Reversible T-wave inversions during left bundle branch area pacing

Jie Geng*, Zhixin Jiang*, Shigeng Zhang, Xiujuan Zhou, Yuanyuan Chen, Meng Chen, Chongchong Chen, Qijun Shan

Department of Cardiology, 1st Affiliated Hospital of Nanjing Medical University, Nanjing, China
*Both authors equally contributed to the study

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Correspondence to:
Qijun Shan, MD, PhD, FHRS, Department of Cardiology, 1st Affiliated Hospital of Nanjing Medical University, Nanjing, 210029, China, phone: +86 13 505 142 015, e-mail: qjshan@njmu.edu.cn

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ABSTRACT

Background: Our clinical observation found that T-wave inversions (TWIs) appeared during left bundle branch area pacing (LBBAP); however, the incidence and influencing factors were unclear. The study aimed to investigate the effects of LBBAP on T-wave and explore possible factors associated with TWIs.

Methods: This was a retrospective cohort study. An electrocardiogram (ECG) was acquired at baseline and after LBBAP. Baseline characteristics, ECG parameters, LBBAP parameters, and troponin T (TnT) levels were compared between the non-TWIs and TWIs groups. Multivariable logistic analyses were performed to adjust for potential confounders to identify the predictive factors of TWIs during LBBAP.

Results: A total of 398 consecutive patients who underwent successful LBBAP were assessed for inclusion between May 2017 and Jan 2021, and 264 (66.3%) patients had TWIs. The mean (standard deviation [SD]) baseline QRS duration (QRSd) was longer in the TWIs group compared to the non-TWIs group (125.9 [34.5] ms vs. 98.2 [18.1] ms; P <0.001). Multivariable logistic regression analysis suggested that QRSd >120 ms was an independent predictor for TWIs. TWIs were partially or completely recovered in 151/172 (87.8%) patients during follow-up, the median (interquartile range [IQR]) follow-up duration was 10 days (7 days to 5.5 months). TWIs in patients with complete left bundle branch block (CLBBB) occurred more frequently in inferior wall leads (II, III, and aVF) and anterior wall leads (V1–V4) (P <0.05). Patients with complete right bundle branch block (CRBBB) were more prone to TWIs in high lateral wall leads (I and aVL) (P <0.05). There were no significant differences in TnT levels between the TWIs and non-TWIs groups.

Conclusions: TWIs during LBBAP were clinically frequent and recoverable. QRSd >120 ms was independently associated with TWIs.

Key words: cardiac memory, complete left bundle branch block, complete right bundle branch block, left bundle branch area pacing, T-wave inversions

INTRODUCTION

We first demonstrated the transient and recoverable T-wave inversions (TWIs) during left bundle branch area pacing (LBBAP) in a patient with prior temporary right ventricular (RV) pacing [1]. TWIs may be caused by cardiac memory (CM). LBBAP is an established treatment option for patients with symptomatic bradycardia [2], especially for patients with heart failure (HF) and a wide QRS complex [3]. Several studies reported the development of TWIs after resumption of normal cardiac conduction in patients undergoing RV pacing or cardiac resynchronization therapy (CRT) [4, 5]. TWIs were a common but infrequently recognized phenomenon, of which many

WHAT'S NEW?

To our knowledge, there are no cohort studies of left bundle branch area pacing (LBBAP) induced T-wave inversions (TWIs), and this is the first report o describe this phenomenon. The main findings are that: (1) TWIs during LBBAP were clinically frequent (66.3%) and recoverable (87.8%); (2) TWIs in patients with complete left bundle branch block (CLBBB) occurred more frequently in inferior and anterior leads; (3) Patients with complete right bundle branch block (CRBBB) were more prone to TWIs in high lateral wall leads; (4) Baseline QRS duration (QRSd) >120 ms predicts TWIs during LBBAP.

clinical practitioners are unaware, particularly in patients during LBBAP.

This study aimed to (1) investigate the epidemiology and characteristics of TWIs during LBBAP and (2) explore possible factors associated with TWIs.

METHODS

Study population

This was a retrospective cohort study conducted between May 2017 and Jan 2021 in the 1st Affiliated Hospital of Nanjing Medical University. Consecutive patients with a pacemaker indication according to the 2013 European Society of Cardiology guidelines [6] and those who also underwent attempts for LBBAP implantation were assessed. The study protocol was approved by the Institutional Review Board of the 1st Affiliated Hospital of Nanjing Medical University (2021-SR-211), and all patients gave written informed consent.

