His or para-His pacing preserves left ventricular function in atrioventricular block: a double-blind, randomized, crossover study


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Aims
To compare left ventricular function after a long-term His or para-His pacing (HP) and right ventricular septal pacing (RVSP) in patients with atrioventricular block (AVB).

Methods and results
We included consecutive patients with AVB, a narrow QRS $< 120$ ms, and a preserved left ventricular ejection fraction (LVEF) $> 0.40$, in a prospective, randomized, double-blinded, crossover design. All patients were treated with 12 months HP and 12 months RVSP. A total of 38 patients [mean age, 67 ± 10 years; 30 (79%) men] were included. The primary endpoint was LVEF, which was significantly lower after a 12 months RVSP (0.50 ± 0.11) than after 12 months of HP (0.55 ± 0.10), $P = 0.005$. We measured the difference in time-to-peak systolic velocity between opposite basal segments in the apical views by using tissue Doppler imaging. In the four-chamber view, the difference was 58 (± 7) ms after RVSP and 49 (± 7) ms after HP, $P = 0.27$; in the two-chamber view, the difference was 45 (± 5) ms after RVSP and 31 (± 4) ms after HP, $P = 0.02$, and in the apical long-axis view, the difference was 63 (± 6) ms after RVSP and 44 (± 7) after HP, $P = 0.03$. There was no difference in New York Heart Association class, 6-min hall walk test, quality-of-life assessments, or device-related complications. The mean threshold was significantly higher in HP leads than in RVSP leads.

Conclusion
His or para-His pacing preserves LVEF and mechanical synchrony as compared with RVSP after 12 months pacing in patients with AVB, narrow QRS, and LVEF $> 0.40$.

Keywords
Pacing • Atrioventricular block • Left ventricular function • His-pacing

Introduction
Right ventricular (RV) pacing impairs left ventricular (LV) function due to a dyssynchronous activation and contraction, and may be associated with an adverse clinical outcome.1,2 Algorithms for minimizing ventricular pacing reduce the percentage of ventricular paced beats,3 but in patients with atrioventricular block (AVB), RV pacing is unavoidable. Most physicians practice RV apical or RV septal pacing (RVSP) due to its ease of access and long-term lead stability. Studies suggest direct His bundle (DHP) or para-His (PHP) pacing as more appropriate to prevent the deleterious effects of RV pacing on LV function.4–9 Most of the patients with AVB and QRS $< 120$ ms have preserved His–Purkinje conduction and therefore represent the majority of potential candidates for His bundle pacing. The aim of the present study was to compare LV function after a long-term His pacing or para-His pacing (HP) with RVSP in consecutive patients with a high-grade AVB, a narrow QRS, and a preserved LV function.

Methods

Study population
The His or para-His pacing in AVB study was a prospective, randomized, double-blinded, single-centre, crossover study. We included consecutive patients with a high-grade AVB and a QRS complex $< 120$ ms referred to our institution between September 2007 and December 2009 for pacemaker implantation. The exclusion criteria were: permanent atrial fibrillation (AF); a life expectancy of $< 2$ years; expecting heart surgery within 2 years; implantable cardioverter-defibrillator or cardiac resynchronization therapy (CRT) indications; LV ejection fraction (LVEF) $< 0.40$; recent myocardial infarction; a history of AV node ablation; and pregnancy. The Central Denmark Regional Ethics Committee and the Danish Data

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What’s new?

- Right ventricular (RV) pacing impairs left ventricular (LV) function due to a dysynchronous activation and contraction, and may be associated with an adverse clinical outcome.
- His and para-His pacing are feasible and could prevent the deleterious effects of RV pacing on LV function.
- Patients with atrioventricular block (AVB) often need frequent RV pacing, and represent the majority of candidates for this pacing mode.
- This study shows that His or para-His pacing preserves LV ejection fraction and mechanical synchrony compared with RV septal pacing in patient with AVB and may be the future pacing strategy to prevent pacing-induced heart failure in selected pacemaker patients.

