Permanent HIS bundle Pacing Feasibility in Routine Clinical Practice: Experience from an Indian Center

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ABSTRACT

There is a paucity of experience regarding His bundle pacing (HBP) at laboratories initially attempting the procedure, especially in the Indian scenario. Patient who underwent HBP were selected for pacing therapy or in lieu of cardiac resynchronization therapy (CRT) at a single center. Among 22 patients attempted, 19 patients underwent successful implant, achieving selective HBP in 14 patients. There was a significant improvement in left ventricular ejection fraction (LVEF) (49.3 ± 9.3 vs. 36.7 ± 9.2) in the LV dysfunction subgroup (n = 6). Over a follow-up of 15 ± 6.5 months, thresholds were stable in all except one patient, and there was no requirement of lead revision. In summary, we found that HBP is a feasible option for achieving physiological pacing.

1. Introduction

Permanent pacing from the right ventricular apex is associated with worsening left ventricular function,1,2 mitral regurgitation,3 and arrhythmia.4 Recent evidence suggests His bundle pacing (HBP) to be a much physiological option with better hemodynamics in patients requiring pacing support.5,6 We report the initial results of permanent HBP in terms of clinical outcomes and lead parameters on follow-up.

2. Methods

2.1. Patient selection

Between January 2017 and January 2019, 22 patients underwent HBP under fluoroscopic guidance in CARE Hospitals, Hyderabad. The inclusion criteria for HBP were as follows: (1) permanent pacing for conduction disease and (2) resynchronization therapy. This study protocol was approved by the institutional ethics committee.

2.2. HBP procedure

The His bundle electrogram was mapped with a deflectable quadripolar catheter placed through the femoral approach under fluoroscopic guidance, and HV interval was measured on the EP Tracer (Cardiotek) recording system at a sweep speed of 100 mm/s. A Select Secure 3830 lead (Medtronic) through Select Site C315 sheath (Medtronic) was advanced into the His bundle region to map the His signal, as described in the study by Vijayaraman et al.7 His capture was assessed by pacing started at 5 V at 1 ms of pulse width (unipolar) before fixation by rotating the lead typically up to 5 turns, with the delivery sheath advanced up to the proximal electrode for guide support. Procedural success was defined as a pacing threshold for a His capture of ≤2.5 V at 1 ms. Acute injury current in the local His and/or ventricular electrogram and thresholds were recorded.

Selective His bundle capture is achieved when an isoelectric segment between the pacing stimulus and the onset of QRS (Stim–QRS) was equal or shorter than the HV interval with rapid onset of QRS activation (Fig. 1).

Nonselective His bundle capture was considered to be present when there was a pseudodelta wave after the stimulus, and the overall electrical axis of the paced QRS will be concordant with the electrical axis of the intrinsic QRS.7

A defibrillator lead was placed into the right ventricle when indicated by standard technique. An atrial lead was placed in the right atrial appendage.

2.3. Follow-up

Patients were followed up in the device clinic for 1 week, 1 month, 3 months, and 6 months and annually thereafter by clinical assessment, device interrogation, and 12-lead ECG.
2.4. Statistical analysis

Comparison between the subgroups was calculated using either the Student t-test or 2-tailed Fisher exact test for continuous and categorical variables, respectively. A two-tailed $p$ value of $< 0.05$ was considered statistically significant.

3. Results

The mean age of the patients in whom HBP was attempted ($n = 22$) was $65.8 \pm 16.9$ years (males, 50%). Among the patients who underwent successful HBP ($n = 19$), there was a history of previous coronary artery disease in 7 (37%) patients. Baseline ECGs
showed bundle branch block (BBB) in 7 patients (left bundle branch block [LBBB], n = 5; right bundle branch block, n = 2) and atrial fibrillation in 11 patients. Six patients had LV dysfunction at baseline (mean LVEF, 37.7 ± 9.2%).

Over 2 years, HBP was successful in 19 patients (86%). The indication includes advanced atrioventricular (AV) block in 3 patients, sick sinus syndrome in 1 patient, and AV junction ablation requiring pacing therapy for 11 patients and 7 patients in lieu of cardiac resynchronization therapy (CRT).

Among our patients who underwent successful HBP, 15 patients had received the CRT device (4 patients with an additional defibrillator lead), 3 patients received dual chamber pacemaker, and only 1 patient received single chamber pacemaker pacemaker. The current of injury was observed in 80% of the patients after the screwing of the lead at the His location. Selective HBP was achieved in 14 patients. The threshold in selective and nonselective HBP is described in each patient in Table 1. Patients with a baseline BBB (n = 7) had a significant narrowing of the QRS complex after HBP compared with patients without baseline BBB (25.7 ± 12.1 vs. 6.0 ± 3.4, p < 0.05).

In 2 patients in whom HBP failed, the His bundle signal could not be recorded, and CRT was performed by implanting a regular LV lead through the coronary sinus in the posterolateral vein. In the third case, HBP failed owing to infra-Hisian AV block.

