Ventricular activation pattern assessment during right ventricular pacing

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Ventricular activation pattern assessment during right ventricular pacing: Ultra-high-frequency ECG study

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Abstract

Background: Right ventricular (RV) pacing causes delayed activation of remote ventricular segments. We used the ultra-high-frequency ECG (UHF-ECG) to describe ventricular depolarization when pacing different RV locations.

Methods: In 51 patients, temporary pacing was performed at the RV septum (mSp); further subclassified as right ventricular inflow tract (RVIT) and right ventricular outflow tract (RVOT) for septal inflow and outflow positions (below or above the plane of His bundle in right anterior oblique), apex, anterior lateral wall, and at the basal RV septum with nonselective His bundle or RBB capture (nsHBorRBBp). The timings of UHF-ECG electrical activations were quantified as left ventricular lateral wall delay (LVLWd; V8 activation delay) and RV lateral wall delay (RVLWd; V1 activation delay).

Results: The LVLWd was shortest for nsHBorRBBp (11 ms [95% confidence interval = 5–17]), followed by the RVIT (19 ms [11–26]) and the RVOT (33 ms [27–40]; $p < .01$ between all of them), although the QRSD for the latter two were the same (153 ms (148–158) vs. 153 ms (148–158); $p = .99$). RV apical capture not only had a longer LVLWd (34 ms (26–43) compared to mSp (27 ms (20–34), $p < .05$), but its RVLWd (17 ms (9–25) was also the longest compared to other RV pacing sites (mean values for nsHBorRBBp, mSp, anterior and lateral wall captures being below 6 ms), $p < .001$ compared to each of them.

Conclusion: RVIT pacing produces better ventricular synchrony compared to other RV pacing locations with myocardial capture. However, UHF-ECG ventricular dyssynchrony seen during RVIT pacing is increased compared to concomitant capture of basal septal myocytes and His bundle or proximal right bundle branch.

KEYWORDS

conductive system, myocardial, pacing, ultra-high frequency ECG, ventricular dyssynchrony

Disclosures: None.

[Correction added on March 23, 2021, after first online publication: the word "Ultrahigh-frequency" has been changed to "ultra-high-frequency ECG" in the proof.]

1 | INTRODUCTION

Right ventricular (RV) apical pacing causes ventricular dyssynchrony with an increased risk of heart failure.¹ Despite attempts to reduce it by changing the pacing location (RV septal vs. RV apex), no clinical benefit has been observed. As was shown, the standard fluoroscopic criteria for exact lead placement are insufficient, and many leads that were considered to be in the septum based on fluoroscopy were, in fact, anchored in the anterior wall.^{2,3} Furthermore, myocardial septal positions might represent a mix of more or less dyssynchronous pacing, which ultimately brings no advantage over RV apical pacing.

His bundle pacing (HBP) results in the most physiological ventricular activation,⁴ however, lead fixation in a region abundant with fibrotic tissue increases the need for pacing lead reinterventions.⁵ Distal HBP has the potential to overcome these shortcomings since the lead is fixed primarily in the septum beyond the annulus of the tricuspid valve.⁶ The nonselective capture of the His bundle and septal myocytes (nsHBp) can lead to an increase in ventricular mechanical dyssynchrony.⁷ The lead placement in ventricular His bundle's location could lead to greater involvement of myocytes in ventricular depolarization with an unknown impact on ventricular electrical synchrony during nsHBp.

Ultra-high-frequency ECG (UHF-ECG, 150–1000 Hz) is an imaging method displaying the activation times and local depolarization characteristics of specific ventricular segments. Transmural information and time resolution in milliseconds are possible thanks to measurement and analysis of the UHF components of ventricular myocyte action potentials.⁸ Its validation in ex vivo and clinical experiments with epicardial, intramural electrical potential mapping, and ECGi showed that it measures intramural electrical activation and provides volumetric depolarization information.⁹ Recent studies showed the ability of UHF-ECG to assess ventricular dyssynchrony¹⁰ and its prognostic importance.¹¹

The aim of the study was to investigate the impact of pacing the various RV locations on the timing of ventricular activation displayed by UHF-ECG.