LBBAP procedure

The technical details of the LBBAP procedure had been described in previous reports [7, 8]. The pacing threshold, sensing and impedance of the 3830 lead (Medtronic, Minneapolis, MN, US) were recorded during operation. Successful LBBAP was defined as unipolar paced QRS with right bundle branch block (RBBB)-like morphology and QRSd ≤130 ms.

Data collection

All patients underwent a full clinical evaluation before the procedure, including their comorbidities (such as hypertension, coronary artery disease, diabetes mellitus, atrial fibrillation [AF], and stroke), indications for permanent pacemaker implantation, ECG parameters, and pacemaker history. Standard 12-lead ECGs were interpreted by two cardiologists. A standard 12-lead ECG was done before LBBAP. ECG data were collected and recorded including native QRS width, heart rate, paced V6 R-wave peak time, native QRS type (narrow, intraventricular conduction disturbance [IVCD], complete left bundle branch block (CLBBB) morphology [including CLBBB and CLBBB-like pattern during RV pacing] and complete right bundle branch block [CRBBB] morphology). All ECG parameters were rechecked and recorded immediately after LBBAP. ECG was performed at baseline and on the first day post-LBBAP.

During the follow-up period, ECG data of patients were collected and compared with the previous ECG. For each recording, T-wave direction of 12-leads was calculated. TWIs were defined as negative or isoelectric T-wave in leads I, II, III, aVL, aVF, V1–V6, or the presence of a positive or isoelectric T-wave in lead aVR. TWIs in two or more contiguous leads were considered significant. Accordingly, patients were divided into two groups: the non-TWIs group and the TWIs group.

Statistical analysis

Continuous variables were expressed as mean (standard deviation [SD]) for normally distributed variables, and categorical variables were expressed as frequencies and percentages. Non-normally distributed variables were expressed as the median with the interquartile range (IQR). Baseline characteristics were compared between patients with and without TWIs, using an independent-samples t-test, pairwise t-test, or Wilcoxon matched-pairs signed-ranks test for continuous data, and the χ^2 test for dichotomous data. Logistic regression analysis was performed to identify the predictive factors of TWIs. All the variables with a *P*-value <0.05 in the univariate analysis (Supplementary material, *Table S1*) were included in the multivariable logistic regression analyses. In the multivariable logistic regression analyses, QRSd <90 ms, 90 ms <QRSd ≤120 ms, 120 ms <QRSd ≤150 ms, and QRSd >150 ms were analyzed separately after adjustment for clinical variables (age, sex, and medical history), and TWIs were presented as odds ratios (ORs) with corresponding 95% confidence intervals (CIs). The association between related factors two-sided *P*-value <0.05 was considered statistically significant. All statistical analyses were performed using the SPSS software (version 23.0).

RESULTS

Baseline characteristics

Overall, a total of 494 consecutive patients were assessed for inclusion between May 2017 and Jan 2021. Based on the definition of success provided above, successful LBBAP was achieved in 447 (90.5%) patients. Forty-nine cases without ECG on the day of operation were excluded (Supplementary material, *Figure S1*). Therefore, the study population consisted of 398 patients for further analysis. The baseline characteristics of study participants are

Table 1. Baseline characteristics of study participants according to TWIs during LBBAP

Patient characteristics	All patients (n = 398)	Non-TWIs group (n = 134)	TWIs group (n = 264)	*P*-value Non-TWIs vs. TWIs
Age, years, mean (SD)	69.8 (11.3)	67.3 (11.0)	71.1 (11.3)	<0.01
Male sex, n (%)	202 (50.8)	63 (47.0)	139 (52.7)	0.29
Medical history, n (%)				
Hypertension	252 (63.3)	78 (58.2)	174 (65.9)	0.13
Diabetes mellitus	78 (19.6)	19 (14.1)	59 (22.3)	0.052
Coronary heart disease	76 (19.1)	20 (14.9)	56 (21.2)	0.13
Atrial fibrillation	121 (30.4)	45 (32.4)	76 (28.8)	0.33
Stroke	63 (15.8)	19 (14.2)	44 (16.7)	0.52
Pacing indication, n (%)				<0.001
SSS	148 (37.2)	65 (48.5)	83 (31.4)	
AVB	200 (50.2)	66 (49.3)	134 (50.8)	
BBB	11 (2.8)	1 (0.7)	10 (3.8)	
Heart failure with CLBBB	39 (9.8)	2 (1.5)	37 (14.0)	
Native QRS type, n (%)				<0.001
Narrow	253 (63.5)	122 (91.0)	131 (49.6)	
IVCD	14 (3.5)	3 (2.2)	11 (4.2)	
CLBBB morphology	87 (21.9)	2 (1.5)	85 (32.2)	
CRBBB morphology	44 (11.1)	7 (5.2)	37 (14.0)	
ECG parameters, mean (SD)				
Intrinsic QRSd (ms)	116.6 (32.7)	98.2 (18.1)	125.9 (34.5)	<0.001
LBBAP QRSd (ms)	107.3 (11.7)	103.6 (11.5)	109.1 (11.4)	<0.001
Pacemaker history, n (%)				
Temporary pacemaker	26 (6.5)	4 (3.0)	22 (8.3)	0.04
DDD/VVI pacemaker	27 (6.8)	1 (0.7)	26 (9.8)	<0.01
CRT/CRTD/BIVP device	6 (1.5)	0	6 (2.3)	0.10