Protection Agency approved the study. The investigation conforms to the principles outlined in the Declaration of Helsinki and all the subjects gave written informed consent to participate in the study. Clinical Trial Registration: NCT01019213 ULR: http://clinicaltrials.gov/show/NCT01019213.

Study design

All the patients received a biventricular pacemaker (Insync III, model 8042, Medtronic Inc.) with a His or para-His lead connected to the LV port, and an RV septal lead connected to the RV port. The implantation procedure was previously described in detail. In short, two convention-al bipolar, active-fixation pacing leads were positioned in the right atrial appendage and on the RV septum. Guided by a diagnostic electrophysiology (EP) catheter, DHP or PHP was obtained by placing a 4.1 Fr bipolar, fixed-screw lead (SelectSecure, model 3830; Medtronic Inc.) in or close to the His bundle using a 8.4 Fr steerable catheter as an introducer tool (SelectSite, model C304-S59 or C304-L69, Medtronic Inc.). We defined the DHP as both of the following: (i) the paced QRS morphology and duration should be identical with the intrinsic QRS, and (ii) the pace–ventricular interval (P–V) should be identical with the His–ventricular interval (H–V). We defined PHP as both of the following: (i) His-potential sensing in the permanent lead identical with the His potential in the EP catheter. (ii) QRS shortening at a high output pacing. We primarily aimed for the DHP, but if the criteria were not met within a reasonable time along with the number of lead positioning attempts we aimed for PHP. If both the criteria for PHP were not met, the leads were implanted in the high septal region in anatomical proximity with the His bundle, obtaining an electrical activation similar to PHP and with a QRS as narrow as possible. The lead positions were confirmed by fluoroscopy. After the implantation, patients were randomly assigned in a ratio of 1:1 by using a block randomization of 10, and opaque envelopes. When the patient was randomized to RVSP, the HP lead was deactivated. When the patient was randomized to HP, the RVSP lead was activated with an interventricular delay of 80 ms (HP first), with the RVSP lead as back-up in the case of an exit block during HP. After 12 months, all patients crossed over to the opposite pacing mode. The pacemaker was programmed in DDD, and paced and sensed AV delays were programmed to 150 and 120 ms during RVSP and PHP, and 90 and 60 ms during the DHP, respectively. The treatment allocations were blinded to all patients and health personnel, except for two pacemaker technicians. All patients had follow-up visits at 3, 12, 15, and 24 months, respectively. At 12 and 24 months, echocardiograms were obtained, quality-of-life (QOL) assessment with the use of the 36-item Short-Form General Health Survey (SF-36), a 6 min walk hall test (6-MH), and an assessment of New York Heart Association (NYHA) class were performed. At 3 and 15 months follow-up, all the patients underwent a 24 h electrocardiogram (ECG) monitoring. At all follow-up visits, the pacemaker was interrogated and tested and the device diagnostics were retrieved.

Echocardiograms were recorded at a rate of 80 beats to reduce the variation of echocardiographic measures within each patient over time (Vivid Seven; General Electric) and included a two-dimensional (2D) grey scale and tissue Doppler imaging (TDI) echocardiograms of the apical four-chamber, two-chamber, and long-axis views. Real-time full-volume 3D echocardiograms of the LV were obtained by using a 3-V transducer. Left ventricular ejection fraction, LV end-diastolic volume (LVEDV), and LV end-systolic volume (LVESV) were measured by real-time 3D or the Simpsons biplane method if the 3D images could not be obtained. The LV intraventricular dyssynchrony was measured with a TDI. The sample volume was placed in the basal parts of the LV in the apical long-axis, two-, and four-chamber views. The time interval between the onset of the QRS complex and the peak systolic velocity was derived from each region and the difference between the earliest and the latest peak systolic velocity was calculated. All echocardiograms were analysed offline (Echopac 7.1w, General Electric-Vingmed, and LV Analysis, TomTec) by one person blinded to the pacing modality in random order according to the study period and the individual.

