Ventricular activation patterns during different pacing modes. An insight from electroanatomical mapping

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Abstract

Background: Biventricular pacing (BiV) is employed as the current standard for cardiac resynchronisation therapy. Other pacing modalities have been proposed as alternatives; however, data on changes in electrical activation sequence caused by pacing from various sites are limited.

Aim: To describe changes in activation patterns during different ventricular pacing modes in patients with chronic heart failure.

Methods: A total number of 20 patients (mean age 59.6±8years) with chronic heart failure, intraventricular conduction abnormality (QRS >130ms) or complete AV block were studied. Endocardial activation maps of both ventricles (CARTO™, Biosense-Webster) were obtained during spontaneous rhythm and biventricular (BiV, n=9), right ventricular bifocal (BiF, n=7) and single-site left ventricular (LV, n=4) pacing. The following parameters were assessed: activation pattern, total LV endocardial activation time (LVAT) and electrical interventricular delay (IVD).

Results: Right ventricular apical pacing was associated with the longest LVAT (145±24 ms). On the contrary, both BiV and BiF pacing shortened LVAT with BiV being superior in the degree of LVAT reduction (89±13 vs 103±10 ms, $p<0.05$). BiV pacing also significantly shortened IVD and modified the LV activation sequence in a complex manner. Such changes were not observed during BiF pacing. In the presence of fusion with spontaneous activation, single-site LV pacing was comparable to BiV pacing.

Conclusions: Among the different pacing modes, BiV pacing and single-site LV pacing with fusion resulted in the most pronounced changes in ventricular activation that appear to be a prerequisite for successful resynchronization of both ventricles.

Key words: cardiac pacing, ventricular activation, electroanatomical mapping, cardiac resynchronisation therapy, chronic heart failure

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Introduction

Cardiac resynchronisation therapy (CRT) has become a valid treatment modality for patients with congestive heart failure (CHF) and intraventricular conduction abnormalities. To accomplish this task, simultaneous pacing of both ventricles (i.e. biventricular pacing, BiV) is employed as the current standard [1, 2]. However, other pacing modalities such as right ventricular bifocal (BiF) [3] or single-site left ventricular (LV) pacing [4] have been proposed as alternatives. Several studies have evaluated the haemodynamic performance of different pacing modalities both in human beings [5-7] and in animal models [8, 9]. Despite this, only limited information is available on changes in electrical activation sequence caused by pacing from various sites and about the role of underlying conduction defects. Therefore, based on our previous experiences with endocardial mapping during conduction abnormalities [10], the aim of this study was to describe and quantify changes in ventricular activation patterns during different pacing modes.

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Methods

Patients

The study population consisted of 20 patients (4 females) with advanced CHF despite optimal drug therapy and LV conduction disturbances, who underwent implantation of a BiV or BiF pacing system in our institution between May 2001 and June 2003 (Table I). Patients were divided into three subgroups according to the principal pacing mode (i.e. BiF pacing, BiV pacing and single-site LV pacing). In order to describe the influence of baseline conduction abnormalities on activation sequence during each ventricular pacing, patients with a distinct ECG pattern (i.e. complete LBBB, intraventricular conduction disturbance and complete AV block) were randomly included in each of the subgroups. The mean age of the patients was 59.6±8 years. Aetiology of CHF was ischaemic heart disease (IHD) in 7, dilated cardiomyopathy (DCM) in 11 and a combination of both in 2 patients. The mean LV ejection fraction and end-diastolic diameter (EDDLV) reached 22±5 % and 75±7 mm, respectively. Additionally, we included a control group of four patients without conduction abnormality (mean age of 32±10 years), who underwent mapping after radiofrequency ablation of concealed left-sided accessory pathway. The study protocol was approved by the local ethics committee, and all patients consented to undergo the mapping procedure.

Implant procedure

The BiV and LV pacing subgroups included 13 patients who underwent implantation of the BiV pacing system (Insync III 8042 or 8040, Medtronic Inc, Minneapolis, MN). The right ventricular (RV) lead was positioned either in the RV apex (RVA, n=11) or in the RV midseptal position (n=2). The LV lead was implanted transvenously and positioned in the lateral or posterolateral branch of the coronary sinus in all but one patient with anterolateral position of the lead. The BiF subgroup consisted of 7 patients who underwent implantation of BiF pacing because of failure of the LV lead placement (n=4) or they required pacemaker