2 | METHODS

2.1 | Study population

Consecutive patients with an indication for pacemaker implantation due to bradycardia, under the care of a single physician between September 2019 and February 2020, were included. The project was approved by the Ethics Committee of the Faculty Hospital Kralovske Vinohrady, Prague, CZ, and all subjects signed informed consent before enrollment.

2.2 | Pacemaker implantation

The left subclavian approach was preferred per study protocol. After the venous system was reached, the atrial pacing lead was

temporarily placed in the apex of the right ventricle. Then the His bundle region was mapped using a SelectSecure™ lead (model 3830, 69 cm, Medtronic Inc.), which was delivered through a fixed-curve sheath (C315 HIS, Medtronic), as previously described.¹² Once the His bundle signal was identified, to ensure the ventricular position of the lead tip, it was moved to the right ventricle, beyond the hinge of the septal leaflet of the tricuspid valve. An X-ray and an electrogram (EGM) of the atrial, His bundle, and ventricular signals were used to navigate the lead tip to the area of the basal interventricular septum. At this location, atrial signals significantly decreased or disappeared, but His bundle signals were present before lead fixation – Figure 1. In case of uncertainty, an injection of contrast agent through a C315 HIS sheath was performed to verify ventricular lead tip placement. Once the proper ventricular septal position of the lead tip was achieved, it was fixed using 3–5 clockwise rotations, which was followed by pacing with outputs between 0 and 5 V at 0.5 ms. The nonselective His bundle or right bundle branch capture (nsHBorRBBp) was confirmed when wide, notched, or slurred QRS complexes present during low output pacing narrowed and had a reduced R wave peak time at V6 (left ventricular activation time = LVAT) during high output pacing. Thereafter the atrial lead with a pre-shaped stylet was moved from the RV apical position and temporarily placed in pre-specified locations in the right ventricle (RV septum, anterior wall, and lateral wall). For appropriate lead placement in these locations, the SelectSecure™ lead and C315His sheath, already positioned in the basal septal region, were used as landmarks – Figure S1. For all pacing locations, X-ray cine loops in two projections (right anterior oblique [RAO] 20–30° and left anterior oblique [LAO] 20–30°) were generated and stored for further analysis. In patients with an indication for a dual-chamber pacemaker, the atrial lead was removed from the ventricular position and was fixed in the right atrium at the end of the procedure. Stored ventricular pacing positions of the atrial lead were later retrospectively reviewed by an experienced implanter blinded to the intended position (PS). The exact location of the lead tip was determined using this approach: first, based on the LAO projection, the lead position was classified as RV myocardial septal – mSp, anterior wall, or lateral wall. Then, mSp positions were further classified as (a) the septal right ventricular inflow tract (RVIT) or (b) the septal right ventricular outflow tract (RVOT) based on the RAO projection – Figure 2.

The distances between cardiac silhouette lines ($a + b$) and the lead with nsHBorRBBp capture to the lower cardiac silhouette (a) were measured in mm – Figure 2, panel B. The location of the region with ventricular nsHBorRBBp capture, with respect to the diameter of the heart silhouette, was calculated as $(a/a + b) \times 100$ and reported as percentages. All ECG recordings before and during pacings of the RV septum were reviewed retrospectively using EP Labssystem software at 25, 100, and 200 mm/s, as needed. HV intervals (calculated as the mean value between two consecutive beats), LVAT, the types of ventricular capture, and the ratio between the atrial and ventricular signal amplitudes in mV (A/V amplitude ratio) were measured after lead fixation in the ventricular HB area during the spontaneous rhythm and pacing at 100 mm/s.