A total of 15 patients have completed a median follow-up time of 5.2 months (range, 1–6 months), and 9 patients have completed a median follow-up of 12.3 months (range, 6–22 months). Two patients died of noncardiac causes, and 2 patients lost to follow-up.

During follow-up, we observed significant improvement in EF% (37.67 ± 9.18 vs. 49.33 ± 9.29) in patients (n = 6) with LV dysfunction.

There was no rise in the threshold of the His bundle lead (except in 1 patient) or lead dislodgement with a mean duration of battery longevity of 76.9 ± 26.5 months on follow-up (range, 3–22 months).

4. Discussion

The main observations of this study are that permanent HBP (1) is a safe and feasible procedure, (2) is a suitable alternative to conventional CRT, especially in patients with tachycardia-related cardiomyopathy in whom AV Junction ablation is planned, and (3) may not be suitable always with infra-Hisian block. Desmukh and Romanyshyn first showed feasibility of HBP (selective His pacing) in atrial fibrillation who underwent AV Junction ablation.

In our present study, 11 (who underwent HBP) of 19 patients underwent AV Junction, with all of them having clinical improvement in terms of symptoms and EF similar to the study by Huang et al. We observed a lower threshold in nonselective HBP than in selective HBP, resembling the study result of Zanon et al. Vijayaraman et al have reported that His capture thresholds at implant remained stable during 5-year follow-up. Similarly, we have observed a stable pacing threshold in the majority of individuals during follow-up so far.

Studies by Ploux et al have shown that CRT only minimizes the ventricular activation timing by fusion in LBBB, whereas physiological BBB correction is plausible by HBP alone, as demonstrated in all 7 patients in our study. As the block is mostly located proximal to the His bundle, the fibers of the left and right bundle are predestined in the penetrating bundle of HIS as explained by the longitudinal dissociation of fibers of the His bundle.

| Patient no. | Morphology of QRS | QRSd (baseline) | QRSd (paced) | Selective/Non-selective His pace | Type of device | Mode | Threshold (His) (unipolar/bipolar) | Impedence |
|-------------|-------------------|-----------------|--------------|---------------------------------|----------------|------|-----------------------------------|-----------|
| 1           | LBBB              | 169             | 124          | SHP                             | CRT-D          | DDD  | 1.5 V at 1.0 ms (unipolar)         | 493       |
| 2           | Normal            | 106             | 123          | SHP                             | CRT-P          | DDD  | 0.75 V at 0.80 ms (bipolar)        | 456       |
| 3           | RBBB              | 146             | 113          | SHP                             | CRT-D          | VVIR | 2.5 V at 1 ms (unipolar)           | 456       |
| 4           | Normal            | 89              | 93           | SHP                             | DDDR           | DVI  | 0.875 V at 0.76 ms                | 490       |
| 5           | LBBB              | 156             | 94           | SHP                             | CRT-P          | VVIR | 1.8 V at 1 ms                     | 350       |
| 6           | Normal            | 146             | 126          | Non-SHP                         | CRT-D          | DDD  | 0.75 V at 1 ms (unipolar)/1.5V at 1 ms (bipolar) | 437       |
| 7           | LBBB              | 108             | 114          | SHP                             | CRT-P          | VVIR | 0.75 V at 1 ms—both unipolar and bipolar | 563       |
| 8           | Normal            | 92              | 107          | Non-SHP                         | CRT-P          | VVIR | Bipolar 1 V at 0.4 ms             | 448       |
| 9           | Normal            | 108             | 110          | Non-SHP                         | DDD            | DVI  | 0.7 V at 1 ms (unipolar)/0.7 V at 1 ms (bipolar) | 532       |
| 10          | Normal            | 164             | 141          | Non-SHP                         | CRT-P          | DDD  | Bipolar 0.5 at 1 ms/unipolar 0.5 at 1 ms | 650       |
| 11          | RBBB              | 116             | 129          | SHP                             | CRT-P          | DDD  | 3 V at 1 ms (unipolar)/2.5 V at 1 ms (bipolar) | 586       |
| 12          | Normal            | 165             | 134          | SHP                             | CRT-P          | DDD  | 0.5 V at 0.6 ms (unipolar)/0.5 V at 0.6 ms (bipolar) | 450       |
| 13          | LBBB              | 105             | 104          | Non-SHP                         | CRT-P          | VVIR | 2.5 V at 1 ms (unipolar)/bipolar same thresholds as unipolar | 400       |
| 14          | Normal            | 81              | 142          | SHP                             | DDDR           | DVI  | 1.50 V at 0.8 ms (bipolar)         | 680       |
| 15          | Normal            | 110             | 124          | SHP                             | CRT-P          | DDD  | Bipolar 1.5 V at 0.8 ms           | 398       |
| 16          | Normal            | 128             | 123          | SHP                             | CRT-D          | DDD  | Bipolar 1.25 V at 0.8 ms          | 520       |
| 17          | RBBB              | 108             | 105          | SHP                             | Single chamber pacemaker | VVIR | Unipolar and bipolar 1.0 V at 1.0 ms | 615       |
| 18          | LBBB              | 87              | 95           | SHP                             | CRT-P          | DDD  | Bipolar 1.8 V at 1 ms             | 685       |
| 19          | Normal            | 82              | 84           | SHP                             | CRT-P          | DDD  | Unipolar 0.5 V at 0.6 ms and 0.75 V at 0.6 ms in bipolar | 560       |
We noticed significant improvement in mean LVEF in 6 patients with baseline LV dysfunction, similar to the study conducted by Ajijola et al.\(^\text{15}\)

