Left bundle branch pacing compared to left ventricular septal myocardial pacing increases interventricular dyssynchrony but accelerates left ventricular lateral wall depolarization

Karol Curila, MD, PhD, MSc,* Pavel Jurak, MSc, PhD,† Marek Jastrzebski, MD, PhD,‡ Frits Prinzen, PhD,§ Petr Waalduf, MD, PhD,‡ Josef Halamek, MSc, PhD,† Kevin Vernooy, MD, PhD,‖ Radovan Smíšek, MSc,†* Jakub Karch, MSc,* Filip Plesinger, MSc, PhD,† Pavel Moskal, MD, PhD,‡ Marketa Susankova, MSc,* Lucie Znojilova, MSc,* Luuk Heckman, MD,‖ Ivo Viscor, MSc, PhD,† Vladimír Vondra, PhD,† Pavel Leinveber, MSc,†† Pavel Osmancík, MD, PhD* From the *Cardiocenter, Third Faculty of Medicine, Charles University and University Hospital Kralovské Vinohrady, Prague, Czech Republic, †Institute of Scientific Instruments, the Czech Academy of Sciences, Brno, Czech Republic, ‡First Department of Cardiology, Interventional Electrocardiology and Hypertension, Jagiellonian University, Medical College, Krakow, Poland, §Department of Physiology, Cardiovascular Research Institute Maastricht, Maastricht University Medical Center, Maastricht, the Netherlands, ‖Department of Anesthesia and Intensive Care, Charles University and University Hospital Kralovské Vinohrady, Prague, Czech Republic, ††Department of Cardiology, Cardiovascular Research Institute Maastricht (CARIM), Maastricht University Medical Center, Maastricht, the Netherlands. **Brno University of Technology, Faculty of Electrical Engineering and Communication, Department of Biomedical Engineering, Technická 12, Brno, Czech Republic, and †††International Clinical Research Center, St. Anne’s University Hospital, Brno, Czech Republic.

BACKGROUND Nonselective His-bundle pacing (nsHBp), nonselective left bundle branch pacing (nsLBBp), and left ventricular septal myocardial pacing (LVSP) are recognized as physiological pacing techniques.

OBJECTIVE The purpose of this study was to compare differences in ventricular depolarization between these techniques using ultra-high-frequency electrocardiography (UHF-ECG).

METHODS In patients with bradycardia, nsHBp, nsLBBp (confirmed concomitant left bundle branch [LBB] and myocardial capture), and LVSP (pacing in left ventricular [LV] septal position without proven LBB capture) were performed. Timings of ventricular activations in precordial leads were displayed using UHF-ECG, and electrical dyssynchrony (e-DYS) was calculated as the difference between the first and last activation. Duration of local depolarization (Vd) was determined as width of the UHF-QRS complex at 50% of its amplitude.

RESULTS In 68 patients, data were collected during nsLBBp (35), LVSP (96), and nsHBp (55). nsLBBp resulted in larger e-DYS than did LVSP and nsHBp [−24 ms (−28; −19) vs −12 ms (−16; −9)] vs 10 ms (7; 14), respectively; \( P < .001 \). nsLBBp produced similar values of Vd in leads V2-V6 (36–43 ms vs 38–43 ms; \( P = \) NS in all leads) but longer Vd in leads V1-V4 (47–59 ms vs 41–44 ms; \( P < .05 \)) as nsHBp. LVSP caused prolonged Vd in leads V2-V6 compared to nsHBp and longer Vd in leads V4-V6 compared to nsLBBp (44–51 ms vs 36–43 ms; \( P < .05 \)) regardless of R-wave peak time in lead V6 or QRS morphology in lead V1 present during LVSP.

CONCLUSION nsLBBp preserves physiological LV depolarization but increases interventricular electrical dyssynchrony. LV lateral wall depolarization during LVSP is prolonged, but interventricular synchrony is preserved.