Table I. Patient's characteristics

Pacing mode	Pt	Age [years]	12-lead QRS morphology	Basal PQ interval	Dg.	EF LV	EDDLV [mm]
Bifocal pacing	1	73	IVCD	235 ms	IHD, Inf IM	20%	71
	2	69	LBBB	193 ms	DCM	20%	83
	3	57	LBBB	177 ms	DCM	20%	73
	4	44	LBBB	173 ms	DCM	20%	78
	5	60	n.a.	AV III	IHD	20%	69
	6	60	n.a.	AV III	DCM	40%	67
	7	60	n.a.	AV III	DCM	25%	84
Biventricular pacing	8	54	LAH+RBBB	161 ms	DCM	20%	74
	9	52	LAH+RBBB	203 ms	DCM	25%	85
	10	64	LAH+RBBB	245 ms	DCM	20%	73
	11	70	LAH+RBBB	202 ms	IHD, ant MI	20%	68
	12	52	IVCD	163 ms	IHD+DCM	25%	80
	13	55	LBBB	240 ms	DCM	20%	80
	14	55	LBBB	164 ms	DCM	20%	91
	15	56	n.a.	AV III	IHD, ant MI	25%	74
	16	78	n.a.	AV III	IHD, inf MI	20%	72
Left ventricular pacing	17	60	LAH+RBBB	193 ms	DCM	20%	76
	18	55	IVCD	166 ms	IHD, ant MI	25%	68
	19	52	LBBB	175 ms	IHD, inf IM	18%	79
	20	66	n. a.	AV III	IHD + DCM	15%	63

Abbreviations: *AVB III – complete heart block; Ant, inf MI – anterior, inferior myocardial infarction; DCM – dilated cardiomyopathy; EF – ejection fraction; EDDLV – end diastolic diameter of the LV; IHD – ischaemic heart disease; IVCD – intraventricular conduction delay; LAH – left anterior hemiblock; LBBB – left bundle branch block; n.a. – not applicable; RBBB – right bundle branch block*

implantation after AV node ablation for drug refractory atrial fibrillation (n=3). In these patients the two RV leads with active fixation were implanted in the RVA and high in the right ventricular outflow tract (RVOT) as described by Pachon et al. [3].

Electroanatomical mapping

In each subject, detailed LV and RV endocardial electroanatomical activation mapping (CARTO™, Biosense Webster, Haifa, Israel) [11] was performed after pacemaker implantation during BiF (n=7), BiV (n=9) or single-site LV pacing (n=4). Femoral access was used for both RV and LV mapping procedures. An electroanatomical mapping catheter with a 4 mm distal electrode (Navistar, Biosense Webster) was employed. In all cases the LV was entered retrogradely via the aortic valve and a bolus dose of heparin (100 IU/kg) was administered for LV mapping. Subsequently, remapping was conducted during baseline sinus rhythm (n=14) with the pacemaker programmed to VVI mode with stimulation rate of 40 bpm. In patients with complete AV block remapping was performed during RVA pacing (n=5) or BiV pacing (n=1). In the control group, mapping was performed only in sinus rhythm. No complications were noted during the mapping procedures.

Data analysis

All 3-D isochronal activation maps were analysed by the same operator. The local activation time was annotated at each point as the onset of the first high-frequency component of the bipolar signal. Whenever low voltage (below 0.5) and/or fragmented signals were present (duration >50 ms), the following additional criteria were applied: a) the steepest negative slope of the unipolar signal, b) continuity of propagation with other neighbouring points. Sites with very low voltage potentials (below 0.2 mV) and no pacing capture were labelled as a dense scar. Both the earliest and the latest LV endocardial activation sites

Table II. Activation intervals during different pacing modes

	Pt	Baseline rhythm	QRS [ms]		LVAT [ms]		IVD [ms]	
			Basal	BiF	basal	BiF	basal	BiF
Bifocal pacing (BiF)	1	IVCD	161	169	132	96	19	73
	2	LBBB	171	161	87	95	82	58
	3	LBBB	155	150	105	102	52	39
	4	LBBB	167	155	97	94	59	66
	5	RVa pace	200	162	188	125	10	39
	6	RVa pace	175	165	127	108	26	42
	7	RVa pace	210	176	123	102	83	71
			Basal	BiV	basal	BiV	basal	BiV
Biventricular pacing (BiV)	8	LAH+RBBB	130	100	121	74	-28	26
	9	LAH+RBBB	165	115	170	88	-51	19
	10	LAH+RBBB	155	123	137	84	-46	31
	11	LAH+RBBB	141	119	79	94	-54	11
	12	IVCD	160	140	125	90	-30	13
	13	LBBB	185	132	125	89	60	43
	14	LBBB	185	120	109	74	74	42
	15	RVa pace	169	161	132	123	23	22
	16	RVa pace	190	111	156	75	31	33
			Basal	LVp	basal	LVp	basal	LVp
Left ventricular pacing (LVp)	17	LAH+RBBB	183	144	128	78	-79	26
	18	IVCD	134	100	85	74	-7	8
	19	LBBB	174	131	119	101	60	32
	20	BiV	204	244	97	186	30	31

Abbreviations: *LVAT – left ventricular activation time; IVD – interventricular delay; rest of the abbreviations as in Table I.*

had to be recorded in at least two separate points in order to minimise potential error in calculation of total left ventricular activation time.

