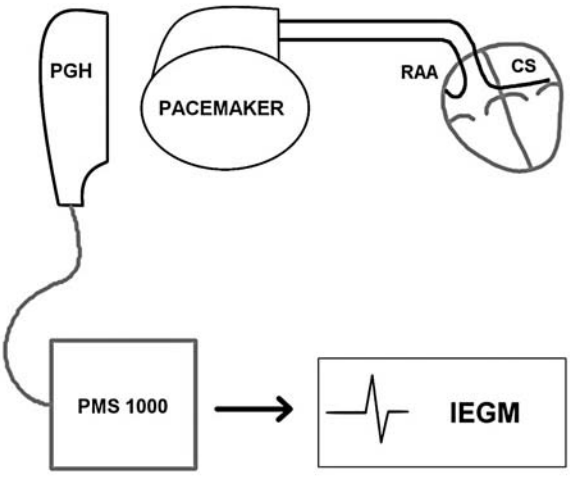

Figure 2. The internal electrogram (IEGM) was recorded from the right atrial appendage (RAA) and coronary sinus (CS) leads connected to a dual-chamber pacemaker via telemetry.

Interatrial conduction evaluation with IEGM recordings

The internal electrogram (IEGM) was recorded from 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}); 2) interatrial conduction time (IACT) measured from the onset of A wave in RAA signal to the onset of A wave in CS signal; 3) total atrial activation time (TAAT) measured from the onset of P wave in lead II ECG to the end of atrial activation in CS signal (Fig. 3).

Statistical analysis

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

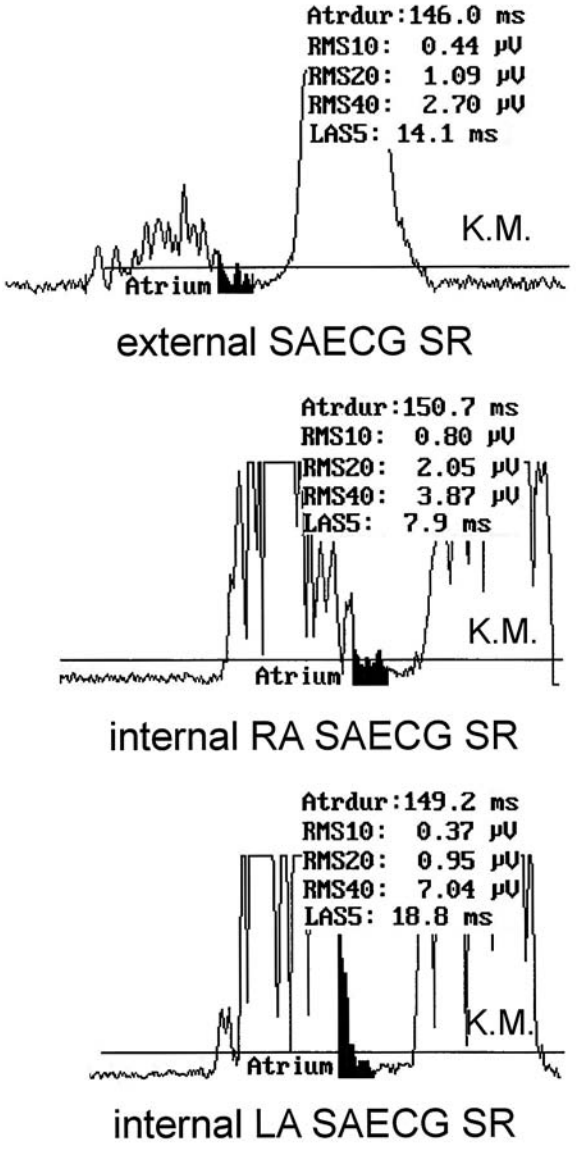

Figure 3. Signal-averaged electrogram obtained from external and intraatrial leads in the same patient.

Bioethical Committee approval

The study was accepted by the Bioethical Committee at the Medical University of Lublin (approval no KE-0254/70/2003).

Results

The results are presented in Table 1.

The average P wave duration was 158 ms at SR and 152 ms at CS pacing. There was a change in atrial activation, assessed by interatrial conduction time (104 *vs*. 115 ms), with no apparent change in TAAT (182 *vs*. 179 ms), SA-ECG P wave duration (156 *vs*. 161 ms) or SA-IEGM A wave duration in RA (175 *vs*. 183 ms). There was, however, a significant shortening of SA-IEGM A wave duration in the left atrium (175 ms *vs*. 143 ms) at CS pacing. There was no clear influence of this pacing mode upon the RMS20 and LAS5 values. Table 2 demonstrates the statistical evaluation of the above data.

