Case Report

Manifest cardiac memory after biventricular pacing in a super-responder patient: Is memory the sign of a 'forgotten' electrical ventricular pattern?

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Abstract We present the case of a super-responder patient with a basal left bundle branch block, who underwent cardiac resynchronization therapy and showed, during biventricular pacing, typical electrocardiographic signs of cardiac memory.

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Introduction

'Cardiac memory' (CM) is a term used clinically to describe a specific form of remodeling seen as an altered electrocardiographic T wave [1]. It is characterized by an altered T wave on electrocardiogram (ECG) recorded during sinus rhythm (or any rhythm with normal ventricular activation) and induced by a preceding period of altered electrical activation. CM has been initially described following stimuli such as right ventricular electrical pacing, and later after intermittent left bundle branch block (LBBB), paroxysmal tachycardia, and preexitation [2].

In this article, we present the case of a patient who underwent cardiac resynchronization therapy (CRT) and showed, during biventricular pacing (BiV), ECG signs of CM.

Case report

A 65-year-old woman with a diagnosis of primary dilated cardiomyopathy was admitted because of effort intolerance and dyspnea [New York Heart Association (NYHA) class III] despite optimal medical therapy. A 12-lead surface ECG showed a sinus rhythm and a persistent LBBB (Fig. 1, LBBB baseline). Echocardiography documented a left ventricle dilatation with severe systolic dysfunction [left ventricular ejection fraction (LVEF) 30%]. According to current guidelines, the patient was a candidate for CRT. A CRT device (Promote Q®, St Jude Medical, Sylmar, CA, USA) was implanted in the left prepectoral region and connected with a right atrial pacing lead (Model 5594, Medtronic Inc., Minneapolis, MN, USA), a right ventricular dual coil defibrillating lead (Durata™ 7120, St Jude Medical) and a left ventricular tetrapolar pacing lead (Quartet™ 1458Q, St Jude Medical), introduced through the coronary sinus into a lateral additional CS branch (Fig. 2). The device was programmed in DDD

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Discussion

Rosenbaum et al. [1] introduced the term cardiac memory in 1982. It is characterized by an altered T wave on ECG during sinus rhythm (or any rhythm with normal ventricular activation) and induced by a preceding period of altered electrical activation. Following the return to sinus rhythm after an interval of ventricular pacing or arrhythmia, the T wave vector persists in tracking the vector angle and amplitude of the QRS complex that characterized the paced or arrhythmic state. According to Rosenbaum et al. [1], the sinus T wave 'remembered' the paced QRS complex. Although other forms of abnormal activation inducing memory (as intermittent left bundle branch block, ventricular arrhythmias, and atrioventricular bypass tracts) have been described, the preponderance of examples of CM derive from cardiac pacing which, as long as it alters ventricular activation, consistently induces memory [2].

In the present case, we observed an original manifestation of the phenomenon. Indeed, our patient showed signs of CM immediately after BiV. The electrocardiographic changes induced by BiV on QRS complex have been reported [3–5], but no data on the effects of CM have been yet described. The paced ventricular activation in CRT is due to different wave fronts: one generated by RV pacing and primarily located at endocardial layers of the myocardial wall and the other one generated by left ventricular pacing which spreads mostly at the epicardium and myocardium [6]. The ECG result is, thus, a significant change of QRS duration and axis in the frontal plane with a switch toward right superior quadrant [7,8].

With regard to ventricular repolarization pattern, as known, the T wave on ECG is the summed signal of action potential repolarization in the cells of the ventricle. More specifically, the gradient of repolarization within the ventricles gives rise to the electrocardiographic ST segment and T wave [3]. The sources of gradients in the ventricle are complex and include: that between apex and base, such that repolarization time is shorter basally than apically; that between left and right ventricle; and that which is transmural (at any site epicardial action potentials are shorter than endocardial). Whether the change in apico-basal gradients is the sole determinant of T wave changes during short-term memory or other gradient changes occur as well to explain the T waves of short- and long-term memory is still unclear. For example, the possibility of changes in the right—left ventricular gradient induced by CRT has not been studied in memory [9].