Before the mapping procedure both the sensed and paced AV delays were set at 120 and 150 ms, respectively. Patients thus had during mapping a variable individual PR interval but constant programmed AV delay. This approach was used to enable identification of the longest spontaneous PR interval for the fusion between spontaneous conduction and pacing. Optimisation of the AV interval was performed after the mapping procedure under echocardiographic guidance.

Definitions

LV activation was described with regard to direction and number of depolarising wavefronts. Left ventricular activation time (LVAT) was defined as the interval between the earliest and the latest LV endocardial activation. Interventricular delay (IVD) was defined as the difference between onsets of right and left ventricular endocardial activation (being negative when activation of the LV preceded the RV). The transseptal conduction interval was calculated for different RV pacing sites as the interval between the pacing stimulus and the earliest activation at corresponding LV septal site. Fusion between paced and spontaneous rhythm was considered to be present, when, besides fascicular terminations, additional separated LV endocardial breakthrough was observed that corresponded to the RV lead and was not present during spontaneous rhythm.

Follow-up

After implantation, patients were followed on a regular basis every three months. The follow-up visits consisted of clinical evaluation, assessment of functional NYHA class and echocardiographic measurement. Patients evaluated in the single-site LV pacing subgroup were reprogrammed to BiV pacing after the procedure. Thus, no long-term follow-up data on this pacing modality are available.

Statistical analysis

All numerical data are presented as the mean ± SD. Quantitative assessment of different pacing modes was obtained by comparison of individual activation intervals. Statistical analysis was performed using an unpaired or paired t test, whenever appropriate. A p value <0.05 was considered statistically significant.

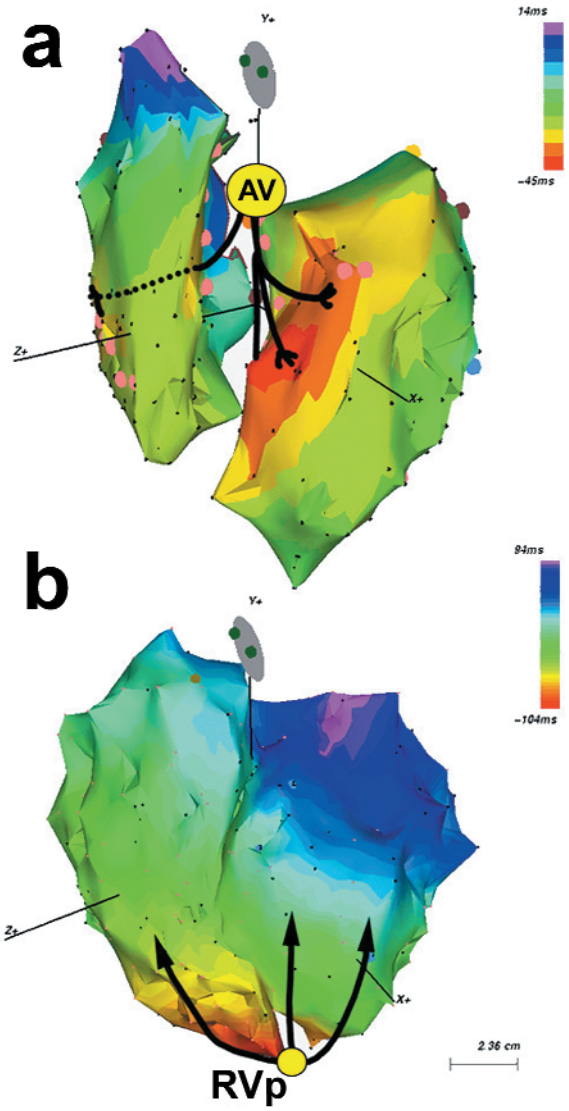


Figure 1. Examples of electroanatomical isochronal maps of both ventricles (left anterior oblique view rotated cranially) showing spread of activation in normals (A) and during RVA pacing (B). Activation times are colour-coded (the earliest activation depicted as red and the latest as violet). Note the very rapid (LVAT 50 ms) and homogeneous spontaneous activation in the control. In contrast, RVA pacing results in reversal of LV activation and in pronounced dyssynchrony between apex and base. AV – AV node; RVp – RVA pacing site

Results

A mean number of 128 and 158 sites were mapped within the RV and LV, respectively. Visual analysis of isochronal maps identified specific activation patterns of the LV both in normals and in each pacing modality.

