Electrophysiologic and Clinical Aspects of Permanent Biatrial and Lone Atrial Pacing Using a Standard DDD Pacemaker

A. KUTARSKI, K. OLESZCZAK, M. WÓJCIK, K. POLESZAK, T. WIDOMSKA-CZEKAJSKA
Department of Cardiology, University Medical Academy, Lublin, Poland

Summary

A biatrial pacing system was implanted in 47 patients who had recurrent arrhythmias and interatrial conduction disturbances (IACD). The right atrial appendage (RAA) lead was usually connected to the atrial port, and the coronary sinus (CS) lead, to the ventricular port of a DDD pacemaker (Dromos or Physios, BIOTRONIK). During examinations, RAA (AAI programming), CS (VVI programming), and biatrial (DDD programming with an AV delay of 5 ms) pacing were applied. The latter was then left as the permanent setting. Sinus rhythm (SR): RAA pacing offered the best sensing conditions in the bipolar (BP) configuration (A-wave amplitude: 4 mV, atrioventricular (A/V) ratio: 14.4). Unipolar (UP) and BP CS sensing showed satisfactory A-wave amplitudes (2.6 to 3.2 mV), but the ventricular amplitude was similar, and the AV ratio was about 1. The excitation front was sensed in the right atrium (UP and BP configurations) 40 ms after P-wave onset; in the proximal part of the CS after 90 ms (BP configuration), and in the mid part of the CS (tip sensing) 100 ~ 110 ms after P-wave onset. Left atrial A-wave duration was the shortest in the RAA (80 ms) and the longest in the CS in the BP configuration (110 ms). RAA pacing prolonged AV conduction to up to 230 ms (in comparison to SR with an AV delay of 110 ms). P duration (165 ms vs. 150 ms during SR), intraatrial conduction (140 ms vs. 100 during SR), and total atrial activation time (TAAT) (220 vs. 195 ms during SR) were all prolonged. Lone CS pacing at the pacing threshold did not influence AV-conduction time and TAAT; the values were similar to those obtained during SR). High-energy BP CS pacing shortened AV conduction (by 20 ms), P duration (by 10 ms), and TAAT (by 10 ms) in comparison to SR. The differences were very clear and significant when comparing the effects of RAA and CS pacing. BiA pacing showed the same influence on AV conduction but a normalized P-wave duration (120 ms vs. 150 ms during SR) and TAAT (140 ms vs. 195 ms during SR and 225 ms during RAA pacing). Intracardiac electrocardiograms (IEGM) obtained with the BiA pacing system via telemetry were very useful for a more precise diagnosis of atrial arrhythmias. In the majority of patients, atrial flutter could be stopped by a burst of CS pacing.

Key Words

Biatrial pacing, right atrial pacing, coronary sinus pacing, electrophysiologic effects

Introduction

Classical right atrial (RA) based pacing modes have proved to be effective in preventing atrial arrhythmias (with or without drugs) in most patients [1-2]. Excellent results can be obtained in patients with bradycardia and/or pause-dependent atrial fibrillation (AF) and vagally mediated AF [2-3]. However, in some patients, RA pacing can increase the frequency of recurring arrhythmia [4-6], especially if the RA screw-in lead was implanted into the free RA wall [4-5]. In patients with single-lead VDD pacing systems, episodes of AF were significantly less common [5-6]. It was hypothesized that the mechanical effect produced by the contact of the RA electrode tip with the atrial wall was the reason for this phenomenon [6].

More and more, inter/intra atrial conduction disturbances (IACD) are recognized for the role they play as substrates for re-entrant atrial arrhythmias [7-9]. Resynchronizing atrial pacing modes (biatrial, dual-site RA, Bachman's bundle, and septal pacing) have created new therapeutic options for patients suffering

from such atrial arrhythmias [10-12]. Electrophysiologic (EP) effects of different atrial pacing modes in patients with IACD were evaluated during electrophysiologic studies (EPS) [13-25]. All reports pointed to unfavorable EP effects of RAA pacing in comparison to sinus rhythm (SR), pacing of the proximal part or the ostium of the coronary sinus (CS), and septal pacing [13-23]. The most popular permanent biatrial (BiA) pacing systems (split bipolar (BP) with the cathode in the right atrium, or dual cathodal unipolar (UP) pacing) do not permit unifocal UP and BP CS pacing, and EP effects of different permanent atrial pacing modes have not been compared.

We decided to implant resynchronizing systems as described in 1988 by Markewitz and Osterholzer [26-27]. The authors showed the usefulness of standard DDD pacemakers (with the shortest possible AV delay programmed) for resynchronizing donor and recipient atria in patients after orthotopic heart transplantation and sinus node insufficiency.

From September 1996 to September 1997, we implanted the Markewitz and Osterholzer resynchronizing BiA pacing system in a group of 47 patients. So far, the results have been promising [20][22][28].

The purpose of this study is to present our experience with clinical and EP aspects of BiA pacing, as well as lone right and left atrial pacing, using standard DDD pacemakers.

Material and Methods

Patient selection

The following inclusion criteria were applied:

I. Accepted indications for permanent pacing due to brady-tachy syndrome.
II. Evidence of IACD: a PII duration of more than 125 ms, either during sinus rhythm (29 patients) or during pacing (18 patients).
III. Drug-resistant symptomatic atrial arrhythmia (flutter or fibrillation) recurring more than once per month. Eighteen of 47 patients were previously permanently paced from the right atrium (15 patients) or the CS (3 patients), and in all of them single-site atrial pacing significantly increased IACD.
IV. No history of AV conduction disturbances and a Wenckebach point of over 130 bpm.

Atrial arrhythmias

In 33 patients, AF usually was recorded in the ECG before DC cardioversion; 14 patients suffered from typical atrial flutter (AFl). In most of them, an analysis of Holter monitoring records showed the presence of short episodes of AFl.

Pacing systems

Standard "J"-shaped BP leads, classically implanted in the right atrial appendage, were usually connected (in 37 patients) to the BP atrial port of a standard DDD pacemaker. Standard straight ("ventricular") BP leads were implanted in a middle position in the CS, and one to three tines were removed [21][28-30] for better contact of the electrode tip with the CS wall (33 patients); or we used specially designed BIOTRONIK leads (14 patients).

The CS lead was usually connected (in 37 patients) to the ventricular BP port of the same pacemaker (Dromos DR and Physios (01 or TC 01), BIOTRONIK). In 10 patients with predominantly left-atrial (LA) arrhythmias, we used inverted connections. This pacing system allows excellent, simultaneous RA and LA sensing (in the atrial and ventricular channel) in UP and BP configuration, as well as during sinus