KEYWORDS Depolarization duration; Dyssynchrony; His-bundle pacing; Left bundle branch pacing; Left ventricular septal myocardial pacing; Ultra-high-frequency electrocardiography

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Introduction
In recent years, new pacing techniques such as His–Purkinje conduction pacing system have emerged. In addition to His-bundle pacing (HBP), left ventricular septal myocardial pacing (LVSP) and direct capture of the left Tawara bundle (left bundle branch block pacing [LBBP]) using the transseptal approach have been described.1,2

Both LVSP and LBBP are characterized by a pseudo–right bundle branch block (pseudo-RBBB) morphology in lead V1. They also share similar characteristics regarding lead placement in the left septum; however, LBBP is characterized by left septal subendocardial lead placement and confirmation of left bundle branch (LBB) capture using pacing maneuvers.3 In contrast, left ventricular (LV) septal myocardial capture is achievable over a larger area and with shallower lead positions.4

Ultra-high-frequency electrocardiography (UHF-ECG) analyzes the ultra-high-frequency components of ventricular myocyte action potentials. It shows the time sequence of ventricular depolarization and describes ventricular electrical dyssynchrony. It also displays local depolarization durations under individual chest leads, with shorter values indicating rapid conduction within specific ventricular segments.5 Using UHF-ECG, we previously showed that nonselective His-bundle pacing (nsHBP) produces a physiological pattern of ventricular activation that is similar to intrinsic depolarization in patients with narrow QRS.6

The impact of LBBP and LVSP on electrical dyssynchrony and local depolarization durations, as seen using UHF-ECG, is not known. This work aimed to better understand the depolarization characteristics of both ventricles during LBBP and LVSP and to compare them to nsHBP, which was considered to represent physiological ventricular activation.

Methods
Study design and study population
In this prospective study, consecutive patients with an indication for pacemaker implantation due to bradycardia were included in the Faculty Hospital Kralovske Vinohrady. The project was approved by the Ethics Committee, and all subjects signed informed consent before enrollment.

Pacemaker implantation
The left subclavian approach was preferred per study protocol. The His-bundle region was mapped using a SelectSecure™ lead (model 3830, 69 cm, Medtronic Inc., Minneapolis, MN), delivered through a fixed-curve sheath (C315HIS, Medtronic), and His-bundle signal was identified. Pacing in this location was performed to obtain nonselective His-bundle capture (nsHBP) as described previously.7

The lead was then moved toward the right ventricle (RV), along a line between the His-bundle region and the RV apex, and screwed deep into the septum to obtain a position in the left side of the interventricular septum showing a paced QRS morphology of RBBB/pseudo-RBBB in lead V1. The process of screwing the lead into the interventricular septum was occasionally interrupted to check the paced QRS morphology and impedance. Once the paced QRS morphology (during unipolar pacing with 5V output at 0.5 ms) demonstrated a terminal r/R or rs/Rs morphology in lead V1, the pacing output was decreased until capture was lost. During decreased pacing output, QRS morphology and changes in R-wave peak time (RWPT) in V5, which represents the time between the pacing artifact and the moment the electrical wavefront reaches the epicardium under lead V5,8 were checked and recorded. Two distinct capture types in the left septal area were described (Figure 1):

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Parameters measured during each type of ventricular capture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of capture</td>
<td>( V_5 ) (ms)</td>
</tr>
<tr>
<td>nsLBBp ((n=35))</td>
<td>70 (66;73)</td>
</tr>
<tr>
<td>LVP (n = 96)</td>
<td>68 (84;89)</td>
</tr>
<tr>
<td>nsHBp (n = 55)</td>
<td>88 (86;91)</td>
</tr>
</tbody>
</table>

* HBpo = His-bundle potential; LBBpo = left bundle branch potential; LVSP = left ventricular septal myocardial pacing; nsHBp = nonselective His-bundle pacing; nsLBBp = nonselective left bundle branch pacing; QRSd = QRS duration; RWPT = R-wave peak time.

1 \( p < .01 \) vs LVSP and nsHBp.
2 \( p < .001 \) vs LVSP.
3 \( p < .001 \) vs nsLBBp and LVSP.

Table 1 | Patient characteristics |
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>78 ± 9</td>
</tr>
<tr>
<td>Male</td>
<td>42 (61)</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>14 (21)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>27 (40)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>32 (47)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>54 (79)</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>56 ± 7</td>
</tr>
<tr>
<td>Septal thickness</td>
<td>11 ± 2</td>
</tr>
<tr>
<td>Pacing indications</td>
<td></td>
</tr>
<tr>
<td>AV block</td>
<td>39 (57)</td>
</tr>
<tr>
<td>SSS</td>
<td>20 (29)</td>
</tr>
<tr>
<td>Bifascicular/trifascicular block</td>
<td>15 (22)</td>
</tr>
<tr>
<td>Atrial fibrillation with planned AV junctional ablation</td>
<td>3 (4)</td>
</tr>
<tr>
<td>QRS morphology</td>
<td></td>
</tr>
<tr>
<td>LBBB</td>
<td>9 (13)</td>
</tr>
<tr>
<td>RBBB</td>
<td>21 (31)</td>
</tr>
<tr>
<td>Isolated left Tawara fascicular blocks</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Left Tawara fascicular blocks with RBBB</td>
<td>15 (22)</td>
</tr>
<tr>
<td>NIVCD</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Narrow QRS</td>
<td>32 (47)</td>
</tr>
</tbody>
</table>

