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Cardiac memory in cardiac resynchronization therapy: A vectorcardiographic comparison of biventricular and left ventricular pacing☆

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Abstract

Introduction: "Cardiac memory" (CM) refers to a change in repolarization induced by an altered pathway of activation, manifested after resumption of spontaneous ventricular activation (SVA).

Aims: To investigate for the first time in humans the effects of left ventricular (LV) pacing on CM development through vectorcardiography (VCG).

Methods: We studied 28 patients with heart failure (HF) and left bundle branch block (LBBB) treated with cardiac resynchronization therapy (CRT). Fourteen patients underwent biventricular (BIV) stimulation; the other 14 underwent LV stimulation only. VCG was acquired during SVA at baseline and during AAI and DDD pacing immediately after and 7 and 90 days after the implant.

Results: At baseline, in both groups, the QRS and T vectors angles were those specific of LBBB pattern. During DDD pacing, QRS vector angle changed to the right and upward in BIV patients while no significant differences were observed in LV patients.

During AAI pacing, T vector angle changed significantly in BIV patients, following the direction of the paced QRS and amplitude significantly increased. In LV patients no significant differences in T vector angles were observed. Only T vector amplitude significantly increased at 7 days ($p = 0.03$) and at 90 days ($p = 0.008$ vs baseline).

Conclusion: In patients with LBBB, BIV pacing induces cardiac memory development as a significant change in T vector magnitude and angle, while LV pacing doesn't induce significant modifications in QRS and T vector angles and CM is manifested only as a significant T vector amplitude change.

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Keywords: Cardiac memory; Cardiac resynchronization therapy; Vectorcardiography; T-wave; Left ventricular pacing

Introduction

"Cardiac memory" (CM) refers to a change in myocardial repolarization induced by an altered pathway of activation, including pacing, transient left bundle branch block, accessory pathways, and tachyarrhythmias [1–3]. It has been described after restoration of normal ventricular excitation as a change of T-vector direction, approaching that of the abnormally activated QRS complex, and as an increased T-vector amplitude [3]. These features are persistent and related to the duration and frequency of the abnormal activation and the phenomenon is known as "accumulation" [3].

Several studies reported the development of CM after resumption of normal cardiac conduction in patients undergoing right ventricular pacing [4,5] but only two studies recently investigated CM during cardiac resynchronization therapy (CRT) [6,7]. Based on biventricular pacing, CRT is an established treatment option for patients with moderate-to-severe heart failure (HF) and left ventricle electrical dyssynchrony as indicated by a wide QRS complex [8–10]. Also single-site left ventricular (LV) pacing has been shown improve LV function in acute hemodynamic and in long term follow-up studies [11–13].

While the repolarization changes of CM have been observed in CRT patients as an increase in amplitude and a direction change of the T vector after one week [6,7], the induction of CM by left ventricular (LV) pacing was examined only in experimental studies [14–17].

Abbreviations: CM, cardiac memory; CRT, cardiac resynchronization therapy; VCG, vectorcardiography; SVA, spontaneous ventricular activation; BIV, biventricular pacing; LV, left ventricular; LBBB, left bundle branch block; HF, heart failure.

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The aim of this study is to investigate for the first time the development of CM in the human heart during LV pacing in patients with heart failure (HF) and left bundle-branch block (LBBB) using a vectorcardiographic approach.

Methods

Patients

We enrolled 28 patients with symptomatic HF (III-IV NYHA functional class) on optimal medical therapy (22 M; mean age 70 ± 9 years) who underwent CRT with defibrillator in our Centre, according to current guidelines for cardiac pacing [18]. All patients had left ventricular dysfunction (ejection fraction <35% assessed by echocardiography) and were in sinus rhythm with a QRS duration ≥ 120 ms and a LBBB morphology. Underlying etiology of HF was ischemic in 11 patients, and idiopathic in 17; patients were excluded if they had history of atrial fibrillation or sustained ventricular tachyarrhythmias.

The study conforms to the principles outlined in the Declaration of Helsinki and was approved by the Institutional Review board. All patients gave written informed consent.

All the 28 patients received a CRT-D device with three transvenous pacing leads implanted. The right atrial lead and right ventricular lead were positioned, respectively, at the right atrial appendage and at the right ventricular apex. The left ventricular lead was implanted through the coronary sinus in the lateral or posterolateral vein.