Abbreviations: AVB, atrioventricular block; BBB, bundle branch block; BIVP, bi-ventricular pacing; CLBBB, complete left bundle branch block; CRBBB, complete right bundle branch block; CRT, cardiac resynchronization therapy; CRTD, CRT with defibrillator; DDD, dual-chamber pacemaker; ECG, electrocardiogram; IVCD, intraventricular conduction disturbance; LBBAP, left bundle branch area pacing; QRSd, QRS duration; SD, standard deviation; SSS, sick sinus syndrome; TWIs, T-wave inversions; VVI, single-chamber pacemaker

summarized in Table 1. The mean (SD) age of these patients was 69.8 (11.3) years, and 202 (50.8%) were men. The patients had a high proportion of comorbidities, including hypertension (63.3%), diabetes mellitus (19.6%), coronary heart disease (19.1%), AF (30.4%), and stroke (15.8%). Two hundred and sixty-four of 398 (66.3%) patients had TWIs during LBBAP. The mean (SD) age of patients in the TWIs group was higher (71.1 [11.3] years vs. 67.3 [11.0]) years; *P* <0.01). The percentages of CLBBB (85/264 vs. 2/134; *P* <0.001) and CRBBB (37/264 vs. 7/134; *P* <0.01) were significantly higher in the TWIs group than that in the non-TWIs group. The mean (SD) of intrinsic QRSd (125.9 [34.5] ms vs. 98.2 [18.1] ms; *P* <0.001), and LBBAP QRSd (109.1 [11.4] ms vs. 103.6 [11.5] ms, *P* <0.001) were significantly longer in patients with TWIs than in patients without TWIs. As for the background pacemaker history, TWIs group patients received a higher proportion of temporary pacing (*P* = 0.04) and permanent pacemaker (*P* <0.01). There were no significant differences between the two groups in terms of sex or medical history.

LBBAP procedural and R-wave peak time in V6 characteristics

For 398 patients with successful LBBAP, the mean (SD) threshold was 0.6 (0.2) v, mean (SD) sensing was 12.5 (6.6) mv, and the mean (SD) impedance was 784.4 (269.0) Ω during implantation. There was no statistically significant difference in the parameters of LBBAP between the non-TWIs and TWIs groups. The mean (SD) paced V6 R-wave peak time (84.7 [13.5] ms vs. 78.2 [14.9] ms; *P* <0.001) was significantly longer in patients with TWIs than in patients without TWIs. The implantation procedure-related characteristics of the included patients are given in Table 2.

TWIs during LBBAP

Two hundred and sixty-four of 398 (66.3%) patients had T-wave changes after LBBAP. TWIs occurred immediately during LBBAP operation (Figures 1–3). Table 3 summarized TWIs on 12-lead ECG under treatment with LBBAP in patients with CLBBB or CRBBB. These patients were divided into two subgroups according to baseline QRS morphology (G1 — CLBBB morphology and G2 — CRBBB morphology). Compared with G2, TWIs occurred more frequently in leads: II (41/85 vs. 7/37; *P* <0.001), III (41/85 vs. 9/37; *P* <0.001), aVF (41/85 vs. 9/37; *P* <0.001),V1 (43/85 vs. 1/37; *P* <0.001),V2 (61/85 vs. 10/37; *P* <0.001), V3 (71/85 vs. 15/37; *P* <0.001), and V4 (70/85 vs. 21/37; *P* <0.001). TWIs occurred more commonly in leads I (16/85 vs. 22/37; *P* <0.001) and aVL (12/85 vs. 23/37; *P* <0.001) in patients with CRBBB than in patients with CLBBB.