FIGURE 1 A description and presentation of the method used to place the pacing lead in the area of the ventricular portion of the His bundle. In this patient, the His bundle potential with HV interval was 62 ms in the first position – left panel. The QRS amplitude was 1.9 mV, and atrial activity could be easily identified on the EGM. The lead was then moved towards the right ventricle, beyond the level of the tricuspid valve – middle panel. Atrial activity was no longer recognizable on the EGM, the QRS amplitude increased to 2.8 mV, and the HV interval shortened to 42 ms. Pacing with an output between 1.0–3.5 V at 0.5 ms led to myocardial capture of the ventricles (nsHB capture was present with outputs above 3.5 V at 0.5 ms). The position seen in the right panel was obtained after three lead rotations beyond that in the middle panel position. Since the lead tip advanced closer to the His bundle after fixation, the nsHB capture lowered to 1.25 V at 0.5 ms (myocardial capture was present between outputs of 0.75–1.25 V at 0.5 ms). Asterisks show the first position of the lead

FIGURE 2 Panel A: The method used to distinguish between right ventricular (RV) septal, anterior, and lateral wall positions in the left anterior oblique (LAO) projection. Panel B: The method used to differentiate between RVIT and RVOT lead positions within mSp using the lead in the nsHBorRBBp area as the landmark in the right anterior oblique (RAO) projection. Panel A: Using the LAO projection, a horizontal line at the level of the lowest part of the pacing lead was drawn. Then two lines with angles of 25° to the perpendicular line delineated the anterior wall in the LAO projection. The individual segments of the right ventricle are shown in Panel A. Panel B: Parallel lines on the upper and on the lower heart silhouette were drawn in the RAO projection. Then a third line parallel to the other two was drawn through the lead tip in the ventricular nsHBorRBBp area, which divided the RV septum into the RVIT (on or below the line) and the RVOT (above the line). The distance between the middle and lower line was marked as “a,” and the distance between the middle and upper line was marked as “b.” The height of the ventricular nsHBorRBBp area was, in this case, 46% of the heart silhouette ($a = 26$, $b = 31$, $26/(26 + 31) \times 100 = 46\%$). LAO, XXX; RAO; RVIT, right ventricular inflow tract; RVOT, right ventricular outflow tract

2.3 | UHF-ECG data acquisition and analysis

A VDI monitor (Ventricular Dyssynchrony Imaging monitor, ISI Brno, Cardion, FNUSA, CZ, 2018) was used to record and analyze the 5 kHz 14-lead ECG signals with a 3 nV resolution and a frequency range of 1.5 kHz. UHF-ECG data were collected during 2–3 min of the RV apical, nsHBorRBBp, mSp, anterior wall, and lateral wall pacing at 100–120 beats per minute. Signal processing and UHF-ECG map construction are described in detail elsewhere.⁸ Shortly, the median amplitude envelopes were computed for 16 frequency bands (150–1000 Hz) for each of the V1–8 chest leads. The broad-band QRS complex (UHF-QRS) was then constructed as the average of the 16 normalized median amplitude envelopes, which were displayed as a color map for each lead. To describe the time course of depolarization propagation in the ventricles, the centers of mass (Mxc) under specific leads (i.e., Vx) were determined. Mxc was calculated as the center of mass of UHF-QRS amplitudes above the 50% threshold of the baseline to peak magnitude – Figure 3, panel A. To quantify the delay of the LV and RV lateral wall after the pacing, time delays from the earliest center of mass (first activation) to M8c and M1c were measured in ms (LVLWd and RVLWd, respectively). To describe the duration of LV lateral wall depolarization, which reflects the speed of its depolarization, the time delay between M5–8c (V5–8d) was measured – Figure 3. The onset and the end of the QRS complex were marked manually using custom-made software, providing high-resolution QRS signals. Global QRS durations (QRSd) were measured from the earliest to the latest deflection in any of the 12 leads (V7 and V8 were excluded from QRSd calculations). During ventricular pacing, the beginning of the QRS was measured from the highest amplitude of the pacing artifact.