Statistics

The sample size was estimated on the basis of the postulated difference in the LVEF of 5 percentage points (absolute) between HP and RVSP after 12 months of treatment. The estimated sample size for this study was 35 to show a difference of 5 percentage points with a standard deviation (SD) of 8%, a drop out of ~20%, giving a 5% two-sided significance level, and a power of 90%. The primary data analysis was intention-to-treat analysis. A supplementary on-treatment analysis was made including patients who met all the criteria for DHP or PHP and received pacing for >50% of the time. Furthermore, an exploratory stratified analysis of the primary outcome was performed for patients (on-treatment) with baseline LVEF <50% during RVSP and for patients with/without arterial hyper-tension at baseline. The differences in continuous variables between the study periods were evaluated with the paired t-test and a shift in NYHA class was evaluated with the Wilcoxon rank-sum tests. To test for the period effect, analysis of variance was used for normally distributed data. Intra- and inter-observer variations of LVEF were calculated in 15 patients by dividing the SD of the difference with the mean of the two measurements and were 6.5 and 8.3%, respectively. All P values are two-sided and nominal. A P < 0.05 was considered statistically significant. All statistical analyses were performed by using STATA software (STATA for Windows, version 12.0).

Results

Study group

During the study period, 982 patients were referred for primary pacemaker implantation. Of these, 333 patients had sinus node disease, 107 patients had chronic AF, 437 patients had AVB (217 with a narrow QRS), and 105 patients received a pacemaker for other indications. Of the patients with AVB and a narrow QRS complex, 90 patients met one or more of the exclusion criteria (21 patients had permanent AF, 7 patients had undergone AV node ablation, 9 patients were <18 years, 6 patients had LVEF <0.40,
16 patients had a recent myocardial infarction or heart surgery, 4 had significant aortic stenosis, 20 patients had a life expectancy shorter than 2 years, and 7 patients had neurocardiogenic syncope. Furthermore, 65 patients underwent acute implantation, and 24 patients refused to participate. A total of 38 patients [mean age, 67 ± 10 years; 30 (79%) men] were included in the study. According to the criteria above, we achieved permanent DHP in 4 patients and PHP in 28 patients. In six patients, the criteria for DHP and PHP could not be met, and the leads were implanted in the high septal region in anatomical proximity with the His bundle. All patients were paced in the ventricle for >99% of the time. However, six patients had fusion or pseudofusion on the 24 h ECG monitoring, all for >50% of the time, despite a >99% ventricular pacing reported by the pacemaker. These patients were not sufficiently exposed to the deleterious effects of RV pacing, and were excluded from the on-treatment analysis. During the first study period, one patient died of sepsicaemia, not related to the implantation, and during the second period, two patients died, one from pneumonia and one...

Figure 1 Flowchart of the His or para-His pacing in AVB study. Right ventricular septal pacing (RVSP) and His or para-His pacing (HP).
died suddenly. One patient was excluded from analysis of the primary endpoint due to inadequate image quality. A total of 34 patients were included in all echocardiographic analyses, and in 2 of these patients LVEF was measured with the Simpson’s biplane method. Of the 35 patients who completed both study periods, 10 patients were unable to perform a 6-MHW test due to physical limitations, in 1 patient NYHA class could not be evaluated due to immobilization, and 7 patients did not complete QOL assessments for both periods. The flowchart of the study is shown in Figure 1 and the baseline characteristics of the patients are shown in Table 1.