Values are given as mean ± SD or n (%).
AV = atrioventricular; LBBB = left bundle branch block; LV = left ventricle; NIVCD = nonspecific intraventricular conduction delay; RBBB = right bundle branch block; SSS = sick sinus syndrome.
1. Nonselective left bundle branch capture (nsLBBp), that is, concomitant myocardial and LBB capture, defined by a pseudo-RBBB morphology with the terminal r/R in lead V1, which occurred after increasing the pacing output up to 5 V at 0.5 ms from selective left bundle branch capture (sLBBp) or myocardial LV septal capture. During the transition from sLBBp, changes in QRS complex and EGM morphology (narrowing of R' in lead V1 and disappearance of a discrete LBB electrogram) were seen; however, V5 RWPT remained the same. During the transition from myocardial capture to nsLBBp, V5 RWPT shortened >10 ms and, usually, the amplitude of R in lead V1 increased or changed from rs/Rs to r/R morphology.

2. Left ventricular septal myocardial capture (LVSP), that is, pure myocardial capture of the left septum in the location where LBB capture was not observed with output up to 5 V at 0.5 ms, was defined by a narrower paced QRS (compared to pacing from the RV septum) and a terminal R/r or rs/Rs morphology in lead V1. While decreasing the pacing output from 5 V to loss of capture, either no changes or only minor changes in QRS morphology and V5 RWPT (<10 ms) were observed.

We aimed to fix the lead in a location providing nsLBBp with a normal QRS axis and the transition to sLBBp or myocardial pacing during each lead deployment. If the "drilling effect"10 or impedance values close to 500 Ω prohibited deeper lead deployment, the lead was withdrawn and deployed in a different location. If nsLBBp was not observed in the second tested location, no further attempts were made.

**UHF-ECG data acquisition and analysis of other measured parameters**

A ventricular dyssynchrony imaging (VDI) monitor (ISI Brno, Cardion, FNUSA, Czech Republic) was used to record and analyze the 5-kHz, 14-lead ECG signals with 3-nV resolution and a frequency range of 1.5 kHz. Standard V1-V8 chest lead positions were used, except for lead V1, which was moved from the 4th to 5th right parasternal intercostal space to obtain better signals from the lateral RV wall. UHF-ECG data for all captures were collected during 2–3 minutes of VVI pacing at 110 bpm. Signal processing and UHF-ECG map construction were described previously.11 In brief, median amplitude envelopes were computed for 16
frequency bands (150–1000 Hz) for each chest lead. The broad-band QRS complex (UHF-QRS) was constructed as the average of the 16 normalized median amplitude envelopes and displayed as a color map for each lead. Local activation times were calculated as the center of mass above 50% threshold of the baseline-to-peak amplitude, and Vxd determined as depolarization duration at 50% threshold of the baseline-to-peak amplitude in V6 and V3 (V6d and V3d). B: Twelve-lead electrocardiogram (ECG). C: Ventricular depolarization map with visualization of M1-8c, electrical interventricular dysynchrony (e-DYS), and V1-8d values. (For details, see Jurak et al.) In C, the dark line connects the center of masses (solid points) under the specific lead (displayed on the y-axis). Time (ms) is displayed on the x-axis. In this case, the first activation occurred under V7 (M7c) and the last under V1 (e-DYS = −57 ms). The width of depolarization under V3 is indicated by the green arrow (V3d) and under V6 by the yellow arrow (V6d). The numerical parameters of the local depolarization duration (under each lead) are shown on the right.