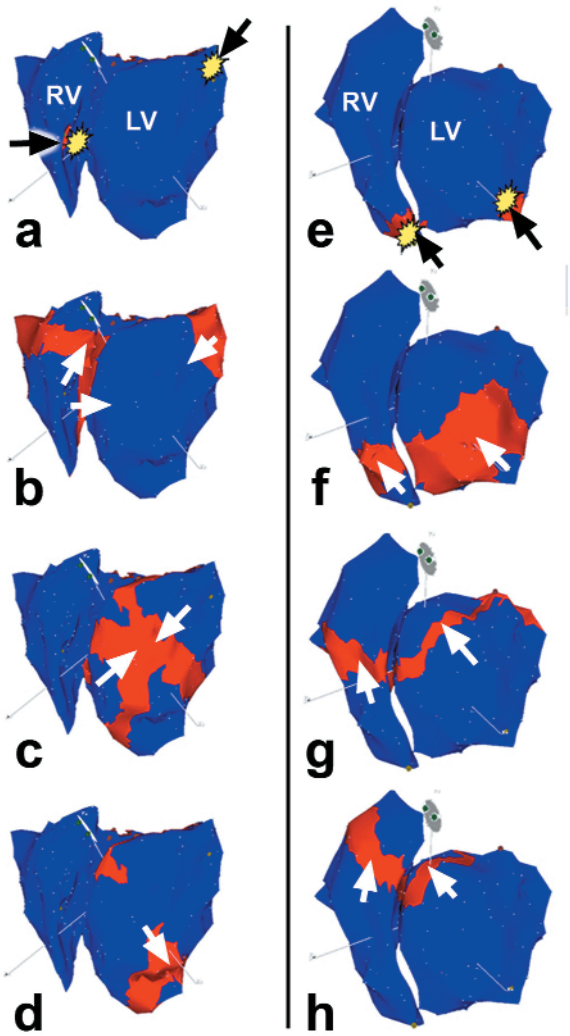


Figure 2. The left panels (a-d) depict the spread of activation (left anterior oblique view rotated cranially) during BiV pacing (patient no. 16) with the RV lead in the RV midseptal position and the LV lead in the posterolateral region (black arrows). This arrangement of the leads results in a merger of activation wavefronts in the middle part of the LV and resembles a normal activation pattern. Right panels (e-h) show propagation of paced wavefronts in a corresponding view during BiV pacing in a patient (no. 12) with the RV lead positioned in the RV apex and the LV lead placed distally in the anterolateral vein (black arrows). The proximity of both leads (approx. 40 mm) results in early fusion of both pacing wavefronts and subsequent LV activation in an apicobasal direction. This was associated with accentuated left intraventricular delay (LVAT 146 ms, QRS 161 ms)

Activation patterns

In the control group, LV activation started in two or three separate regions of the septum, reflecting branching of the left bundle, and then propagated rapidly towards the apex and the lateral wall (Figure 1a). During RVA pacing the LV was activated with a substantial delay and in an apicobasal direction (Figure 1b). The latest activation occurred at the lateral basal wall of the LV. BiV pacing was associated with a specific activation pattern that reflected LV activation via two opposite wavefronts that merged together. Of note, considerable differences were observed among individual patients and the resulting activation pattern was greatly influenced by the respective lead positions (Figure 2). BiF pacing was characterised by delayed LV endocardial activation via two septal wavefronts (superior and inferior) that corresponded to the RVOT and RVA pacing sites. In the LV itself, both wavefronts fused relatively early with an activation pattern resembling the complete left bundle branch block (LBBB) (Figure 3). Single-site LV pacing produced an activation sequence that was highly variable, which depended on the degree of fusion with spontaneous conduction (Figure 4). If fusion was present, a similar pattern of two opposing wavefronts as in BiV pacing was observed. In the absence of fusion, LV activation originated only from the LV pacing site and was followed by delayed septal activation. Therefore, the resulting LV activation pattern resembled a mirror image pattern of complete LBBB.

Activation intervals

Analysis of activation intervals among different patient groups underlined the above-mentioned differences in activation patterns (Figure 5). The control group had the shortest LVAT. RVA pacing produced the longest intervals in both patient groups. BiF pacing changed neither the overall QRS duration nor LVAT when compared to basal spontaneous rhythm (NS). However, it was associated with a lesser degree of prolongation of both intervals when compared to single-site RVA pacing ($p<0.01$ for QRS and $p<0.005$ for LVAT). BiV pacing resulted in a more pronounced shortening of both the QRS and LVAT when compared to BiF pacing (both $p<0.05$). Comparable intervals were also observed during single-site LV pacing in patients with preserved AV conduction (NS).

No IVD (i.e. right-to-left transseptal conduction) was observed in control subjects as LV activation preceded activation of the RV by 5±2 ms. In patients with CHF the IVD during spontaneous activation varied according to the presence of conduction via left and right bundle,