Table 2. Parameters of left bundle branch area pacing and V6 R-wave peak time

	All patients (n = 398)	Non-TWIs group (n = 134)	TWIs group (n = 264)	*P*-value Non-TWIs vs. TWIs
Threshold, v, mean (SD)	0.6 (0.2)	0.6 (0.3)	0.6 (0.2)	0.97
Sensing, mv, mean (SD)	12.5 (6.6)	12.2 (6.7)	12.7 (6.5)	0.42
Impedance, Ω, mean (SD)	784.4 (269.0)	756.1 (164.4)	798.7 (308.1)	0.14
V6 RWPT, ms, mean (SD)	82.5 (15.1)	78.2 (14.9)	84.7 (13.5)	<0.001

Abbreviations: RWPT, R-wave peak time; other — see Table 1

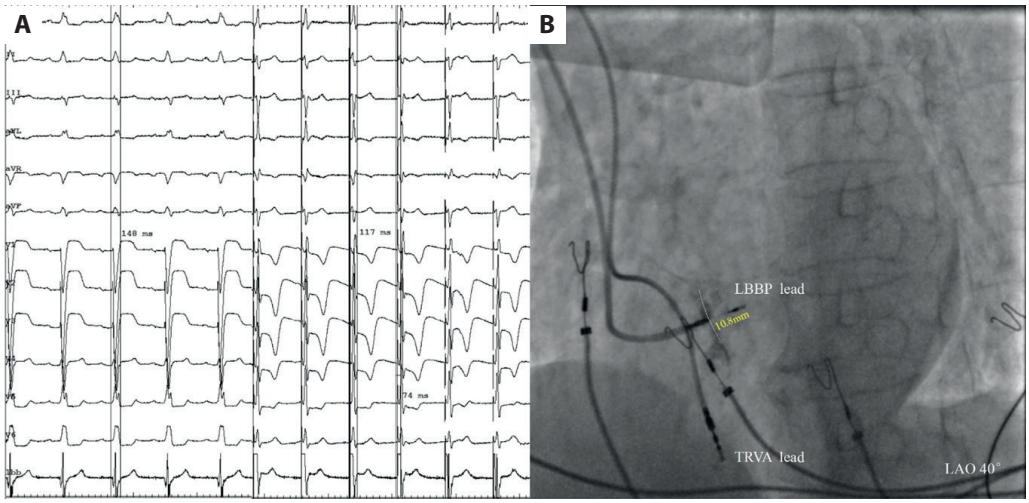

Figure 1. Immediate T-wave inversions on electrocardiogram (ECG) (V1–V5) during left bundle branch area pacing (LBBAP) in a patient with complete left bundle branch block (LBBP). We attempted to perform LBBAP on a patient (83 years/male) with heart failure and left bundle branch block (QRS duration [QRSd], 148 ms). Before the procedure, his computer tomography angiography work-up showed normal coronary arteries; the New York Heart Association (NYHA) class was III, and his left ventricular ejection fraction was 41.9%. During the procedure, when the tip reached the area of LBB, the unipolar pacing showed right bundle branch block liked morphology with QRSd 117 ms and Sti-LVAT 74 ms (**A**, continuous ECG and intracardiac electrogram record with speed 25 mm/sec). The implant depth of LBBAP lead was 10.8 mm (**B**, sheath angiography in the left anterior oblique [LAO] 40° view). After the procedure, his heart failure symptoms (NYHA I) significantly improved at follow-up of 3 months