2.4 | Statistics

Exploratory data analysis was performed for all parameters. Unpaired measurement comparisons were made using the unpaired *t*-test. Repeated measurement comparisons of QRSd, and Mxc during different RV captures were made using a linear mixed effect model (LMEM) with the Tukey multiple comparison test. The results of these models are presented as means with 95% confidence intervals (CIs) and comparisons as mean differences with 95% CIs and *p*-values. The value of *p* < .05 was considered statistically significant. RStudio version 1.2.1335 with R version 3.6.1 was used to perform statistical analyses. LMEM was done using lme4 version 1.1–21. If not specified, all values are shown as means (95% CI). All analyses are available as a PDF R markdown document in appendices.

3 | RESULTS

A total of 300 different ventricular activations (51 spontaneous rhythms, 46 nsHBorRBBp, 61 mSp, 48 RV anterior wall, 48 RV lateral wall, and 46 RV apical captures) were recorded in 51 patients – their characteristics are shown in Table 1. The average procedural time

was 98 ± 23 min, and one procedural complication was observed (a pericardial effusion that resolved spontaneously). Other procedural characteristics are shown in Table 1. Of 48 RV anterior wall positions labeled during the procedure, 9 were found to be septal during the retrospective analysis since the lead tip exceeded the 25° angle to the horizontal plane in the LAO (all of them were classified as RVOT based on the lead position in the RAO). Of the 60 mSp positions, 30 were classified as RVIT and 30 as RVOT. Two records (one marked as mSp and one as the lateral wall) were not analyzed since the X-ray images were not stored. As a result, definitive analyses were performed on 46 nsHBorRBBp, 69 mSp (30 RVIT and 39 RVOT), 39 RV anterior wall, 47 RV lateral wall, 46 RV apical captures, and 32 spontaneous rhythms without a bundle branch block (a narrow QRS group representing a physiological ventricular activation pattern).

The average height of the position with nsHBorRBB capture was $40 \pm 5\%$ of the heart silhouette in the RAO. The mean A/V ratio was $4 \pm 4\%$, and in 29 of 46 patients with nsHBorRBBp, the atrial signal amplitude was lower than 0.05 mV on the EGM in the nsHBorRBBp location. A wide range of HV intervals was observed (30–83 ms), with an average value of 50 ± 13 ms. There were seven patients with HV intervals shorter than 35 ms. Their LVLWd and RVLWd were similar compared to those with HV > 35 ms; 19 ms (10–27) versus 10 ms (7–14), *p* = .14 for LVLWd and 3 ms (–6 to 12) versus 1 ms (–3 to 5), *p* = .70 for RVLWd, but QRSd was significantly shorter (126 ms (112–139) versus 141 ms (135–146); *p* = .02). Their UHF-ECG maps with measured parameters are shown in Supplementary Figure 2.

Ventricular nsHBorRBBp showed superior depolarization patterns compared to all other pacing locations – Figures 4 and S3. QRSd and LVLWd were significantly shorter during nsHBorRBBp than during RVIT (*p* < .01 for both) and RVOT pacing (*p* < .001 for both). Although both RVIT and RVOT had virtually the same QRSd (*p* = .99), the LVLWd and V5–8d were significantly longer for RVOT capture (*p* < .001 for LVLWd and *p* < .05 for V5–8d). nsHBorRBB captures resulted in wider QRS complexes compared to the “narrow QRS” group, but RVLWd and LVLWd were similar (*p* = .2 and *p* = .6, respectively). nsHBorRBBp and RVIT capture caused the same V5–8d as the spontaneous narrow group (*p* > .99) – Figure 5 and Table 2.

Regarding RV myocardial pacing locations, the shortest LVLWd was observed during mSp, followed by RV apical (*p* = .02 between them), lateral and anterior wall capture being the longest. Compared to each of the others, the longest RVLWd was observed during RV apical capture (*p* < .001). The V5–8d was significantly shorter in mSp compared to other RV myocardial captures (*p* < .001 to each of them) – Figure 6 and Table 3.