Coronary sinus pacing had no significant influence upon the analyzed parameters except the SA-IEGM A wave duration, which was radically shorter (–32 ms) at CS pacing. Although the differences in RMS20 values were not significant, they tended to be higher for CS pacing in external (+1.1 μV) and internal leads in the right (+0.5 μV) and left (+0.4 μV) atrium. The LAS5 values were slightly shorter in external and left-atrial leads at CS pacing. The results suggest that CS pacing has no negative influence on atrial activation synchrony, which is consistent with clinical observations (Fig. 4).

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 P wave (RMS20) and the duration of low amplitude signal < 5 μV (LAS5). Short Pdur and LAS5, with high RMS20, characterize the homogenous atrial activation [24–29]. The coincident abnormal values of Pdur and RMS20 lead to detection of the atrial late potentials (ALP) — a widely accepted predictor of atrial arrhythmias. Table 3 presents the rate of ALP criteria occurrence in the external and intraatrial leads at SR and CS pacing.

ALP 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. CS pacing drastically reduced the occurrence of ALP criteria in left atrial leads, which confirms that although CS pacing does not prolong the atrial potential it still fails to restore the homogeneity of activation in the right atrium in patients with interatrial conduction disturbances.

Recently we described the possibility of recording signal-averaged intracardiac electrograms (SA-IEGM) separately from the right and left atrium with the use of standard signal-averaging equipment designed to perform external SA-ECG analysis [42–44]. The described technique allowed us to record an additional 20 ms of atrial activation, omitted in external SA-ECG, confirming its value as a diagnostic and scientific tool. We observed less homogenous extinction of atrial activation in the right atrium in the group of patients with recurrent atrial arrhythmias [42]. Therefore, the next step was to compare the SA-ECG/IEGM parameters from external and intraatrial leads during CS pacing. The differences between parameters obtained from the

Table 1. Results. Values of atrial conduction parameters and high gain SA P and A wave time domain during sinus rhythm (SR) and coronary sinus pacing (CSP).

Parameters	Sinus rhythm						Coronary sinus pacing					
	N	Aver.	Median	Min.	Max.	SD	N	Aver.	Median	Min.	Max.	SD
High res. ECG	24	157.7	159.0	125.0	199.0	15.9	24	151.7	145.0	118.0	227.0	25.7
IACT (IEGM)	24	103.7	99.5	55.0	148.0	22.6	24	114.7	108.5	72.0	200.0	32.8
TAAT (IEGM)	24	181.5	182.0	144.0	225.0	22.4	24	179.2	181.0	108.0	260.0	35.7
Ext. SA ECG Pdur	24	156.2	155.4	119.3	186.8	16.8	24	160.7	156.7	112.0	243.4	32.0
Ext. SA ECG RMS20	20	2.17	1.96	1.09	3.69	0.76	24	2.15	2.12	0.83	3.94	0.85
Ext. SA ECG LAS5	24	6.68	5.50	0.00	26.70	7.48	24	10.21	8.65	0.00	31.40	8.02
Int. RA SA Adur	24	174.7	168.8	144.4	224.5	24.1	24	183.3	179.2	136.0	244.9	29.7
Int. RA SA RMS20	23	1.76	1.69	0.75	3.46	0.72	16	1.95	1.82	1.04	3.20	0.69
Int. RA SA LAS5	23	12.90	11.00	0.00	33.00	7.91	16	10.36	9.45	3.10	20.10	4.94
Int. LA SA Adur	23	175.3	169.6	138.2	237.1	26.6	24	143.5	139.9	108.3	204.1	26.1
Int. LA SA RMS20	15	2.33	1.78	0.98	4.81	1.19	22	4.35	2.82	1.36	17.67	4.03
Int. LA SA LAS5	15	9.0	9.40	0.00	18.80	6.17	21	6.36	6.30	0.00	14.10	4.08

Table 2. Interatrial conduction and high gain SA P and A wave parameters during sinus rhythm (SR) and coronary sinus pacing (CSP). Comparison and statistical evaluation.