Abbreviations: TRVA, temporary right ventricular apex

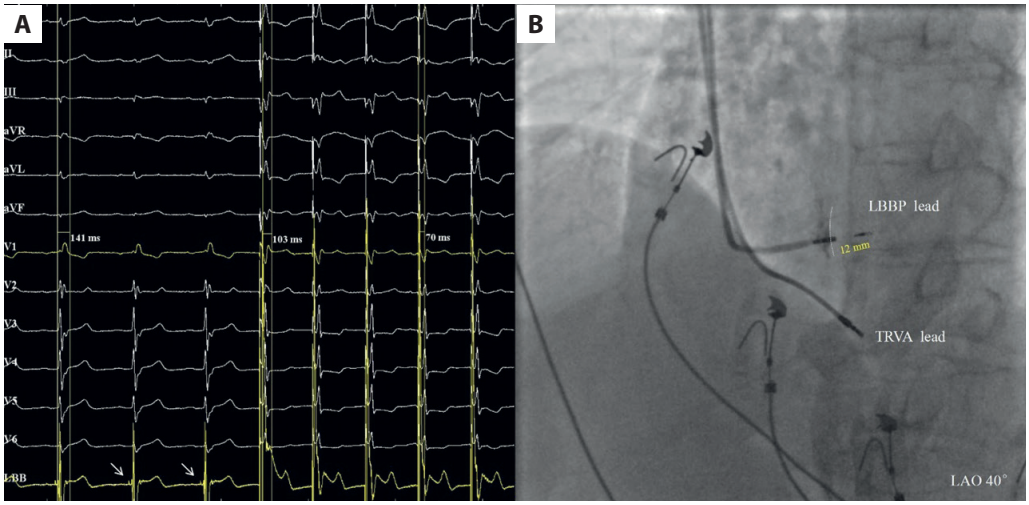

Figure 2. Immediate T-wave inversions on ECG (I, III, aVL, aVR, and V3–V6) during LBBAP in a patient with CRBBB. An 86-year-old man was admitted to our institution with recurrent syncope. Holter showed an intermittent AVB with a QRS complex morphology of CRBBB (QRSd, 141 ms). Given the documented symptomatic conduction trouble at the level of the AV node (HV interval 75 ms) and the existence of CRBBB, the patient was considered to be indicated for LBBAP. During the procedure, a sharp LBB potential pre-QRS was seen (the arrow) when conduction occurs *via* the left bundle, resulting in a narrow complex. When the tip reached the area of LBB, the unipolar pacing showed RBBB-like morphology with QRSd 103 ms and Sti-LVAT 70 ms (**A**, continuous ECG and intracardiac electrogram record with speed 50 mm/sec). Sheath angiography in the LAO 40° confirmed deep insertion (12 mm) of the LBBP lead into the septum (see **B**). No more syncope occurred during the follow-up

Abbreviations: CRBBB, complete right bundle branch block; other — see Figure 1

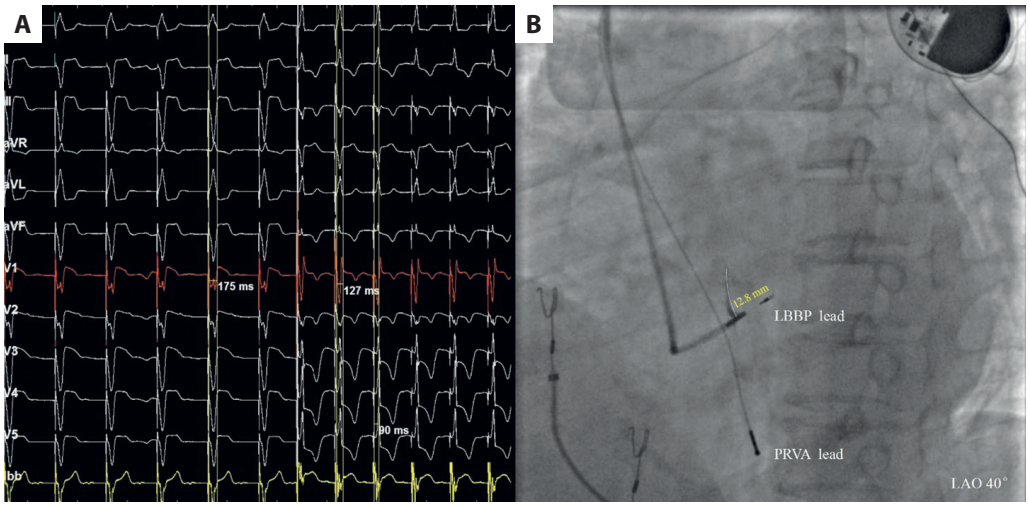

Figure 3. Immediate TWIs on ECG (II, III, aVL, aVR, aVF, and V1–V6) during LBBAP in a patient with RV pacing. A 60-year-old female with advanced AVB had undergone implantation of a single-chamber pacemaker about 9 years earlier. Holter indicated that the rate of RV pacing was 100 percent in external hospital. Given the important interventricular dyssynchrony due to RV pacing, as well as serious tricuspid valvular regurgitation, we upgraded RV pacing to LBBAP. **A.** Surface ECG and intracardiac electrograms from the LBBAP lead are shown at a sweep speed of 25 mm/sec. Intrinsic electrogram showed CLBBB-like morphology with QRS duration 175 ms; unipolar pacing electrogram of LBBAP showed RBBB-like morphology with QRS duration 127 ms and Sti-LVAT 90 ms. **B.** Sheath angiography in the LAO 40° view demonstrated the depth of the LBBP lead (12.8 mm) inside the septum (the dotted line)