Development of automatic threshold algorithms to identify His capture thresholds would be an important step to extend battery longevity, which is currently limited by the fact that selective His capture is not associated with an evoked potential.

The limitations of this study are a small sample size and short follow-up. Owing to the lack of guidelines at present, HBP was attempted in patients based on the physician’s discretion.

5. Conclusion

HBP can be performed safely and is associated with good clinical outcome. Improved understanding of lead performance in different patients’ substrate and anatomy is needed to guide patient selection for HBP.

5.1. What is already known?

HBP has been proposed to represent the most physiologic mode of ventricular pacing and has shown good results in a few experienced centers.

5.2. What this study adds?

This is the first reported study from India demonstrating good feasibility of permanent HBP in a heterogeneous study cohort.

Conflicts of interest

All authors have none to declare.

References

1. Nahtlawi M, Waligora M, Spies SM, Bonow RO, Kadiash AH, Goldberger JJ. Left ventricular function during and after right ventricular pacing. \textit{J Am Coll Cardiol}. 2004;44:1883–1888.
2. O’Keeffe Jr JH, Abuissa H, Jones PG, et al. Effect of chronic right ventricular apical pacing on left ventricular function. \textit{Am J Cardiol}. 2005;95:771–773.
3. Barold SS, Owsyshcher EL. Pacemaker-induced mitral regurgitation. \textit{Pacing Clin Electrophysiol}. 2005;28:357–360.
4. Gardiwal A, Yu H, Oswald H, et al. Right ventricular pacing is an independent predictor for ventricular tachycardia/ventricular fibrillation occurrence and heart failure events in patients with an implantable cardioverter–defibrillator. \textit{Europace}. 2008;10:358–363.
5. Scherlag BJ, Kosowsky BD, Damato AN. A technique for ventricular pacing from the His bundle of the intact heart. \textit{J Appl Physiol}. 1967;22:584–587.
6. Dabrowski P, Kleinsrok A, Kozluk E, Opolski G. Physiologic resynchronization therapy: a case of his bundle pacing reversing physiologic conduction in a patient with CHF and LBBB during 2 years of observation. \textit{J Cardiovasc Electrophysiol}. 2011;22:813–817.
7. Vijayaraman P, Dandamudi G, Zanon F, et al. Permanent His bundle pacing: recommendations from a multicenter His bundle pacing collaborative working group for standardization of definitions, implant measurements, and follow-up. \textit{Heart Rhythm}. 2018;15:460–468.
8. Deshmukh P, Romaniushyn M. Direct His bundle pacing: present and future. \textit{PACE (Pacing Clin Electrophysiol)}. 2004;24:862–887.
9. Huang W, Su L, Wu S, et al. Benefits of permanent His bundle pacing combined with atrioventricular node ablation in atrial fibrillation patients with heart failure with both preserved and reduced left ventricular ejection fraction. \textit{J Am Heart Assoc}. 2017 Apr 1;6.e005309.
10. Zanon F, Baracca E, Aggio S, et al. A feasible approach for direct His bundle pacing using a new steerable catheter to facilitate precise lead placement. \textit{J Cardiovasc Electrophysiol}. 2006;17:29–33.
11. Vijayaraman P, Naperkowski A, Subzposh FA, et al. Permanent His bundle pacing: long-term lead performance and clinical outcomes. \textit{Heart Rhythm}. 2018;15:696–702.
12. Ploux S, Eschalier R, Whinnett ZL, et al. Electrical dyssynchrony induced by biventricular pacing: implications for patient selection and therapy improvement. \textit{Heart Rhythm}. 2015;12:782–791.
13. Sharma Parikshit S, Naperkowski Angela, Bauch Tery D. Permanent his bundle pacing for cardiac resynchronization therapy in patients with heart failure and right bundle branch block. \textit{Circ Arrhythm Electrophysiol}. 2018;11.e006613.
14. Narula OS. Longitudinal dissociation in the His bundle: bundle branch block due to asynchronous conduction with in the His bundle in man. \textit{Circulation}. 1977;56:996–1006.
15. Ajijola OA, Upadhayay GA, Macias C, Shivkumar K, Tung R. Permanent His-bundle pacing for cardiac resynchronization therapy: initial feasibility study in lies of left ventricular lead. \textit{Heart Rhythm}. 2017;14:1353–1361.