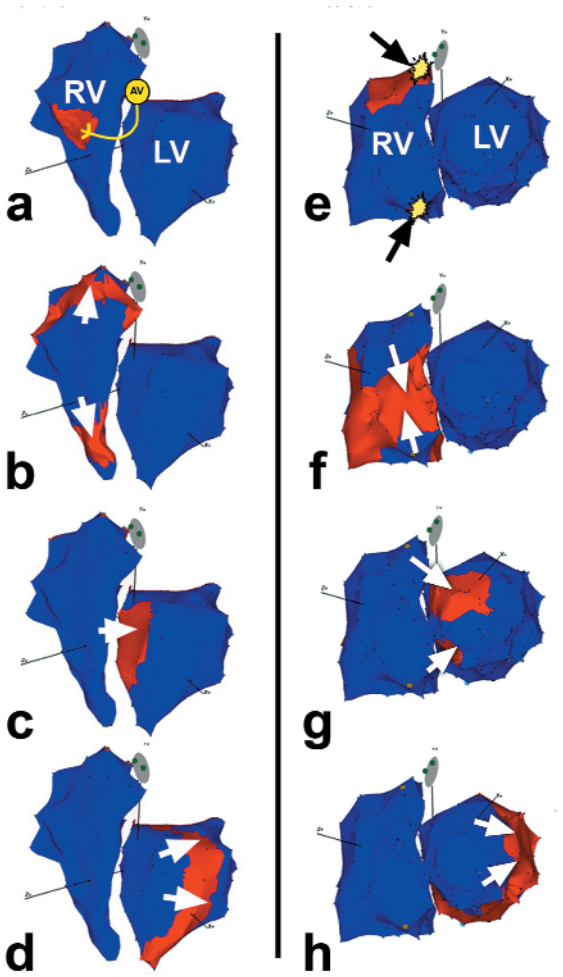


Figure 3. The panels depict an activation pattern during LBBB and after implantation of the BiF pacing system (left anterior oblique view). Left panels (a-d) show the propagation map during LBBB, when the activation begins on the right lateral free wall and the septum (termination of the right fascicle), then is slowly conducted towards the LV via the interventricular septum. Activation of the LV begins 61 ms after the beginning of QRS onset on the midseptum. The right panels (e-h) show LV activation in the same projection during BiF pacing. Note the similar LV activation pattern with the latest activation on the lateral wall

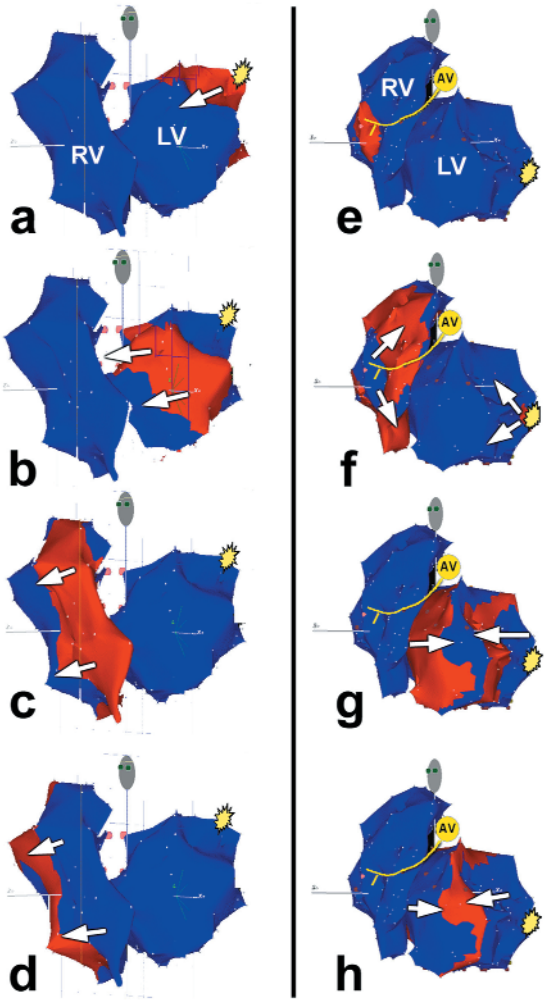
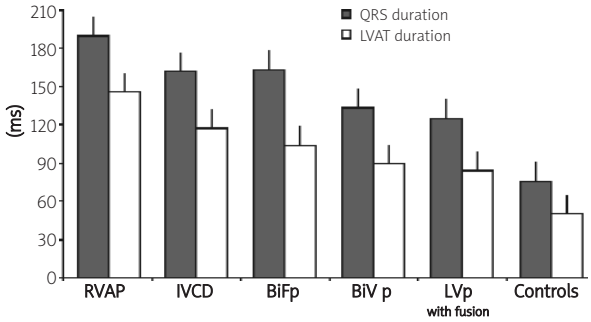


Figure 4. Panels depict the ventricular activation sequence during single-site LV pacing in a patient with complete AV block (a-d) and in a subject with normal PR interval leading to the phenomenon of fusion (e-h). In the left panels, activation propagates in a left-to-right direction, creating a 'reversed LBBB' pattern. In the right panels, spontaneous activation via the fascicular system causes early septal activation and the LV is activated from two opposing wavefronts as observed during BiV pacing

reaching 55±10 ms for the LBBB. During RV pacing the transseptal conduction interval was relatively constant (53±9 ms for RVA pacing site and 64±11 ms for pacing at RVOT, p=NS). As a result, all BiF patients presented a significant IVD of 55±14 ms, and thus with a considerable interventricular electrical dyssynchrony. On the contrary, BiV pacing resulted in shortening of IVD to 27±11ms ($p<0.05$). IVD in these patients represents not only right-to-left transseptal conduction but comprises to some degree the transmural activation delay for activation from the epicardially-located LV lead to its endocardial breakthrough.