Abbreviations: PRVA, permanent right ventricular apex; other — see Figure 1

Table 3. Incidence of T-wave inversions on 12-lead electrocardiogram

	CLBBB group (n = 85)	CRBBB group (n = 37)	*P*-value
Limb leads			
I	16 (18.8%)	22 (59.5%)	<0.001
II	41 (48.2%)	7 (18.9%)	<0.01
III	41 (48.2%)	9 (24.3%)	0.01
aVR	49 (57.6%)	24 (64.9%)	0.46
aVF	41 (48.2%)	9 (24.3%)	0.01
aVL	12 (14.1%)	23 (62.2%)	<0.001
Precordial leads			
V1	43 (50.6%)	1 (2.7%)	<0.001
V2	61 (71.8%)	10 (27.0%)	<0.001
V3	71 (83.5%)	15 (40.5%)	<0.001
V4	70 (82.4%)	21 (56.8%)	<0.01
V5	63 (74.1%)	28 (75.7%)	0.86
V6	49 (57.6%)	24 (64.9%)	0.46

Abbreviations: aVR, augmented vector right; aVF, augmented vector foot; aVL, augmented vector left; other — see Table 1

Predictors for TWIs

After multivariable adjustment for the confounding factors such as sex, age, and comorbidities (hypertension, diabetes mellitus, coronary heart disease, AF, and stroke), 120 ms <QRSd ≤150 ms (OR, 7.59; 95% CI, 2.88–19.96; *P* <0.001) and QRSd >150 ms (OR, 28.06; 95% CI, 8.40–93.71; *P* <0.001) independently predicted TWIs during LBBAP (Table 4).

Recovery of TWIs

One hundred and seventy-two of 264 patients had LBBAP-ECG at 3 days post LBBAP or during the follow-up. Figure 4 showed dynamic changes of T-wave on ECG after LBBAP in a patient with CLBBB. During the follow-up period, the median (IQR) follow-up duration was 10 days (7 days to 5.5 months). One hundred and fifty-one (87.8%) patients were found to have partial or complete recovery from TWIs, while 21 (12.2%) patients still had TWIs at follow-up (Supplementary material, *Figure S2*).

Effect of LBBAP on troponin T

Troponin T (TnT) was tested in 123 of 398 patients at baseline and 12 hours after operation, 83 of 123 had TWIs during LBBAP. Compared with those before implantation, the mean (SD) levels of TnT in all patients (11.9 [7.7–18.8] ng/l vs. 56.3 [37.7–120.1] ng/l; *P* <0.001), the non-TWIs group (9.6 [6.6–16.2] ng/l vs. 56.3 [35.0–124.7] ng/l; *P* <0.001), and the TWIs group (12.6 [8.5–20.6] ng/l vs. 53.8 [39.3–115.4]

Table 4. Multivariable logistic analysis for T-wave inversions

Intrinsic QRSd (ms)	Non-TWIs (n = 134)	TWIs (n = 264)	Multivariable analysis	
			OR (95% CI)	*P*-value
QRSd ≤90, n (%)	49 (36.5)	38 (14.4)	1.00	
90 <QRSd ≤120, n (%)	73 (54.5)	95 (36.0)	1.73 (0.88–3.38)	0.11
120 <QRSd ≤150, n (%)	8 (6.0)	54 (20.4)	7.59 (2.88–19.96)	<0.001
QRSd >150, n (%)	4 (3.0)	77 (29.2)	28.06 (8.40–93.71)	<0.001

The multivariable logistic regression analyses model adjusted for clinical variables (age, sex, and medical history)