Left ventricular function

In the intention-to-treat analysis, the mean LVEF after 12 months of RVSP was 0.50 (± 0.11) and significantly lower than after 12 months of HP 0.53 (± 0.10), with a difference of 0.05 (± 0.09), P = 0.005 (Figure 2). The LVEDV was 95 (± 36) mL after RVSP and 91 (± 31) mL after HP, with a difference of 4 (± 27) mL, P = 0.41, and the LVESV was 49 (± 26) mL after RVSP and 42 (± 21) mL after HP, with a difference of 7 (± 18) mL, P = 0.03. In the on-treatment analysis, excluding six patients with a >50% fusion or pseudofusion during the 24 h ECG monitoring, the mean LVEF was 0.48 (± 0.10) after RVSP and 0.54 (± 0.10) after HP, with a difference of 0.06 (± 0.09), P = 0.0007 (Figure 3A). In the patients on-treatment who met all the criteria for DHP or PHP (n = 23), the mean LVEF was 0.49 (± 0.09) after RVSP and 0.55 (± 0.10) after HP, with a difference of 0.06 (± 0.09), P = 0.004. The changes in the LVEF according to the pacing site during HP are shown in Figure 3B. In the subgroup on-treatment analysis, the effect of HP on LVEF was more pronounced in patients with a reduced LVEF (0.50 during RVSP [n = 15, 0.50 (± 0.09) vs. 0.41 (± 0.06), P = 0.001], as compared with patients who had preserved LVEF > 0.50 during RVSP [n = 13, 0.60 (± 0.08) vs. 0.56 (± 0.05), P = 0.2] (Figure 3C). Also, in patients with arterial hypertension (n = 14), LVEF differed significantly between RVSP 0.46 (± 0.10) and HP 0.54 (± 0.11), P = 0.006, whereas this was not the case for patients without hypertension (n = 14), 0.50 (± 0.09) during RVSP vs. 0.55 (± 0.09) during HP, P = 0.06 (Figure 3D). The difference in time-to-peak systolic velocity (TPV) between opposite basal segments in the apical four-chamber view was 58 (± 40) ms after RVSP and 49 (± 39) ms after HP, with a difference of 9 (± 48) ms, P = 0.27. In the two-chamber view, there was a significant difference in TPV after RVSP 45 (± 29) ms, and HP 31 (± 26) ms, with a difference of 14 (± 33) ms, P = 0.02, as well as in the apical long-axis view where the difference was 63 (± 43) after RVSP and 44 (± 38) after HP, with a difference of 19 (± 50) P = 0.03. There was no significant interaction between the effect of the different pacing modes and the randomized order or period in any of the echocardiographic outcomes.

Clinical endpoints

The distribution of NYHA class (I/II/III/IV) was 21/6/7/0 after a 12 months RVSP and 24/6/4/0 after a 12 months HP, P = 0.06. The distance covered by a 6-MHW test was not different between the two treatment periods, 558 (± 109) m after RVSP vs. 560 (± 97) m after HP, P = 0.84. In the assessment of QOL, there was no significant difference in any parameter after the two treatment periods. Three patients developed persistent AF and underwent cardioversion in the RVSP period as compared with none during HP. Three patients crossed over to HP during the period where they received RVSP earlier than scheduled (3–6 months), due to development of heart failure symptoms and a significant reduction in LVEF. All endpoint measures are shown in Table 2.

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**Table 1** Baseline characteristics of patients randomized to RVSP and His or para-His pacing (HP) in the first study period, and in all patients

<table>
<thead>
<tr>
<th>Patient characteristics (n/%)</th>
<th>Right ventricular septal pacing</th>
<th>His or para-His pacing</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>66.5 (± 10)</td>
<td>67.8 (± 10)</td>
<td>67 (± 10)</td>
</tr>
<tr>
<td>Men</td>
<td>15 (79)</td>
<td>15 (79)</td>
<td>30 (79)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>3 (16)</td>
<td>1 (5)</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (58)</td>
<td>8 (42)</td>
<td>19 (50)</td>
</tr>
<tr>
<td>Paroxysmal atrial fibrillation</td>
<td>1 (5)</td>
<td>0 (0)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>6 (32)</td>
<td>2 (11)</td>
<td>8 (21)</td>
</tr>
</tbody>
</table>