4 | DISCUSSION

This UHF-ECG study describes in detail the time delays between the activation of ventricular segments during RV pacing and shows: (1) pacing of the basal interventricular septum with conductive system engagement produces the smallest ventricular dyssynchrony compared to other myocardial captures of the right ventricle and (2) pacing of RV septal inflow area results in less LV ventricular

FIGURE 3 Presentation of the UHFQRS complex construction in the single chest lead and Mxc calculation is shown in Panel A. Schematic representation of the calculated parameters from the UHF-ECG maps during RVIT (Panel B) and RV apical (Panel C) pacing and corresponding 12-lead ECGs. Complete information on the methodology for calculating UHF-ECG parameters can be found in 8. The dark line connects the center of masses (solid points) under the specific lead, which is displayed on the y-axis. Time (ms) is displayed on the x-axis. During RVIT capture shown, the first activation occurred under V4 (M4c). RVLWd = M4c to M1c, LVLWd = M4c to M8c, and V5 to V8d = M5c to M8c in ms. During RV apical capture, the first activation occurred under V5 (M5c). RVLWd = M5c to M1c, LVLWd = M5c to M8c = V5 to V8d in ms. RVIT, right ventricular inflow tract; UHF, ultra-highfrequency

dyssynchrony than septal RVOT pacing, although no differences in QRSd were observed between them.

4.1 | UHF-ECG dyssynchrony during septal pacing

The pacing of the RV septum has been considered to be more physiological than apical pacing.¹³ However, its clinical benefit was never

shown in a prospective randomized trial.^{2,3} But the RV septum is not a homogenous structure. It consists of an inflow and outflow tract, and also the His bundle and its ramification are localized in its basal part. The pacing in the basal septal area with concomitant myocardial and conduction tissue engagement produced the most physiological UHF-ECG depolarization pattern in our study. Even, if it was not the intention, in seven patients, the lead was placed in the proximal RBB instead of the His bundle, as their HV intervals were shorter

TABLE 1 Patients and procedural characteristics

Age (years), mean $\pm$ SD	78 $\pm$ 8
Male gender, <i>n</i> (%)	35 (67)
Comorbidities	
Heart failure, <i>n</i> (%)	11 (22)
Coronary heart disease, <i>n</i> (%)	20 (39)
Diabetes mellitus, <i>n</i> (%)	25 (49)
Hypertension, <i>n</i> (%)	41 (80)
LV ejection fraction (%), mean $\pm$ SD	56 $\pm$ 7
LV wall thickness ((septum + posterior wall)/2) (mm), mean $\pm$ SD	11 $\pm$ 1
Pacing indications	
AV block, <i>n</i> (%)	29 (57)
SSS, <i>n</i> (%)	10 (20)
Bi-, trifascicular block, <i>n</i> (%)	8 (15)
Atrial tachy-fibrillation with planned AV junctional ablation, <i>n</i> (%)	4 (8)
QRS morphology	
LBBB, <i>n</i> (%)	5 (10)
RBBB, <i>n</i> (%)	14 (27)
Without BBB, <i>n</i> (%)	32 (63)
Procedural characteristics	
A/V amplitude ratio (%), mean $\pm$ SD	4 $\pm$ 4
HV interval (mm), ^a mean $\pm$ SD	50 $\pm$ 13
Procedural duration (min), mean $\pm$ SD	98 $\pm$ 23
LVAT during nsHBorRBBp (ms), mean $\pm$ SD	96 $\pm$ 9
LVAT during myocardial basal septal capture of ventricles (ms), mean $\pm$ SD	118 $\pm$ 11
Height of basal septal region with nsHBorRBBp to heart silhouette diameter (%), mean $\pm$ SD	40 $\pm$ 5

Abbreviations: AV, atrioventricular; BBB, bundle branch block; LBBB, left bundle branch block; RBBB, right bundle branch block; SSSy, sick sinus syndrome; UHF-ECG, ultra-high-frequency ECG.

^aHV – His to ventricle (RBB potential-ventricle intervals in patients with HV < 35 ms are included), LVAT – R wave peak time in V6, nsHBorRBBp – nonselective His bundle or right bundle branch capture.

than 35 ms. QRSd in these seven patients was significantly shorter; however, the difference in the LVLWd did not reach statistical significance compared to other nsHBorRBBp. This was possibly due to the proximal position of the pacing lead within the right Tawara bundle in most cases.