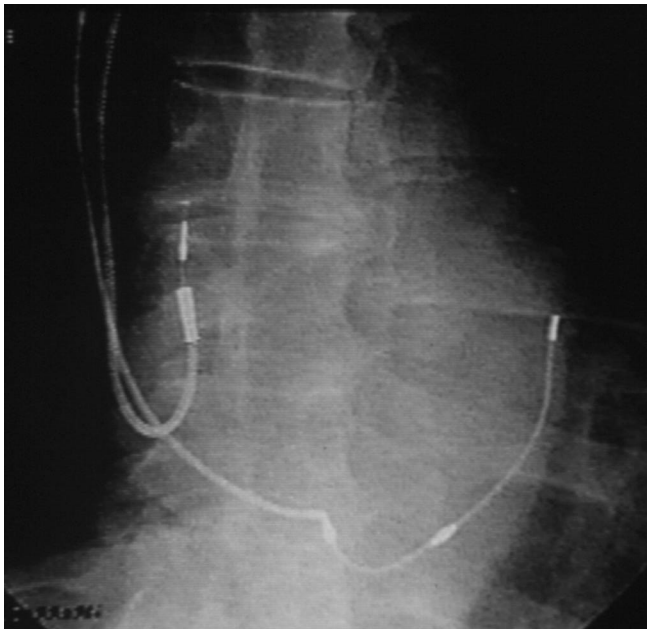


Figure 1. X-ray of the BiA pacing system. A standard J-shaped BP lead located in the RAA and the specially-designed CS lead in the CS. The distant anchoring part of the lead located in the cardiac vein only has only the purpose of ensure stable positioning. Both rings of the lead are designed for LA sensing/pacing in UP (distal ring) and BP (proximal ring as anode) configuration.

rhythm. It also enables lone RA or CS pacing (in UP and BP configuration), as well as simultaneous pacing of both atria. Excellent filtered intracardiac electrograms (IEGM), recorded simultaneously with a standard ECG lead, allowed for an evaluation of IACD parameters.

Pacemakers

For the simultaneous pacing of both atria with standard DDD pacemakers, we selected Dromos DR and Physios (01 or TC 01) devices, mainly because they offer the option of programming an ultra-short AV delay (15 ms) and excellent telemetry possibilities including filtered IEGM transmission.

Follow-up

Acute pacing and sensing conditions after final lead fixation were evaluated using the standard BIOTRONIK pacing threshold analyzer ERA 300.

Intraoperatively, during implantation of the atrial leads, we recorded IEGMs on paper for a "manual" measurement of AV duration, atrial (A)- and ventricular (V)-wave amplitudes, and, most importantly, calculation of the A/V ratio. The latter parameter seems to be more important than an automatic measurement of lone A and V amplitudes.

Pacing/sensing conditions during the short-term and long-term postoperative period were controlled via telemetry using the PMS 1000 programmer (BIOTRONIK). The first examination was performed during the patient's hospital stay, 3 to 5 days after the operation; the following controls were performed monthly up to 6 months after the operation. During follow-up, the patients were questioned about the exact number of arrhythmia recurrences and the mode of restoration of sinus (paced) rhythm, unknown heart palpitations (which were verified using pacemaker Holter recordings), and antiarrhythmic therapy.

Non-invasive atrial EPS

During follow-up, we evaluated the following for SR and RAA, CS, and BiA pacing (in UP and BP configurations):

I. The interval between PII onset (or atrial spike) and the onset of the QRS complex (P/S-QRS interval).
II. The sinus and paced PII or PIII duration.
III. The time from PII onset or atrial spike to the onset of the A-wave in the opposite atrium. In the BP LA sensing configuration, this parameter expresses mainly the intraatrial conduction time as long as care was taken to fixate the CS electrode tip in the middle of the CS and to locate the electrode ring near the CS ostium.
IV. The triggered response time: the time from PII onset to the RA and LA triggered spike. This parameter expresses the time from the onset of RAA activation to the appearance of an A-wave amplitude sufficient to be sensed by the pacemaker; its values are 5 to 10 ms longer than the spike of A-wave onset.
V. The total atrial activation time (TAAT): the time from the onset of P (or the atrial spike) to the end of the A-wave in the opposite (later activated) atrium. The CS electrode tip, which senses the potential, is located in the low posterior part of the left atrium, and the results are similar to (or a few ms less than) those obtained with esophageal LA lead location. Differences were not significant. In some

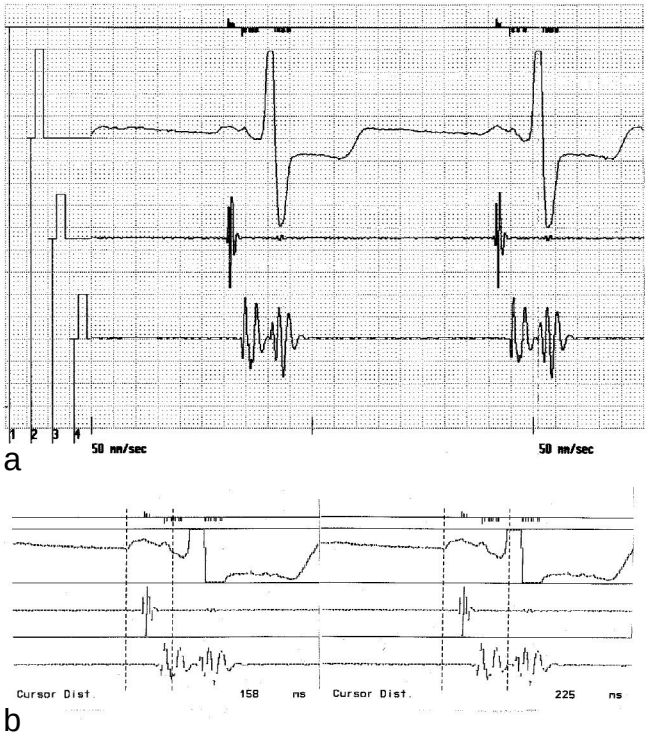


Figure 2. Measurements of atrial timing from IEGMs of the BiA pacing system. Atrial timing (of the right and left atrium) can be measured on a paper printout (a) or directly on the screen using cursors (b). a) PII duration: 160 ms; PII-onset RA: 60 ms; RA duration: 55 ms; PII-onset LA: 120 ms; LA duration: 120 ms; PII-end LA (TAAT): 220 ms. b) Cursors show PII duration (158 ms) and TAAT duration (225 ms).

Inter/intra atrial conduction parameters			Acute (implantation) values from ERA 300 printout			Subacute - after implantation, before discharge IEGM obtained with telemetry				AAT (VVT) pacing program
			A-V duration (ms)	A Ampl (mV)	A/V ratio	A duration (ms)	P_{II}-onset A (ms)	TAAT (ms)	A(RA)-A(CS) (ms)	P_{II}-spike (ms)
RAA	UP	mean		3.2	2.0		31.4			38.9
		Sd		1.4	1.1		13.4			16.7
	BP	mean	190.8	4.1	14.4	76.3	31.0			38.0
		Sd	26.7	0.9	11.4	23.5	15.7			11.4
CS	UP	mean	98.4	2.6	0.9	92.0	110.0	196.0	89.2	125.6
		Sd	29.3	1.2	0.7	24.9	25.2	25.3	26.1	20.1
	BP	mean	110.0	3.2	1.1	112.1	93.7	194.5	81.3	116.1
		Sd	30.4	1.4	0.7	21.6	24.2	27.1	28.8	26.7

Table 1. Acute and subacute intra and interatrial conduction parameters on sinus rhythm.

of the patients, the non-invasive atrial EPS was repeated using different pacing energies and frequencies.

Table 1 presents acute and subacute atrial sensing and timing parameters during SR recorded via telemetry from permanently implanted BiA pacing systems.