	RVAP	IVCD	BiFp	BiV p	LVp with fusion	Controls
QRS (ms)	189±15	161±18	163±8	133±29	125±18	75±5
LVAT (ms)	145±24	117±23	103±10	89±13	84±12	50±5
IVD (ms)	53±9	#	55±14	27±11	22±10	-5±2

Figure 5. Relationship between duration of QRS and LVAT during different pacing modes and spontaneous conduction abnormalities (IVCD). The mean QRS, LVAT and IVD for each mode are demonstrated. RVA pacing produced the longest QRS and LVAT activation. BiF pacing did not differ significantly from the basal spontaneous rhythm; however, it seemed to be superior to RVA pacing. BiV pacing was superior compared to other modes in the degree of achieved electrical synchrony. Single-site LV pacing in patients with preserved AV conduction was comparable to BiV pacing (see text for discussion). # – IVD during spontaneous activation varied accordingly to the presence of RBBB or LBBB.

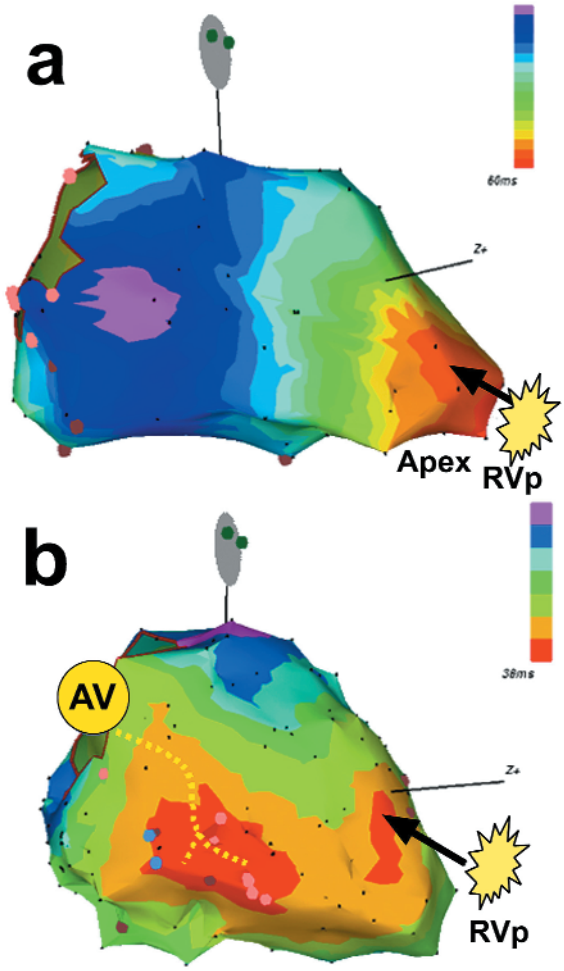


Figure 6. The panels depict activation maps of the LV septum (right oblique view) during BiV pacing without (a) and with (b) fusion. Activation times are colour-coded (red as the earliest, violet as the latest). In panel A the LV septum is activated only from the RV pacing lead positioned in the apex with a delay in activation of the basal part. In panel B the septum is activated from two separate breakthroughs – the majority is activated via the left fascicle while the apical portion is activated from the RV pacing lead

Effect of basal conduction abnormality on activation pattern during pacing

Activation patterns in each pacing group were further analysed with respect to spontaneous conduction abnormality. The character of activation during pacing was affected mostly by conduction defects localised on the ventricular level that corresponded to the sites of scarring after myocardial infarction or fibrosis. Additionally, considerable influence on activation pattern during pacing was observed in the case of fusion with spontaneous conduction. Mainly in patients with preserved conduction via the left posterior fascicle the early activation of the inferior wall shifted the latest activation region within the LV anteriorly. In the absence of fusion the conduction defects within the principle fascicles did not affect the resulting activation.

The phenomenon of fusion

As already mentioned, the resulting LV activation sequence during pacing was influenced by the presence of fusion with spontaneous conduction. Its occurrence depended mostly on spontaneous AV conduction. In patients with the higher degree of AV block, no fusion was present and both ventricles were activated solely from the pacing sites (Figure 6a). In those with a normal PR interval, spontaneous conduction led to early septal activation that competed with RV pacing (Figure 6b).

In addition, the degree of fusion varied with respect to the underlying intraventricular conduction defect. In patients with preserved conduction via the left bundle (mainly via the posterior fascicle), early activation of the

LV septal endocardium was observed, which enabled fusion even in patients with a relatively longer spontaneous PR interval. On the other hand, the absence of conduction through the left bundle (i.e. complete LBBB) resulted in slow transseptal right-to-left conduction and late activation of the LV septal endocardium (occurring 55±12 ms after QRS onset). Under such circumstances, fusion occurred in a much shorter spontaneous PR interval. Finally, the occurrence of fusion was more probable during single-site LV than during BiV or BiF pacing. The main reason was that LV lateral wall pacing allowed a long time interval for spontaneous septal conduction to occur.