An important shortcoming of studies comparing the clinical benefit of RV septal and apical pacing involved inadequate definitions of lead placement in the septum. They were often based on ECG or X-ray criteria in postero-anterior or LAO projections, which have been shown to be inaccurate.^{2,14,15} This led to incorrect placement of pacing leads towards the anterior wall (e.g., in the anteroseptal groove) in a substantial percentage of patients.^{2,3} To overcome this shortcoming, we used the position of the lead in the ventricular HB area displayed in the LAO and RAO

projections as a landmark. Its position in the RAO projection was used to approximate the position of the crista supraventricularis.¹⁶ Pacing in the RVOT region above this level has been shown to lead to significantly worse UHF-ECG LV dyssynchrony compared to pacing on or below the level. Interestingly the mean QRSd was the same in RVIT and RVOT captures, which corroborates previous studies showing QRSd to be an imperfect marker of ventricular dyssynchrony during ventricular pacing.¹⁷ The difference in the LVLWd between these two pacing locations is highly likely a result of different distances from the left ventricular subendocardial Purkinje system. Since it is located in the lower third of the LV septum and LV free wall, the time to its activation is necessarily shorter during RVIT pacing, which is anatomically directly opposite. Once it is activated, its contribution to left

FIGURE 4 Impact of lead placement on the pattern of ventricular depolarization in different RV locations in one patient. The plane of the tricuspid valve is highlighted using a contrast agent or a dashed white line. Please note the distance of the lead during nsHBp from the tricuspid annulus, which was approximately 15 mm

ventricular depolarization is greater during RVIT capture, which was also reflected by a shorter V5-8d compared to RVOT captures.

4.2 | UHF-ECG dyssynchrony during myocardial RV pacing

The shortest LVLWd was observed with mSp, follow by RV apical capture, and was similar during RV lateral and anterior wall captures. Interestingly, the V5-8d, which reflects the speed of depolarization in the LV lateral wall, was similar during RV apical, anterior, and lateral wall pacings, but in all of them significantly longer compared to mSp capture. This is likely a reflection of the differences in electrical wavefront propagation in the LV during RV pacing. RV apical capture relies mostly on cell-to-cell LV activation, contrary to RV septal

capture, which also uses (after trans-septal signal transition) the left ventricular Purkinje system to activate the left ventricular mass.¹⁸ Our results suggest that similar to RV apical capture, during both RV anterior and lateral wall captures, the left ventricular mass under V5-V8 is being activated mainly through slower myocardial conduction. However, the longer distance from the RV lateral wall and anterior wall to the LV lateral wall under V8 is responsible for the significantly longer time delay between the first activation and activation under the V8 compared to RV apical capture. This accented electrical LV dyssynchrony may be the cause of worse outcomes in patients observed in some clinical studies in which the pacing lead was placed in the RV anterior wall or lateral wall.^{3,19} The LVLWd during RV apical capture was only slightly longer compared to all mSp captures, but as was discussed before, significant differences exist between septal locations (RVIT vs. RVOT). Noteworthy was that significant RVLWd, during RV apical pacing, was documented.

FIGURE 5 Mixed effect model for QRSd (Panel A), mean Mxc delay from the first activated segment (placed on 0 ms; Panel B) and mean M6-8c delays (Panel C) from the M5c (placed on 0 ms) during nsHBorRBBp, RVIT, RVOT captures in all studied patients and 32 spontaneous rhythms in patients without bundle branch blocks. RVIT, right ventricular inflow tract; RVOT, right ventricular outflow tract

It reflects concomitant RV dyssynchrony during apical pacing; our observation was very similar to the one documented using an ECGi in patients with heart failure.²⁰

