Short- and Long-Term Effects of Left Bundle Branch Area Pacing on the T Wave of the ECG

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Abstract

Although left bundle branch area pacing (LBBAP) provides more physiological ventricular activation than conventional pacing techniques, its impact on ventricular repolarization remains unclear. This study investigates the temporal changes of the T wave in patients with bradycardia treated with LBBAP, using vectorcardiographic analysis of 12-lead ECGs. Standard 12-lead ECGs were collected from 23 healthy subjects and 58 patients with bradycardia who underwent LBBAP. ECGs were recorded at baseline for the two cohorts and at three additional time points after LBBAP for the bradycardia group. The principal direction of the T wave was obtained from vectorcardiographic analysis and classified into 4 quadrants (q_x). In healthy controls, all T wave vectors were located in q_1. In patients with bradycardia and narrow QRS, 80% of the T wave vectors lied in q_1 at baseline. A reduction in the number of T wave vectors belonging to q_1 was observed immediately after LBBAP (45%), followed by a significant increase in this number at 24 hours (75%) and again at one year post-implantation (80%) ($p<0.05$). Qualitatively similar results were obtained for patients with wide QRS at baseline. These findings suggest that, although LBBAP transiently alters the direction of ventricular repolarization, the T wave vector progressively realigns to a physiological pattern over time.

1. Introduction

Patients with bradycardia often require pacemaker implantation as part of antibradycardia therapy to maintain an adequate heart rate. Left bundle branch area pacing (LBBAP) is a recent technique that directly stimulates the left bundle branch of the cardiac conduction system. This results in more physiological ventricular depolarization [1] compared to conventional right ventricular pacing in which the myocardium is activated first and leads to slower and dyssynchronous ventricular activation.

Although extensive research has been conducted to assess the effects of LBBAP on the ventricular activation sequence and morphology of the QRS [2–4], its influence on ventricular repolarization, reflected in the T wave of the electrocardiogram (ECG), has received far less attention. To the best of our knowledge, few studies have reported changes in the T wave following LBBAP [5]. In published work, T-wave inversions were observed immediately after pacemaker implantation, but these inversions progressively resolved over time.

This study investigates the effects of LBBAP on ventricular repolarization over time by analyzing the T wave using standard 12-lead ECG recordings. Short- and long-term effects are studied, from immediately after pacemaker implantation to after one year of pacing, to evaluate changes in ventricular repolarization.

2. Methods

2.1. Study population

ECG signals were collected from two different populations: a healthy control group and a cohort of patients with bradycardia and indications for pacemaker implantation. The control group consisted of 23 healthy male volunteers, with a mean age of 63±3 years. The patient cohort comprised 58 patients who underwent LBBAP as antibradycardia therapy at Lozano Blesa Clinical University Hospital (Zaragoza, Spain), with a mean age of 77±6 years, of whom 29% were women. The primary indication for pacing in this population was type II atrioventricular block followed by complete atrioventricular block (34% and 29% of patients, respectively). Comorbidities included dyslipidemia (57%), hypertension (69%), and diabetes mellitus (36%). Patients were stratified according to the baseline QRS morphology and duration into three subgroups: narrow QRS, right bundle branch block (RBBB), and left bun-

dle branch block (LBBB). All healthy subjects presented a narrow QRS complex.

For both groups, 12-lead ECG recordings were obtained at baseline. Furthermore, in the bradycardia group, additional ECG recordings were collected at three time points after implantation: immediately after pacemaker implantation, 24 hours after implantation, and one year after implantation. ECGs were recorded with a sampling frequency of 1000 Hz and an amplitude resolution of 3.75 µV.

2.2. Signal processing and median beat computation

ECG preprocessing included removal of baseline wander and low-pass filtering with a cutoff frequency of 50 Hz. In the paced ECGs of patients with bradycardia, spikes from pacemaker activity were removed from the ECG. For that purpose, the strategy described in [6] was implemented, which involves identifying the start and end of the spikes using a threshold-based criterion on the ECG derivative and replacing them with linear interpolation.

The preprocessed ECG signals were delineated using a wavelet-based multilead approach [7], allowing the identification of QRS fiducial points for all cardiac beats. RR intervals were calculated from consecutive QRS fiducial points. Beats with RR intervals within a 20 ms window centered on the RR mode were selected to compute an initial median beat. To remove extrasystole beats and include only beats with similar QRS morphology, only cardiac beats whose QRS complexes exhibited a Pearson correlation coefficient greater than 0.95 with the initially calculated median beat were included in the final QRS selection. A refined median beat was computed from those selected beats.

The time instants corresponding to the fiducial point, onset and end of the QRS complex, and the end of the T wave were identified in the final median beats using the same multilead wavelet-based approach [7].