The height of the lead tip on the septum in the right anterior oblique (RAO) projection was measured with respect to heart silhouette diameter and reported in percentages as reported recently (Supplemental Figure 1). Global QRS duration (QRSd) values were measured using an electrophysiological system (LabSystem Pro, Boston Scientific, USA), from the earliest to the last deflection in any of the 12 leads. During pacing, the beginning of the QRS was measured from the pacing artifact (QRSd) and from the first deflection identified after the pacing artifact (actual QRSd). Paced V5 RWPT was measured from the pacing artifact to the maximum positive QRS amplitude in lead V5. All measurements were obtained at 200 mm/s using 2 consecutive beats, and their average values were taken. During the procedure, 2–3 mL of contrast agent was injected through a C315HIS sheath in the left anterior oblique (LAO) projection. Lead depth inside the septum for all pacing locations was measured with an xViewer (Vidis, Prague, Czech Republic) using the distance between the tip and the anodal ring of the 3830 lead (10.8 mm) in LAO as a reference. QRS axis in the frontal plane was calculated and considered to have deviated to the left if axis \( \leq -30^\circ \), normal (axis \( -29^\circ \) to \( 105^\circ \)), or deviated to the right (axis \( \geq 105^\circ \)).
Statistical analysis

Exploratory data analysis was performed for all parameters. Unpaired comparisons of continuous and categorical variables were made using the unpaired Student t test and $\chi^2$ test. Repeated measurement comparisons were made using a linear mixed effect model and the Tukey multiple comparison test. The results of these models are given as mean (95% confidence interval) and comparisons as mean differences (95% confidence interval) and $P$ values (Figures 3–6). $P < .05$ was considered significant. RStudio Version 1.2.1335 with R Version 3.6.1 was used to perform statistical analyses. The linear mixed effect model was calculated using lme4 Version 1.1–21. If not specified, all values are given as mean (95% confidence interval).

Results

In a total of 68 patients with bradycardia, we recorded UHF-ECG during nsLBBp (n = 35), LVSP (n = 96), and nsHBp (n = 55). Narrow QRS with duration $\leq 120$ ms was present during spontaneous rhythm in 47% of patients. In 32 patients, LVSP was investigated. Patient characteristics are listed in Table 1.

The shortest $V_5$ RWPT was observed during nsLBBp, followed by LVSP and nsHBp. During nsLBBp, the lead tip was deeper in the septum than during LVSP, and the LBB potential was present in the majority of nsLBBp (28/35) compared to only 7 of 96 LVSP (Table 2). All LVSP with a visible LBB potential had paced $V_5$ RWPTs that were $> 10$ ms longer than their LBB potential RWPTs in $V_5$ during spontaneous rhythm, which indicated that LBB was not captured (Supplemental Figure 2).

A comparison of the sequence of ventricular activation between nsLBBp, LVSP, and nsHBp is shown in Figure 3A. The timing of ventricular depolarization under leads $V_4$–$V_8$ was remarkably similar during both nsLBBp and LVSP. However, more delayed activation under leads $V_1$–$V_3$ during nsLBBp resulted in greater left-to-right interventricular dysynchrony with a more negative e-DYS than during LVSP (Figure 3B). Compared to both left septal captures, mean e-DYS during nsHBp was 10 ms (7;14) ($P < .001$). Negative e-DYS (indicating delayed RV depolarization) was observed in 7 of 55 nsHBp (in 5 due to incomplete RBBB correction), 34 of 35 nsLBBp, and 75 of 96 LVSP.

nsHBp resulted in the shortest QRSd and $V_d$ mean (Figures 4A and 4B). There was no difference in QRSd and $V_d$ mean between nsLBBp and LVSP, but significant differences in $V_d$ for $V_1$–$V_8$ were observed. Local depolarization durations associated with the LV lateral wall ($V_5$–$V_8$) were shorter; however, depolarization duration in $V_1$–$V_3$ was longer during nsLBBp than LVSP. $V_5$–$V_8$ values observed in nsLBBp were statistically the same as those in nsHBp, but both nsLBBp and LVSP had longer $V_1$–$V_4$ compared to nsHBp (Figure 4C).

$V_5$ RWPT values overlapped during LVSP (range 52–114 ms) and nsLBBp (52–88 ms), with 51 of the LVSP (53%) having $V_5$ RWPT $\leq 88$ ms. To determine whether shorter $V_5$ RWPT values during LVSP reflect faster LV lateral wall depolarization, we compared LVSP with the shortest and longest $V_5$ RWPTs divided by quartiles. Mean $V_5$ RWPT was 69 ms in the 1st quartile vs 101 ms in the 4th quartile. We found that only 2 of 24 captures from the 1st quartile had a normal axis compared to 17 of 24 from the 4th quartile ($P < .001$). Those from the 1st quartile had more significant interventricular dysynchrony [e-DYS $-20$ ms (−27;−14) vs −8 ms (−14;−2); $P = .007$], but individual $V_1$–$V_8$ values were the same (Figure 5).