The table shows that BP RAA sensing offered the best sensing conditions (mean A-wave amplitude: 4.1 mV, A/V ratio: 14.4). The worst, but still acceptable, sensing conditions were found during UP LA sensing (2.6 and 0.9 mV, respectively). The time from A-wave onset to V-wave onset (AV duration), recorded from IEGMs during the implantation of both leads, indicated that the left atrium was activated 80 to 90 ms later than the RAA during SR. The duration of the LA A-wave (recorded via telemetry) was longer than that of the RA A wave (112 and 76 ms, respectively). The polarity of RA sensing (UP and BP) had no influence on the interval between PII and ARA onset; the RA excitation front was sensed 31 ms after PII wave onset in the RA channel. The pacemaker channel connected to the CS lead sensed the excitation front more than 10 ms earlier in BP configuration (ring sensing) than in UP configuration (tip sensing). Average and median values of the TAAT were similar in both sensing configurations. Furthermore, the pacemaker spike during atrial-triggered pacing points exactly to the moment when the excitation front is sensed, and those examinations confirmed the previous results. Right atrial appendage excitation was sensed nearly 40 ms after the onset of the PII wave (in both configurations). This front was sensed earlier in the proximal part of the CS (BP sensing configuration, ring sensing) than in its middle part (UP configuration, tip sensing) (116 and 126 ms, respectively, after PII-wave onset).

Table 2 presents some EP effects recorded during RA, LA, and BiA pacing, and the respective values recorded for comparison during SR.

The table indicates that RAA pacing significantly prolongs the atrial spike-QRS (S-QII) duration in comparison to SR (230 vs. 200 ms, respectively). This effect was not noted during BiA pacing (200 ms). Right atri-

Place and polarity of pacing*			S-Q_{II} interval (ms)	P_{II} duration (ms)	S-onset of opposite atrium (ms)	TAAT (S-end of opposite atrium (ms)
RA	UP	mean	234.4	165.0	141.5	226.9
		Sd	40.0	20.3	32.3	32.2
	BP	mean	227.9	161.7	136.8	216.9
		Sd	30.8	16.4	34.4	26.4
CS	UP	mean	215.9	154.8	149.9	197.6
		Sd	47.4	29.1	29.5	38.0
	BP	mean	196.4	147.7	131.3	187.3
		Sd	41.6	30.3	35.9	41.1
BIA	UP	mean	215.1	130.4		139.6
		Sd	37.8	19.5		30.0
	BP	mean	200.0	123.8		140.0
		Sd	49.0	19.0		26.8

* pacing with moderate frequency (+/- 10 bpm over sinus rhythm) and moderate energy (+/- 50% over pacing threshold)

Sinus rhythm		P-Q_{II} (ms)	P_{II} dur. (ms)	P-onset of LA (ms)	TAAT end of LA (ms)
	mean	197.1	151.9	92.0	194.5
	Sd	38.0	31.3	24.9	27.1

Table 2. Electrophysiologic effects of RA, LA and BiA pacing (moderate frequency and moderate energy).

Place and energy of pacing			P(S)-QRS$_I$ interval (ms)	P$_I$ duration (ms)	P(S)-end of opposite atrium (TAAT) (ms)	P$_{II}$-onset of later activated atrium (BP) (ms)
Sinus rhythm		mean	197.1	151.9	194.5	93.7
		Sd	38.0	31.3	27.1	24.2
RAA pacing (UP)		mean	234.4	165.0	226.9	141.5
		Sd	40.0	20.3	32.2	32.3
	UP	mean	215.9	154.8	197.6	149.9
		Sd	47.4	29.1	38.0	29.5
CS pacing	BP low energy	mean	196.4	147.7	192.8	131.3
		Sd	41.6	30.3	41.3	35.9
	BP high energy	mean	178.4	137.3	180.0	122.6
		Sd	30.1	29.5	36.6	32.4
BiA pacing BP		mean	200.0	123.8	140.0	
		Sd	49.0	19.0	28.8	

Table 3. Pacing energy and electrophysiologic effects of CS pacing.

al pacing similarly prolongs the PII duration as compared to SR (165 vs. 150 ms). Coronary sinus pacing showed no influences on these intervals, but BiA pacing significantly normalized PII duration (120 ms). The time between the pacemaker's atrial spike and the A-wave onset in the opposite atrium (S-onset) and the TAAT (spike-completed A wave in the opposite atrium) were significantly longer during RA pacing (140 and 225 ms) than during SR (92 and 195 ms) and CS pacing (130 and 195 ms). Biatrial pacing effected a normalization of the TAAT (140 ms).

Table 3 presents some EP effects of CS pacing using different polarity (UP and BP configuration) and different energies.

The results presented in Table 3 show that high-energy CS pacing significantly shortened (by over 20 ms) the spike-QRS duration, as well as the PII-wave duration (by over 10 ms), the TAAT, and the interatrial conduction time (by over 10 ms). These results indicate some resynchronizing effect of CS pacing if higher values of energy are applied. This slight resynchronizing effect is more visible when compared to RAA pacing: S-QRS 180 vs. 197 ms, PII duration 137 vs. 152 ms, and TAAT 180 vs. 195 ms.

Table 4 presents effects of the atrial pacing rate on interatrial conduction.

The table indicates that the TAAT was slightly longer if the RAA pacing rate was relatively high (246 vs 220 ms). This effect of the pacing rate was less visible during CS pacing (190 vs. 180 ms).

Table 5 presents antiarrhythmic effects of permanent BiA pacing during the first 6 months of follow-up.

Discussion

The very promising results obtained by Daubert's [10] [31] and Saksena's teams [11][32] inspired us to implant BiA pacing systems. Some years ago, "Y" connectors were not available on the market, so we decided to start BiA pacing using DDD pacemakers and the Osterholzer-Markewitz configuration [26-27] (inverted or not). This pacing system permits sensing/pacing of a single atrium (from the atrial or ventricular channel) and also of both atria simultaneously (using DDD programming with an ultra-short AV delay). Programming of the UP or BP configuration was possible, and IEGMs obtained via telemetry enabled the measurement of atrial timing parameters. This BiA pacing system proved to be a very valuable tool for non-invasive atrial EPS, especially for interatrial conduction evaluation.

Because a BiA designed pacemaker is not currently available, standard pacemakers with two different main modes of lead connections must be used to

Atrial pacing mode		Frequency of pacing slow*	med**	fast***
RA BP pacing	mean	220.2	231.4	245.6
	Sd	22.9	24.8	31.1
CS BP pacing	mean	177.9	185.0	188.2
	Sd	20.4	19.3	21.1

* slow: 5-10 bpm over sinus rhythm
** med: median between "slow" and "fast"
*** fast: 10-15 bpm under Wenckebach point or 130 bpm

Table 4. Frequency of pacing and measured total atrial activation time.

Antiarrhythmic effect of BiA pacing			Months of observation					
			1	2	3	4	5	6
		patients	40	36	34	30	31	34
Well working BiA (DDD) pacing system	excellent (no arrhythmias)	no. of patients	24 60.0%	22 61.1%	24 70.6%	19 63.3%	24 77.4%	25 73.5%
	good (significant decrease of number of arrhythmias)	no. of patients	5 12.5%	7 19.4%	5 14.7%	6 20.0%	4 12.9%	4 11.8%
	week (slide decrease, recurr. of arrhythmias)	no. of patients	7 17.5%	7 19.4%	4 11.8%	2 6.7%	1 3.2%	2 5.9%
	no effect (no change of arrhythmia frequency)	no. of patients	3 7.5%	0 0%	1 2.9%	2 6.7%	1 3.2%	2 5.9%
	appearance of chronic AF	no. of patients	1 2.5%	0 0.0%	0 0.0%	1 3.3%	1 3.2%	1 2.9%

Table 5. Antiarrhythmic effect and functionality of BiA pacing system during six-month follow-up.