2.3. Vectocardiograhy analysis

The vectocardiogram (VCG), including leads X, Y, and Z, was derived from the calculated 12-lead median beat using the Kors regression method [8]. The Euclidean norm of the VCG (n_{XYZ}) was calculated at each instant in time. The onset of the T wave was identified by searching the time point with the minimum value in n_{XYZ} within a predefined window of 20 to 40 ms after the end of the QRS complex. This strategy was adopted to improve delineation accuracy given the challenges associated with identifying the onset of the T wave. Finally, the VCG signal was baseline-corrected by subtracting the mean of the values

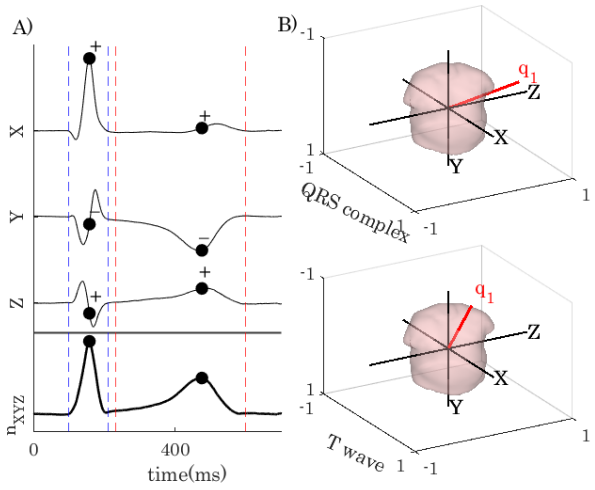


Figure 1. (A) X, Y, and Z leads derived from 12-lead standard ECG using the Kors regression method, along with the computed vector magnitude, n_{XYZ}. Black dots mark the time points corresponding n_{XYZ} maximum during QRS complex and the T wave. Blue dashed lines indicate the boundaries of the QRS complex, while red dashed lines indicate the T wave boundaries. (B) 3D vector representation of the principal direction of the QRS complex (top panel) and T wave (bottom panel) in q_1 indicated by the red vectors.

of n_{XYZ} at QRS_{onset}, QRS_{end}, T_{onset}, and T_{end} and subsequently normalized by the maximum value of n_{XYZ}.

The principal directions for the QRS complex and the T wave were calculated from the VCG by identifying the time point where n_{XYZ} took the maximum value within the corresponding wave window (Figure 1A). The vectors associated with the principal directions of the QRS complex and the T wave were classified into four spatial quadrants q_x, based on the x and z coordinates: q_1 for positive x and z; q_2 for negative x and z; q_3 for positive x and negative z; and q_4 for negative x and positive z (see Figure 1B).

The angle between the X-axis and the projection of the T wave dominant vector into the X-Z plane (transversal plane) was calculated for each ECG recording of healthy volunteers and bradycardia patients and denoted as θ^{T}.

2.4. Statistics

For each quadrant q_x, the proportion of ECG recordings in which the principal direction of the QRS complex (T wave, respectively) fell within that quadrant was calculated. The χ^2 test of independence was performed to evaluate the association between percentages in a given quadrant in different temporal states. The Wilcoxon rank-sum test was used to analyze differences in θ^{T} between healthy

subjects at baseline and patients with bradycardia after a year with LBBAP. P-values < 0.05 were considered statistically significant.

3. Results and discussion

3.1. Principal directions at baseline

In the healthy group, 91% of the principal directions of the QRS complexes and 100% of the T wave vectors were found to be in q_1, indicating a left anterior direction.

Among patients with bradycardia, the principal direction of the QRS complex varied depending on the morphology of the baseline QRS. Patients with narrow QRS showed the highest percentage of principal directions of the QRS in q_1 (75%), as observed for healthy individuals (p=0.15). In contrast, patients with RBBB and LBBB showed broader distributions across q_1 and q_3 due to conduction abnormalities. For the T wave, 80%, 70% and 64% of patients with narrow QRS, RBBB and LBBB, respectively, had the principal direction in q_1 at baseline (Figure 2).

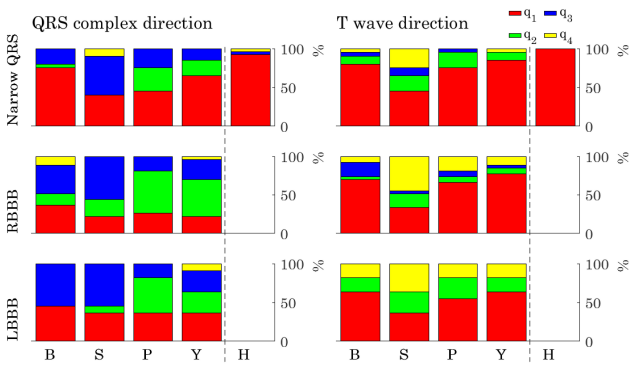


Figure 2. Percentage of the principal direction of the QRS complex (left) and T wave (right) across quadrants q_x, (with q_1 in red, q_2 in green, q_3 in blue, and q_4 in yellow) for bradycardia cohort at baseline (B), immediately after (S), after 24 hours (P), after one year (Y) and healthy population (H).