At implant, the patients were prospectively allocated to 1 treatment group in a nonrandomized 1:1 fashion: in 14 patients the CRT-D device was programmed as in conventional biventricular stimulation (BIV) while 14 patients underwent atrial-triggered left ventricular epicardial pacing (LV). In both groups, a DDD pacing mode with low base rate has been chosen, in order to allow sequential AV pacing with intrinsic atrial activity.

Protocol

VCG was recorded on a Marquette MAC 12/15 software (Marquette Electronics, Inc Milwaukee, WI) using a 14-wire Marquette acquisition module that acquires the Frank lead set orthogonal XYZ ECG, with a sampling frequency of 500 Hz. The recordings were acquired before pacemaker implantation (baseline) during sinus rhythm, immediately after and 7 and 90 days after the implantation.

In both the arms (BIV and LV), the CRT-D pacemaker was programmed with a short atrio-ventricular delay in order to avoid fusions and to obtain maximum ventricular stimulation. All patients underwent an atrioventricular delay optimization according to maximum diastolic filling time [19] and the AV delay was therefore selected after testing a wide range of AV delays, using steps of 20 ms. In BIV group, the biventricular pacing was programmed synchronous (V-V delay = 0 ms).

During each visit, VCG was recorded in DDD mode during BIV stimulation or LV stimulation, respectively, and in AAI mode (at the same rate), to allow spontaneous ventricular activation.

Table 1
Patient characteristics.

	All (n = 28)	BIV Group (n = 14, 50%)	LV Group (n = 14; 50%)
Age (y), mean ± SD	70 ± 9	72 ± 8	68 ± 10
Sex (M), n (%)	22 (78)	11 (78)	11 (78)
Ischemic Etiology, n (%)	11 (39)	6 (54)	5 (36)
LVEF, %	29 ± 4	26 ± 4	31 ± 2
TAPSE (mm), mean ± SD	19 ± 4	19 ± 6	19 ± 4
SBP (mmHg), mean ± SD	119 ± 8	120 ± 8	119 ± 9
PQ baseline (ms), mean ± SD	190 ± 45	205 ± 57	176 ± 26
Paced AV (ms), mean ± SD	114 ± 18	120 ± 19	104 ± 13
QRS baseline duration (ms), mean ± SD	162 ± 30	150 ± 30	170 ± 24
Paced QRS width (ms), mean ± SD	146 ± 22	145 ± 20	147 ± 26
QTc baseline (ms), mean ± SD	465 ± 47	486 ± 53	446 ± 35
Paced QTc (ms), mean ± SD	474 ± 50	477 ± 52	469 ± 52

Clinical, demographic and ECG characteristics of study population. LVEF: left ventricular ejection fraction; SBP: systolic blood pressure; TAPSE: tricuspid annular plane systolic excursion; QTc: corrected QT interval.

Vector loops of QRS and T wave were recorded on frontal, transversal and right sagittal plane and were analyzed offline quantitatively by two different investigators (G.P., M.N.). For each recording, the maximum QRS and T vectors in each plane were defined and expressed as amplitude (or magnitude) in microvolts (mV); their direction in space was calculated. The vector angles were expressed as azimuth (the angle in the transverse plane, defined as 0° when the vector is pointing to the left, +90° to the front, -90° to the back, 180° to the right) and elevation (the angle between the vector and an axis perpendicular to the transverse plane, with 0° being the vector pointing downward and 180° pointing in the cranial direction) [4–7,20].

Statistical analysis

Data are presented as mean ± standard deviation or median and 25^{th}–75^{th} percentiles (pct). Data were compared using Student's t test for paired samples and Wilcoxon matched-pairs test. A p value <0.05 was considered significant.

Results

All the patients in both groups achieved nearly 100% ventricular pacing during the study and the mean paced AV delay was 114 ± 18 ms.

There were no significant differences between the two groups for all the baseline characteristics (Table 1).

Baseline

At baseline, in both groups, the QRS vectors angles were those typical of the LBBB pattern [20], pointing backward and to the left (Azimuth BIV: −62°[−75°; −43°] vs LV: −65° [−78°; −42°]; p = N.S.) and situated in the transversal plane or a little below (Elevation BIV: 82°[75°; 93°] vs LV: 55°[48°; 95°] p = N.S.) (Table 2, Figs. 1–2). Accordingly, also T wave vectors were similar in both groups (Table 2, Figs. 1–2).

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