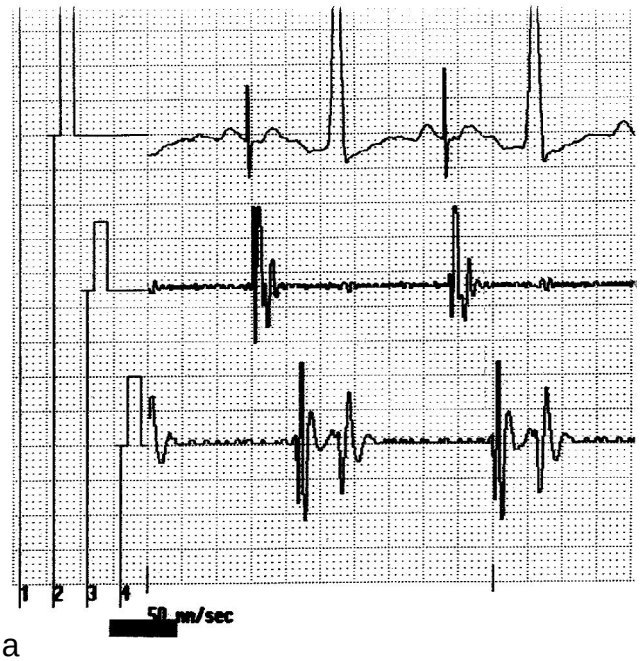
enable successful BiA pacing. The first system was introduced in 1990 in Rennes by Daubert [33] and later by Saksena in New York [34]. In this unique, newly developed pacing system, both atrial leads are connected in a series (split); the cathode is connected to the RA lead, and the anode to the CS lead. The advantages of this pacing configuration are: relatively low energy consumption in spite of moderately high pacing threshold values, and good sensing conditions. Disadvantages of the proposed system are: anodic (potentially pro-arrhythmic) pacing of the left atrium and risk of problems related to high impedance (two electrode tips in the same circuit). The second system, proposed primarily by Cazeau (for permanent BiV pacing), is based on a parallel connection of leads, and the cathodic current is "divided" between two leads via a "Y" connector [35]. The advantages of this pacing configuration are: relatively low pacing thresholds, very low global lead impedance, and avoidance of potential pro-arrhythmic high-energy anodic pacing. The main disadvantage of this lead connection system is a relatively high consumption of energy [31-42]. Neither of these BiA pacing systems permits lone pacing/sensing of the left atrium, and some aspects of permanent atrial and BiA pacing/sensing were not described.

Our present findings and observations can be divided into five groups:

I. AV conduction during different atrial pacing modes
II. Sensing conditions and atrial timing during different atrial pacing modes
III. EP effects of RA pacing
IV. EP effects of CS pacing
V. Clinical, antiarrhythmic, and EP effects of permanent BiA pacing

AV conduction during different atrial pacing modes

It is known that standard RAA pacing prolongs the SQ interval. Unfavorable AV prolongation remains the main limitation of rate-responsive, single-chamber, atrial pacing [43]. It is interesting that the CS was the first location for permanent atrial pacing [44]. Moss was the first to report that the CS paced atrial spike-QRS interval is shorter than the P-QRS interval during SR [45]. We confirmed this observation in our first reports on permanent CS pacing [46]. In our present examination of over 40 patients, we found significantly longer S(P)-QRS intervals during RAA pacing (230 ms) than during CS, BiA pacing and SR (200 ms) (Table 3). These observations confirmed our previously reported suggestion [47-49] that the S-QRS interval during CS pacing becomes shorter (by 20 ms) if higher pacing energies are applied. Our impression (based on over 3 years of experience with more than 150 patients with BiA pacing systems) is that prolonged



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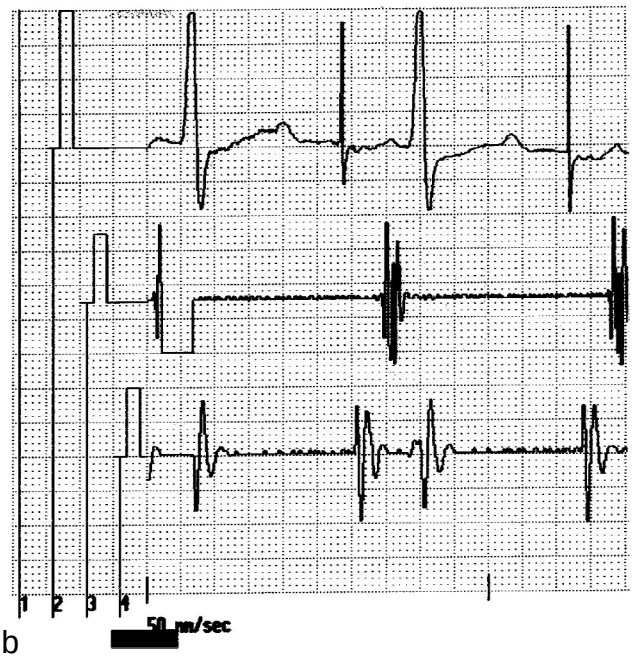

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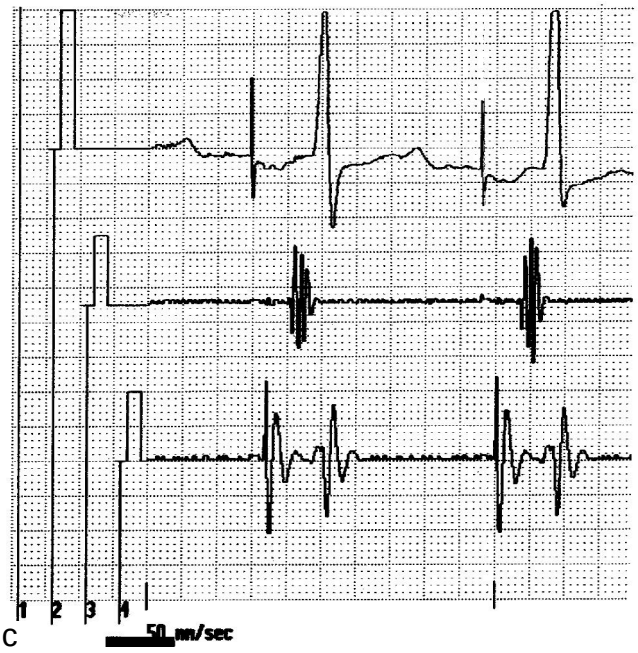

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Figure 3 a-c. EP effects of high-energy CS pacing. A comparison of different atrial pacing modes in the same patient. a) BP RAA pacing: spike-QRS: 225 ms; PII duration: 160 ms; spike-onset CS: 120 ms; spike-end CS (TAAT): 240 ms. b) UP CS pacing (routine energy of about 1.5 times the threshold energy): spike-QRS: 200 ms; PII duration: 160 ms; spike-onset RA: 120 ms; spike-end RA (TAAT): 200 ms. c) BP CS pacing ("high" energy-over 3 times the threshold energy): spike-QRS: 160 ms; PII duration: 120 ms; spike-onset RA: 105 ms; spike-end RA (TAAT): 180 ms. The significant difference between the effects of RAA and BP CS pacing has been shown.