3.2. LBBAP effects on QRS complex and T wave principal directions

The principal direction of the paced QRS complex showed differences in the three pacing states compared to baseline, reflecting the altered ventricular activation pattern induced by LBBAP. After a year, the group of patients with narrow QRS recovered up to 65% of their depolarization vectors located within q_1, approaching the distribution observed at baseline (p=0.57). In contrast, the groups of patients with RBBB and LBBB showed broader distributions of QRS complex vectors, with 22% and 36% in q_1 (Figure 2).

Regarding the T wave, in the group of bradycardia patients with narrow QRS, the number of patients who had the direction of the T wave within q_1 showed a decrease immediately after LBBAP, falling from 80% to 45% (p=0.07). After 24 hours of pacing, the percentage increased to 75% (p=0.03). After one year, it exceeded the percentage at baseline and reached 85% (p=0.003) (Figure 3).

In patients with bradycardia and RBBB, the percentage of patients with the principal direction of the T wave within q_1 decreased from 70% at baseline to 33% immediately after pacemaker implantation (p=0.02). After 24 hours and a year of implantation, the proportion increased to 67% (p=0.008) and 78% (p<0.001) (Figure 3).

Patients with bradycardia and LBBB exhibited a reduction in the percentage of the principal directions of the T wave within q_1 right after pacemaker implantation, decreasing from 64% at baseline to 36% immediately after pacemaker implantation (p = 0.20). This percentage increased in the following temporal states, reaching 55% after 24 hours and returning to baseline levels (64%) after a year of pacing (Figure 3). No significant differences in the percentage of vectors in q_1 were observed, possibly due to the small sample size of the LBBB subgroup in our cohort.

In general, for all subgroups, LBBAP modified the QRS complex vector immediately after pacemaker implantation. However, while patients with narrow QRS partially recovered their initial vector distribution over time, this recovery was not observed in the RBBB and LBBB cohorts, which maintained a distinct QRS complex quadrant distribution compared to their distributions at baseline.

Regarding the T wave, LBBAP induced acute changes in its direction immediately after pacing in all subgroups of patients. However, in all cases, the T wave vector progressively realigned with a more physiological direction, with the highest percentage of T wave directions observed in q_1 after 24 hours and a year of LBBAP, comparable to the direction seen in the healthy population.

Repolarization abnormalities are known to potentially induce arrhythmias. In this study, the T wave was affected immediately after LBBAP, but it progressively returned to a more physiological pattern over time suggesting a ventricular adaptation with long-term pacing. These findings are consistent with those reported in the study by Geng et al. [5], where T wave inversions were frequently observed after LBBAP. Inversions of T waves occurred in approximately 66% of the patients and were shown to be transient, with partial or complete recovery in almost 88% of the cases during follow-up.

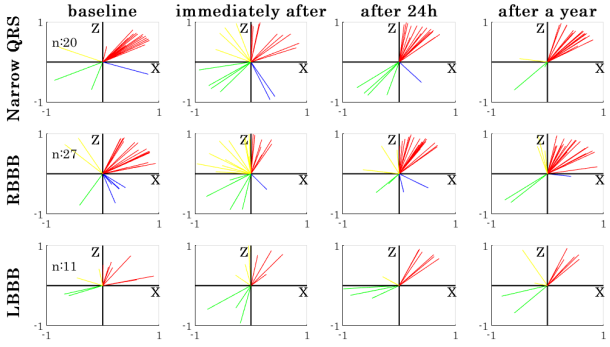


Figure 3. T wave principal direction in the X-Z plane for bradycardia cohort at baseline and after LBBAP (inmediatly after pacemaker implantation,after 24 hours and after a year) for narrow QRS, RBBB and LBBB patients. Each line represents an individual T wave vector, with direction color-coded according to its quadrant assignment: q_1 in red, q_2 in green, q_3 in blue, and q_4 in yellow.

3.3. $\theta^{\mathbf{T}}$ after a year of LBBAP

The angle θ^{T}, defined as the angle between the X axis and the projection of the T wave vector onto the X-Z plane, was $39\pm10°$ in the healthy group, reflecting a physiological repolarization pattern. This value was significantly lower ($p<0.001$) than the values measured in patients with bradycardia and narrow QRS ($53\pm13°$), RBBB ($55\pm17°$) and LBBB ($59\pm8°$) after one year of LBBAP. This suggests that, despite directional similarity, repolarization after pacing remains quantitatively different from that in healthy individuals.

Our findings with respect to θ^{T} indicated that although most T wave vectors aligned in the direction with those of healthy individuals (predominantly within q_1), its orientation in terms of the values of θ^{T} remained quantitatively distinct. In particular, the increased angle values after LBBAP suggest that repolarization does not fully achieve a physiological state despite apparent directional similarity.

4. Conclusion

This study evaluated the short- and long-term impact of LBBAP on ventricular repolarization by analyzing the temporal evolution of the T wave dominant vector from the ECG. Our findings indicate that, although LBBAP induces acute alterations in the polarity of the T wave in patients with bradycardia, it gradually reverts to a physiological pattern over time. After one year of pacing, the T wave displays a qualitatively similar directional profile to that found in healthy subjects, even if some quantitative differences are still observed.

Acknowledgments

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