Parameters	Rhythm	N	Aver.	SE	p
P duration High res. ECG	SR	24	157.7	3.26	0.133
	CSP	24	151.7	5.24	
TAAT (IEGM)	SR	24	181.5	4.58	0.671
	CSP	24	181.5	4.58	
SA Pdur Ext. leads	SR	24	156.2	3.44	0.370
	CSP	24	160.7	6.54	
RMS20 Ext. leads	SR	20	2.17	0.17	0.635
	CSP	20	2.28	0.19	
LAS5 Ext. leads	SR	24	6.68	1.53	0.135
	CSP	24	10.21	1.64	
SA Adur Int. RA leads	SR	24	174.8	4.92	0.103
	CSP	24	183.3	6.06	
RMS20 Int. RA leads	SR	16	1.48	0.11	0.094
	CSP	16	1.95	0.17	
LAS5 Int. RA leads	SR	16	15.42	1.92	0.199
	CSP	16	10.36	1.23	
SA Adur Int. LA leads	SR	23	175.3	5.56	0.000
	CSP	23	142.9	5.53	
RMS20 Int. LA leads	SR	12	2.09	0.27	0.201
	CSP	12	2.51	0.21	
LAS5 Int. LA leads	SR	12	9.82	1.61	0.683
	CSP	12	8.37	1.01	

external and (separately) intraatrial leads are illustrated in Table 4.

The RAA paced SA atrial potential recorded from right-atrial leads was significantly longer (approx. 20 ms) than the external SA P wave recorded from Frank's leads. There was a significant difference between SA-IEGM A wave duration recorded from the right and left atria, which seems to be