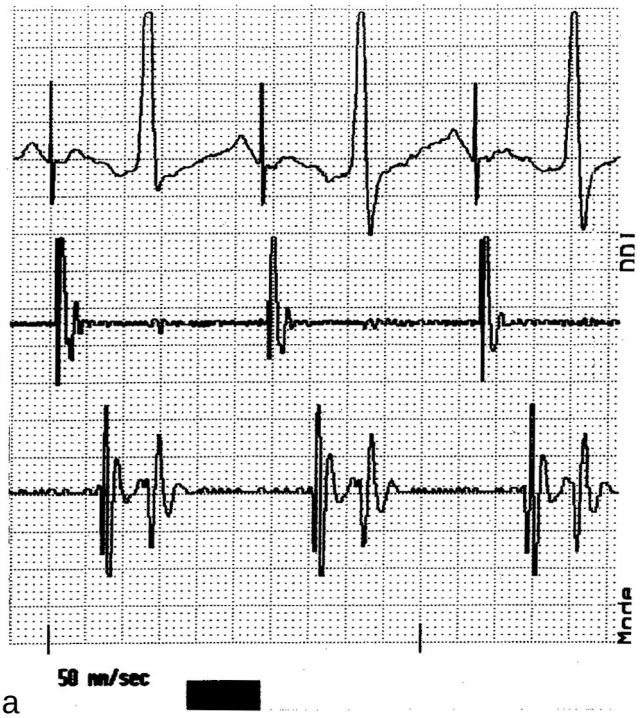
AV conduction (first degree of AV block) is a very rare finding in patients with BiA pacing systems as compared to those with standard RAA pacing systems. This impression will have to be confirmed in future.

Atrial sensing conditions and atrial timing during different atrial pacing modes

Our present observations are in agreement with the general consensus that BP RAA sensing offers the best sensing conditions. With CS sensing, V waves cannot be eliminated even in the BP sensing configuration, due to the anatomical location of the CS (Figures 2-8). It is of great importance for atrial resynchronizing pacing modes that the excitation front during SR reaches the RAA after more than 30 ms and can be sensed by the atrial channel 40 ms after PII onset (AAT pacing findings in Table 1). Our findings seem to confirm the idea that the CS region plays a conductive role [50]. The excitation front is sensed 10 ms earlier by the CS electrode ring (BP sensing) than by the electrode tip (UP sensing). As a result, the CS paced LA A-wave duration sensed in BP configuration is about 10 ms longer (Table 1: AV duration, AAT pacing, and A duration).

Electrophysiologic effects of RA pacing in patients with IACD

The most popular RAA based pacing modes are sufficient to prevent pause- or bradycardia-dependent atrial



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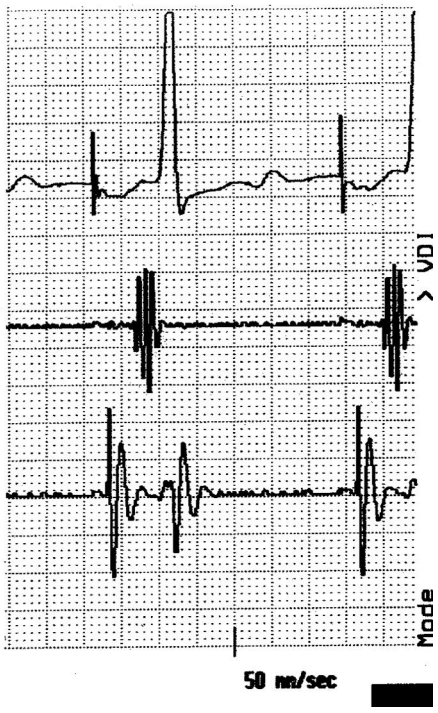

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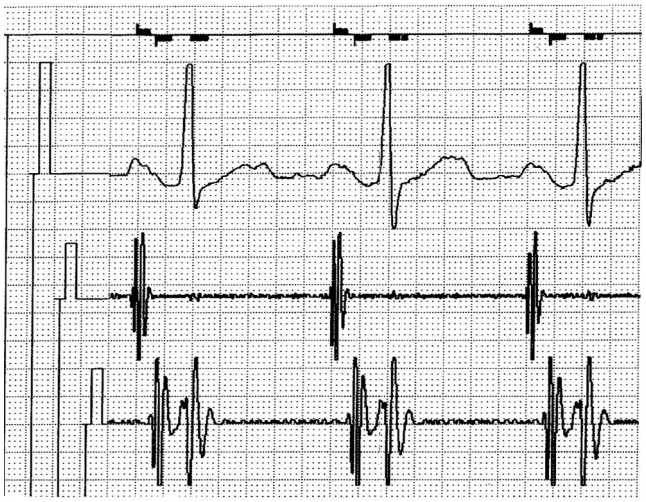
Rate	Atrial pacing mode	Spike-QRS interval	P_{II} duration	TAAT
90 bpm	RAA	220	170	220
90 bpm	CS	160	140	200
110 bpm	RAA	250	180	220
110 bpm	CS	180	140	200
134 bpm	RAA	260	180	220
134 bpm	CS	200	160	200

c

Figure 4 a-c. EP effects of the pacing rate on atrial timing during different atrial pacing modes. a) RAA pacing. b) CS pacing. All atrial timing parameters are longer during RAA than during CS pacing. c) Increased pacing rates prolong mainly the spike-QRS and PII duration. The influence of the pacing rate on the TAAT is less visible in this patient (a precise definition of the end of LA excitation during RAA pacing can be difficult).

arrhythmias, as well as vagal AF episodes. Right atrial overdrive pacing modes (hyperchronotropic sensor response programming, continuous atrial pacing algorithms, etc.) can depress the most common arrhythmia trigger-atrial premature excitations-and prevent "short-long-short" triggered AF [2-3]. On the other hand, it is known that RAA pacing can increase the frequency of arrhythmia episodes in some patients. Even AF recurrences can appear after implantation of a permanent RAA pacing system [4-6]. Such effects were often observed if straight screw-in leads were fixed to the lateral wall of the right atrium. Atrial arrhythmia episodes were less common in patients with single-lead VDD pacing systems [5-6]. These observations were explained as the result of mechanical irritation caused by the physical contact between the electrode tip and RA wall [6]. Recently, unfavorable EP effects of RAA pacing on interatrial conduction were also taken into consideration [13-23]. The biatrial pacing system that we used proved to be very helpful for evaluating EP effects of RAA pacing. The results showed a prolongation of the PII-wave duration by 10 to 20 ms during RAA pacing in comparison with SR. Right atrial appendage pacing prolonged the intraatrial conduction time, expressed as the time between atrial spike and onset of the LA A-wave recorded from the CS electrode ring (BP configuration) located near the CS ostium. During RAA pacing, this time was about 40 ms longer compared to SR (onset PII-onset LA A wave). Similarly, the TAAT was 30 ms longer during RAA pacing than during SR. An increase in the atrial pacing rate was accompanied by an additional prolongation of the TAAT. All findings are comparable to those obtained by other authors during EPS [13-25].