In the present case, the ECG result of CRT was really impressive, not only for the ventricular depolarization pattern (i.e. significant modification of QRS duration and axis from baseline toward a “pseudonormal” activation pattern) but also for the repolarization pattern (Fig. 1). During BiV, ECG showed an electrocardiographic T wave vector change, (Fig. 1, Day 1 and Day 7) reflecting the QRS complex vector during prior periods of spontaneous LBBB, followed by a complete normalization at 1 month (Fig. 1, Day 30), without further changes at 6 month (Fig. 1, Day 180; Fig. 3). Besides depolarization/repolarization pattern restoration, during follow up the patient showed a super response to CRT.

Figure 1 Twelve-lead electrocardiogram before (baseline) and during (post-implantation, Day 7, Day 30; Day 180) BiV. BiV pacing induces dynamic modifications of the depolarization and repolarization patterns. Depolarization pattern modifications included QRS shortening and axis rotation toward right superior quadrant in the frontal plane (Day 1–Day 30). Dynamic repolarization modifications manifested as the T wave vector persisting in tracking the vector angle and amplitude of the spontaneous QRS complex that characterized the LBBB, with a slow and gradual normalization at the 30th day and no further changes at the 6 months' follow up (day 180), compatible with a cardiac memory phenomenon: BiV, biventricular pacing; LBBB, left bundle branch block.
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Figure 2  Fluoroscopic image session showing CS angiography in LAO (A) and RAO (B) projections, and the final CS pacing lead site in LAO (C) and PA (D) projections: CS, coronary sinus; LAO, left anterior oblique; PA, postero-anterior; RAO, right anterior oblique.

Figure 3  12-Lead electrocardiogram recording during BiV pacing (CRT on) and during transient CRT inhibition (CRT off). At Day 180, temporary CRT inhibition (CRT off) documents a persistent LBBB, confirming that the QRS shortening and pattern is not a fusion beat complex of BiV pacing and spontaneous recovered conduction (i.e. intermittent LBBB). Of note, with respect to baseline LBBB, Day 180 LBBB showed a slightly modified QRS axis with a switch toward the left superior quadrant and positive T waves in V4–V6 (see also Figure 1). In this case, T wave vector does not persist in tracking the vector angle and amplitude of the spontaneous QRS complex (almost negative in V4–V6) that characterized the BiV Day 180, thus not supporting a phenomenon of ''reverse type of manifest cardiac memory''. More likely, LBBB Day 180th QRS and ST–T modifications compared to the LBBB baseline might be related to left ventricular remodeling after CRT: BiV, biventricular pacing; CRT, cardiac resynchronization therapy; LBBB, left bundle branch block.
As known, identifying reliable predictors of effectiveness of CRT remains a major challenge in clinical practice [10]. Although, on the basis of the known pathophysiology of electromechanical disorders, the change in QRS duration produced by BiV should represent the quality of electrical resynchronization and indirectly reflect the degree of correction of electromechanical abnormalities, the predictive role of QRS shortening is still debated [11,12]. The main problem is that individual reduction in QRS duration after CRT varies substantially among patients and therefore accurate prediction based on direct QRS reduction after CRT is not possible.

As repolarization changes according to depolarization pattern modifications, a manifestation of CM requires a “common” substantial change of QRS duration, from a longer one to a shorter one (for example from right pacing induced LBBB pattern toward a normal AV conduction). In the present case we observed a similar phenomenon in a super-responder patient, with CM manifestation only after an “uncommon” substantial change in QRS pattern and duration (i.e. moving from a spontaneous persistent LBBB to a shorter “pseudonormal” BiV induced QRS). From this point of view, CM after BiV could represent a secondary indirect ECG sign of “effective” modification of a primary pathologic (i.e. LBBB) ventricular electrical activation pattern toward a “pseudonormal” BiV one, with a final ventricular mechanical dyssynchrony readaptation.

More recent investigation has shown that memory accumulates during abnormal ventricular activation which is often hard to appreciate and requires careful measurements of QRS/T vectors or QRS/T isoareas. The reason that memory usually is not easily appreciated during pacing is explained by secondary T wave modulation. This report is remarkable in that T wave memory can easily be appreciated during biventricular paced rhythm. Noteworthy is that the QRS duration during biventricular paced rhythm in this patient is extremely narrow allowing us to see overt evidence of memory that should also be present in other patients with wider QRS complexes but is concealed by secondary T wave modulation.

In conclusion, is memory in BiV, then, the sign of reacquisition of a ‘forgotten’ normal activation/resynchronization ventricular pattern? Further studies could evaluate whether memory in BiV has an incremental predictive value for CRT response in addition to conventional ECG parameters.

References