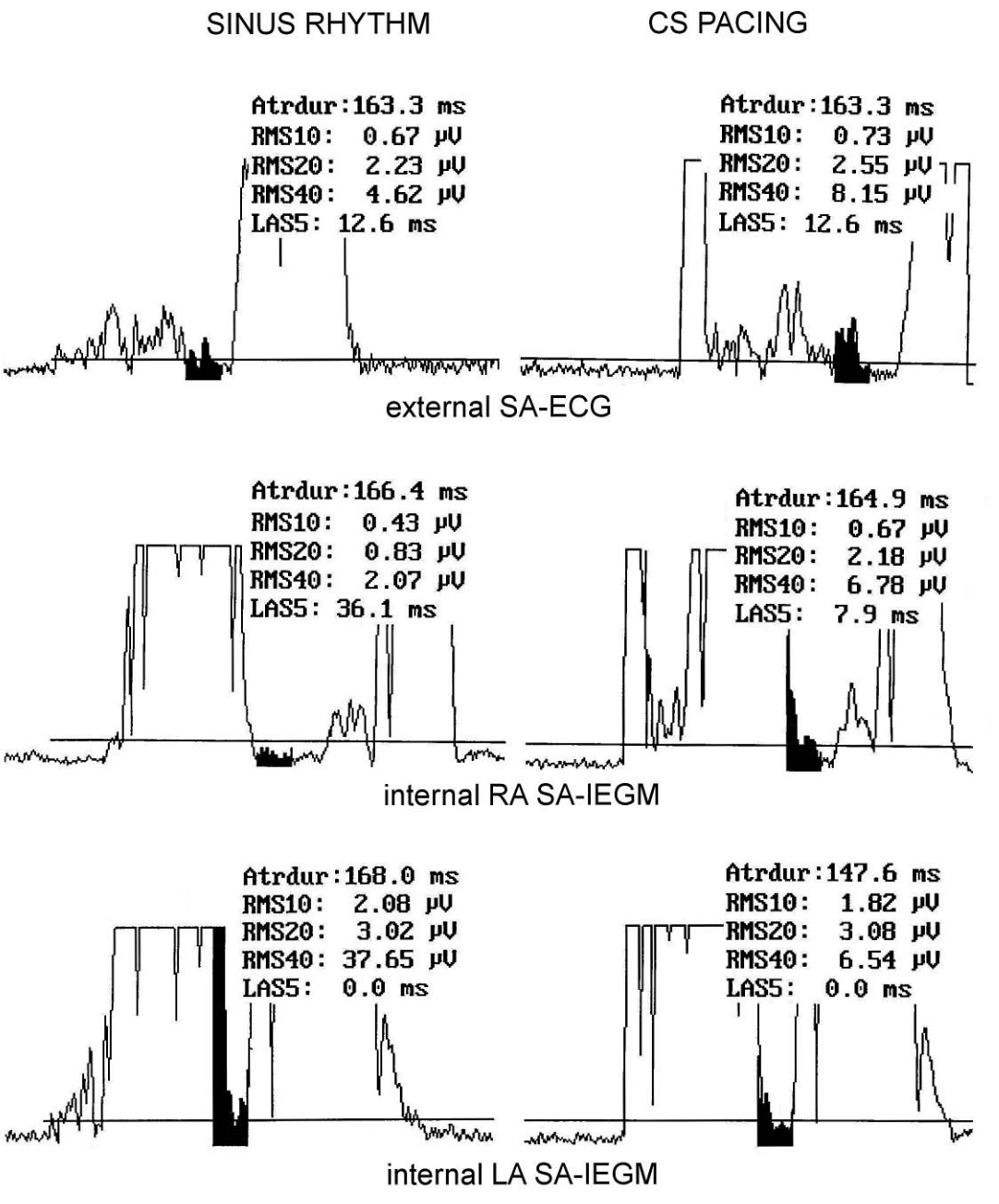

Figure 4. Signal-averaged electrogram obtained from external and intraatrial leads during sinus rhythm and coronary sinus pacing.

a specific effect of CS pacing (the depolarization wave spreads through the posterior atrial wall). The RMS20 values were significantly higher in LA SA-IEGM compared to RA and external leads; the LASA5 values were significantly lower in LA compared to external leads. This may suggest more homogenous termination of atrial activation in the left atrium; on the other hand it confirms the limitations of external SA-ECG.

There was a strong correlation ($r = 0.83$) between CS-paced P wave duration in external and right-atrial leads; however, the latter was 22 ms shorter (Table 5). Interestingly, there was no correlation between the duration of right- and left-atrial signal-averaged potential.

There were no correlations between RMS20 values in external and intraatrial leads, as illustrated in Table 6.

There was a correlation between LAS5 values in external and right-atrial leads ($r = 0.6$), as well as between right- and left atrial leads ($r = 0.5$) (Table 7).

Table 3. Presence of ALP criteria during sinus rhythm (SR) and coronary sinus pacing (CSP) in conventional and intraatrial SA ECG/IEGM.

Rhythm	Leads	Ext.	Int. RA	Int. LA
SR	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%
CSP	ALP Yes	21/24 87.5%	22/24 91.7%	9/24 37.5%
	ALP No	3/24 12.5%	2/24 8.3%	15/24 62.5%
	Lack*	0/24 0%	0/24 0%	0/24 0%

*Impossible for evaluation (terminal part of A wave cancelled in V wave)

Discussion

In our previous study, the electrophysiological effects of CS pacing (evaluated by the IEGM obtained via pacemaker telemetry) were dependent upon the pacing configuration (unipolar/bipolar) and its energy. Unipolar CS pacing increased the asynchrony of atrial activation in comparison to sinus rhythm: A-V conduction was prolonged by 17 ms, P wave duration by 9 ms and IACT by 47 ms, with no apparent change in TAAT. Bipolar CS pacing has no influence on AV conduction or P wave duration although it slightly shortened the TAAT value [21, 23]. The most essential observation was the clinical antiarrhythmic effect of this pacing mode compared to RAA pacing [24, 25]. Those results confirmed previous observations by Moss [1–3], Greenberg [4] and ECG morphology analysis carried out by Man [30]. He reported a shorter P wave with prominent negative phase when he paced proximal CS with high amplitude, in comparison with low-energy distal CS pacing. We explained the described "resynchronizing" effect of high-energy bipolar CS pacing by the phenomenon of dual-site pacing (additional anodal pacing from the ring of the lead in contact with the coronary sinus wall). The histological examinations demonstrated that the muscular sleeve surrounding the proximal section of coronary sinus consists of mixed muscular fibres coming from the left atrium, inferior part of the right atrium and interatrial septum [46, 47, 52].