**Electrocardiographic characteristics**

| QRS intrinsic (ms) | 93 (± 11) | 94 (± 20) | 93 (± 16) |
| QRS RVSP (ms)      | 156 (± 11) | 150 (± 13) | 153 (± 12) |
| QRS HP (ms)        | 115 (± 20) | 107 (± 18) | 111 (± 19) |
| Second-degree AVB  | 6 (31)     | 5 (26)    | 11 (29)   |
| Third-degree AVB   | 10 (53)    | 6 (32)    | 16 (42)   |
| Second- and third-degree AVB | 3 (16) | 8 (42) | 11 (29) |

**Medication at implantation**

| Beta-blockers | 4 (21) | 3 (16) | 7 (18) |
| ACE inhibitors| 5 (26) | 5 (26) | 10 (26) |
| Loop diuretics| 4 (21) | 1 (5)  | 5 (13)  |
| Aldosterone antagonists | 2 (11) | 1 (5) | 3 (8) |

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**Figure 2** Mean left ventricular ejection fraction with SD after 12 months right ventricular septal pacing and His/para-His pacing.
Lead stability and complications

The median threshold measured on the day after the implantation was 0.5 (0.5–0.5) V in the RVSP leads and 1 (0.5–2.5) V in the HP leads, \( P < 0.001 \). At the end of both the study periods, there was no change in thresholds in the RVSP leads, but in the HP leads there was an increase to median 1.5 (0.75–2.9) V, \( P = 0.008 \). One patient developed an exit block on the PHP lead at the 15-month follow-up visit during the RVSP period, and two patients had an intermittent loss of capture for seconds on the HP lead documented on the 24 h ECG monitoring. There were no other major complications during the implantation or study period and no lead dislocations.

Discussion

This study shows that His or para-His pacing can prevent the detrimental effect of a long-term RV pacing on LV systolic function in patients with AVB, a narrow QRS width, and LVEF \( < 0.40 \), by preserving LVEF and reducing LV dyssynchrony.

Right ventricular pacing induces an abnormal and prolonged electrical and mechanical activation of the ventricles, where an early contraction at the pacing site occurs thus stretching the opposite wall that contracts later causing a dyssynchronous contraction.7 This impairs LV systolic and diastolic functions and can cause a mitral regurgitation.13,14 Patients with sinus node disease without AVB, can be treated with pacemaker algorithms reducing RV pacing. In patients with a high-grade AVB, who need ventricular pacing to avoid bradycardia, such algorithms have not been proven to be beneficial or safe. Another strategy to avoid or reduce ventricular dyssynchrony caused by RV pacing is to use alternative pacing site(s).

Depolarization of the ventricles through the His–Purkinje system induces a normal synchronous activation, and is therefore the ideal pacing site to avoid dyssynchrony in patients with a preserved His–Purkinje conduction who need frequent pacing. Achieving DHP in patients with AV conduction disturbances is technically challenging9,10,15 and only a few small randomized studies have been published evaluating the effect of DHP. One acute study found no difference in LV function between DHP and RV pacing,16 and one crossover study including 12 patients for 3 months follow-up periods showed an increased myocardial perfusion, decreased mitral regurgitation, and decreased dyssynchrony during DHP as compared...
with RV apical pacing. Since the QRS complex during DHP is identical to the QRS complex during an intrinsic rhythm, any differences in LV performance between an intrinsic ventricular activation and DHP are not related to an abnormal ventricular activation, but must be owing to AV timing and LV filling. Achieving PHP, on the other hand is much easier, and shortens the QRS complex compared with traditional RV pacing sites. We have in an acute study shown that PHP preserves LV function and synchrony. One previous randomized crossover study compared 6 months of PHP with 6 months of RV apical pacing in 16 patients with chronic AF who underwent AV node ablation. During PHP, they found an improvement in NYHA class, an increased 6-MHW, a decreased mitral and tricuspid regurgitation, and a reduced interventricular dyssynchrony. In contrast, we documented that HP preserves LVEF compared with RVSP, but found no differences in clinical parameters. These differences may be explained by the larger sample size and longer follow-up period in our study, different definitions of PHP employed in the two studies, or different thresholds in patients with AVB. PHP is more clinically applicable compared with DHP in patients with AVB. We found no statistical interaction between the order of study periods and the effect on LVEF from the different pacing sites, which suggests that the changes of LV contraction pattern induced by each pacing site are reversible within the 12-month study periods. It cannot be ruled out that a longer term pacing may cause structural and irreversible changes. Longer term parallel studies are needed for detecting such changes between different pacing sites.