LVSP with left-axis deviation (51/96) had shorter $V_5$ RWPT of 80 ms (77;83) compared to those with normal axis of 92 ms (89;95) ($P < .001$). To study whether LVSP
with left-axis deviation were placed more inferiorly on the septum, such as those with a normal axis, we calculated the height of the lead tip in the RAO projection with respect to the height of the heart silhouette. Stored RAO projections were available in 17 LVSP with left-axis deviation and 14 LVSP with normal QRS axis. Lead tip positions resulting in LVSP with left-axis deviation were in RAO placed lower than those with normal axis [42% vs 50%; mean difference –8% (–13;–3); P = .004] and had shorter V5 RWPT [81 ms vs 91 ms; mean difference –10 ms (–18;–2); P = .02].

To understand how V1 QRS morphologies influence ventricular depolarization patterns, we compared 75 LVSP having terminal r/R and 21 LVSP with terminal rs/Rs morphology in V1. Captures with terminal rs/RS morphology had more shallow lead tip insertions [10 mm (8;11) vs 12 mm (11;12); P = .01] and less interventricular dyssynchrony [e-DYS –3 ms (–9;4) vs –16 ms (–19;–13); P < .001] compared to those with terminal r/R morphology. However, their Vd in V2–V8 was the same (Figure 6). Eleven of 21 captures with terminal rs/RS morphology had a positive e-DYS compared to 10 of 75 captures with terminal r/R morphology (P < .001).

No differences in e-DYS, Vd\textsubscript{mean}, QRS\textsubscript{d}, and local V1–8d were observed when comparing left septal pacing with left-axis deviation (n = 66) to those with normal paced QRS axis (n = 62), although V5 RWPT was significantly shorter with left-axis deviation [76 ms (72;80) vs 85 ms (82;89); P < .001]. The proportions of nsLBBp and LVSP in both groups were the same: 27% nsLBBp and 73% LVSP in the left-axis deviation group vs 26% and 74%, respectively, in the normal axis group (P = .99) (Supplemental Figure 3).
Discussion
This study showed that, in bradycardia patients, HBP provides more physiological activation than pacing from the left side of the interventricular septum, and that differences in UHF-ECG–derived ventricular synchrony and local depolarization duration during pacing from the left side of the interventricular septum depend on LBB capture. nsLBBP creates greater interventricular dyssynchrony compared to LVSP. LV lateral wall depolarization durations in nsLBBp are similar to those in nsHBP due to direct capture of His–Purkinje tissue with rapid electrical wavefront propagation. The study also showed that left septal myocardial pacing without confirmed LBB capture (LVSP) has slower LV lateral wall depolarization compared to nsLBBp and nsHBP irrespective of RWPT in V5 or V1 QRS morphology. Finally, both nsLBBp and LVSP compared to nsHBP resulted in longer depolarization durations in leads placed above the septum and the RV. This likely is a result of the greater contribution of slower myocardial conduction in their activation.

In past years, novel pacing strategies have been introduced to avoid nonphysiological ventricular activation associated with RV myocardial pacing. Shortcomings linked to HBP led to the development of transseptal pacing methods.1,2 Both nsLBBp and LVSP reduce the transseptal conduction time associated with RV myocardial pacing. They also share other similarities, such as the lead tip being fixed in the left septal area, and delayed activation of the RV leading to a typical QRS morphology with the terminal r/R in lead V1.3 However, direct capture of the LV subendocardial His–Purkinje system during LBBp results in earlier electrical wavefront propagation through the LV cavity compared to myocardial capture of the LV septum. This is reflected by a shorter V5 RWPT compared to pure myocardial LV septal capture, in which the left septal myocytes are activated first after pacing and then, after some delay, the LV His–Purkinje system is activated. Due to the ability to display local depolarization durations using UHF-ECG, we were able to show that LVSP caused statistically significant prolongation of local depolarization durations under V5–V8 compared to nsLBBp. This likely results from the more significant contribution of slower myocardial cell-to-cell electrical wavefront propagation through the LV during LVSP. However, V5d–V8d values during nsLBBp were the same as during nsHBP, which shows that the latency between the pacing artifact and LBB capture in LBBp does not play a significant role in LV lateral wall depolarization. The interventricular dyssynchrony (expressed as e-DYS) was significantly shorter during LVSP than during nsLBBp. Better RV–LV depolarization interplay during LVSP was a result of the time delay between left septal myocyte capture and activation of the LV His–Purkinje system. The longer the delay, the less interventricular dyssynchrony is present because left-to-right transseptal depolarization occurs.