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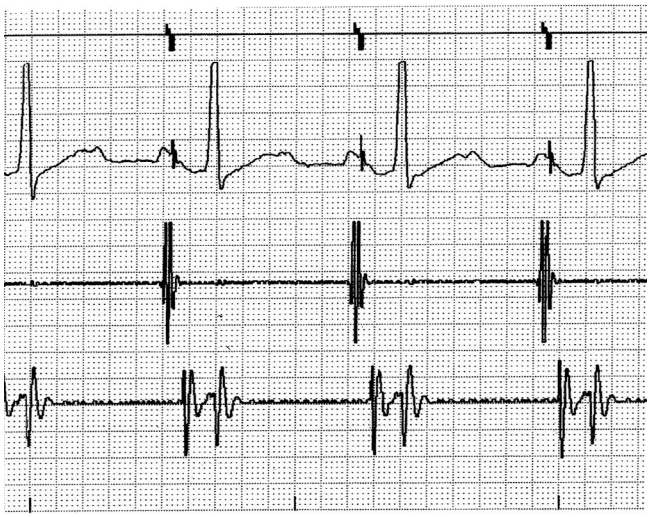

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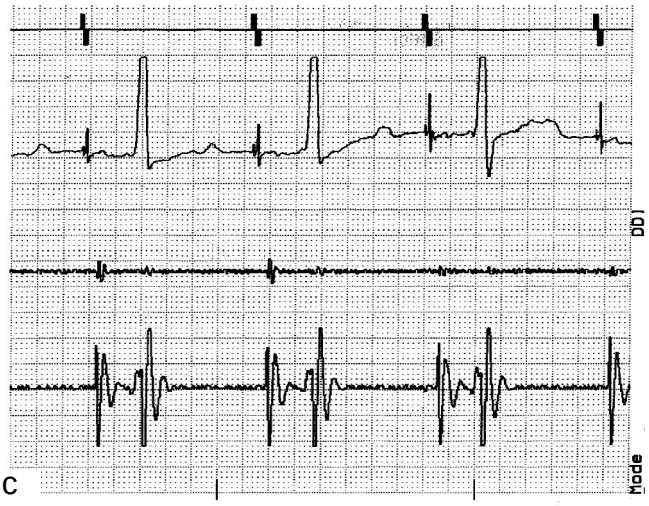


c

Figure 5 a-c. Atrial timing during LA pacing triggered by BiA- and RAA-potentials. a) SR. PII: 150 ms; onset PII-onset RA: 40 ms; TAAT: 220 ms. b) LA-triggered pacing (DDD mode with a programmed AV delay of 15 ms). The A-wave in the RAA is sensed about 45 ms after PII onset (see marker for atrial channel sensing), and the LA pacing pulse (from the ventricular channel) was delivered 60 ms after P-wave onset. The resynchronization obtained in the end is not so excellent. PII duration: 135 ms; TAAT: 180 ms. c) BiA pacing. Pacing of both atria from the atrial and the ventricular channel with an AV delay of 15 ms. TAAT: 160 ms; flat PII - difficult to evaluate.

Electrophysiologic effects of CS pacing in patients with IACD

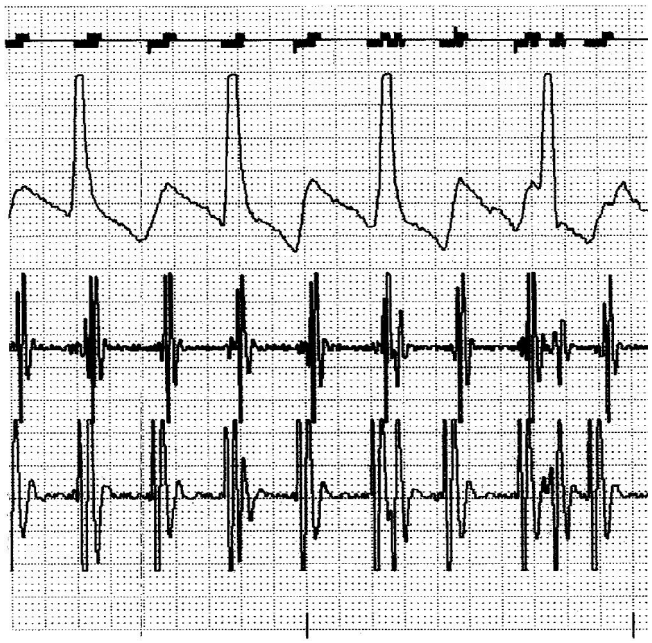
Moss [44] introduced the CS as a single site for atrial pacing in 1968, and many important observations were made in the early 70s. The most important one seems to be Greenberg's findings in 1978 [51]:

I. "in some patients with intermittent atrial fibrillation, CS pacing can provide an atrial impulse that minimizes the tendency to return atrial fibrillation".

II. "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".

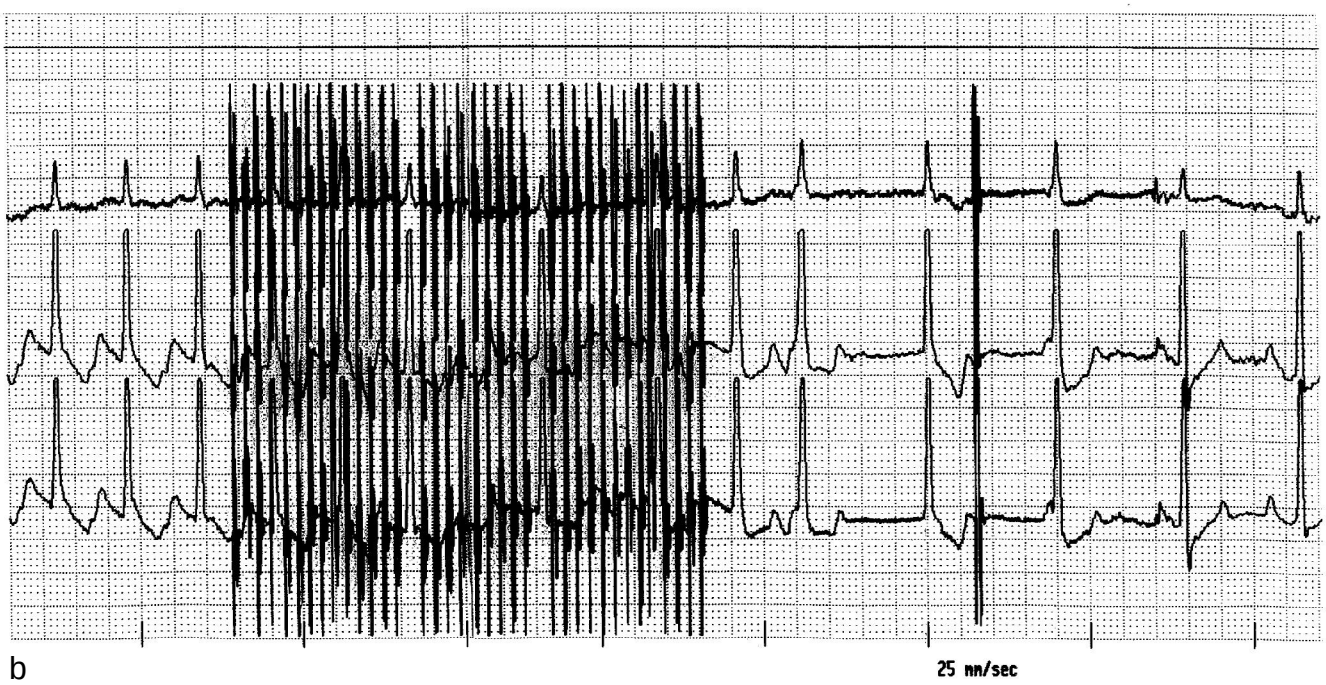
III. "in our experience, patients with prolonged P-R interval, bundle branch block and three-fascicular block and 2° A-V block on atrial pacing below rate of 130 were successfully treated.