A subgroup analysis showed that in patients with a reduction in LVEF, the effect of HP was more pronounced, as was the case in patients with hypertension. The clinical experience is that the majority of patients who have preserved LVEF tolerate RV pacing for years without developing overt heart failure. However, in a small minority of these patients, RV pacing will cause a deterioration of the LV function and heart failure. Identifying these patients with an increased propensity for developing pacing-induced heart failure is challenging. The present results indicate that LVEF < 50% or arterial

<table>
<thead>
<tr>
<th>Endpoint measures according to treatment period</th>
<th>Right ventricular septal pacing</th>
<th>His or para-His pacing</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricular function</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>0.50 (± 0.11)</td>
<td>0.55 (± 0.10)</td>
<td>0.005</td>
</tr>
<tr>
<td>LV end-diastolic volume (mL)</td>
<td>95 (± 36)</td>
<td>91 (± 31)</td>
<td>0.41</td>
</tr>
<tr>
<td>LV end-systolic volume (mL)</td>
<td>49 (± 26)</td>
<td>42 (± 21)</td>
<td>0.03</td>
</tr>
<tr>
<td><strong>TPV</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Four chamber (ms)</td>
<td>58 (± 40)</td>
<td>49 (± 39)</td>
<td>0.27</td>
</tr>
<tr>
<td>Two chamber (ms)</td>
<td>45 (± 29)</td>
<td>31 (± 26)</td>
<td>0.02</td>
</tr>
<tr>
<td>Apical long axis (ms)</td>
<td>63 (± 43)</td>
<td>44 (± 38)</td>
<td>0.03</td>
</tr>
<tr>
<td><strong>Clinical endpoints</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NYHA class (III/IV)</td>
<td>21/6/7/0</td>
<td>24/6/4/0</td>
<td>0.06</td>
</tr>
<tr>
<td>NYHA class (mean)</td>
<td>1.6</td>
<td>1.4</td>
<td>0.84</td>
</tr>
<tr>
<td>6 min hall walk test (m)</td>
<td>558 (± 109)</td>
<td>560 (± 97)</td>
<td>0.03</td>
</tr>
<tr>
<td><strong>Quality-of-life SF-36 score</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical function</td>
<td>69 (30)</td>
<td>74 (27)</td>
<td>0.20</td>
</tr>
<tr>
<td>Role—physical</td>
<td>59 (43)</td>
<td>58 (45)</td>
<td>0.90</td>
</tr>
<tr>
<td>Bodily pain</td>
<td>73 (25)</td>
<td>74 (26)</td>
<td>0.90</td>
</tr>
<tr>
<td>General health</td>
<td>58 (24)</td>
<td>65 (24)</td>
<td>0.09</td>
</tr>
<tr>
<td>Vitality</td>
<td>55 (23)</td>
<td>63 (28)</td>
<td>0.09</td>
</tr>
<tr>
<td>Social function</td>
<td>85 (21)</td>
<td>84 (24)</td>
<td>0.86</td>
</tr>
<tr>
<td>Role—emotional</td>
<td>69 (42)</td>
<td>65 (42)</td>
<td>0.62</td>
</tr>
<tr>
<td>Mental health</td>
<td>75 (19)</td>
<td>78 (16)</td>
<td>0.35</td>
</tr>
</tbody>
</table>