The electrophysiological studies also help to explain the antiarrhythmic effect of CS pacing. In 1975 Leon [32] confirmed the observations published by Moss, that the mid CS pacing is conducted faster to the His bundle in comparison to HRA pacing (93 *vs*. 139 ms). Gitanidou et al. [33] demonstrated that pacing at HRA results in prolonged interatrial conduction compared to CS pacing; moreover, it promotes AF induction. Permanent CS pacing reduced the coupling window for AF induction by right-atrial extrastimuli, as demonstrated by Hill [31]. Papageorgiu et al. [34, 35] emphasized the antiarrhythmic effect of permanent preexcitation of the triangle of Koch by CS pacing, which blocked AF induction by HRA extrastimuli in his study. In 1997 Yu [36] proved that premature extrastimuli from HRA produced a markedly prolonged atrial potential recorded from infero-posterior interatrial septum during HRA pacing in comparison to CS pacing, which prevented AF induction [37]. Comparable results were published by Ng et al. [38]. Wood et al. [39] demonstrated (on isolated rabbit hearts) that left atrial pacing decreased the dispersion of atrial repolarization; the normalization of effective atrial refractory period by CS pacing in

Table 4. The comparison of right atrial appendage (RAA) paced atrial potential duration and its RMS20 and LAS5 in recordings obtained from external (Frank's) and intraatrial leads.

Examined parameters	N	Leads	Average	SE	Analysis of variance	LSD test Groups	Compa -rison	Aver. diff.	SD of diff.	p
SA ECG/IEGM	24	Extern.	160.7	6.54	F = 1032.8	1	1 *vs*. 2	–22.6	5.6	0.000
P&A wave duration	24	Int. RA	183.3	6.06	P = 0.000	2	1 *vs*. 3	17.2	5.6	0.003
	24	Int. LA	143.5	5.33		3	2 *vs*. 3	39.8	5.6	0.000
SA ECG/IEGM	16	Extern.	2.09	0.23	F = 56.115	1	1 *vs*. 2	0.14	0.6	0.814
P&A RMS20	16	Int. RA	1.95	0.17	P = 0.000	2	1 *vs*. 3	–1.5	0.6	0.016
	16	Int. LA	3.58	0.78		3	2 *vs*. 3	–1.6	0.6	0.009
SA ECG/IEGM	16	Extern.	12.16	2.12	F = 68.610	1	1 *vs*. 2	1.8	1.7	0.291
P&A LAS5	16	Int. RA	10.36	1.23	P = 0.000	2	1 *vs*. 3	5.2	1.7	0.004
	16	Int. LA	6.96	1.00		3	2 *vs*. 3	3.4	1.7	0.051

Table 5. Examination of mutual correlations between high gain SA P duration obtained from external (conventional) leads and A duration measured from RA and LA intraatrial recordings during coronary sinus pacing (CSP).

Variables (x, y) during CSP	N	Average	SD	*r* (X, Y)*	t	p
Ext. SA Pdur	24	160.7	32.0	0.84	7.253	0.000
Int. SA RA Adur		183.3	29.7			
Ext. SA Pdur	24	160.7	32.0	0.48	2.593	0.016
Int. SA LA Adur		143.5	26.1			
Int. SA RA Adur	24	183.3	29.7	0.34	1.679	0.107
Int. SA LA Adur		143.5	26.1			

*Pearson's *r* test

Table 6. Examination of mutual correlations between high gain SA P RMS20 obtained from external (conventional) leads and A duration measured from RA and LA intraatrial recordings during coronary sinus pacing (CSP).

Variables (x, y) during CSP	N	Average	SD	*r* (X, Y)*	t	p
Ext. RMS20	16	2.09	0.91	0.62	2.954	0.010
Int. RA RMS20		1.95	0.69			
Ext. RMS20	22	2.19	0.87	0.40	1.936	0.078
Int. LA RMS20		4.35	4.03			
Int. RA RMS20	16	1.95	0.69	0.38	1.555	0.142
Int. LA RMS20		3.58	3.11			

*Pearson's *r* test

Table 7. Examination of mutual correlations between SA P LAS5 obtained from external (conventional) leads and A duration measured from RA and LA intraatrial recordings during coronary sinus pacing (CSP).

Variables (x, y) during CSP	N	Average	SD	*r* (X, Y)*	t	p
Ext. LAS5	16	12.16	8.48	0.61	2.877	0.012
Int. RA LAS5		10.36	4.94			
Ext. LAS5	21	10.54	8.24	0.32	1.474	0.157
Int. LA LAS5		6.35	4.09			
Int. RA LAS5	16	10.36	4.94	0.56	2.527	0.024
Int. LA LAS5		6.96	4.02			

*Pearson's *r* test

patients with recurrent AF was demonstrated by Ishimatsu et al. [40]. Conversely, in the paper published by Gilligan et al. [41], CS pacing shortened the P wave duration only in healthy individuals but not in patients with recurrent atrial arrhythmias.