As a result, fusion occurred in 66% of patients with preserved conduction via the left bundle (n=6) during BiV and BiF pacing (in those with a basal PR interval up to 203 ms). In patients with complete LBBB (n=5), fusion occurred only in one with a very short basal PR interval of 164 ms. In the remaining patients the LV septum was activated only from the RV pacing site with a significant delay (Figure 6A). During single-site LV pacing, fusion occurred in all three patients with preserved AV conduction (with a spontaneous PR interval up to 193 ms). However, it may be expected to occur even in patients with much longer spontaneous PR intervals.

Clinical follow-up

All 9 patients from the BiV pacing group were available for the follow-up. When compared to baseline, the mean NYHA class decreased at six months from 3.1 to 2.5 ($p<0.05$). Similarly, the mean EDDLV decreased from 79.1 to 76.5 mm ($p<0,05$) and LV ejection fraction tended to improve from 21 to 23% (p=NS). In the BiF pacing group, 6 of 7 patients were available for follow-up visits and no sustained clinical benefit was observed. At six months, the mean NYHA class remained unchanged, 2.8 vs 2.6, together with EDDLV (75.8 vs 75.4mm) and LV ejection fraction (21% *vs* 22%, all NS).

Discussion

Despite the fact that several studies have addressed activation patterns during different pacing modes, most of them were experimental [12] and used animal models of CHF with artificially created LBBB [8, 13]. Such models do not reflect the spectrum of conduction abnormalities observed in typical CHF patients. Only recently, Auricchio et al. [14] using non-contact endocardial mapping have proposed that variability of activation patterns in LBBB patients is generated by individual functional block. Labiase et al. [7] using a similar system showed that the outcome of CRT may be adversely affected by placing the LV lead within the region of slow conduction. Both these studies concluded that the resynchronisation therapy should be tailored to the individual electromechanical characteristics of each patient. In this context, our study provides further description of factors affecting ventricular activation such as baseline conduction abnormality, PR interval, pacing modality and lead positioning, and thus illustrates the complexity of LV dyssynchrony and the array of possible variables determining the success of CRT.

One of the original contributions of this study appears to be the description of the characteristics/factors affecting the degree of fusion between pacing and spontaneous conduction. The presence of fusion has been shown to be crucial for the benefit of single-site LV pacing in the experimental model [13]. Similarly, van Gelder et al. [15] found that the optimal haemodynamic effect of LV pacing occurs at longer AV delays that allow fusion of pacing with spontaneous conduction; and Auricchio et al. [16] demonstrated that single-site LV pacing is haemodynamically inferior to BiV pacing in patients with complete AV block.

Based on our observations, fusion may also play a significant role in patients with BiV and/or BiF pacing (especially those with preserved conduction via the left bundle and normal AV delay) as it results in rapid septal activation. Secondly, when present, it may shift the latest activated segment within the LV (e.g. in the case of fusion and preserved conduction via the left posterior bundle, the latest activation within LV shifts more anteriorly). This may have a clinical impact as a substantial proportion of patients undergoing CRT have a normal basal PR interval and the character of the LV conduction defect is highly variable [10, 17], ranging from complete LBBB to preserved but slow conduction via the left bundle.

Our study also provides a direct comparison of ventricular activation patterns between BiV and BiF pacing. It clearly demonstrated that only BiV pacing produced the most fundamental changes in LV activation. It shortened both LVAT and IVD, and thus corrected both inter- and intra-ventricular conduction delays. On the contrary, RV BiF pacing resulted only in moderate shortening of LVAT, and the degree of LV resynchronization achieved through BiF stimulation was outbalanced by prolongation of the right-to-left interventricular conduction. In this respect, earlier RV than LV activation may lead to ventricular interaction and constraint to LV filling [18]. Relief of this constraint by early activation of the LV has been shown to be one of the mechanisms underlying the hemodynamical improvement after instalment of CRT

[19, 20]. Such may not be expected during BiF pacing, and for all these reasons, this pacing modality cannot be considered a comparable alternative to BiV pacing. The long-term clinical outcome of the patients is in concordance with this hypothesis.

Finally, our data emphasise the clinical problem of unnecessary RVA pacing, which was observed to produce the longest duration of LVAT and significant IVD, and thus significant inter- and intra-ventricular asynchrony. These observations are consistent with clinical data about the detrimental effect of RVA pacing on cardiac function [5, 9, 21]. Similarly, recent results of the DAVID trial showed approximately 60% higher relative probability of death or hospitalisation for new or worsened heart failure in ICD patients randomised to DDDR mode as compared to back-up VVI pacing [22].