Abbreviations: see Table 1

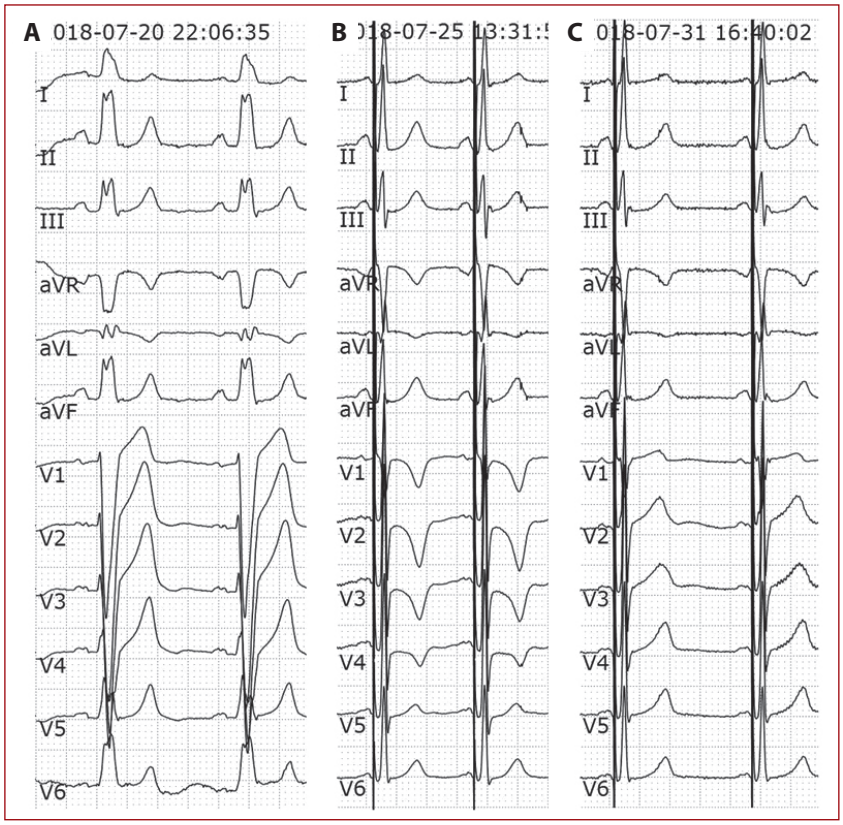

Figure 4. Dynamic changes of T-wave on ECG after LBBAP in a patient with CLBBB. **A.** Baseline ECG showed LBBB. **B.** ECG on first day post-LBBAP showed T-waves inverted in V1–V4; **C:** ECG 6 days post-procedure showed that T-waves returned to normal

Abbreviations: see Figure 1

ng/l; *P* <0.001) increased significantly after LBBAP. On the other hand, there was no statistical significance in TnT levels between the non-TWIs and TWIs groups.

DISCUSSION

To our knowledge, there are no cohort studies of LBBAP-induced TWIs, and this is the first report to demonstrate this phenomenon. The main findings are that: (1) 66.3% of patients have TWIs during LBBAP; (2) the prevalence of TWIs in inferior leads (II, III, and aVF) and anterior leads (V1–V4) is significantly higher than high side leads (I and aVL) in CLBBB patients, which is contrary to patients with CRBBB; (3) baseline QRSd >120 ms predicts TWIs during LBBAP; (4) TWIs are reversible in 87.8% patients; and (5) TWIs during LBBAP may be unrelated to acute myocardial ischemia.

TWIs and CM

Rosenbaum et al. [9] attributed TWIs to CM, as a change in myocardial repolarization manifested by a persistent change in the T-wave axis after restoration of normal cardiac excitation. They also described the property of accumulation in cardiac memory cells, where the magnitude of TWIs increased with repetitive activation by pacing or CLBBB and persisted for longer periods with increasing duration of the altered activation. TWIs due to CM is a frequently encountered electrical phenomenon that appears after the cessation of a period of abnormal ventricular depolarization. It occurs in response to several conditions including Wolff-Parkinson-White syndrome [10], ventricular arrhythmia [11], and ventricular pacing [12]. Previous studies showed that 59% of patients with Wolff-Parkinson-White syndrome had TWIs after undergoing successful catheter ablation [13] and TWIs after termination of idiopathic left ventricular tachycardia (ILVT) in 9/16 (56%) patients [14]. Grimm [12] has reported TWIs observed in one-third of patients following pacemaker implantation, with more than four-fifths of patients developing CM if the ventricular stimulation burden was 75% or greater. In the present

study, 264 of 398 (66.3%) patients had TWIs during LBBAP. This suggests that T-wave changes following LBBAP are not uncommon.

TWIs during LBBAP

LBBAP has recently emerged as a new promising pacing modality. During LBBAP, the His-Purkinje system was swiftly recruited by advanced activation of the area of the LBB trunk or proximal left anterior or posterior fascicle, which leads to good electrical synchrony and a short-paced QRS duration. Hou et al. [15] showed that cardiac electrical and LV mechanical synchrony of LBBAP were superior to that of right ventricular systolic pressure (RVSP) and similar to that of His bundle pacing. Previous studies also demonstrate that LBBP maintains ventricular synchrony at a level close to normal [16]. CM occurred after the ventricular activation altered or returned to normal because the changes in repolarization remained. Successful LBBAP can achieve narrow QRS complexes and maintain good LV electrical and mechanical synchrony, especially for patients with wide QRS complexes before operation, which can change ventricular activation sequence. This may be the reason for TWIs in patients during LBBAP.