In the spotlight of the presented literature [1–4, 30–39] and our own experience we postulated the broad use of permanent CS pacing in patients with brady-tachycardia syndrome [24, 25]. We continued the studies on the electrophysiological effects of this pacing mode with the use of signal-averaged ECG technique [26, 27]. We demonstrated that although unipolar mid CS pacing does not change significantly the signal-averaged P wave duration (in comparison to sinus rhythm), it significantly decreases P wave dispersion (22 *vs*. 15 ms), increases RMS20 value (2.1 *vs*. 2.3 ms) and modifies (91% *vs*. 73%) ALP occurrence [26]. In the next study we demonstrated that CS pacing significantly shortens signal-averaged P wave duration, increases RMS20 value (2.0 *vs*. 2.3 ms), shortens LAS5 (15 *vs*. 10 ms) and reduces (75% *vs*. 50%) ALP occurrence in comparison to RAA pacing [27]. There was no possibility to assess the effect of bipolar CS pacing since the study was

performed in a group of patients implanted with biatrial pacing systems based on "Y" connectors [48].

The present study, derived from intraatrial averaged signal analysis, demonstrated that CS pacing did not change, in comparison to the sinus rhythm, the parameters (P_{II}, TAAT, external and right-atrial P/A wave), reflecting the duration of atrial activation, except the SA-IEGM A wave in left atrium, which was significantly shorter. CS pacing increased (but not significantly) the RMS20 both in external and intraatrial leads and to some extent decreased LAS5 values in intraatrial leads, which may confirm its favourable effect on the homogeneity of atrial activation. Moreover, CS pacing reduced the occurrence of ALP criteria in left-atrial leads with no apparent change in right-atrial and external leads. There was a significant difference between SA-IEGM A wave duration and its RMS20 values recorded from the right and left atria, which seems to be a specific effect of CS pacing: increased homogeneity of activation in left atrium with little influence on the right atrium.

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 [49, 50], none of them concentrating on the effects of CS pacing. Keane et al. [49] analyzed signal-averaged P wave time- and frequency-domain parameters at sinus rhythm and RAA pacing, which caused prolongation of the SA P wave duration with no significant differences in the RMS20. Yamada et al. [50] published the results of RAA and Bachmann's bundle (BB) pacing, assessed with the use of SA-ECG time-domain parameters. In his study BB pacing reduced (in comparison to SR) SA P wave duration and increased RMS20; RAA pacing prolonged the SA P wave, with no influence on RMS20 — the authors emphasized the arrhythmogenic effect of RAA pacing. The presented literature and our data advocate the choice of an alternative atrial pacing site in patients with interatrial conduction disturbances and recurrent atrial arrhythmias — coronary sinus pacing may be a reasonable choice [53].

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, frequently observed in the studied group. The applied "left atrial" lead system is not purely left atrial — introduction of multipolar catheter to the left atrium via transseptal 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 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 the selected group of patients with recurrent atrial arrhythmia and considerable conduction disturbances within the atria; thus, our conclusions must not be applied to the general population of patients with paroxysmal atrial fibrillation.

Conclusions

1. Coronary sinus pacing, in comparison to the sinus rhythm, does not change significantly the P wave duration, TAAT, SA-ECG Pdur or SA-IEGM RA Adur (except for significant shortening of the LA Adur), leading to a general assumption that CS pacing does not prolong the atrial activation.
2. Coronary sinus pacing increases (not significantly) the RMS20, both in external and intraatrial leads, and to some extent decreases LAS5 values in intraatrial leads, which suggests that this pacing mode does not increase the asynchrony of atrial activation in comparison to the sinus rhythm, which corresponds with the observed clinical antiarrhythmic effect.
3. Coronary sinus pacing reduced the occurrence of ALP criteria in left-atrial leads, with no evident change in right-atrial and external leads, which confirms that although CS pacing does not prolong the atrial activation, it fails to eliminate the right-atrial ALP criteria in patients with interatrial conduction disturbances and recurrent atrial arrhythmias.
4. The duration of SA-IEGM A wave recorded from the right atrium was significantly longer in comparison to the external leads. Moreover, there was a significant difference between SA-IEGM A wave duration recorded from the right and left atria, which seems to be a specific effect of CS pacing.
5. The CS-paced RMS20 values were significantly higher in LA SA-IEGM in comparison to RA and external leads, which suggests less homogenous termination of atrial activation in the right atrium; then again, it confirms the limitations of external SA-ECG.

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