The spike R interval tends to remain stable at follow-up. Patients with normal spike intervals tend to remain normal. Those with prolonged intervals tend to remain the same or decrease to normal" [51]. Moss' and Greenberg's observations, forgotten for many years, were confirmed during EPS 20 years later [13-15]. We were surprised to find primarily a suppression of atrial arrhythmia recurrence by BP CS pacing in many patients with severe tachy-brady syndrome, especially if a higher pacing energy was programmed [47-49]. Our observations were confirmed by Papageorgiou [24], who proved that lone pacing of the CS eliminates the propensity of high RA extrasystoles to induce AF because the lower right atrium is activated prior to the upper right atrium during CS pacing. Recent observations by Ng from EPS even indicate that lone CS pacing can be more efficacious than multisite pacing in completely suppressing AF onset [25]. Both authors suggest that continuous CS pacing in patients with



a

[28-30][46-47][49]. During UP (tip) CS pacing, the excitation front reaches the RAA in the same time (over 140 ms) as during opposite way; probably, the most arrhythmogenic low-posterior RA region can be excited earlier. But during lone BP CS pacing, the respective time is 10 ms shorter (ring pacing effect?). A comparison of the TAAT during RAA and CS pacing is most interesting (Table 3). During RAA pacing, the TAAT values were the longest (220 to 230 ms); significantly shorter (by about 30 ms) values of this parameter were obtained during CS pacing (190 to 200 ms). The comparison suggests a more synchronous atrial activation during CS pacing than during RAA pacing (Figures 3 and 4). The observation confirmed our other previous suggestion that "high-energy" BP CS pacing shows no specific slight synchronizing effect [47-49]. Intraatrial conduction time (spike-onset of A-wave in the opposite atrium) and TAAT (spike-end of A-wave in the opposite atrium) were shorter by



b

Figure 6 a-b. Atrial flutter sensed by BiA pacing system. a) Flutter A-waves (260 bpm) are sensed earlier in the proximal CS than in the RAA IEGM. The middle part of the record shows that several additional wave fronts, recorded earlier in the proximal CS, are accompanied by changes in flutter wave morphology in the standard ECG (lead II). b) A CS burst of 820 bpm terminated the arrhythmia. Earlier RAA bursts of 300 to 1000 ppm were ineffective.

paroxysmal AF may reduce the fibrillatory potential of RA extrasystoles. Our present observation confirmed our previous suggestion that there are differences between the effects of BP and UP CS pacing [20-22] 10 to 15 ms or more during CS than during RA pacing (Table 3). Similarly, the pacing rate has a less unfavorable influence on IACT and TAAT during CS than RA pacing.

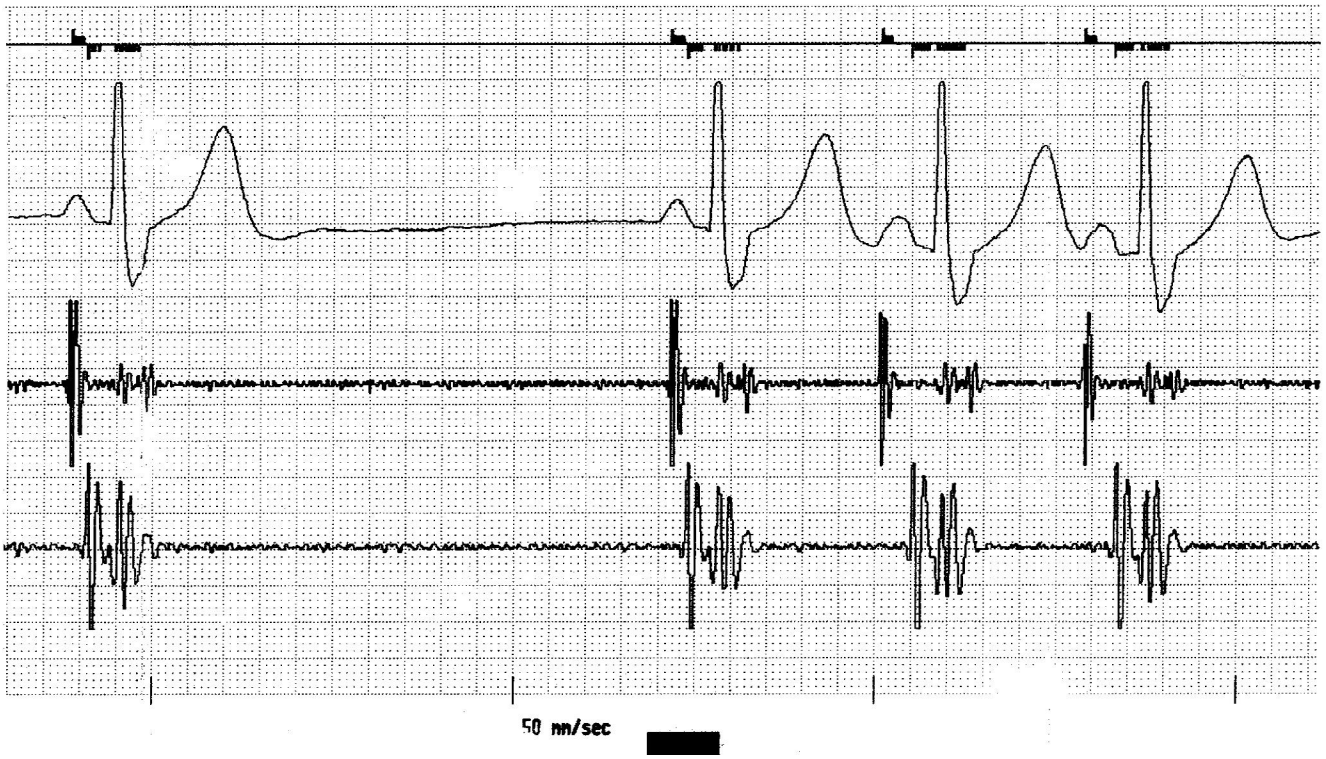


Figure 7. Sinus bradycardia and high RA extrasystoles. During SR (37 bpm): PII: 85 ms; onset PII-onset RA: 30 ms; PII-onset CS: 80 ms; RA-CS: 40 ms; TAAT: 140 ms. No signs of IACD are present. During RA extrasystoles (focus located nearly RAA lead): PII: 100 ms; onset PII-onset RA: 20 ms; PII-onset CS: 90 ms; RA-CS: 90 ms; TAAT: 160 ms. RAA extrasystoles increase the asynchrony of atrial excitation, and their EP effects are similar to those observed during RAA pacing (Figures 5 and 6).