Study limitations

The relatively small number of patients with heterogeneous basal characteristics limits to some extent the value of this study. However, the unselected patient population more resembles a spectrum of typical CHF patients undergoing CRT, and no previous study has in detail described the role of variable spontaneous conduction abnormalities during different pacing modes.

Another limitation of the study is that only one pacing mode was evaluated in each patient. However, evaluation of all modes in each patient would make the study unethical, because of the time-consuming nature of high density mapping. On the other hand, the observed activation sequences were relatively consistent and specific for each conduction abnormality and/or pacing modality.

Finally, as the study was focused predominantly on electroanatomical mapping, no haemodynamic correlates were obtained. Shortening of QRS duration and LVAT were thus considered to be a marker of achieved resynchronisation. In this respect, it has to be emphasised that the correlation of QRS duration to efficacy of CRT is ambiguous. On one side, there are data suggesting that the benefits of CRT correlate positively with the degree of QRS shortening and this parameter could be considered a marker of achieved resynchronisation [23, 24]. However, more recent studies using experimental models suggest that the haemodynamic benefit of different pacing modes depends on LV activation sequence rather than on overall electrical synchronicity as represented by QRS duration [8, 9, 13, 21, 25]. On the contrary, electrical synchronicity of LV as represented by endocardial LVAT has shown to correlate highly with haemodynamic performance [12]. This observation supports the key role of the endocardium for LV activation, reflecting the presence of the subendocardial Purkinje system with very rapid conduction (the estimated conduction is up to four times faster than intramyocardial conduction) [25]. For these reasons, we believe that quantification of changes in timing and sequences of endocardial LV activation patterns is crucial for understanding the mechanisms responsible for the benefit of CRT.

Conclusions

Different modes of CRT lead to characteristic patterns of LV activation in patients with CHF and intraventricular conduction abnormalities. BiV pacing shortens LV activation, minimizes interventricular delay and modifies the LV activation pattern in a complex manner. Single-site LV pacing is associated with similar characteristics provided by fusion of pacing wavefronts with spontaneous septal activation if present. Right ventricular BiF pacing results in a decrease in LVAT at the expense of an increase in IVD. Single-site RVA pacing causes reversed LV activation and leads to the highest degree of inter- and intra-ventricular asynchrony.

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Sposób aktywacji komór podczas różnych typów stymulacji – wnioski z elektro-anatomicznego mapowania serca

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Streszczenie

Wstęp: Terapia resynchronizacyjna (CRT) stała się skuteczną metodą leczenia u chorych z przewlekłą niewydolnością serca (CHF) i zaburzeniami przewodzenia śródkomorowego. Zwykle wprowadza się dwie osobne elektrody do stymulacji prawej i lewej komory (stymulacja dwukomorowa – BiV), ale istnieje kilka innych możliwości umiejscowienia elektrod, które do tej pory nie były odpowiednio zbadane.

Cel: Zbadanie zmian w sposobie aktywacji mięśnia komór podczas stymulacji z różnych miejsc w komorach u chorych z CHF.

Metodyka: Grupa badana składała się z 20 chorych (średni wiek 59.6±8 lat) z CHF, zaburzeniami przewodzenia śródkomorowego (QRS >130 ms) lub całkowitym blokiem przedsionkowo-komorowym. Mapy aktywacyjne wsierdzia obu komór (CARTO™, Biosense-Webster) skonstruowano podczas samoistnego rytmu zatokowego oraz podczas stymulacji BiV (n=9), dwuogniskowej prawokomorowej (BiF, n=7) i jednoogniskowej lewokomorowej (LV, n=4). Oceniano następujące parametry: sposób aktywacji, całkowity czas aktywacji wsierdzia lewej komory (LVAT) oraz elektryczne opóźnienie międzykomorowe (IVD).

Wyniki: Stymulacja wierzchołka prawej komory wiązała się z najdłuższym LVAT (145±24 ms). W przeciwieństwie do tego, zarówno stymulacja BiV jak i BiF powodowała skrócenie LVAT, przy czym największy stopień redukcji tego parametru był osiągnięty podczas stymulacji BiV (89±13 *vs* 103±10 ms, p <0.05). Stymulacja BiV skróciła również istotnie IVD i zmodyfikowała w sposób złożony sekwencję aktywacji LV, czego nie obserwowano podczas stymulacji BiF. W przypadku obecności pobudzeń zsumowanych z samoistna aktywacją mięśnia komór, stymulacja jednoogniskowa LV była porównywalna do stymulacji BiV.

Wnioski: Spośród różnych trybów stymulacji, stymulacja BiV oraz stymulacja jednoogniskowa lewej komory podczas pobudzenia zsumowanego powodowały najsilniejsze zmiany w sposobie aktywacji mięśnia komór, konieczne do uzyskania skutecznej resynchronizacji pracy obu komór.

Słowa kluczowe: stymulacja serca, aktywacja komór, maping elektroanatomiczny, resynchronizacja serca, przewlekła niewydolność serca

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