Figure 5  Local depolarization durations (Vd in V1–V8) during LVSP with the shortest and longest RWPT in V5 (1st quartile vs 4th quartile). Abbreviations as in Figures 1 and 2.
immediately after pacing; however, LV conduction system activation with the fastest LV depolarization is delayed. Criteria to differentiate between LBBp and left septal myocardial pacing are still not established, although a few attempts have been made. Lead tip penetration into the septum leads to distinct paced QRS morphology changes in V1. The notch, present in the nadir location in the QRS complex during right septal pacing, moves upward as the lead progresses into the septum. Finally, formation of the pseudo-RBBB in V1 usually is considered a sign that the left septal area was reached. However, it should not be considered a marker of optimal LV depolarization because it just reflects delayed RV depolarization compared to the relatively faster depolarization of the LV. As we showed in our study, pseudo-RBBB morphology in V1 was also present in more shallow LV septal positions, where direct LBB capture was not proven with pacing output up to 5 V at 0.5 ms. Although they resulted in improved interventricular synchrony, their LV lateral wall depolarization durations were longer compared to nslBBp. Another parameter commonly used during left septal lead placement is V5 RWPT. It represents the time between the pacing artifact and the moment the electrical wavefront reaches the LV lateral wall epicardium, and its shorter values could assume faster LV activation.

However, as we showed, shorter V5 RWPTs during LVSP were not a reflection of faster LV lateral wall depolarization but rather were the result of lead deployment closer to the left posterior LBB fascicle with resultant left-axis deviation. Lead placement in more inferior septal areas with preferential activation of the His–Purkinje tissue of the left posterior fascicle shorten V5 RWPT because the distance to the LV lateral wall epicardium is shorter compared to pacing the LBB trunk area in more superior locations.

The most balanced LV–RV depolarization was seen during LVSP with the terminal rs/Rs morphology in V1 due to reduced transseptal left-to-right conduction time because of more shallow lead placement compared to deeper LVSP lead positions with r/R V1 morphology. It not only resulted in less interventricular dyssynchrony but also produced shorter depolarization durations under V1. Compared to nslHBp, both types of left septal pacing led to significant prolongations of depolarization under the leads located above the interventricular septum and RV. This is caused by left-to-right electrical wavefront propagation through the septum during nslBBp and LVSP, and delayed RV activation due to slowed conduction associated with myocardial cell-to-cell transmission. Both the septum and the RV are depolarized in an unphysiological manner during nslBBp and LVSP.

Figure 6 Local depolarization durations (Vd in V1–V8) during LVSP and with terminal r/R morphology and rs/Rs morphology in V1. Abbreviations as in Figures 1 and 2.
which is in contrast to the physiological activation seen during nsHBp.

**Study limitations**

This study was performed during the actual implant procedures. UHF-ECG measurements were taken immediately after the lead was placed in a predefined position and ventricular capture type was confirmed. We cannot rule out that the resultant damage to conductive and myocardial tissue could have influenced the paced ventricular depolarization patterns. Data were not compared to that of any other invasive or noninvasive electrocardiographic methods, and no hemodynamic or echocardiographic measurement of mechanical dyssynchrony during the procedure was performed. The only method used to confirm LBB capture was the decrease in pacing amplitude, which could have led to misclassification of some nsLBB captures as LVSP captures. The low QRS signal quality did not allow construction of UHF-ECG maps in 1 patient, and this patient was excluded from the study. Because of the low number of patients with LBBB during spontaneous rhythm included in the study, the differences in ventricular depolarization between nsLBBp and LVSP cannot be generalized to the population of patients with LBBB.

**Conclusion**

This UHF-ECG study comparing left septal pacing with HBP showed that both types of left septal pacing cause less physiological ventricular depolarization compared to nsHBp. Nonselective capture of the left bundle preserves fast LV lateral wall depolarization but at the costs of deterioration in interventricular synchrony and slower conduction in the septum and RV. LV septal lead placement, without proven LBB capture, produces less interventricular dyssynchrony than nsLBBp. However, the contribution of rapid conduction in all individual ventricular segments is smaller compared to the physiological ventricular activation seen during nsHBp. If the most physiological pattern of LV activation is the goal of the implant procedure, then nsHBp and nsLBBp should be preferred over LVSP because LVSP results in slower LV lateral wall depolarization, irrespective of V5 RWPT or paced QRS morphology in lead V1.

**Appendix**

**Supplementary data**

Supplementary data associated with this article can be found in the online version at [https://doi.org/10.1016/j.hrthm.2021.04.025](https://doi.org/10.1016/j.hrthm.2021.04.025).

**References**