5 | LIMITATIONS

This was a UHF-ECG analysis of ventricular depolarization patterns from different types of ventricular pacing during implant procedures. The data acquisition for UHF-ECG analysis was performed immediately after lead fixation in a predefined location and confirmation of ventricular capture. It is possible that during lead fixation in the ventricular His bundle or RBB area, damage to the conductive tissue occurred in some patients, which could have influenced the paced ventricular depolarization pattern. Data were not compared to any other invasive or noninvasive electrocardiographic methods, and no hemodynamic or echocardiographic measurement of mechanical dyssynchrony was performed. No studies on the relationship

between UHF-ECG electrical dyssynchrony and mechanical ventricular dyssynchrony have been published yet. Furthermore, the lead location with RV myocardial pacing was assessed using fluoroscopy. Despite best efforts and the use of the lead in the ventricular His bundle region as a landmark, inadequate lead placement, in some cases, cannot be excluded. It is also possible, that during the myocardial septal pacing (mSp) the unrecognized concomitant capture of septal myocytes and RBBB occurred.

SUMMARY

Ultra-high-frequency ECG (UHF-ECG) differentiates ventricular depolarization patterns during right ventricular (RV) septal, apical, anterior wall, and lateral wall pacing and between different locations on the RV septum. Right ventricular inflow tract (RVIT) pacing was showed to produce better ventricular dyssynchrony than other RV pacing locations with myocardial capture. However, ventricular

	Spontaneous narrow	nsHBorRBBp	RVIT	RVOT
LVLWd	8 (1–16) ms	11 (5–17) ms	19 ^{*,†} (11–26) ms	33 (27–40) ms
RVLWd	6 (–1 to 13) ms	1 (–5 to 7) ms	6 (–1 to 14) ms	6 (–1 to 13) ms
V5-8d	2 (–4 to 8) ms	2 (–3 to 7) ms	1 (–5 to 8) ms	9 ^{‡,§} (3–14) ms

Abbreviations: LVLWd, left ventricular lateral wall delay; RVLWd, right ventricular lateral wall delay; RVIT, right ventricular inflow tract; RVOT, right ventricular outflow tract.

**p* < .001 versus RVOT.

†*p* < .01 versus nsHBorRBBp.

‡*p* < .01 versus nsHBorRBBp.

§*p* < .05 versus RVIT.

TABLE 2 Mixed effect model values for LVLWd, RVLWd, V5-8d, during nsHBorRBBp, RVIT, RVOT captures, and 32 spontaneous rhythms without bundle branch blocks (for detailed statistics, see Appendix B)

FIGURE 6 Mixed effect model for QRSd (Panel A), mean Mxc delay from the first activated segment (placed at 0 ms; Panel B) and mean M6-8c delays (Panel C) from the M5c (placed at 0 ms) during nsHBorRBBp, mSp, RV anterior, lateral wall, and apical captures. RV, right ventricular

TABLE 3 Mixed effect model values for LVLWd, RVLWd, V5-8d, during nsHBorRBBp, mSp, RV anterior, lateral wall, and apical wall captures (for detailed statistics, see Appendix C)

	nsHBorRBBp	mSp	Anterior wall	Lateral wall	RV apex
LVLWd	11 (3–20) ms	27 ^{*,†} (20–34) ms	56 (47–65) ms	59 (51–67) ms	34 (26–43) ms
RVLWd	1 (–7 to 9) ms	6 (–1 to 13) ms	2 (–7 to 11) ms	0 (0–0) ms	17 [‡] (9–25) ms
V5-8d	5 (–5 to 9) ms	5 [§] (0–11) ms	21 (14–29) ms	17 (10–24) ms	24 (17–31) ms

Abbreviations: LVLWd, left ventricular lateral wall delay; RVLWd, right ventricular lateral wall delay.

* $p < .05$ versus RV apex.

[†] $p < .001$ versus anterior and lateral wall.

[‡] $p < .001$ vs each of the others.

[§] $p < .001$ vs anterior, lateral wall, and RV apex.

dyssynchrony seen during the RVIT was still worse compared to the pacing of the basal interventricular septum with conductive tissue engagement. RVIT pacing should be considered as an alternative for ventricular pacing when pacing the His-Purkinje conduction system is not possible or available. Our results show that UHF-ECG provides detailed and additional information on electrical ventricular dyssynchrony during RV pacing.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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