Electrophysiologic and clinical effects of permanent BiA pacing using standard DDD pacemakers

Theoretically, BiA pacing should offer simultaneous pacing of both atria during pacing, SR and extrasystoles. A disadvantage of this pacing system is the impossibility of resynchronizing premature LA excitations if the "standard" lead connection was applied. This fact seems to be very important; due to known LA arrhythmias, 10 of 47 patients received an inverted lead configuration. The CS lead was connected to the atrial channel, and the RA lead to the ventricular channel. In another 5 patients, LA extrasystoles were the reason for changing the BiA pacing system to a split bipolar system during the follow-up period. Atrial P-on-T extrasystoles seem to be frequently found in patients with atypical AFl, and co-existence of LA extrasystoles seems to most the important problem in patients with atrial arrhythmias. When interpreting our clinical effects of BiA pacing, it is important to remember that the excitation wave front was sensed with a delay of about 40 ms in the RAA during SR. An additional limitation to "full" resynchronization is the often limited possibility of programming an ultra-short AV delay. Three years ago, Logos (BIOTRONIK) pacemakers (with 0 ms AV delay) were not available on our market, and we had to use devices of the Physios and Dromos family, which offer a minimum AV delay of 15 ms [20][22][28]. As the result, the left atrium was paced with an average delay of 55 ms during SR and high RA extrasystoles. We can only speculate that these wasted 55 ms could have critical significance and could be responsible for the unsatisfying results of BiA pacing in some patients (Figure 5). For proper LA resynchronization during SR, sensing the RA activation as early as possible plays a key role, and the location of the RAA lead is very important. It has to be located in the RAA as high as possible. Unfortunately, in 18 of our 47 patients, the RAA lead was implanted a long time ago when this aspect was not yet taken into consideration during implantation of

the atrial lead. Finally, especially during atrial extrasystoles, the resynchronization that was attained was probably not sufficient to prevent recurrence of atrial reentry. In spite of this, our clinical results are comparable with those presented earlier by Daubert [10][31] and Saksena [11][32]. However, after becoming more experienced with BiA pacing systems we received better results using the modified version of Daubert's pacing configuration [23][29][37][39][40][52].

The value of BiA IEGM recordings for the diagnosis, better understanding, and treatment of atrial arrhythmias

During routine follow-ups, we found a high number of atrial arrhythmias, mainly when BiA pacing was switched off or if the pacemaker was temporarily programmed to RAA pacing. In 7 of 11 patients, episodes of atrial arrhythmia (AFl or AF) occurred during follow-up examinations. Three of these episodes had to be reverted by electrical cardioversion. On the other hand, we interrupted 14 of 21 episodes of paroxysmal or sustained spontaneous episodes of typical AFl using CS burst pacing; earlier RA burst pacing was ineffective in these cases (Figure 6).

We found that in most patients with arrhythmias that are induced by LA extrasystoles, this BiA pacing system does not show significant results. Some examples of atrial arrhythmias are presented in Figures 7 and 8.

Conclusions

1. Right atrial pacing prolongs AV duration and both intraatrial and interatrial conduction times, and

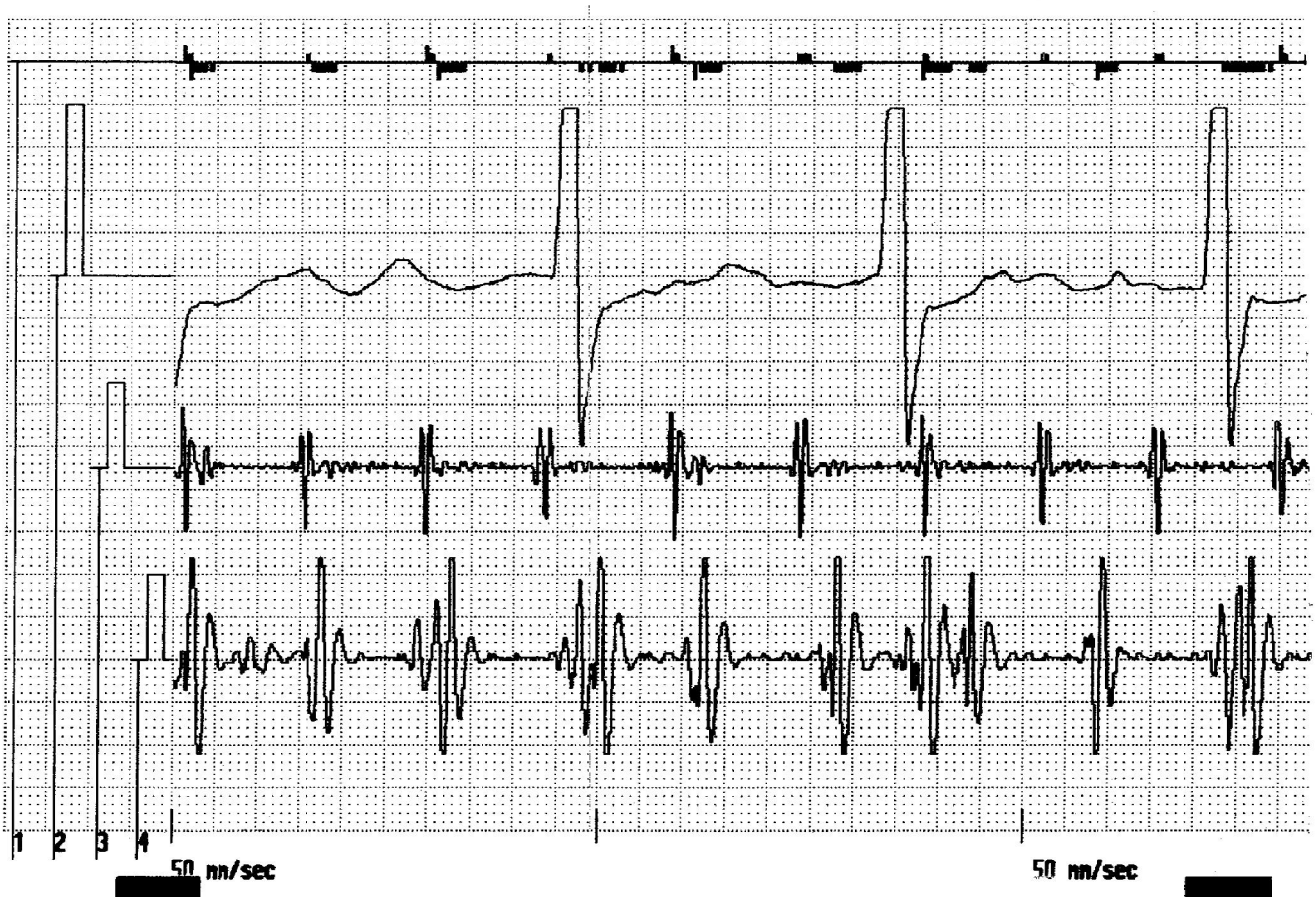


Figure 8. Atrial fibrillation (atypical flutter). Regular RA A-waves 215 bpm. A lone RAA IEGM can suggest AFl. Slower rhythm (apparently, its irregularity is caused by simultaneously sensed V-waves). Different duration of LA A-waves independent of the atrial rhythm (rate 70 to 80 bpm). The picture suggests the presence of two independent atrial circuits; this is consistent with the typical appearance of AF in a standard ECG.

increases asynchrony of excitation. It is possible that it has some proarrhythmic effect in patients with IACD.
2. Lone mid/proximal CS pacing does not increase intra/interatrial conduction time as significantly as RAA pacing. It shows slight synchronizing effects if high energy is applied.
3. Biatrial pacing normalizes atrial timing parameters and shows a marked atrial synchronizing effect. Thus, it helps to solve the problem of frequent arrhythmia recurrence in most patients with IACD.
4. Intracardiac electrocardiograms obtained via telemetry from BiA pacing systems are very useful for a more precise diagnosis of atrial arrhythmias. In many patients, AFl can be terminated by burst CS pacing.

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