CM had the property of accumulation, where the magnitude of the T-wave changes increased with repetitive activation by pacing or LBBB and persisted for longer periods with increasing duration of the altered activation [9]. During follow-up, we found that TWIs were partially or completely recovered in 151/172 (87.8%) patients. This was a retrospective study, and some patients only had a follow-up ECG 3 days to one week after LBBAP. This may be one reason why TWIs remained in 12.2% of patients. CM due to ventricular stimulation is benign and should not be confused with similar T-wave inversions due to acute coronary syndrome, ventricular hypertrophy, or myocarditis.

In our research, we found that TnT increased observably after LBBAP. The results are similar to our previous reports [17]. Meanwhile, we observed there were no significant differences in TnT levels and coronary heart disease prevalence between the TWIs and non-TWIs groups at baseline. These findings may indicate that TWIs may be unrelated to coronary heart disease.

TWIs in different leads

Another important result of the study is the observation of the QRS morphology before LBBAP, resulting in TWIs occurring in different ECG leads. Jeyaraj [18] found that there was mild action potential prolongation in the myocardial region that was close to the site of pacing (early activated), while significant action potential prolongation was noted in the myocardial region that was farthest from the site of pacing (late activated). The amplification of repolarization gradients between segments of the left ventricle is the electrophysiological basis for T-wave memory. The LV was activated only through transmyocardial conduction starting after the RV activation onset, and then the activation spread centrifugally over the anterior and inferior wall in patients with LBBB-like patterns [19]. The maximum repolarization gradients in patients with CLBBB are the anterior and inferior walls. Thus, TWIs in patients with CLBBB before LBBAP occurred more frequently in inferior leads (II, III, and aVF) and anterior leads (V1–V5). A previous study also found that patients with intermittent left bundle branch block frequently have TWIs in right and mid-precordial leads during normal conduction [20]. On the other hand, ventricular activation started in the inferior wall or lower septum of the LV and propagated toward the LV anterior or anterolateral walls in patients with RBBB [21]. Hence, patients with CRBBB were more prone to TWIs in high lateral wall leads (I and aVL).

Predictors of TWIs during LBBAP

Previous studies showed memory T-waves can be triggered by any conditions which produce wide QRSd transiently [5]. In this study, we also found that T-wave alterations occurred more frequently in patients with ventricular conduction abnormalities. One hundred and twenty-two of 264 (46.2%) patients had obvious conduction abnormalities (CLBBB morphology: 85 patients, CRBBB morphology: 37 patients), which is more frequent than that in the non-TWIs group (9/134, 6.7%). It was observed that the mean (SD) QRSd was longer (98.2 [18.1] ms vs. 125.9 [34.5] ms; P <0.001), and a higher percentage of CLBBB (2/134 vs. 85/264; P <0.001) was in the TWIs group. Multiple regression analysis showed that QRSd >120 ms was an independent predictor of TWIs during LBBAP. We also observed that the wider the baseline QRSd, the higher was the predictive value of TWIs. This may be because QRSd can reflect abnormal ventricular conduction. In the present study, dynamic T-wave changes during LBBAP also occurred in patients with a normal QRS complex before operation. The phenomenon cannot be explained by known mechanisms of CM.

Limitations

First, this was a retrospective study evaluating TWIs changes in patients with LBBAP. And the ECG analysis was qualitative and not quantitative. Second, coronary angiography was not performed during operation; it was difficult to exclude the damage to interventricular septum branches during the LBBAP procedure. According to the earlier studies, the prevalence of this complication was quite low, so it could not explain the high occurrence of TWIs during LBBAP. Additionally, the ECG leads related to TWIs were different from those caused by septum ischemia. Third, LBBP and left ventricular septal pacing (LVSP) were not further differentiated in the study. Through direct activation of the left bundle branch, LBBP was more physiological than LVSP. As a result, a higher prevalence of TWIs recovery could be expected after LBBP. Furthermore, the duration of follow-up varied, which may have influenced the recovery rate of TWIs. The results should be confirmed in future prospective cohort studies with a larger patient number.

CONCLUSION

This study demonstrated that nearly two-thirds of patients had TWIs during LBBAP. T-wave changes during LBBAP occurred more frequently in inferior leads (II, III, and aVF) and anterior leads (V1–V5) in patients with CLBBB. Patients with CRBBB were more prone to T-wave inversion in high side leads (I and aVL). QRSd >120 ms could predict TWIs during LBBAP. These findings could be used to avoid unnecessary testing for myocardial ischemia in patients with T-wave changes during LBBAP.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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