Difference in time from QRS onset to peak systolic longitudinal velocity in opposite basal segments (TPV). The SF-36 scores (36-Item Short-Form General Health Survey) range from 0 to 100, with higher scores indicating a better health status.
hypertension might predict a worse outcome with constant ventricular pacing. However, larger studies are needed before implementing any consequences of these findings into the clinic. Comparative studies have reported conflicting results with respect to the optimal RV pacing site.16–21 whereas biventricular pacing (CRT) preserves the LVEF and LV synchrony.1,22 The recent BLOCK-HF was the first larger trial to show a clinical benefit of the biventricular pacing over standard RV pacing in patients with reduced LVEF (<0.50) and AVB.23 These findings support the use of CRT in patients with a need for ventricular pacing and LVEF < 50%. The BLOCK-HF study included patients with impaired LV function and a wide QRS, both of which were the exclusion criteria in our study. However, the beneficial effect of CRT on LVEF in the BLOCK-HF population was very similar to our findings with HP.1,22 Similar to HP there are non-negligible proportions of complications and unsuccessful lead implantations with CRT. One of the major concerns for using HP in pacing-dependent patients is the higher threshold and the increased risk of failure to capture. Pacemaker treatment is already very effective and safe, so it is important to focus on reducing the complications and side effects. Therefore, any evaluation of new pacing strategies requires randomized trials taking into consideration benefits as well as risks and complications. As seen for LV pacing through the coronary sinus, we would expect that the technology for His or para-His pacing can be improved to reduce implantation and fluoroscopy times, lower thresholds, and improve lead stability. Since the feasibility, complication rate, and benefit with HP are comparable with CRT, HP may in the future develop to become the preferred pacing site in patients who are expected to be paced frequently and who have an increased propensity for developing pacing-induced heart failure.

Limitations

The sample size of our study was moderate with respect to the evaluation of the secondary endpoints, and the follow-up period was limited to 12 months. Our study group was selected from a larger group of patients with AVB, which may affect the generalizability of the findings. However, this is the largest randomized study of His or para-His pacing, and even if pacemaker therapy in most cases lasts over 12 months, some of the deleterious effects of RV pacing are present within the first 12 months.19,20 The RV septal lead positions were verified by fluoroscopy, with the uncertainties associated with this method;20 however, we achieved a shorter QRS duration with these positions than with apical pacing.10 There is no reason to believe that the benefit documented from His or para-His pacing would have been smaller had we used an RV apical position for control RV pacing, and RVSP used in this trial was standard therapy at our institution.

Conclusion

His or para-His pacing preserves LVEF and mechanical synchrony compared with RV septal pacing in patients with a high-grade AVB, a narrow QRS width, and LVEF > 0.40. In the selected patients, His or para-His pacing might potentially prevent pacing-induced heart failure.

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References


**EP CASE EXPRESS**

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Developing a crush: acute implantable cardioverter-defibrillator lead insulation break in a patient with multiple leads

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A 71-year-old man with ischaemic cardiomyopathy had a dual-chamber pacemaker inserted via the subclavian vein for complete heart block. He was pacing dependent. Nine years later, he was upgraded to a cardiac resynchronization defibrillator. Three new leads were implanted via the subclavian, and the old leads capped. Six weeks later, he presented with falls. Left arm elevation precipitated dizziness and ventricular standstill. The extracted defibrillator lead showed extensive insulation damage (see Figure). The old right ventricular and atrial leads were explanted, and a new defibrillator lead implanted. This case illustrates an acute crush injury in a physically inactive patient. The patient had five leads in his subclavian vein, which may contribute to lead compression; however, there is currently no strong evidence to extract abandoned leads. This patient was pacing dependent, hence became symptomatic during pacing inhibition due to oversensing. A defibrillator lead crush injury could have resulted in undersensing of ventricular fibrillation. Caution needs to be taken with using the subclavian route to place all leads, lead extraction may become necessary, and regular follow-up is necessary to minimize the potentially serious consequences of lead failure.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/Developing-a-crush.pdf.

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